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Effects of resistance training on endurance capacity and muscle fiber composition in young top-level cyclists

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Equivocal findings exist on the effect of concurrent strength (S) and endurance (E) training on endurance performance and muscle morphology. Further, the influence of concurrent SE training on muscle fiber-type composition, vascularization and endurance capacity remains unknown in top-level endurance athletes. The present study examined the effect of 16 weeks of concurrent SE training on maximal muscle strength (MVC), contractile rate of force development (RFD), muscle fiber morphology and composition, capillarization, aerobic power (VO_{2max}), cycling economy (CE) and long/short-term endurance capacity in young elite competitive cyclists ($n = 14$). MVC and RFD increased 12–20% with SE ($P < 0.01$) but not E. VO_{2max} remained unchanged. CE improved in E to reach values seen in SE. Short-term (5-min) endurance performance increased (3–4%) after SE and E ($P < 0.05$), whereas 45-min endurance capacity increased (8%) with SE only ($P < 0.05$). Type IIA fiber proportions increased and type IIX proportions decreased after SE training ($P < 0.05$) with no change in E. Muscle fiber area and capillarization remained unchanged. In conclusion, concurrent strength/endurance training in young elite competitive cyclists led to an improved 45-min time-trial endurance capacity that was accompanied by an increased proportion of type IIA muscle fibers and gains in MVC and RFD, while capillarization remained unaffected.

Equivocal findings exist on the effect of concurrent strength (S) and endurance (E) training on endurance performance and muscle morphology. Some studies have reported attenuated cardiovascular and musculoskeletal improvements with combined S and E training regimes (Hickson et al., 1980; Dudley & Djamil, 1985; Kraemer et al., 1995; Glowacki et al., 2004; Izquierdo et al., 2005). In contrast, others have shown that concurrent SE training can lead to similar cardiovascular or musculoskeletal adaptations compared with S or E training alone (Bell et al., 1991; McCarthy et al., 1995, 2002; Izquierdo et al., 2005), or that concurrent SE training may increase endurance performance beyond that achieved by endurance training alone (Hoff et al., 1999, 2002; Østerås et al., 2002; Støren et al., 2008; Rønnestad et al., 2009; Rønnestad et al., 2010).

Studies on untrained persons and moderate-to-well-trained endurance athletes indicate that both short-term (Hickson et al., 1980, 1988; Hoff et al., 1999, 2002; Østerås et al., 2002; Støren et al., 2008; Losnegard et al., 2010) and long-term endurance capacity (Hickson et al., 1980, 1988; Marcinik et al., 1991) can be increased by the use of strength training (resistance exercise). While the influence of resistance training on long-term endurance performance (>30 min) has been examined almost exclusively in untrained-to-moderately trained individuals (Hickson et al., 1980, 1988; Marcinik et al., 1991, Bishop et al., 1999), only few studies have evaluated this effect in highly trained endurance athletes (Paavolainen et al., 1999; Bastiaans et al., 2001). Specifically, the effect of concurrent S and E training on long-term endurance capacity in top-level endurance athletes remains unknown.

Although not a universal finding (Häkkinen et al., 2003; Rønnestad et al., 2010), concurrent SE training may result in elevated maximal muscle strength in the absence of muscle fiber hypertrophy (Hickson et al., 1988, Bishop et al., 1999) where the increase in strength likely appears as a result of neuromuscular adaptation (Aagaard, 2003). The potential lack of muscle hypertrophy with concurrent SE training may be particularly interesting to top-level endurance athletes in sports where muscle forces are generated to support the body mass against gravity (i.e. running, uphill cycling) and where gains in body mass are undesirable because they are believed to impede

optimal endurance performance. The potential gain in maximal muscle force in the absence of muscle hypertrophy is likely to result in an enhanced exercise performance in highly trained competitive endurance athletes. Further, longitudinal study data obtained in previously untrained subjects have shown that resistance training may *per se* provide a stimulus for capillary neoformation (Hather et al., 1991; Green et al., 1998; Kadi & Thornell, 2000). Representing a novel stimulus, it is possible therefore that resistance training could induce angiogenesis even in highly trained endurance athletes.

Strength training in untrained individuals typically causes the proportion of type IIA muscle fibers to increase at the expense of a reduced fraction of type IIX fibers (Kraemer et al., 1995; Andersen & Aagaard, 2000). It remains unknown, however, if strength training leads to an increased proportion of type IIA muscle fibers in top-level endurance athletes, representing a shift toward a powerful yet fatigue-resistant muscle phenotype. Potentially, such a shift could have a positive effect on the long-term performance in endurance events requiring high levels of sustained muscle force and power output, i.e. in time-trial cycling.

