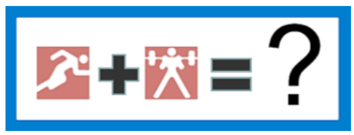
5th Congress of the Hellenic Society of Biochemistry and Physiology of Exercise
Athens · November 6-7th, 2015

Effects of resistance training on endurance capacity in Top Level Athletes



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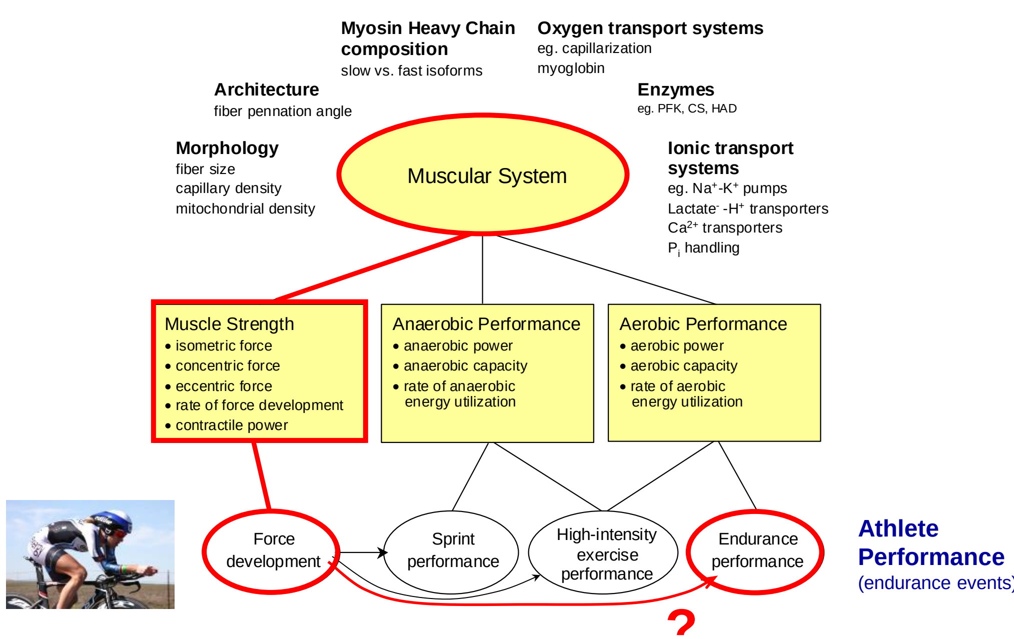


Aagaard & Bangsbo in "ACSM's Advanced Exercise Physiology"
American College of Sports Medicine, 2005

Combined endurance and strength training

Compromised or synergistic adaptation?

- sedentary individuals
- moderately trained individuals
- highly trained top level athletes

Cardiovascular and muscular adaptations with concurrent S+E training

Previous study findings: contradictory data

Diminished cardiovascular and musculoskeletal adaptations to endurance (E) and strength (S) training when training regimes are combined

Hickson 1980, Dudley et al 1985, Hunter et al 1987, Nelson et al 1990, Kraemer et al 1995

Concurrent E+S training does not impair cardiovascular or musculoskeletal adaptations compared to either training regime alone

Bell et al 1991, McCarthy et al 1995, Izquirdo et al 2005

Concurrent E+S training increases endurance performance more than either training regime alone

Marcinik et al 1991, Hickson et al 1988, Hoff et al 2002, Rønnestad et al 2009, Aagaard et al 2011

Strength training and running performance



Strength training and running performance

Trained runners (VO_{2max} ~60 ml O_2/minkg):
↑ leg muscle strength (1RM squat)
Improved running economy (reduced O_2 uptake at 70% VO_2max)
↑ time to exhaustion at max aerobic speed, No change in VO_{2max}

Støren et al 2008

Well Trained triathletes (VO_{2max} ~70 ml O_2/minkg):
14 wks of combined strength and running training (E+S)
↑ leg muscle strength
Improved running economy at 75%VO_{2max} (reduced ml O_2/km) following E+S training compared to E training alone
↑ Run speed at VO_{2max} in E+S only (19.5 → 20.0 km/h)
No change in VO_{2max}

Millet et al. 2002

Improved running economy in well-trained runners

Well trained runners (VO_2max: 65 ml/kg min)

Training: 32% of the normal running training volume was replaced by short sprints, plyometric exercises, overload-jumping, high-speed low-resistance weight training ('power training'), Training duration: 9 weeks (n=10), 8-9 hours/week

Results: 5 km run time improved about 30 seconds (18.20 to 17.50 min)
Improved performance in anaerobic run test (MART)
Improved running economy (8% reduced VO_2 at 16 km/h)
Shortened ground contact times during the stance phase

No changes observed in the control group (n=8)

Paavolainen et al. 1999

Strength training and cycling performance

Strength training and cycling performance

Cycle-specific resistance training (“strength training” or “power pedalling” performed on the bike):

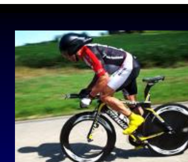
cycling with a low cadence (e.g. 40 rpm) with relatively high force

has <u>no effect</u> on neither maximal force capacity of the legs nor cycling performance*

Kristoffersen et al. 2014, Mujika et al. 2015

* Aerobic capacity, 30-min cycling performance, power output at lactate threshold, cadence, gross efficiency

Strength training and cycling performance



<u>Untrained subjects:</u>

↑ Leg muscle strength

↑ Long term cycling performance at 75% VO_{2max} (26→35 min, +33%)

↓ Blood lactate at same absolute workload (-30%)

Elevated blood lactate threshold (↑ Watts at 3.5 mM, +12%)

No change in VO_{2max}

Marcinik et al. 1991

Training regime:

- 12 wks resistance training,
- 3 sessions per week, 36 sessions in total
- knee extension, knee flexion, leg press, parallel squat + upperbody exercises
- 8-12 RM and 15-20 RM loads
- No endurance (E) training

Strength training and cycling performance

Well trained subjects:

↑ Leg muscle strength (30%)

↑ Short term cycling performance (6-8 min, +11%)

↑ Long term cycling performance at 80% VO_{2max} (69→85 min, +20%)

No change in VO_{2max}

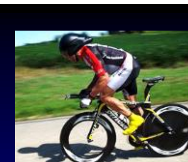
No increase in body weight

No increase in muscle fibre area (VL muscle) !

