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Review

Effects of strength training on endurance capacity in top-level endurance athletes

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The effect of concurrent strength (S) and endurance (E) training on adaptive changes in aerobic capacity, endurance performance, maximal muscle strength and muscle morphology is equivocal. Some data suggest an attenuated cardiovascular and musculoskeletal response to combined E and S training, while other data show unimpaired or even superior adaptation compared with either training regime alone. However, the effect of concurrent S and E training only rarely has been examined in top-level endurance athletes. This review describes the effect of concurrent SE training on short-term and long-term endurance performance in endurance-trained subjects, ranging from moderately trained individuals to elite top-level athletes. It is concluded that strength training can lead to enhanced long-term (>30 min) and short-term (<15 min) endurance capacity both in well-trained individuals and highly trained top-level endurance athletes, especially with the use of high-volume, heavy-resistance strength training protocols. The enhancement in endurance capacity appears to involve training-induced increases in the proportion of type IIA muscle fibers as well as gains in maximal muscle strength (MVC) and rapid force characteristics (rate of force development), while likely also involving enhancements in neuromuscular function.

Equivocal reports exist for the effect of concurrent strength (S) and endurance (E) training on adaptive changes in aerobic capacity, endurance performance, maximal muscle strength and muscle morphology. Thus, previous studies have reported a diminished range of cardiovascular and musculoskeletal adaptation, respectively, when S and E training regimes were combined (Hickson et al., 1980; Dudley & Djamil, 1985; Hunter et al., 1987; Nelson et al., 1990; Kraemer et al., 1995; Glowacki et al., 2004; Izquierdo et al., 2005). In contrast, other data have suggested that concurrent endurance and strength (resistance) training can lead to similar cardiovascular or musculoskeletal adaptations compared with either training regime alone (Bell et al., 1991; McCarthy et al., 1995, 2002; Izquierdo et al., 2005), or that concurrent endurance and strength training may even 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., 2010; Losnegaard et al., 2010). However, the effect of concurrent S and E training on endurance capacity in relation to muscle morphology, fiber type composition and contractile muscle function have not been examined previously in top-level endurance athletes.

Previous studies conducted in untrained individuals and moderately to well-trained endurance athletes have indicated that both short-term endurance capacity (Hickson et al., 1980, 1988; Hoff et al., 1999, 2002; Østerås et al., 2002; Støren et al., 2008; Losnegaard et al., 2010) and long-term endurance capacity (Hickson et al., 1980, 1988; Marcinik et al., 1991) can be improved in response to strength training. In contrast, the effect of concurrent strength training on the endurance capacity during long-term exercise (>30 min) only very rarely has been addressed in top-level endurance athletes.

Changes in short-term endurance capacity

Concurrent SE training has been reported to lead to improved short-term (<15 min) endurance capacity measured as an increased time to exhaustion during treadmill running or ski ergometer testing in untrained subjects (Hickson et al., 1980), well-trained recreational endurance athletes (Hickson et al., 1988), well-to-highly trained cross-country skiers (Hoff et al., 1999, 2002; Østerås et al., 2002; Mikkola et al., 2007b; Losnegaard et al., 2010) and well-trained distance runners (Støren et al., 2008) and competitive cyclists (Sunde et al., 2009). Using a longitudinal comparative setting, the gain in short-term endurance performance was observed to be

greater following concurrent SE training than E training alone (Hoff et al., 1999, 2002; Støren et al., 2008; Sunde et al., 2009). A few of these studies examined competitive athletes (Hoff et al., 2002; Østerås et al., 2002; Sunde et al., 2009; Losnegaard et al., 2010); however, on a group basis, these individuals could not strictly be characterized as absolute top-level endurance athletes since demonstrating a VO_{2max} below 70 mL O_2/min/kg. A borderline exception might be the study by Hoff et al. (2002) who looked at male cross-country skiiers with an average VO_{2max} of 69–70 mL O_2/min/kg (SD: 2.3).

Early studies by Hickson et al. (1980) reported that the time to exhaustion during treadmill running and ergometer biking at 100% of their pretraining VO_{2max} (<7 min) could be increased by 12 and 47%, respectively, in previously untrained individuals in response to 10 weeks of strength training that left VO_{2max} unaffected. These data were among the first to demonstrate that short-term endurance capacity can be positively affected by strength training. In a subsequent classical study, short-term endurance capacity increased by 11–13% during standardized maximal treadmill running and cycling exercise (4–8 min duration) in well-trained recreational endurance athletes (VO_{2max} ~ 60 mL/min/kg) when a regime of multi-exercise heavy-resistance strength training was performed concurrently with their regular endurance training for 10 weeks (Hickson et al., 1988). Similarly, recent data obtained in well-trained male and female runners (VO_{2max} ~ 56–61 mL/min/kg) showed 21% increased time to exhaustion (5.6–6.8 min) during treadmill running at a speed corresponding to 100% of VO_{2max} when heavy-resistance strength training was added to the scheme of endurance training over an 8-week period (Støren et al., 2008).

Similar to the above findings in runners, adaptive changes in short-term endurance capacity with concurrent strength training have been demonstrated in well-trained to highly trained cross-country skiers. Thus, strength training for 8 weeks led to an improved time to exhaustion (from 6.5 to 10.2 min) during all-out testing on a double poling ski ergometer at W_{max} (velocity at VO_{2max}) in highly trained male cross-country skiers (VO_{2max} ~ 70 mL/min/kg) (Hoff et al., 2002). Similar findings were reported for well-trained female cross-country skiers (VO_{2max} ~ 55 mL/min/kg) in response to 9 weeks of concurrent S and E training (Hoff et al., 1999).