It was the aim of the present study, therefore, to examine the effect of concurrent strength and endurance (SE) training on muscle morphology and fiber-type composition, capillarization, maximal muscle strength (MVC) and contractile rate of force development (RFD) and long/short-term endurance performance in young male National Team cyclists. We hypothesized that SE training would result in enhanced long-term endurance performance compared with E training alone, which would be accompanied by increased type IIA muscle fiber proportions, increased capillarization and elevated contractile RFD and MVC.

Material and methods

Subjects

Fourteen young male elite cyclists (U-23 National Team, non-professionals only), 19.5 ± 0.8 years (± SD), 70.7 ± 5.8 kg, 180.7 ± 5.4 cm volunteered to participate in the study, which was approved by the local Ethics Committee of Copenhagen. Written informed consent was obtained from all participants. The subjects were randomly assigned to performing either concurrent endurance (E) and strength (S) training (SE) ($n = 7$) or endurance training alone (E) ($n = 7$). Body composition (percentage body fat, lean body mass) was evaluated by measurements of skinfold thickness (Durnin & Womersley, 1974), which were performed by the same skilled lab technician in all subjects.

Training

Concurrent strength and endurance training (SE group)

Concurrent strength and endurance training was performed for 16 weeks. SE training included 40 strength training sessions, initiated by 2 weeks of preparatory strength training (training loads: 10–12 RM) (for more details, see Table 1). Four sets of four exercises (isolated knee extension, incline leg press, hamstring curls, calf raises) were performed in each session (two to three sessions per week) using a periodized loading pattern (Table 1). Rest time between exercises was 1–2 min, and rest time between exercise series was 2–3 min. For safety precautions, all weights were lifted and lowered in a controlled manner, i.e. using non-explosive contraction efforts (Andersen & Aagaard, 2000). Restitution time between sessions was ≥ 48 h. In addition, the SE subjects performed 10–18 h of endurance (cycling) training each week according to a periodized scheme (Table 2).

Endurance training alone (E group)

A total of 16 weeks of endurance training was performed, consisting of 10–18 h of endurance (cycling) training per week (Table 2). The amount and intensity [heart rate (HR) monitored] of E training did not differ from that performed by SE.

Assessment of aerobic power and cycling economy (CE)

Standard cycle ergometer tests were performed to evaluate submaximal and maximal aerobic power (oxygen uptake rate: VO_2) according to the procedures commonly used in our lab (Jensen & Johansen, 1998). In brief, maximal aerobic power was measured as the maximal rate of oxygen uptake (VO_{2max}). Before this assessment, a four-step incremental steady-state cycle test (4 × 5 min, at ~ 60%, 70%, 80% and 90% of VO_{2max}) to assess submaximal aerobic power and CE was conducted using the subjects own preferred road bike mounted onto a Ciclo Training Politecnica 80 (Padova, Italy) (Jensen & Johansen, 1998). On-line oxygen uptake (VO_2) (Amis 2001, Innovision, Odense, Denmark), respiratory exchange ratio ($RER = VCO_2/VO_2$), HR and blood lactate (drawn from finger) were obtained at the fourth minute of each 5-min time interval.

Blood lactate was analyzed in whole blood samples (500 μL) that was immediately transferred to a vial containing 25 IU heparin. Plasma was separated by centrifugation at 4 °C and analyzed for lactate content using a YSI 2300 STAT

Table 1. Strength training (SE group): weekly training frequency (Freq) and loading intensity (Loads, expressed in RM units) during 16 weeks of periodized progressively adjusted resistance training

Week	1	2	3	4	5	6	7	8
Freq	3	2	3	2	3	2	3	2
Loads (RM)	10–12	8–10	8–10	6–8	6–8	5–6	5–6	5–6
Week	9	10	11	12	13	14	15	16
Freq	3	2	3	2	3	2	3	2
Loads (RM)	5–6	5–6	5–6	5–6	5–6	5–6	5–6	5–6

More details are given in text.

Table 2. Endurance training (E group and SE group): hours of training per week during the 16 weeks of periodized endurance (cycling) training

Week	1	2	3	4	5	6	7	8
Hours	14	15	15	11	16	17	17	10
Week	9	10	11	12	13	14	15	16
Hours	16	17	18	18	10	18	18	18

More details are given in text.

PLUS analyzer (YSI Inc., Yellow Springs, OH, USA). Resting blood hematocrit (Hct) was obtained by the micro-hematocrit method.

CE was calculated as the cycling power (Watt/kg) divided by the rate of oxygen uptake (mL O_2/min/kg where both parameters were obtained at 75% VO_{2max} by linear interpolation between successive values obtained during the submaximal incremental test (Jensen & Johansen, 1998).

After completion of the submaximal test and 15-min rest, the subject performed a 5-min all-out test on the cycle ergometer in which the goal was to cover a distance as great as possible in the 5-min period. VO_2, VCO_2, power production and HR were continuously recorded (Jensen & Johansen, 1998). VO_{2max} was identified as peak VO_2 (15-s average) recorded during the 5-min test, and total distance, total work (joule) and average power production were calculated (Jensen & Johansen, 1998). Blood lactate was obtained acutely after end of the 5-min test, and at 1, 2 and 5 min of recovery.