Hickson et al. 1988

Training regime:
- 10 wks concurrent S+E training,
- 3 sessions per week, 36 sessions in total
- knee extension, knee flexion, parallel squat
- 8-10 RM (80% 1 RM), 3-4 sets of 5 reps
- E training: running and/or cycling 3-5 days/wk

Strength training and cycling performance



In well trained cyclists (VO_{2max} ~ 66-70 ml O_2 min^{-1} kg^{-1}): cycling economy was improved to a greater extent by concurrent S+E training than E training alone during the final hour of a 185-min long cycling test Rønnestad et al 2011

Training regime:
- 12 wks concurrent resistance training,
- 2 sessions per week, 24 sessions in total
- half squat, unilateral leg press
- 8-10 RM → 5-6 RM, 3 sets, max speed
- E training: cycling or xc-skiing ?hrs/wk

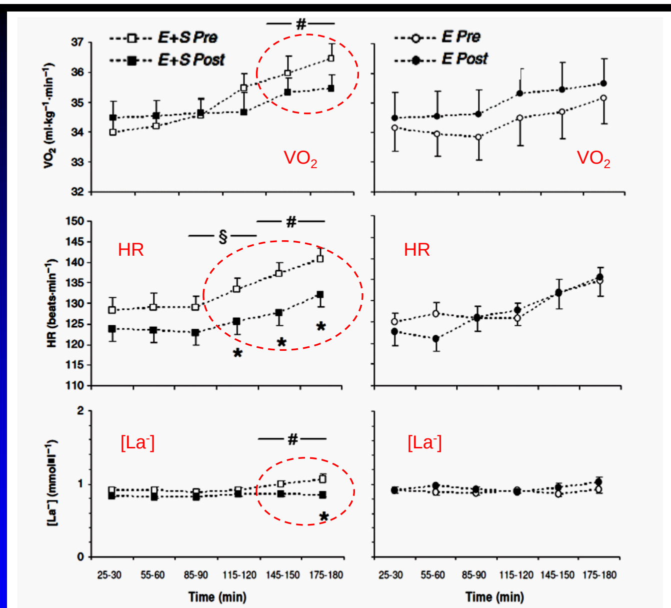
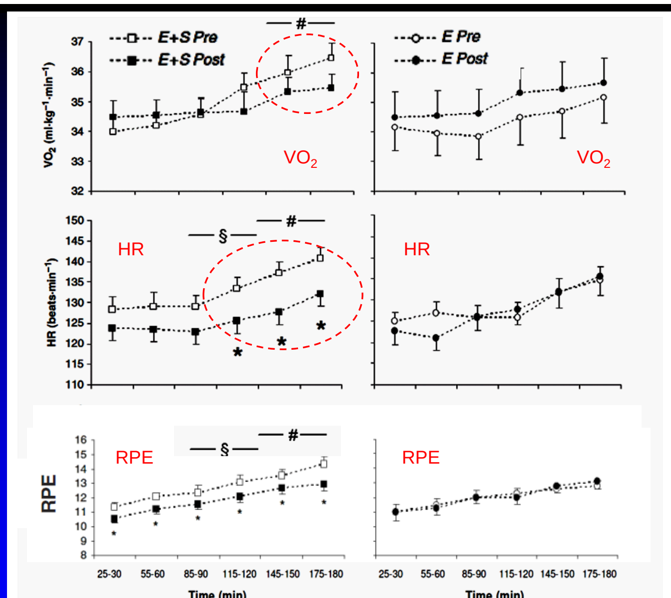


Fig.1 Oxygen uptake (VO_2; top panel), heart rate (HR; mid panel) and blood lactate concentration ([La^-]; bottom panel) during 180 min of cycling at 44% of baseline Wmax before (Pre) and after (Post) 12 weeks of concurrent endurance (cycling) and strength training (E+S; left panels) and endurance (cycling) training alone (E; right panel). Wmax was calculated a the mean power output during the final two 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 (§, $P<0.01$).

Rønnestad et al,
Scand J Med Sci Sports 2011

Graph from
Aagaard & Andersen 2010



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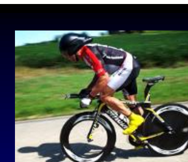
Rønnestad et al,
Scand J Med Sci Sports 2011

Graph from
Aagaard & Andersen 2010

Strength training and cycling performance

In conclusion, heart rate and blood lactate responses were reduced during the final hour of a 185-min long cycling test following concurrent S+E training compared to E training alone Rønnestad et al 2009

Strength training and cycling performance



In conclusion, heart rate and blood lactate responses were reduced during the final hour of a 185-min long cycling test following concurrent S+E training compared to E training alone Rønnestad et al 2009

Notably, all-out cycling performance measured during a 5-min max test at the finish of 185 min cycling was improved following concurrent S+E training (8% increased power production) while unaffected by endurance training alone.

⇒ sprint capacity in the final phase of a race can be enhanced by concurrent SE training

Rønnestad et al 2009

Strength training and cross-country skiing

Improved economy in cross-country skiing

Male elite cross-country skiers (n=9)

VO_{2max} ~ 69 ml O_2/ min kg

Strength training: 3 x 6 RM, 3 times/wk, 8 wks + ski training

Results: Increased muscle strength and RFD,

Improved endurance performance - Time to exhaustion:

6.5 → 10.2 min = +20% vs controls

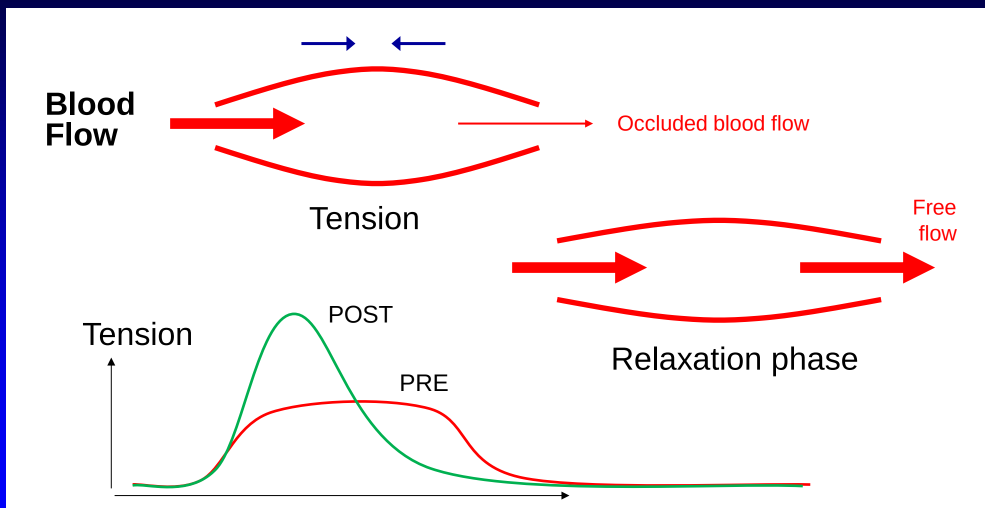
Improved economy: 1.02 → 0.74 ml O_2 $kg^{-0.67}$ min^{-1}

Proposed adaptive mechanism (Hypothesis):

Increased endurance and improved economy due to enhanced muscle blood perfusion

Hoff et al. 2002
Østerås, Helgerud, Hoff 2002

Hypothetical changes in Blood flow perfusion due to changes in RFD



Hoff et al. 1999, 2002
Østerås, Helgerud, Hoff 2002

Improved economy in cross-country skiing

<u>Female trained cross-country skiers</u> (n=8)

VO_{2max} ~ 55 ml O_2/ min kg

Strength training: 3 x 6 RM, 3 times/wk, 9 wks + ski training

Results: Increased muscle strength and RFD,
Improved endurance performance - Time to exhaustion:
5.2 → 12.3 min (vs 4.0 → 6.3 min in C) = +80% vs controls
Improved economy: 1.42 → 1.10 ml O_2 $kg^{-0.67}$ min^{-1}

Proposed adaptive mechanism (Hypothesis):
Increased endurance and improved economy due to enhanced muscle blood

Hoff et al. 1999

SUMMARY

Changes in short-term endurance performance induced by concurrent S+E training

Concurrent SE training

⇒ improved short-term (<10 min) endurance capacity measured as an increased time to exhaustion during treadmill running, cycling ergometer testing, ski ergometer testing in...

- untrained subjects Hickson et al. 1980
- recreational moderately trained individuals Hickson et al. 1988
- well-to-highly trained cross-country skiers and cyclists

Hoff et al. 1999, 2002, Østerås et al. 2002, Mikkola et al. 2007, Støren et al. 2008, Psilander et al. 2015

SUMMARY

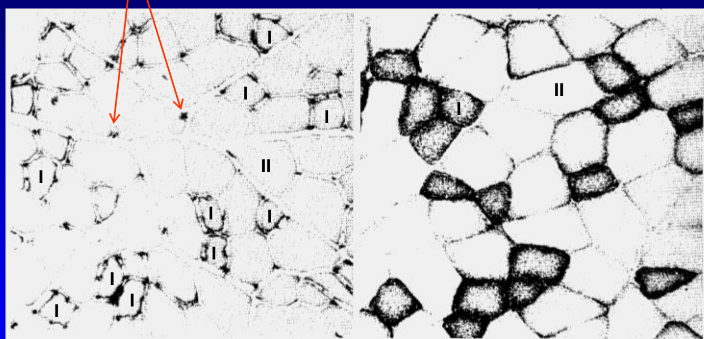
Changes in LONG-TERM endurance performance induced by concurrent S+E training

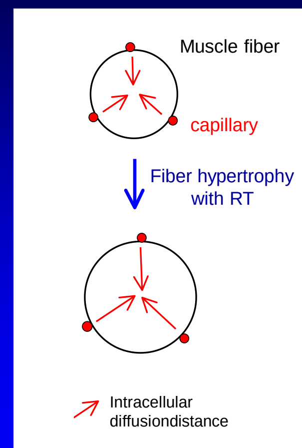
Not well examined -
only very few data exist!!!

Muscle capillarization and myofiber size

Effect of ↑ fiber CSA on intracellular diffusion distance?

Muscle capillary





Capillary staining

ATPase staining:
fibre type composition

Scand J Med Sci Sports 2011: 21: e298–e307
doi: 10.1111/j.1600-0838.2010.01283.x

SCANDINAVIAN JOURNAL OF MEDICINE & SCIENCE IN SPORTS

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

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Accepted for publication 29 November 2010

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 muscu- 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

Aagaard et al, Scand J Med Sci Sports 2011

Material and Methods

Subjects
14 young amateur elite cyclists (Danish U-21 National Team)
No strength training for at least 8 months prior to the study

Training groups
E + S : combined endurance and strength training (n=7)
E : endurance training alone (n=7)

Training Group	n	Age, years	Heigth, cm	Weigth, kg
E + S combined endurance and strength	7	20.1 ± 0.9	182.4 ± 6.2	69.2 ± 5.8
Control group, E endurance only	7	19.0 ± 0.8	179.0 ± 4.5	72.3 ± 6.0

Aagaard et al, Scand J Med Sci Sports 2011

Study design

Test 1 (pre)
biopsy sampling

Test 2 (post)
biopsy sampling

Test 3 (post 2)
biopsy sampling

combined training (E+S)
endurance training (E)
16 weeks

endurance training
[both groups]
8 weeks

November
start of combined training

February
end of combined training

May
follow-up

Aagaard et al, Scand J Med Sci Sports 2011

Material and Methods...

Endurance training (E+S and E groups)
- controlling hours of training, not intensity
- training diary

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

Aagaard et al, Scand J Med Sci Sports 2011

Material and Methods...