Changes in long-term endurance capacity with strength training

The effect of strength training on long-term (>30 min) endurance performance almost exclusively have been examined in untrained-to-moderately trained individuals (i.e. Hickson et al., 1980, 1988; Marcinik et al., 1991; Bishop et al., 1999), and only a single study have involved highly trained endurance athletes (Bastiaans et al., 2001). In the latter study, long-term endurance capacity (60-min time trial) increased to a similar extent in well-trained competitive cyclists in response to concurrent SE training or E training alone, respectively (Bastiaans et al., 2001). An improved 5K running time (18.3–17.8 min) was observed in well-trained runners (VO_{2max} of 68 mL/min/kg) when about 30% of their normal running training was changed into explosive-type strength training; however, no measures of long-term (>30 min) endurance capacity were obtained (Paavolainen et al., 1999).

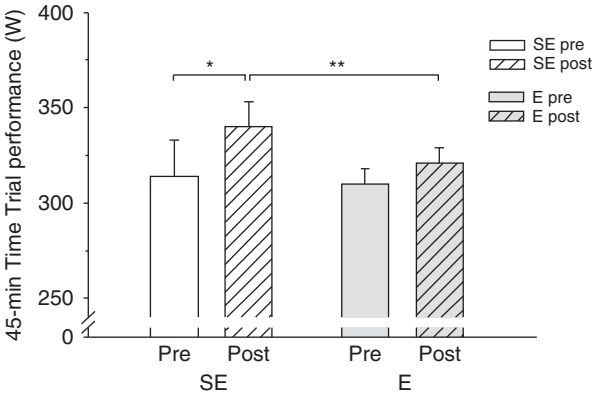

Fig. 1. Long-term endurance performance measured as average biking power output (± SD) during an all-out 45-min time trial in elite (National Team) road cycling athletes pre- and post 16 weeks of concurrent strength and endurance training (SE) or endurance training alone (E). Average power (biking speed) increased 8% after SE training and was not affected by E training alone. *Post > pre ($P<0.05$), **SE > E ($P<0.01$). Data adapted from Aagaard et al. (2010).

We recently examined the effect of strength training on short/long-term endurance capacity, mechanical muscle output, skeletal muscle fiber size, fiber type composition and muscle vascularization in top-level endurance athletes, where a regime of heavy-resistance strength training was found to result in enhanced long-term endurance capacity (improved 45-min time trial performance) in highly trained National Team cyclists (VO_{2max} ~ 71–75 mL O_2/min/kg) (Aagaard et al., 2007, 2010) (Fig. 1). These changes were accompanied by an increased proportion of type IIA fibers and reduced proportion of type IIX fibers, elevated maximal muscle strength (MVC) and increased rapid force capacity [elevated rate of force development (RFD)] (Aagaard et al., 2007, 2010). Importantly, the regime of concurrent strength and endurance (SE) training did not result in cellular muscle hypertrophy or reduced muscle capillary density in the group of highly trained endurance athletes (Aagaard et al., 2007, 2010). In

accordance with these recent findings in top-level endurance athletes, improved long-term endurance previously have been observed in untrained-to-moderately trained individuals following a prolonged period of concurrent SE training (Hickson et al., 1980, 1988; Marcinik et al., 1991; Izquierdo et al., 2005), with SE training producing greater improvements than E training alone (Hickson et al., 1980, 1988; Izquierdo et al., 2005). Interestingly, improved long-term endurance performance has failed to be demonstrated in studies using low-volume (<8 weeks duration) and/or low-intensity (<80% 1 RM loadings) strength training, which typically have comprised use of a single exercise, low-training intensity (<60% 1 RM) and/or short-duration exercise periods (Bishop et al., 1999; Bastiaans et al., 2001; Levin et al., 2009). Taken together, the available data suggest that a high muscle loading intensity (85–95% 1 RM) and/or a large volume of strength training need to be performed before a benefit on long-term endurance performance can be achieved. Importantly, maximal (≥ 85% 1 RM) (Hoff et al., 1999, 2002; Aagaard et al., 2007, 2010; Støren et al., 2008; Rønnestad et al., 2010; Sunde et al., 2009) and/or explosive-type (Paavolainen et al., 1999; Mikkola et al., 2007a, b) strength training appears to be more advantageous in endurance athletes (well trained to top level) than the hypertrofic type of training.

Notably, concurrent S and E training can lead to elevated maximal muscle strength even in the absence of muscle fiber hypertrophy (Hickson et al., 1988; Bishop et al., 1999; Aagaard et al., 2007, 2010) or gains in anatomical muscle CSA (Losnegaard et al., 2010). The latter finding is especially important to top-level endurance athletes, who typically intend to avoid gains in muscle mass, as elevated muscle mass is thought to be detrimental for an optimal endurance capacity within endurance sports where muscle forces are generated to support the body mass against gravity (i.e. running, cycling). Also, cellular hypertrophy effects are often undesirable in the top-level endurance athlete because an increased single muscle fiber area would lead to an increased diffusion distance from the exterior to the interior (central portions) of the muscle cell, thereby potentially compromising the transport of glucose and free fatty acid (FFA) from the capillary bed into the muscle cells as well as potentially leading to a reduced removal of excessive heat production from the working muscle(s), altogether resulting in impaired long-term endurance capacity.