Assessment of 5-min and 45-min endurance capacity

Total work production in the 5-min all-out test expressed as an average ergometer work rate (mean Watts) was used as a measure of short-term endurance performance. To evaluate long-term endurance capacity, the subjects performed a 45-min all-out time-trial test on a separate day in the lab using their own preferred racing bike in which the goal was to cover a distance as great as possible. Use of such closed-end (i.e. "time-trial" type) lab tests has been shown previously to provide a reproducible measure of long-term endurance capacity in well-trained cyclists (Jeukendrup et al., 1996). Distance, power production and HR were continuously measured throughout the 45-min time-trial test. These data as well as the elapsed time were kept unknown to the subject throughout the test to minimize pre-to-post differences in subjective pacing ability. Maximal HR and peak blood lactate (at end, 1-min post, 5-min post) were obtained along with average power production (Watts), total distance (m) and work (joule). Time-trial performance measured as the average work rate (Watts) was used as a measure of 45-min time-trial endurance capacity. Body mass, resting blood lactate concentration and Hct were all obtained before warm-up. All subjects were highly familiarized with the above test procedures.

Mechanical muscle strength characteristics

As described previously in detail (Aagaard et al., 2002), maximal isometric quadriceps contraction strength (MVC), and rapid force capacity evaluated as contractile RFD (ΔForce/Δt) and impulse ($\int$ Force dt) were obtained at 70° knee joint angle (0° = full knee extension) using an isokinetic dynamometer (Kinetic Communicator, Chattecx Corp., Chattanooga, TN, USA). All signals were synchronously sampled at a 1 kHz sampling rate using an external 12-bit A/D converter (dt28EZ, Data Translation). In brief, subjects were seated 10° reclined in a rigid chair and firmly strapped at the hip and distal thigh. The rotational axis of the dynamometer was visually aligned to the lateral femoral epicondyle of the subject and the lower leg was attached to the dynamometer lever arm 2 cm above the lateral malleolus. All strength measurements were preceded by a thorough warm-up that included 3-min ergometer cycling at 200 W. During all strength testing, subjects were carefully instructed to contract "as hard and as fast as possible." MVC was obtained as the maximal moment of force (in N m), while the rate of force development (ΔMoment/Δt) was calculated as previously described (Aagaard et al., 2002). The steepest tangential slope of the moment–time curve (i.e. peak contractile RFD) (Aagaard et al., 2002) was chosen as an overall measure of rapid force capacity. While MVC was obtained from the trial with maximal peak moment, peak RFD was obtained from the trial with largest contractile impulse ($\int$ Moment dt) measured in the time interval of 0–200 ms relative to force onset. All recorded force moments were corrected for the effect of gravity on the lower leg (Aagaard et al., 2002).

Muscle morphology and fiber-type composition

Muscle biopsies (100–150 mg) obtained from m. vastus lateralis of the dominant limb were analyzed as described in detail previously (Andersen & Aagaard, 2000). In brief, fiber-type composition was established using myofibrillar ATPase staining at pH 9.4 after both alkaline (pH 10.3) and acid (pH 4.37 and 4.6) preincubation. Fiber-type distribution [type I, IIC (I/IIA), IIA, IIAX, IIX] and fiber cross-sectional area were determined using digital image analysis (TEMA 1.04, Scanbeam, Hadsund, Denmark). Data were collapsed to calculate the mean fiber size for the two major fiber types (I and II) according to the procedures described previously (Andersen & Aagaard, 2000). The mean number of muscle fibers analyzed per subject (mean between pre- and post-training) was 175 ± 86 in ES and 238 ± 107 in E (mean ± SD).

Muscle capillarization

Vascular capillarization was evaluated using double staining for Ulex europaeus agglutinin I lectin combined with collagen IV antibody (Qu et al., 1997) and the following parameters were calculated: (i) number of capillaries per unit of muscle fiber area (cap/mm^2), (ii) number of capillaries divided by the number of muscle fibers within the analyzed cross-section (cap/fiber) and (iii) number of capillaries in contact with each muscle fiber (cap/fiber). For this analysis, 77 ± 25 and 68 ± 29 muscle fibers were analyzed per biopsy in SE and E, in which 291 ± 107 and 283 ± 114 capillaries were identified (± SD), respectively.

Statistical analysis

Differences between subject groups were analyzed using non-parametric ANOVA (Kruskal–Wallis test) with *post hoc* group contrasts evaluated by Mann–Whitney tests (two-tailed). Within-group changes were analyzed by the Wilcoxon signed rank testing (Aagaard et al., 2002). Level of significance was set at $\alpha = 0.05$. All data are expressed as group mean values ± SD unless otherwise stated.