Strength training (E+S group only)
- training diary
- two weeks preparatory strength training
  training loads: 10-12 RM
- heavy-resistance training, restitution ≥ 48 h
- 4 sets of 4 exercises

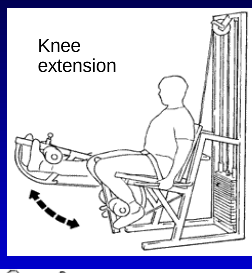
Week	1	2	3	4	5	6	7	8
Freq Loads	3 10-12 RM	2 8-10 RM	3 8-10 RM	2 6-8 RM	3 6-8 RM	2 5-6 RM	3 5-6 RM	2 5-6 RM
Week	9	10	11	12	13	14	15	16
Freq Loads	3 5-6 RM	2 5-6 RM	3 5-6 RM	2 5-6 RM	3 5-6 RM	2 5-6 RM	3 5-6 RM	2 5-6 RM

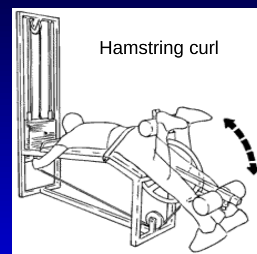
Freq = training sessions per week
Loads = training loads expressed in RM

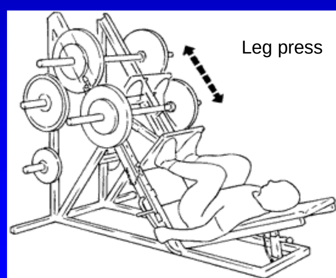
Aagaard et al, Scand J Med Sci Sports 2011

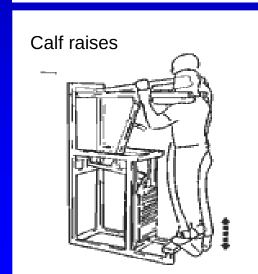
Material and Methods...

Strength training exercises









Results

VO_{2max} remained unchanged in either group

S+E: 73.5 ±8.2 vs 75.0 ±6.0 ml O_2 min^{-1} kg^{-1},

E: 71.5 ±6.0 vs 73.0 ±2.3 ml O_2 min^{-1} kg^{-1}

Cycling Economy (VO_2 at 75% VO_{2max}) remained unchanged in either group

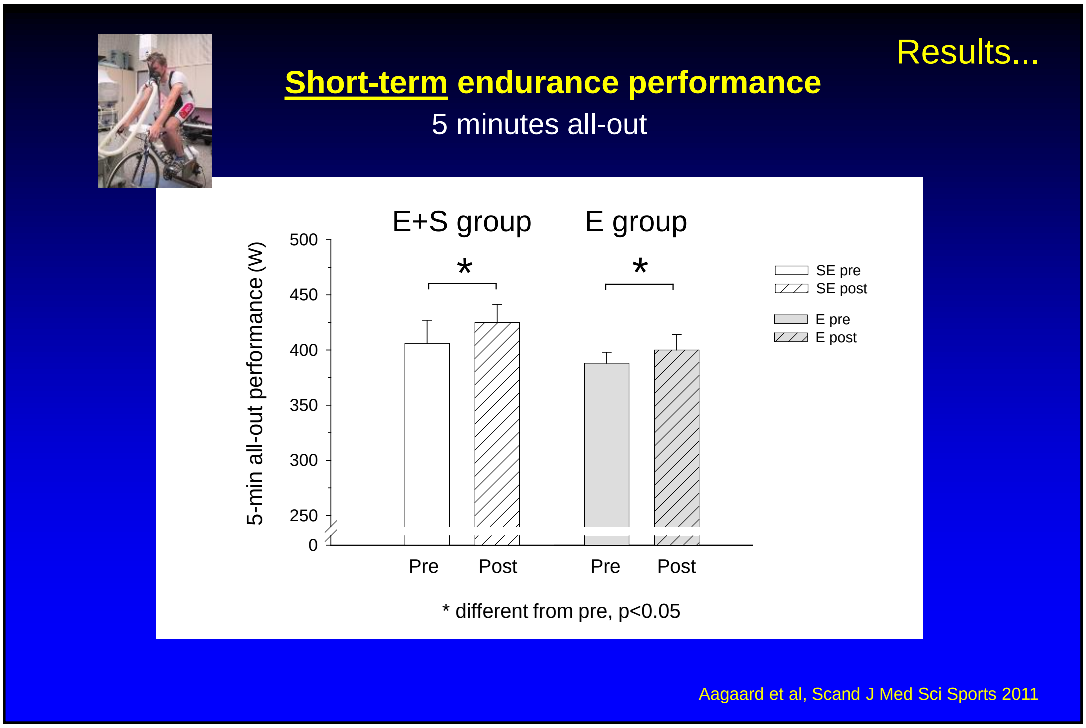
Blood lactate profile obtained during graded submaximal graded testing was unchanged in both groups