Longitudinal data obtained in previously untrained individuals suggest that resistance exercise (i.e. strength training) per se may provide a stimulus for capillary neoformation (Hather et al., 1991; McCall et al., 1996; Green et al., 1998; Kadi & Thornell, 2000) and capillary density (cap/mm^2) (Hather et al., 1991; McCall et al., 1996; Green et al., 1998) appears to remain unchanged or even increase despite the presence of cellular muscle hypertrophy. The above findings suggest that resistance exercise (strength training) may provide an effective stimulus for angiogenesis, which may hold true even for high-level endurance athletes despite that these subjects are already characterized by a very high degree of muscular vascularization. Importantly, concurrent endurance and strength training can diminish or fully blunt the muscle hypertrophy that normally occurs with strength training, while increases in maximal muscle strength are still observed (Hickson et al., 1988; Kraemer et al., 1995; Bishop et al., 1999; Aagaard et al., 2007, 2010), the latter likely as a result of neuromuscular adaptation (Aagaard, 2003). This gain in mechanical muscle output in the absence of cellular hypertrophy may also result in enhanced short-term and long-term endurance performance in highly trained endurance athletes.

Adaptive mechanisms

The likely candidates for the observed improvement in long-term endurance capacity comprise an increased proportion of type IIA muscle fibers (Aagaard et al., 2007, 2010) that are less fatigable and yet highly capable of producing high contractile power (Bottinelli et al., 1999). In addition, concurrent SE training led to substantial gains in maximal muscle strength (MVC) and rapid force capacity (RFD) in top-level endurance athletes (National Team cyclists), which are likely to have contributed to the observed increase in long-term endurance capacity manifested by a 7% increased mean Watt production in a standardized 45-min time trial performed in the lab (Aagaard et al., 2007, 2010). Concurrent SE training in competitive cyclists has failed previously to produce greater gains in long-term endurance capacity (1-h lab time trial) than endurance training alone (Bastiaans et al., 2001).

A similar lack of improved long-term endurance capacity was observed in well-trained cyclists/triathletes (30-km time trial) in response to 6 weeks of strength training using a mix of strength and power exercises (Levin et al., 2009), and in well-trained female cyclists (1-h cycling) after 12 weeks of low-volume (single exercise) strength training (Bishop et al., 1999). However, as discussed above, these negative findings may have been caused by the use of short-term (9 weeks), low-resistance (i.e. "power" type) strength training (Bastiaans et al., 2001), rather than long-term (>12 weeks), heavy-resistance training (Aagaard et al., 2007, 2010) that appears to be more optimal for eliciting neuromuscular adaptation

(Aagaard, 2003) and inducing shifts in fiber type composition (Andersen & Aagaard, 2000). Additionally, the lack of adaptation in long-term endurance capacity could arise from a short duration of training (Levin et al., 2009), and/or a low volume of strength training performed (Bishop et al., 1999).

For cycling athletes, the increase in maximal muscle strength observed following concurrent SE training means that when producing a pedal thrust force of a certain magnitude (corresponding to a given Watt production for a given pedaling cadency), this will represent a reduced relative load (relative to max), which in turn may have contributed to the observed enhancement in long-term endurance performance (Hickson et al., 1988; Aagaard et al., 2007, 2010; Rønnestad et al., 2010). Further, an increase in rapid force capacity (RFD) also was observed following concurrent SE training in top-level cyclists (Aagaard et al., 2007, 2010), which may also contribute to a reduced degree of muscle fiber exhaustion for a given cycle power output during long-term endurance (time trial) events. Thus, the observed increase in RFD following SE training would enable cyclists to more rapidly produce pedal force and thereby allow for a more prolonged relaxation phase in each pedal revolution. Similar arguments have been forwarded to explain the improved short-term performance and economy observed in high-level cross-country skiers after concurrent SE training (Hoff et al., 1999), namely that the observed training-induced increase in RFD would allow for a shorter propulsion phase for a given overall power output, hence allowing for an extended muscle relaxation phase. The prolonged muscle relaxation phase would reduce the time of contraction-induced muscle occlusion, and hence increase the time of muscle perfusion given the prolonged relaxation phase, thereby increasing the mean capillary transit time (MTT). Thus, a prolonged muscle relaxation phase potentially would allow for an increased MTT per pedal revolution. In turn, due to the relatively large molecular size of FFAs, it has been suggested that an increased MTT could enable an increased diffusion of FFA into the muscle cells, hence potentially sparing the rate of muscle glycogen breakdown and thereby delaying the onset of muscle fatigue (Kiens et al., 1993). Further, a longer MTT potentially could lead to an enhanced removal of metabolites produced by the contracting muscle fibers, which might contribute to the enhanced long-term endurance performance that was observed in top-level athletes in response to concurrent SE training (Aagaard et al., 2007, 2010). Because currently based on theoretical speculations, future experiments should examine the potential interaction between concurrent strength-endurance training and altered muscle perfusion dynamics during endurance exercise (longer MTT).