Results

Body composition

Body mass remained unchanged with training (SE: 69.2 ± 5.8 vs 70.0 ± 6.1 kg; E: 72.3 ± 5.9 vs 71.4 ± 5.4 kg). An increase in lean body mass was observed following SE training (60.7 ± 5.2 to 62.7 ± 5.1 kg, $P < 0.05$) but not E training (62.5 ± 5.0 vs 62.6 ± 4.6, NS). Percentage body fat decreased in SE (12.2 ± 1.5 to 10.4 ± 1.8 kg, $P < 0.05$) but not in E (13.3 ± 2.5 vs 12.1 ± 1.2, NS).

5-min and 45-min endurance capacity

Short-term endurance performance measured as average power production (Watts, equivalent to total work) performed in the 5-min all-out cycling test increased 3–4% ($P<0.05$) in both SE (405.4 ± 53.3 to 425.0 ± 39.4 W) and E (388.4 ± 14.1 to 400.4 ± 33.6 W) (Fig. 1). SE tended to perform at a higher work rate (mean power) than E during the 5-min all-out test before the period of training ($P = 0.073$).

Endurance performance assessed by the 45-min time-trial testing increased 8% after SE training (313.7 ± 45.9 to 340.1 ± 33.1 W) ($P<0.05$), while remaining statistically unchanged after E training alone (309.5 ± 20.3 vs 321.0 ± 19.5 W) (Fig. 2). The distance covered (total work, average power production) during the 45-min time trial was greater in SE than E after the period of training ($P<0.01$). Likewise, relative pre-to-post training changes in time-trial performance (total distance, total work, average power production) were greater in SE than E ($P<0.01$).

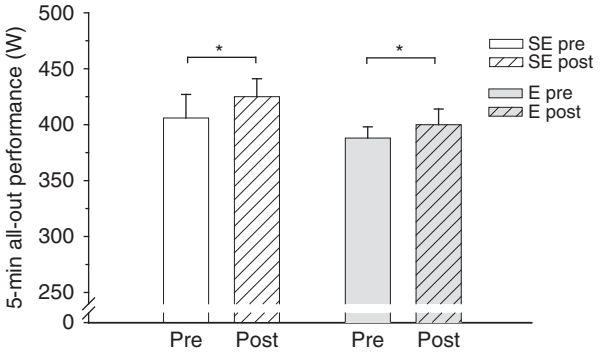

Fig. 1. Short-term endurance performance measured as mean power production during 5-min all-out cycling pre and post 16 weeks of concurrent strength and endurance training (SE) or endurance training alone (E). *Post > pre ($P<0.05$).

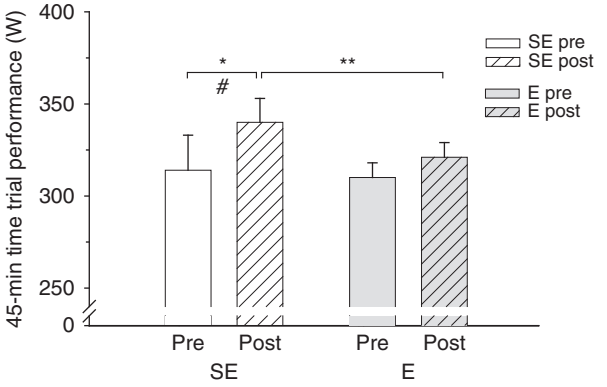

Fig. 2. Endurance performance measured as mean power production during 45-min time-trial testing pre and post 16 weeks of concurrent strength and endurance training (SE) or endurance training alone (E). *Post > pre ($P<0.05$), **SE > E post training ($P<0.01$), #greater relative pre-to-post change in SE than E ($P<0.01$).

Maximal muscle strength and rapid force capacity

Maximal isometric quadriceps muscle strength (MVC) increased 12% after SE training (275.3 ± 42.4 to 307.7 ± 40.4 N m) ($P<0.05$), while no change occurred with E training alone (261.9 ± 45.9 vs 257.9 ± 28.5 N m). The capacity for rapid force production (peak contractile RFD) increased 20% following the period of concurrent SE training (1999.2 ± 684.4 to 2433.1 ± 540.1 N m/s) ($P<0.01$), while remaining unaffected following E training alone (1707.1 ± 814.6 vs 2158.4 ± 559.4 N m/s).

Muscle fiber area and fiber-type distribution

Muscle fiber area and fiber-type distribution did not differ between SE and E groups at baseline. Muscle fiber area was unchanged following SE training and E training (Table 3). Type IIA muscle fiber proportions (area%) increased from 26% to 34% following SE training ($P<0.05$), while type IIX percentage decreased from 5.0% to 0.6% ($P<0.05$) (Fig. 3). Fiber-type composition based on fiber number remained statistically unchanged, although a statistical trend ($P = 0.07$–0.08) increased for IIA (25.3–29.7%) and decreased IIX proportions (4.5–0.8%), respectively, emerged after SE training. Fiber-type composition remained unaltered after E training alone based on either fiber area (Fig. 3) or fiber number.