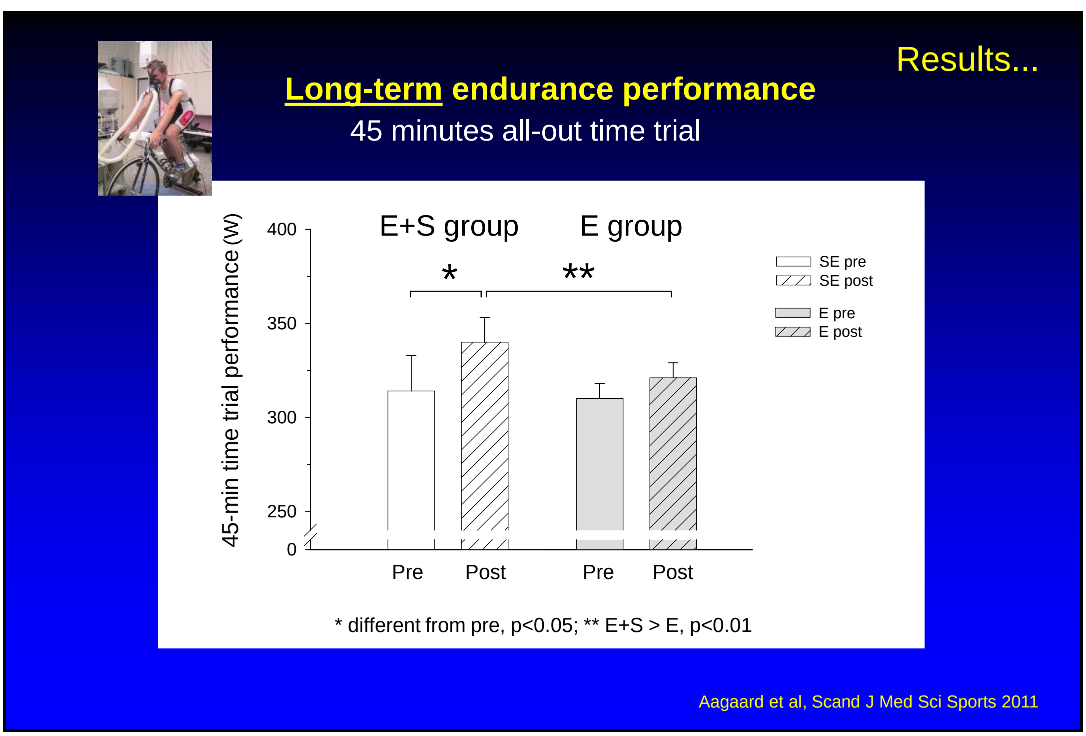
Muscle fiber size and vascularization (cap/fiber, cap/mm^2) did not change with either mode of training