It is likely that the involvement of heavy-resistance exercise over a prolonged period of time in highly trained endurance athletes may evoke changes in neural function that will contribute to the observed gain in maximal muscle strength and rapid force capacity, respectively (Aagaard, 2003). Thus, a high volume of heavy-resistance and/or explosive-type strength training previously have been shown to induce increased neuromuscular activity during maximal muscle contraction efforts that resulted in an elevated MVC and RFD (Häkkinen et al., 1998; Van Cutsem et al., 1998; Aagaard et al., 2002a; Del Balso & Cafarelli, 2007). This improvement in neural function may comprise spinal as well as supraspinal adaptation mechanisms (Aagaard et al., 2002b; Del Balso & Cafarelli, 2007; Duclay et al., 2008). Interestingly, neuromuscular activity (VL muscle) recorded during MVC appeared to remain unchanged following 21 weeks of low-frequency concurrent SE training in untrained individuals (Häkkinen et al., 2003), which suggests that a relatively high volume of S training may be necessary to induce neuromuscular adaptations in response to concurrent training regimes.

Changes in economy of movement

Studies conducted in untrained individuals (Loveless et al., 2005), well-to-highly trained runners (Paavolainen et al., 1999; Østerås et al., 2002; Støren et al., 2008), cross-country skiers (Hoff et al., 1999, 2002; Mikkola et al., 2007b), triathletes (Millet et al. 2002), and moderately to well-trained cyclists (Rønnestad et al., 2010; Sunde et al., 2009) have reported that concurrent SE training or S training alone can lead to improved economy of movement compared with E training alone, although not always a uniform finding (Aagaard et al., 2007, 2010; Mikkola et al., 2007a). Cycling economy did not improve in top-level (National Team) cyclists following 16 weeks of concurrent SE training (Aagaard et al., 2007, 2010). It is possible, however, that cycling economy may already be highly optimized in top-level cyclists and therefore highly 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).

In a recent study conducted in well-trained cyclists ($VO_{2max} \sim 66$–$70\,mL\,O_2/min/kg$), Raastad and colleagues demonstrated that cycling economy was improved to a greater extent by concurrent SE training than E training alone during the final hour of a 185-min long cycling test, which was accompanied by a reduced rise in heart rate and blood lactate, respectively (Rønnestad et al., 2010) (Fig. 2).

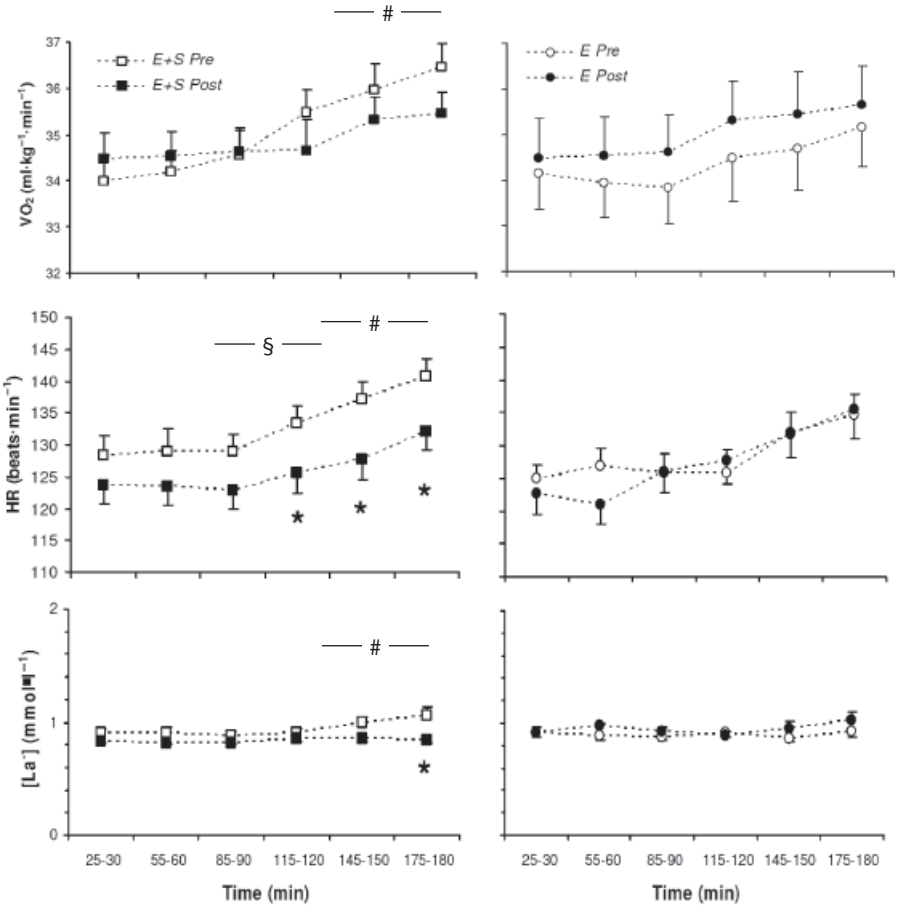

Fig. 2. Oxygen uptake (VO_2; top), heart rate (HR; mid) and blood lactate concentration ($[La^-]$; bottom) during 180 min of cycling at 44% of baseline W_{max} before (Pre) and after (Post) 12 weeks of concurrent endurance (cycling) and strength training (E+S; left) and endurance (cycling) training alone (E; right). W_{max} was calculated as the mean power output during the final 2 min of a short-term all-out incremental VO_{2max} test. *Post different from pre ($P<0.05$), relative changes (pre-to-post training) different between E+S and E at 120–180 min (#, $P<0.01$) and 60–120 min (§, $P<0.01$). Data adapted from Rønnestad et al. (2010).