Muscle capillarization

Muscle fiber capillarization did not change with either SE training or E training (Table 3). Likewise, the number of capillaries in contact with each muscle fiber remained unaltered in SE and E, respectively (Table 3). The number of capillaries in contact with type I and type II fibers also remained unchanged in SE and E (Table 3).

Aerobic power and CE

VO_{2max} remained unaltered following either type of training (SE: 5.08 ± 0.64 vs 5.23 ± 0.44 L O_2/min, E: 5.16 ± 0.39 vs 5.21 ± 0.38 L O_2/min), also when expressed relative to body mass (SE: 73.5 ± 8.2 vs 75.0 ± 6.0 mL O_2/min/kg, E: 71.5 ± 6.0 vs 73.0 ± 2.3 mL O_2/min/kg).

Before the period of intervention, CE unexpectedly was worse in E (0.223 ± 0.015 mL O_2/J) than SE (0.204 ± 0.025 mL O_2/J) ($P<0.05$). In a possible consequence hereof, CE improved in E alone (0.207 ± 0.008 mL O_2/J) ($P<0.05$) to reach a post

Table 3. Muscle fiber area and capillarization (group mean ± SD) in the quadriceps (VL) muscle before (Pre) and after (Post) 16 weeks of concurrent strength and endurance training (SE) or endurance training alone (E)

	Type I CSA (μm^2)	Type II CSA (μm^2)	Cap/mm^2 (cap/mm^2)	Cap/fiber (I) (cap/fiber)	Cap/fiber (II) (cap/fiber)
SE training					
Pre	5874 ± 774	6039 ± 667	608.4 ± 44.8	7.75 ± 0.68	7.19 ± 1.05
Post	4961 ± 1087	5912 ± 1046	603.5 ± 57.4	7.26 ± 0.82	7.10 ± 0.71
E training					
Pre	5200 ± 863	4958 ± 1235	710.9 ± 142.4	7.76 ± 0.51	6.91 ± 0.87
Post	6329 ± 837	5791 ± 772	644.9 ± 208.0	8.55 ± 0.66	7.72 ± 1.08

Type I CSA, type I fiber cross-sectional area; Type II CSA, type II fiber cross-sectional area; Cap/mm^2, number of capillaries per mm^2; Cap/fiber (I), number of capillaries in contact with type I fibers; Cap/fiber (II), number of capillaries in contact with type I fibers.

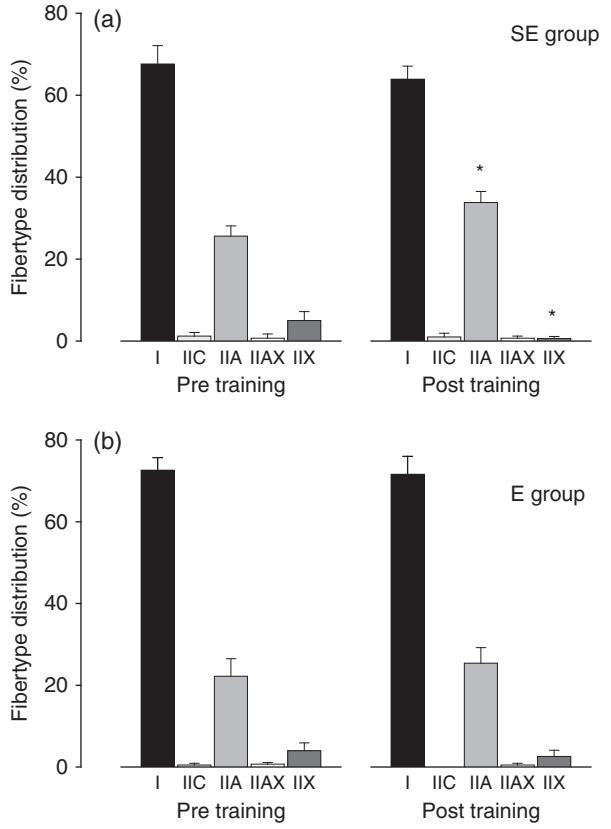

Fig. 3. Muscle fiber-type distribution based on muscle fiber area obtained in the quadriceps muscle (VL) pre and post 16 weeks of concurrent strength and endurance training (SE) (a) or endurance training alone (E) (b). *Post > pre for IIA fibers and post < pre for IIX fibers ($P < 0.05$).

training value that was not different from that seen in SE (0.199 ± 0.014 mL O_2/J). The work rate–lactate curve obtained during the submaximal incremental test did not appear to shift as a result of training in either group, as indicated by an unchanged power production at 4 mM blood lactate (SE: 323.7 ± 44.7 vs 329.0 ± 34.7 W, E: 305.0 ± 20.6 vs 324.0 ± 21.7 W). Likewise, maximal blood lactate did not change with either type of training (SE: 12.6 ± 1.4 vs 13.2 ± 1.1 mM, E: 12.0 ± 1.9 vs 11.9 ± 1.1 mM).