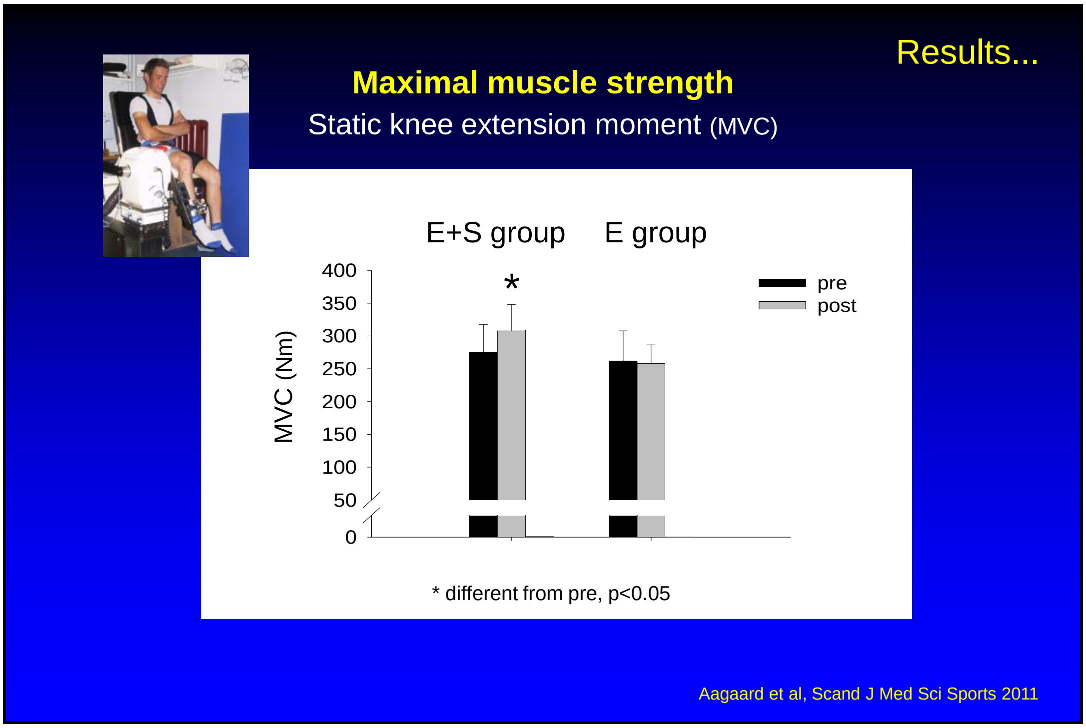
S+E: 7.6 ±0.8 vs 7.2 ±0.8 cap/fiber

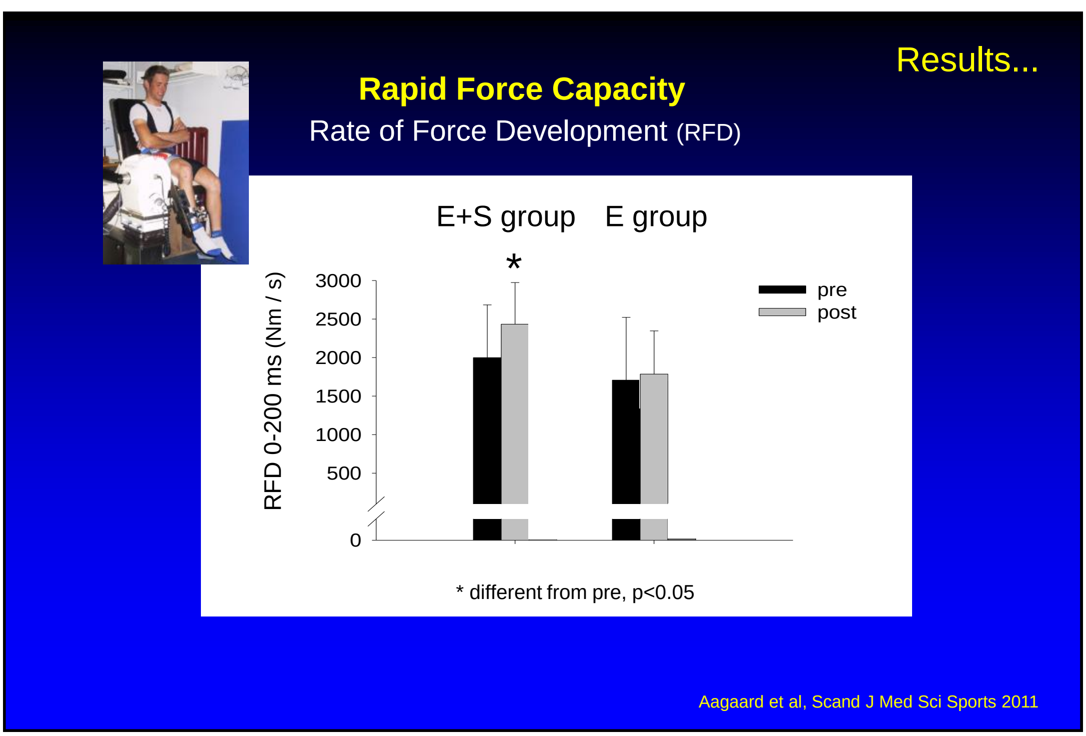
E: 7.6 ±0.6 vs 8.4 ±0.7 cap/fiber

Aagaard et al, Scand J Med Sci Sports 2011

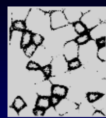

Results...
Short-term endurance performance
5 minutes all-out
E+S group
E group
*
*
5-min all-out performance (W)
500
450
400
350
300
250
0
Pre
Post
Pre
Post
SE pre
SE post
E pre
E post
* different from pre, p<0.05
Aagaard et al, Scand J Med Sci Sports 2011


Results...
Long-term endurance performance
45 minutes all-out time trial
E+S group
E group
*
**
45-min time trial performance (W)
400
350
300
250
0
Pre
Post
Pre
Post
SE pre
SE post
E pre
E post
* different from pre, p<0.05; ** E+S > E, p<0.01
Aagaard et al, Scand J Med Sci Sports 2011

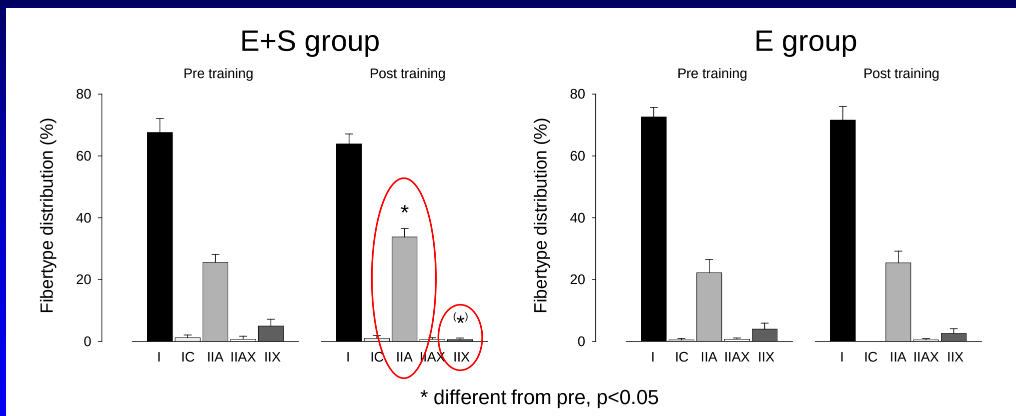

Results...
Maximal muscle strength
Static knee extension moment (MVC)
E+S group
E group
*
pre
post
MVC (Nm)
400
350
300
250
200
150
100
50
0
* different from pre, p<0.05
Aagaard et al, Scand J Med Sci Sports 2011


Results...
Rapid Force Capacity
Rate of Force Development (RFD)
E+S group
E group
*
pre
post
RFD 0-200 ms (Nm / s)
3000
2500
2000
1500
1000
500
0
* different from pre, p<0.05
Aagaard et al, Scand J Med Sci Sports 2011

Results...



Muscle fibre type distribution
evaluated by ATPase histochemistry



Aagaard et al, Scand J Med Sci Sports 2011

Conclusions

Both E training and combined SE training increased (3-4%) short-term cycling performance in young top-level cyclists.

Only combined SE training increased (8%) long-term cycling performance. This parameter remained unchanged after E training alone.

The improvement in long-term endurance performance was ascribed to

(i) an increased proportion of type IIA fibers at the expense of a reduced proportion of IIX fibers,

(ii) training-induced increases in RFD and MVC

Aagaard et al, Scand J Med Sci Sports 2011

Possible explanations for the increase in endurance performance observed with combined E+S training

- Increased proportion of type IIA muscle fibres
  ⇒ more endurant type II muscle fibre profile

  11 wks of heavy-resistance strength training: Change in 40 min all-out performance in female well-trained cyclists correlated with changes in the proportion of IIAX fibers ($r = -0.63$, $p = 0.009$)

Vikmoen et al, ECSS 2015

Possible explanations for the increase in endurance performance observed with combined E+S training

- Increased proportion of type IIA muscle fibres
  ⇒ more endurant type II muscle fibre profile

- Increased ratio of relaxation-to-activation time (reduced ”duty factor”) due to the increased RFD
  ⇒ increased time for restitution in each pedal revolution
  + increased capillary mean transit time (MTT)
  ⇒ increased FFA uptake from blood ⇒ glycogen sparing
  ⇒ reduced fatigue

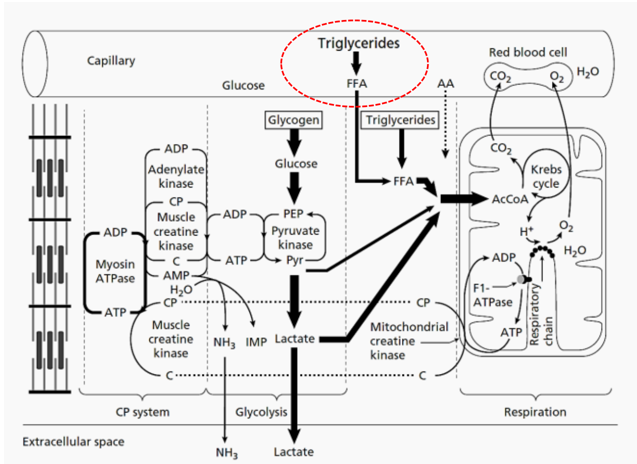
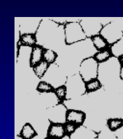


Fig. 4.20 Integrated, schematic view of the pathways of energy supply to the myofibrils. The thickness of the arrows indicates the relative importance of some of the substrate fluxes. The proportion of the produced lactate that is directly oxidized in a fibre's mitochondria is currently a matter of debate. AA, amino acids; AcCoA, acetyl coenzyme A; C, creatine; CP, creatine phosphate; FFA, free fatty acids; IMP, inosine monophosphate; PEP, phosphenolpyruvate; Pyr, pyruvate.