Furthermore, all-out cycling performance measured during a 5-min max test conducted immediately at the end of the 185-min cycling was substantially improved following SE training while unaffected by E training alone (7% improved average power production), suggesting that sprint capacity in the final phase of a race can be enhanced by strength training (Rønnestad et al., 2010) (Fig. 3). Likewise, cycling economy was improved following 8 weeks of concurrent cycling and strength training in well-trained competitive cyclists (VO_{2max} of 58–64 mL O_2/min/kg) (Sunde et al., 2009), while similar findings were demonstrated in moderately to well-trained individuals (VO_{2max} ~ 52–62 mL O_2/min/kg) after 12 weeks of single-mode strength training (Hansen et al., 2007).

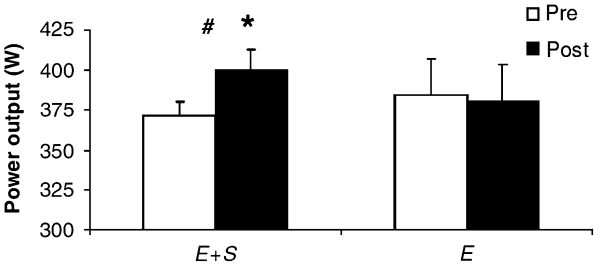

Fig. 3. Mean power output during a 5-min all-out trial performed following 185 min of cycling at 44% of baseline W_{max} before (Pre) and after (Post) 12 weeks of concurrent endurance (cycling) and strength training (E+S) or endurance (cycling) training alone (E). *Post different from pre ($P<0.01$). #Difference between E+S and E in relative change from pre-test to post-test ($P<0.01$). Data adapted from Rønnestad et al. (2010).

Lack of muscle fiber hypertrophy with concurrent training in endurance athletes

As mentioned above, 16 weeks of concurrent SE training in National Team cyclists resulted in altered type IIA fiber proportions (increased % area) in the quadriceps (VL) muscle, which increased from 26 to 34% while remaining unchanged with E training alone (Aagaard et al., 2007, 2010) (Fig. 4). The elevated percentage of type IIA fibers appeared to

occur at the expense of reduced type IIX area. Notably, no signs of quadriceps muscle fiber hypertrophy were detected, despite the prolonged period of heavy-resistance strength training, and muscle fiber capillarization also remained unchanged with either SE or E training (Aagaard et al., 2007, 2010).

In a classical study, Hickson et al. (1988) were also unable to demonstrate any measurable muscle fiber hypertrophy (VL muscle) in moderately trained recreational endurance athletes after an extensive regime of concurrent SE training. In accordance, no signs of muscle fiber hypertrophy were observed in a group of moderately trained female cyclists in response to a protocol of low-volume strength training (Bishop et al., 1999) while anatomical quadriceps muscle CSA remained unaltered after 12 weeks of concurrent SE training in elite cross-country skiers (Losnegaard et al., 2010). Recent studies have shown that distinct cell signaling events involving the Akt/mTOR or AMPK pathways appear to become activated by resistance or endurance training, respectively (Atherton et al., 2005), and that inhibitory cross-talk exists from one pathway to the other (Nader 2006; Baar 2006). Consequently, the endurance training stimuli delivered to the muscle cells during concurrent SE training may effectively blunt the muscle hypertrophy response that is normally observed in response to heavy-resistance strength training alone. In support of this notion, absent or reduced muscle hypertrophy may occur in response to concurrent strength and endurance training in untrained to moderately trained individuals (Kraemer et al., 1995; Bell et al., 2000), although not a universal finding (McCarthy et al., 2002).

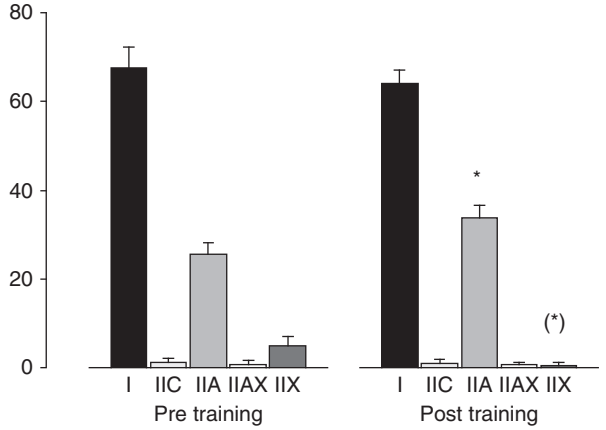

Fig. 4. Muscle fiber type distribution based on muscle fiber area obtained in the quadriceps muscle (VL) pre and post 16 weeks of concurrent strength and endurance training in a group of male elite (National Team) cycling athletes. *Type IIA: post > pre ($P < 0.05$), $^{(*)}$trend for type IIX: post < pre ($P = 0.08$). No changes were observed in cycling athletes who did not perform strength training. Data adapted from Aagaard et al. (2010).