Discussion

The present study is the first to investigate the effect of concurrent strength (S) training and endurance (E) training on short-term (5-min) and long-term (45-min) endurance capacity, mechanical muscle output, skeletal muscle cell size and vascularization in top-level (National Team) endurance athletes. As a main finding, concurrent SE training led to enhanced 45-min endurance capacity (improved time-trial performance) in this group of highly trained ($VO_{2max} \sim 75$ mL O_2/min/kg) athletes. Multiple factors were suggested to attribute to this adaptation, including an increased percentage of type IIA muscle fibers (area%) and training-induced gains in maximal muscle strength and rapid force capacity, respectively. Notably, the regime of concurrent strength and endurance (SE) training did not lead to muscle fiber hypertrophy or cause impaired vascularization.

Improvements in long-term and short-term endurance capacity

Endurance capacity evaluated as 45-min time-trial performance increased by 8% in the present group of endurance athletes (National Team cyclists) in response to 16 weeks of concurrent SE training. Comparable gains in long-term endurance capacity have been observed previously in well-trained cyclists (Rønnestad et al., 2010) and untrained-to-moderately trained individuals following concurrent SE training (Hickson et al., 1980, 1988; Marcinik et al., 1991; Izquierdo et al., 2005), where SE training produced greater improvements than E training alone (Hickson et al., 1980, 1988; Izquierdo et al., 2005; Rønnestad et al., 2010). Notably, in the present 45-min time-trial test, the relative pacing intensity calculated by dividing the 45-min work rate (mean power production) with the power production at 100% VO_{2max} ($Watt_{100\%}$, derived by linear extrapolation of the work rate production obtained at 75% VO_{2max}, $Watt_{75\%}$) increased by 9% after the period of SE training (from 75.9% to 83.1%), while remaining

unaffected by E training alone (80.4% vs 77.0%). Previous study data exist to support that concurrent SE training in high-level endurance athletes can increase long-term endurance capacity. Thus, an improved 5 K running time (from 18.3 to 17.8 min) was observed in well-trained runners when one-third of their normal running exercise was replaced by explosive-type strength training for 9 weeks (Paavolainen et al., 1999). Similarly, all-out cycling performance during a 5-min max test conducted immediately following 185 min submaximal cycling increased by 7% after 12 weeks of concurrent SE training in highly trained cyclists, suggesting that the sprint capacity in the final phase of a long-term endurance event can be enhanced by strength training (Rønnestad et al., 2009).

One likely mechanism for the improvement in 45-min endurance capacity observed in the present study is the increased proportion (area%) of type IIA muscle fibers following the period of concurrent SE training. Type IIA fibers are less fatigable yet highly capable of producing contractile power compared with the type IIX muscle fibers (cf. Bottinelli et al., 1999). In addition, the observed gains in maximal muscle strength (MVC) and rapid force capacity (RFD) are likely to also have contributed to the increase in 45-min endurance capacity (discussed in detail below). Concurrent SE training in competitive cyclists using ''power-type'' exercises with low loading intensity (30 RM loads, $\sim$20% 1 RM) for 9 weeks failed to produce greater gains in long-term endurance capacity (1-h lab time trial) than endurance training alone (Bastiaans et al., 2001). Likewise, an unaltered long-term endurance capacity was observed following low-intensity (50–80% 1 RM), low-volume (single exercise) strength training in well-trained female cyclists (Bishop et al., 1999). Conversely, studies demonstrating improved long-term endurance capacity (present data, Hickson et al., 1980, 1988; Marcinik et al., 1991; Rønnestad et al., 2009; Rønnestad et al., 2010) consistently have used high-volume protocols with heavy training loads ($\geq$ 85% 1 RM) lasting for an extended period of time (12–16 weeks). Notably, the use of such heavy training loads ($\geq$ 85% 1 RM) is known to result in significant neural adaptation (Aagaard, 2003).

In the present group of top-level endurance athletes, short-term endurance capacity measured during 5-min all-out cycle testing increased similarly following SE or E training, respectively (cf. Fig. 1). Maximal blood lactate and VO_{2max} did not differ between training groups and remained unchanged by training. Likewise, power production at 4 mM blood lactate remained unaffected by the present regimes of SE and E training, as recently also reported in well-trained cyclists (2 mM lactate threshold; Rønnestad et al., 2010). Concurrent SE training has been reported previously to lead to improved short-term ($<$10 min) endurance capacity measured as an increased time to exhaustion during treadmill running or ski ergometer testing in untrained subjects (Hickson et al., 1980), moderately trained individuals (Hickson et al., 1988) and well-to-highly trained athletes (Hoff et al., 1999, 2002; Østerås et al., 2002; Mikkola et al., 2007b; Støren et al., 2008; Losnegard et al., 2010).