Billeter & Hoppeler,
Strength & Power in Sports, IOC 2003

Combined endurance and resistance training

Effects on myofiber hypertrophy



Concurrent endurance training may fully or partially inhibit the hypertrophy response normally elicited by resistance training (moderately to highly trained individuals)

Aagaard et al 2011, Hickson et al 1988, Kraemer et al 1995, Rønnestad et al 2012

In fact, single-modality high-volume endurance training may induce substantial muscle fiber atrophy *per se*

Teradros et al 1986, Ratzin Jackson et al 1990,
Kraemer et al 1995, Harber et al 2004, Trappe et al 2006

WHICH CELLULAR MECHANISM(S) ARE INVOLVED in the blunted hypertrophy response?

Skeletal muscle size: Molecular regulatory signalling pathways

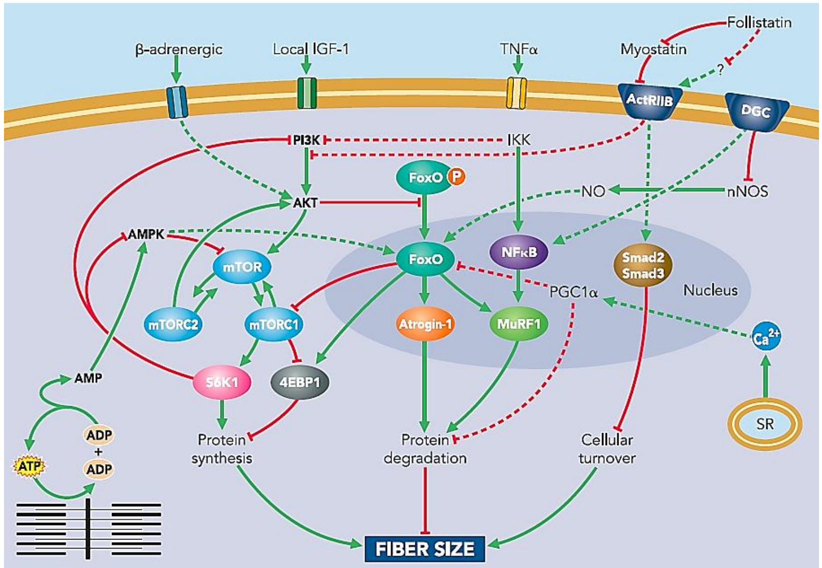


FIGURE 2. **Scheme illustrating the major pathways that control fiber size**
Dotted lines depict pathways whose molecular mechanisms and role in adult skeletal muscle have yet to be completely defined.

Sandri, Physiology 2008

The FASEB Journal express article10.1096/fj.04-2179fje. Published online February 16, 2005.

Selective activation of AMPK-PGC-1α or PKB-TSC2-mTOR signaling can explain specific adaptive responses to endurance or resistance training-like electrical muscle stimulation

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*School of Life Sciences, University of Dundee, UK; [†]Department of Biological Sciences, University of Central Lancashire, UK; [‡]Clinical Physiology Laboratory, University of Nottingham, UK

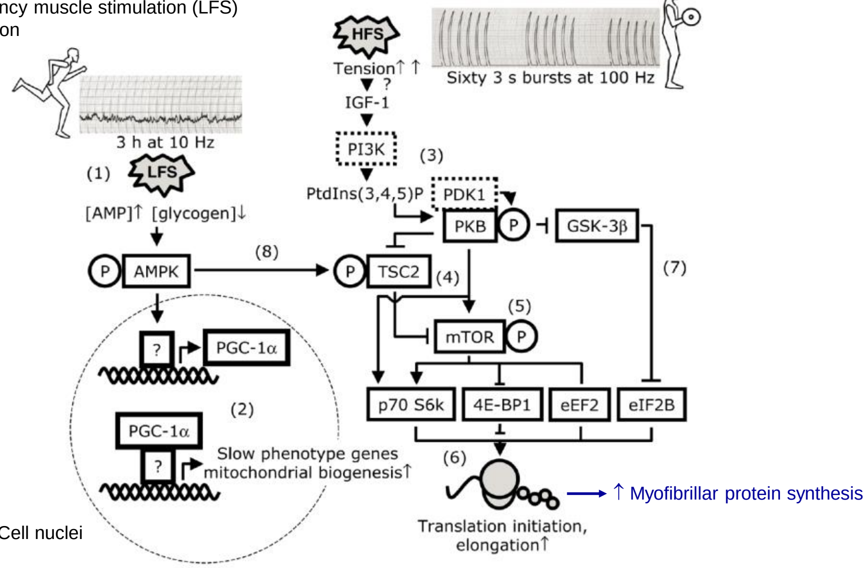
ABSTRACT

Endurance training induces a partial fast-to-slow muscle phenotype transformation and mitochondrial biogenesis but no growth. In contrast, resistance training mainly stimulates muscle protein synthesis resulting in hypertrophy. The aim of this study was to identify signaling events that may mediate the specific adaptations to these types of exercise. Isolated rat muscles were electrically stimulated with either high frequency (HFS; 6×10 repetitions of 3 s-bursts at 100 Hz to mimic resistance training) or low frequency (LFS; 3 h at 10 Hz to mimic endurance training). HFS significantly increased myofibrillar and sarcoplasmic protein synthesis 3 h after stimulation 5.3- and 2.7-fold, respectively. LFS had no significant effect on protein synthesis 3 h after stimulation but increased UCP3 mRNA 11.7-fold, whereas HFS had no significant effect on UCP3 mRNA. Only LFS increased AMPK phosphorylation significantly at Thr172 by ~2-fold and increased PGC-1α protein to 1.3 times of control. LFS had no effect on PKB phosphorylation but reduced TSC2 phosphorylation at Thr1462 and deactivated translational regulators. In contrast, HFS acutely increased phosphorylation of PKB at Ser473 5.3-fold and the phosphorylation of TSC2, mTOR, GSK-3β at PKB-sensitive sites. HFS also caused a prolonged activation of the translational regulators p70 S6k, 4E-BP1, eIF-2B, and eEF2. These data suggest that a specific signaling response to LFS is a specific activation of the AMPK-PGC-1α signaling pathway which may explain some endurance training adaptations. HFS selectively activates the PKB-TSC2-mTOR cascade causing a prolonged activation of translational regulators, which is consistent with increased protein synthesis and muscle growth. We term this behavior the "AMPK-PKB switch." We hypothesize that the AMPK-PKB switch is a mechanism that partially mediates specific adaptations to endurance and resistance training, respectively.