In further support of an atrophy stimulus provided by endurance training, a reduction in muscle fiber CSA (atrophy) has been observed following intensive endurance training (Terados et al., 1986; Ratzin Jackson et al., 1990; Kraemer et al., 1995; Harber et al., 2004; Trappe et al., 2006). From a muscle

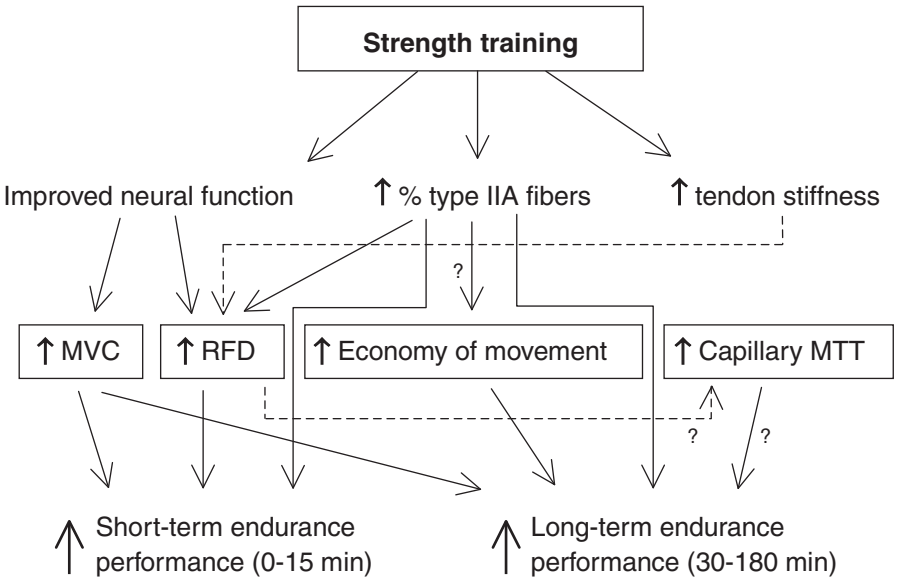

Fig. 5. Proposed mechanisms by which short-term and long-term endurance performance can be increased in well-trained to highly trained endurance athletes by the addition of strength training to ongoing endurance training. MVC, maximal muscle strength; RFD, rate of force development (Δforce/Δtime) or rapid muscle strength; MTT, mean transit time; economy of movement: oxygen uptake rate (VO_2) when moving a certain distance at a certain speed, a reduced VO_2 reflects an improved economy. Full lines indicate a stimulatory effect, and dotted lines indicate proposed potential interactions that await experimental verification with concurrent strength and endurance training. Question mark indicates a potential effect that awaits general experimental verification (see text for more details).

perfusion perspective, this adaptation could be highly important, because it results in an elevated capillary to muscle fiber CSA ratio, which in turn facilitates O_2 delivery and FFA uptake into the muscle cell due to the reduced diffusion distance. In turn, an elevated FFA uptake would result in a reduced rate of glycogen breakdown, which potentially would lead to an enhanced endurance performance (prolonged time to exhaustion). Overall, these data suggest that concurrent strength and endurance training evoke concurrent opposing stimuli for cellular hypertrophy and atrophy, respectively. In consequence, no muscle fiber hypertrophy and no signs of a reduction in capillary density have been observed when strength and endurance training are combined (Hickson et al., 1988; Bell et al., 2000; Aagaard et al., 2007, 2010). It should be noted that in previously untrained individuals, strength training per se appears to increase the number of capillaries per fiber (Hather et al., 1991; McCall et al., 1996; Green et al., 1998; Kadi & Thornell, 2000) or result in unchanged capillarization (Luhti et al., 1986; Tesch et al., 1990; Bell et al., 2000). Likewise, capillary density (cap/mm^2) appears to remain unchanged (Hather et al., 1991; McCall et al., 1996; Green et al., 1998). Muscle capillarization was not improved (nor compromised) when concurrent strength training was performed in elite endurance athletes characterized by a high initial capillary density ($\sim$ 6–700 cap/mm^2, 7–8 cap/fiber) (Aagaard et al., 2009). Thus, concurrent strength training in top-level endurance athletes does not appear to negatively affect muscle perfusion capacity (capillary density), at least when strength training is performed for up to 20 weeks.

Conclusions

Experimental data demonstrate that strength training can lead to enhanced long-term (>30 min) and short-term (<15 min) endurance capacity both in well-trained individuals and highly trained top-level endurance athletes, especially (but not exclusively) when high-volume, heavy-resistance strength training protocols are applied. As summarized in Fig. 5, the enhancement in long-term endurance capacity appears to involve training-induced increases in the proportion of type IIA muscle fibers as well as gains in maximal muscle strength (MVC) and rapid force characteristics (RFD), while also likely involving enhanced neuromuscular function.

Key words: Aerobic capacity, concurrent training adaptions, muscle, neuromuscular function.

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References

Aagaard P. Training-induced changes in neural function. Exerc Sports Sci Rev 2003: 31(2): 61–67.

Aagaard P, Andersen JL, Bennekou M, Larsson B, Olesen J, Crameri R, Magnusson SP, Kjær M. Effects of resistance training on endurance capacity, muscle morphology and fiber type composition in young top-level cyclists. Scand J Med Sci Sports 2010 (in review).

Aagaard P, Bennekou M, Larsson B, Andersen JL, Olesen J, Crameri R, Magnusson PS, Kjaer M. Resistance training leads to altered muscle fiber type composition and enhanced long-term cycling performance in elite competitive cyclists. Med Sci Sports Exerc 2007: 39(5, Suppl.): S448–S449 [abstract].