Improvements in mechanical muscle function

The present study demonstrated increases of 12–20% in maximal muscle strength (MVC) and rapid muscle force capacity (RFD) in response to 16 weeks of concurrent strength and endurance (SE) training in highly trained top-level endurance athletes. Comparable gains in maximal muscle strength have been reported following concurrent SE training in untrained individuals (Marcinik et al., 1991; McCarthy et al., 2002; Häkkinen et al., 2003; Glowacki et al., 2004), moderately trained subjects (Hickson et al., 1988; Kraemer et al., 1995) and well-to-highly trained endurance athletes (Millet et al., 2002; Østerås et al., 2002; Mikkola et al., 2007b, 2007a; Støren et al., 2008), respectively. In contrast, MVC remained unaltered following endurance training alone (E), in accordance with previous data obtained in well-to-highly trained endurance athletes (Østerås et al., 2002; Millet et al., 2002; Mikkola et al., 2007b, 2007a; Støren et al., 2008; Losnegard et al., 2010). The present study further demonstrated that concurrent SE training can lead to improved rapid force characteristics (RFD) in top-level endurance athletes. Similar findings were recently reported after concurrent SE training in well-to-highly trained runners (Mikkola et al., 2007a; Støren et al., 2008) and cross-country skiers (Mikkola et al., 2007b). In contrast, RFD remained unaffected by endurance training alone (E) in the present study, as observed previously (Mikkola et al., 2007b, 2007a; Støren et al., 2008).

Notably, the present gains in maximal and rapid muscle force capacity were observed in the absence of muscle fiber hypertrophy, suggesting that training-induced improvements in neural function were mainly responsible for the observed muscle strength gains (for review, see Aagaard, 2003). Increases in maximal muscle strength and rapid force capacity (RFD) along with elevated neuromuscular activity typically are observed following periods of heavy-resistance strength training (Häkkinen et al., 1998; Aagaard et al., 2002; for a brief review, see Aagaard, 2003). The training-induced rise in RFD to a large part depends on neural adaptation mechanisms such as increased maximal MU firing frequency and increased excitability and/or reduced amounts of

pre/post synaptic inhibition of spinal α motor neurons (Aagaard, 2003). Similar neuronal training effects are likely to have been achieved for the present group of top-level endurance athletes following SE training.

The observed increase in maximal muscle strength (MVC) following SE training suggests that a pedal thrust force of a given absolute magnitude represented a reduced relative load (relative to max) following SE training, which might have contributed to the improvement in 45-min endurance performance. Further, the increase in contractile RFD with SE training may *per se* have contributed to the elevated cycling power output that was observed during the 45-min endurance test. Thus, the increase in RFD would enable to faster reach a high level of pedal force in each pedal thrust, hence producing a greater force–time integral (kinetic impulse) during the effective (i.e. propulsive) 120° pedal arch from top-dead center, which in turn could contribute to the observed gain in 45-min endurance performance. In addition, as previously suggested for cross-country skiers (Hoff et al., 1999) during cyclic muscle actions, the training-induced increase in RFD potentially allows for an elongated muscle relaxation phase with unrestricted blood flow perfusion. Thus, potential changes in muscle perfusion dynamics might have contributed to the enhanced time-trial performance observed in the present study.

Muscle fiber area and fiber-type composition

In the present study, concurrent SE training led to altered type IIA fiber proportions (area%) in the quadriceps (vastus lateralis) muscle, increasing from 26% to 34%, while remaining unchanged with E training alone (cf. Fig. 3). The elevated percentage of type IIA fibers appeared to occur mainly at the expense of a reduced type IIX area percentage. Notably, no signs of quadriceps muscle fiber hypertrophy or impaired capillarization were detected, despite the prolonged period of heavy-resistance training. Similarly, no muscle fiber hypertrophy was observed in the quadriceps (VL) muscle of moderately trained recreational endurance athletes (Hickson et al., 1988) or female cyclists (Bishop et al., 1999) following months of concurrent SE training, while anatomical quadriceps muscle CSA remained unaltered after 12 weeks of concurrent SE training in elite cross-country skiers (Losnegard et al., 2010). Thus, the endurance training stimuli delivered to the muscle cells during concurrent SE training may blunt the muscle hypertrophy response that otherwise is observed with heavy-resistance strength training (Kraemer et al., 1995; Häkkinen et al., 1998; Aagaard et al., 2001).