Aagaard P, Simonsen EB, Andersen JL, Magnusson P, Dyhre-Poulsen P. Increased rate of force development and neural drive of human skeletal muscle following resistance training. J Appl Physiol 2002a: 93: 1318–1326.

Aagaard P, Simonsen EB, Andersen JL, Magnusson SP, Dyhre-Poulsen P. Neural adaptation to resistance training: changes in evoked V-wave and H-reflex responses. J Appl Physiol 2002b: 92: 2309–2318.

Andersen JL, Aagaard P. Myosin heavy chain IIX overshooting in human skeletal muscle. Muscle Nerve 2000: 23: 1095–1104.

Atherton PJ, Babraj J, Smith K, Singh J, Rennie MJ, Wackerhage H. Selective activation of AMPK-PGC-1 alpha or PKB-TSC2-mTOR signaling can explain specific adaptive responses to endurance or resistance training-like electrical muscle stimulation. FASEB J 2005: 19: 786–788.

Baar K. Training for endurance and strength: lessons from cell signaling. Med Sci Sports Exerc 2006: 38: 1939–1944.

Bastiaans JJ, Van Diemen AB, Veneberg T, Jeukendrup AE. The effects of replacing a portion of endurance training by explosive strength training on performance in trained cyclists. Eur J Appl Physiol 2001: 86: 79–84.

Bell GJ, Peterson SR, Wessel J, Bagnell K, Quinney HA. Physiological adaptations to concurrent endurance and low velocity resistance training. Int J Sports Med 1991: 4: 384–390.

Bell GJ, Syrotuik D, Martin TP, Burnham R, Quinney HA. Effect of concurrent strength and endurance training on skeletal muscle properties and hormone concentrations in humans. Eur J Appl Physiol 2000: 81: 418–427.

Bishop D, Jenkins DG, Mackinnon LT, McEniery M, Carey MF. The effects of strength training on endurance

performance and muscle characteristics. Med Sci Sports Exerc 1999: 31: 886–891.
Bottinelli R, Pellegrino MA, Canepari R, Rossi R, Reggiani C. Specific contributions of various muscle fibre types to human muscle performance: an in vitro study. J Electromyogr Kinesiol 1999: 9: 87–95.
Del Balso C, Cafarelli E. Adaptations in the activation of human skeletal muscle induced by short-term isometric resistance training. J Appl Physiol 2007: 103: 402–411.
Duclay J, Martin A, Robbe A, Pousson M. Spinal reflex plasticity during maximal dynamic contractions after eccentric training. Med Sci Sports Exerc 2008: 40: 722–734.
Dudley GA, Djamil R. Incompatibility of endurance- and strength-training modes of exercise. J Appl Physiol 1985: 59: 1446–1451.
Glowacki SP, Martin SE, Maurer A, Baek W, Green JS, Crouse SF. Effects of resistance, endurance, and concurrent exercise on training outcomes in men. Med Sci Sports Exerc 2004: 36: 2119–2127.
Green H, Goreham C, Ouyang J, Ball-Burnett M, Ranney D. Regulation of fiber size, oxidative potential, and capillarization in human muscle by resistance training. Am J Physiol 1998: 276: R591–R596.
Häkkinen K, Alen M, Kraemer WJ, Gorostiaga E, Izquierdo M, Rusko H, Mikkola J, Häkkinen A, Valkeinen H, Kaarakainen E, Romu S, Erola V, Ahtiainen J, Paavolainen L. Neuromuscular adaptations during concurrent strength and endurance training versus strength training. Eur J Appl Physiol 2003: 89: 42–52.
Häkkinen K, Kallinen M, Izquierdo M, Jokelainen K, Lassila H, Mälkiä E, Kraemer WJ, Newton RU, Alén M. Changes in agonist-antagonist EMG, muscle CSA, and force during strength training in middle-aged and older people. J Appl Physiol 1998: 84: 1341–1349.
Hansen EA, Raastad T, Hallén H. Strength training reduces freely chosen pedal rate during submaximal cycling. Eur J Appl Physiol 2007: 101: 419–426.
Harber MP, Gallagher PM, Creer AR, Minchev KM, Trappe SW. Single muscle fiber contractile properties during a competitive season in male runners. Am J Physiol 2004: 287: R1124–R1131.
Hather BM, Tesch P, Buchanan P, Dudley GA. Influence of eccentric actions on skeletal muscle adaptations to resistance training. Acta Physiol Scand 1991: 143: 177–185.
Hickson RC, Dvorak BA, Gorostiaga EM, Kurowski TT, Foster C. Potential for strength and endurance training to amplify endurance performance. J Appl Physiol 1988: 65: 2285–2290.
Hickson RC, Rosenkoetter MA, Brown MM. Strength training effects on aerobic power and short-term endurance. Med Sci Sports Exerc 1980: 12: 336–339.
Hoff J, Gran A, Helgerud J. Maximal strength training improves aerobic endurance performance. Scand J Med Sci Sports 2002: 12: 288–295.
Hoff J, Helgerud J, Wisløff U. Maximal strength training improves work economy in trained female cross-country skiers. Med Sci Sports Exerc 1999: 31: 870–877.
Hunter G, Demment R, Miller D. Development of strength and maximum oxygen uptake during simultaneous training for strength and endurance. J Sports Med Phys Fitness 1987: 27: 269–275.
Izquierdo M, Häkkinen K, Ibáñez J, Kraemer WJ, Gorostiaga EM. Effects of combined resistance and cardiovascular training on strength, power, muscle cross-sectional area, and endurance markers in middle-aged men. Eur J Appl Physiol 2005: 94: 70–75.
Kadi F, Thornell LE. Concomitant increases in myonuclear and satellite cell content in female trapezius muscle following strength training. Histochem Cell Biol 2000: 113: 99–103.
Kiens B, Essen-Gustavsson B, Christensen NJ, Saltin B. Skeletal muscle substrate utilization during submaximal exercise in man: effect of endurance training. J Physiol 1993: 469: 459–478.
Kraemer WJ, Patton JF, Gordon SE, Harman EA, Deschenes MR, Reynolds K, Newton RU, Triplett NT, Dziados JE. Compatibility of high-intensity strength and endurance training on hormonal and skeletal muscle adaptations. J Appl Physiol 1995: 78: 976–989.
Levin GT, McGuigan MR, Laursen PB. Effect of concurrent resistance and endurance training on physiologic and performance parameters of well-trained endurance cyclists. J Strength Cond Res 2009: 23: 2280–2286.
Loveless DJ., Weber CL, Haseler LJ, Schneider DA. Maximal leg-strength training improves cycling economy in previously untrained men. Med Sci Sports Exerc 2005: 37: 1231–1236.
Luhti JM, Howald H, Claasen H, Rosler K, Vock P, Hoppeler H. Structural changes in skeletal muscle tissue with heavy-resistance exercise. Int J Sports Med 1986: 7: 123–127.
Marcinik EJ, Potts J, Schlabach G, Will S, Dawson P, Hurley BF. Effects of strength training on lactate threshold and endurance performance. Med Sci Sports Exerc 1991: 23: 739–743.
McCall GE, Byrnes WC, Dickinson A, Pattany PM, Fleck SJ. Muscle fiber hypertrophy, hyperplasia, and capillary density in college men after resistance training. J Appl Physiol 1996: 81: 2004–2012.
McCarthy JP, Agre JC, Graf BK, Pozniak MA, Vailas AC. Compatibility of adaptive responses with combining strength and endurance training. Med Sci Sports Exerc 1995: 27: 429–436.
McCarthy JP, Pozniak MA, Agre JC. Neuromuscular adaptations to concurrent strength and endurance training. Med Sci Sports Exerc 2002: 34: 511–519.
Mikkola J, Rusko H, Nummela A, Pollari T, Häkkinen K. Concurrent endurance and explosive type strength training improves neuromuscular and anaerobic characteristics in young distance runners. Int J Sports Med 2007a: 28: 602–611.
Mikkola JS, Rusko HK, Nummela AT, Paavolainen LM, Häkkinen K. Concurrent endurance and explosive type strength training increases activation and fast force production of leg extensor muscles in endurance athletes. J Strength Cond Res 2007b: 21: 613–620.
Millet GP, Jaouen B, Borrani F, Candau R. Effects of concurrent endurance and strength training on running economy and VO2 kinetics. Med Sci Sports Exerc 2002: 34: 1351–1359.
Nader GA. Concurrent strength and endurance training: from molecules to man. Med Sci Sports Exerc 2006: 38: 1965–1970.
Nelson AG, Arnall DA, Loy SF, Silvester LJ, Conlee RK. Consequences of combining strength and endurance training regimens. Phys Ther 1990: 70: 287–294.
Østerås H, Helgerud J, Hoff J. Maximal strength-training effects on force–velocity and force–power relationships explain increases in aerobic performance in humans. Eur J Appl Physiol 2002: 88: 255–263.
Paavolainen L, Häkkinen K, Hämäläinen I, Nummela A, Rusko H. Explosive strength training improves 5-km running time by improving running economy and muscle power. J Appl Physiol 1999: 86: 1527–1533.
Ratzin Jackson C, Dickinson AL, Ringel SP. Skeletal muscle fiber area alterations in two opposing modes of resistance-exercise training in the same

individual. Eur J Appl Physiol 1990: 61: 37–41.
Rønnestad BR, Hansen EA, Raastad T. Strength training improves 5-min all-out performance following 185 min of cycling. Scand J Med Sci Sports 2010 (in press).
Støren O, Helgerud J, Støa EM, Hoff J. Maximal strength training improves running economy in distance runners. Med Sci Sports Exerc 2008: 40: 1089–1094.
Sunde A, Støren Ø, Bjerkass M, Larsen MH, Hoff J, Helgerud J. Maximal strength training improves cycling economy in competitive cyclists. J Strength Cond Res 2009. DOI: 10.1519/JSC.0b013e3181aeb16a.
Terados N, Melichna J, Sylven C, Jansson E. Decrease in skeletal muscle myoglobin with intensive training in man. Acta Physiol Scand 1986: 128: 651–652.
Tesch PA, Thorson A, Colliander EB. Effects of eccentric and concentric resistance training on skeletal muscle substrates, enzyme activities and capillary supply. Acta Physiol Scand 1990: 140: 575–580.
Trappe S, Harber M, Creer A, Gallagher P, Slivka D, Minchev K, Whitsett D. Single muscle fiber adaptations with marathon training. J Appl Physiol 2006: 101: 721–727.
Van Cutsem M, Duchateau J, Hainaut K. Changes in single motor unit behavior contribute to the increase in contraction speed after dynamic training in humans. J Physiol 1998: 513(1): 295–305.