In support of this notion, absent or reduced muscle hypertrophy has been reported following concurrent SE training in untrained-to-moderately trained individuals (Kraemer et al., 1995; Bell et al., 2000), although not a universal finding (McCarthy et al., 2002; Häkkinen et al., 2003). In further support of a "atrophy" stimulus provided by endurance training, a reduction in muscle fiber CSA (atrophy) has been reported following extensive endurance training (Kraemer et al., 1995; Harber et al., 2004; Trappe et al., 2006). Recent studies have shown that resistance or endurance training activate distinct cell signaling involving the Akt/mTOR or AMPK pathways, respectively (Atherton et al., 2005), and that inhibitory cross-talk exist from one pathway to the other (Baar, 2006; Nader, 2006). Consequently, SE training may evoke concurrent opposing stimuli for cellular hypertrophy and atrophy, respectively. In result, no or highly attenuated muscle hypertrophy may occur in response to concurrent SE training (present data, Hickson et al., 1988; Kraemer et al., 1995, Bishop et al., 1999, Bell et al., 2000; Losnegard et al., 2010). On the other hand, significant muscle hypertrophy has been observed following concurrent SE training in both well-trained cyclists (Rønnestad et al., 2010) and previously untrained individuals (Häkkinen et al., 2003). The disparate findings may arise from study differences in total resistance training volume where low-volume protocols may result in no or only minor hypertrophy, differences in the temporal distribution ("timing") of resistance vs endurance exercise, differences in the timing of nutritional intake relative to the completion of resistance exercise sessions and/or differences in the genetic makeup of the experimental subjects. Furthermore, in the present study, lean body mass increased (~ 2 kg) following SE training despite that total body mass remained unchanged, suggesting that body composition remodeling and hypertrophy may have occurred for muscles other than the quadriceps (i.e. hip extensors, lower back extensors) – which hypothetically might have contributed to the improvement in 45-min all-out cycling performance observed following SE training.

Capillarization

Reflecting a very high training state, the present subjects demonstrated extreme vascularization characteristics (an average of seven to eight capillaries in contact with each myofiber), which did not differ between type I and II fibers. Importantly, capillarization was not compromised by the regime of concurrent SE training, whether analyzed for type I and II fibers separately or collapsed for all fibers. In line with this finding, the number of capillaries per fiber appears to either increase (Hather et al., 1991;

Green et al., 1998; Kadi & Thornell, 2000) or remain unchanged (Tesch et al., 1990; Bell et al., 2000) following prolonged strength training in untrained subjects. Likewise, capillary density (cap/mm^2) appears to remain unchanged (Hather et al., 1991; Green et al., 1998).

Aerobic power and CE

In the present study, VO_{2max} remained unaltered both with SE training or E training alone. Somewhat unexpectedly, CE was 10% lower in E than SE before the start of the study, which may explain the finding of improved CE (+8%) with E training alone, reaching a value similar to that observed in SE. It is possible that CE is already highly optimized in top-level (National Team) cyclists and when above a certain threshold this parameter is very difficult to improve, at least within weeks and months of training. In support of this notion, net and delta cycling efficiency during graded cycle ergometer testing remained unaffected in a group of competitive cyclists in response to 9 weeks of concurrent SE training (Bastiaans et al., 2001). Similarly, CE remained unchanged in well-trained competitive cyclists in response to 12 weeks of concurrent SE training followed by 13 weeks using strength maintenance training (Rønnestad et al., 2010). In contrast, concurrent SE training has been reported to lead to improved movement economy during running, cycling or skiing in previously untrained individuals (Loveless et al., 2005) as well as in moderate-to-well-trained athletes (Hoff et al., 1999, 2002; Paavolainen et al., 1999; Millet et al., 2002; Østerås et al., 2002; Mikkola et al., 2007b; Støren et al., 2008; Rønnestad et al., 2009), although not consistently demonstrated in all studies (Mikkola et al., 2007a).

Study limitations

The present fiber area data were characterized by a large within-group variance and a somewhat low number of experimental subjects, resulting in a low statistical power for this parameter (fiber CSA). Although difficult to achieve in this population (National Team athletes), future longitudinal biopsy studies should seek to recruit larger numbers of experimental subjects in order to reduce the potential risk of type II errors.

In conclusion, the present study demonstrated that concurrent heavy-resistance strength training and endurance training could elicit an enhanced 45-min time-trial performance in top-level (National Team) endurance athletes. Parallel gains in type IIA muscle fiber proportions, maximal muscle strength (MVC) and rapid force characteristics (RFD), respectively, were suggested to be responsible for the adaptive change in 45-min performance. Notably, muscle fiber size and capillarization were not compromised in this process.

Perspectives

Based on the present findings, it may be recommend to use heavy-resistance strength training in concurrence with ongoing endurance training to induce shifts toward a more fatigue-resistant type II muscle fiber profile and an elevated rapid force capacity in elite endurance athletes. These effects may be achieved without gains in body mass or measurable muscle fiber hypertrophy in prime mover muscles. In a likely consequence hereof, prolonged concurrent strength and endurance training led to an enhanced 45-min time-trial performance in the present group of highly trained young National Team cyclists.

Key words: muscle, endurance, RFD, elite athletes.

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