Atherton et al, FASEB J 2005

RESISTANCE exercise activity
high-frequency muscle stimulation (HFS)
short duration

ENDURANCE exercise activity
low-frequency muscle stimulation (LFS)
long duration



Atherton et al, FASEB J 2005

ACTIVATION OF THE MUSCLE ENDURANCE ADAPTATION PATHWAY

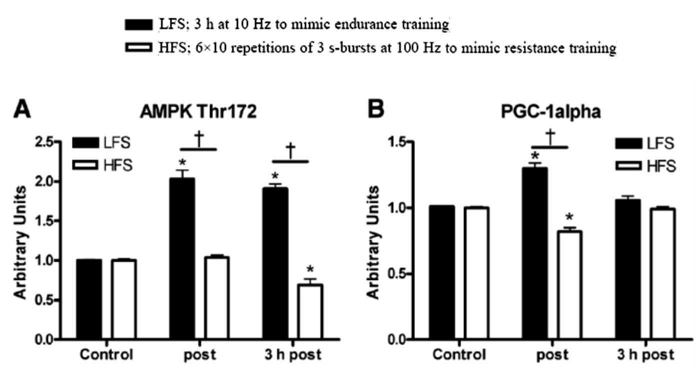
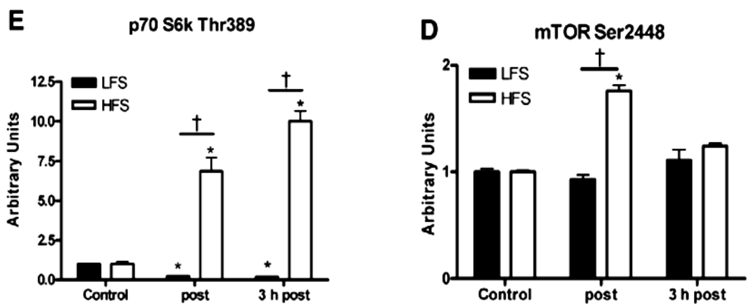


Figure 2. ***A)*** AMPK Thr172 phosphorylation relative to total AMPK. ***B)*** Total PGC-1α (*n*=8; 4 EDL and 4 soleus per bar; mean± SE) of resting muscle (control), directly after (post) and 3 h after (3 h post) LFS and HFS. All values were normalized to the relative intensity of the control bands. *Significantly different from control; †significant difference between LFS and HFS stimulation protocols (ANOVA, Tukey's post hoc, $P<0.05$).

rat EDL muscles Atherton, Babraj, Rennie, Wackerhage et al, FASEB J 2005

ACTIVATION OF THE MUSCLE HYPERTROPHY PATHWAY

LFS; 3 h at 10 Hz to mimic endurance training
HFS; 6×10 repetitions of 3 s-bursts at 100 Hz to mimic resistance training



rat EDL muscles Atherton, Babraj, Rennie, Wackerhage et al, FASEB J 2005

ACUTE CHANGES IN CELLULAR PROTEIN SYNTHESIS

LFS; 3 h at 10 Hz to mimic endurance training
HFS; 6×10 repetitions of 3 s-bursts at 100 Hz to mimic resistance training

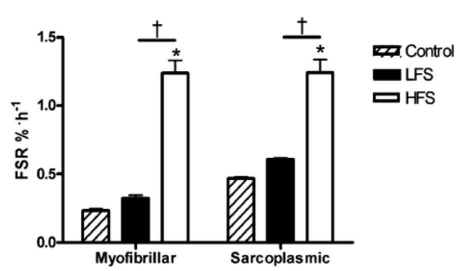
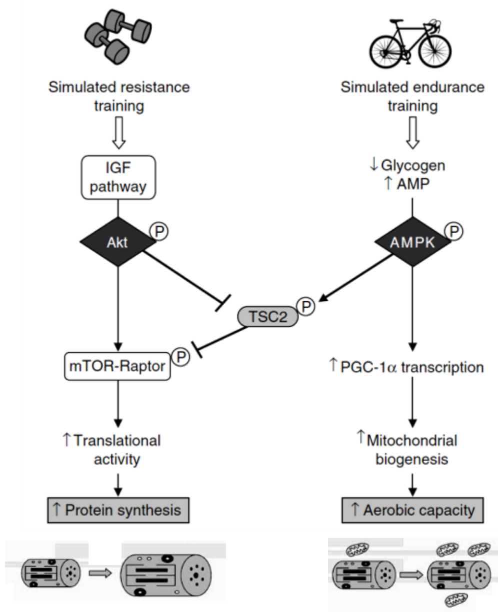


Figure 1. *A)* Myofibrillar and sarcoplasmic protein synthesis in rat EDL muscles incubated without stimulation and 3 h after LFS or HFS, respectively (*n*=6 EDL per bar; mean±SE).

rat EDL muscles Atherton, Babraj, Rennie, Wackerhage et al, FASEB J 2005



Sports Med 2007; 37 (9): 737-763
0112-1642/07/0009-0737/$44.95/0

The Molecular Bases of Training Adaptation

Vernon G. Coffey and *John A. Hawley*

Fig. 7. Putative adaptive pathways in response to endurance- and resistance-like stimuli. Bars denote inhibition and arrows denote activation. **AMP** = adenosine monophosphate; **AMPK** = adenosine monophosphate kinase; **IGF** = insulin-like growth factor; **mTOR** = mammalian target of rapamycin; **P** = phosphorylation; **PGC-1α** = peroxisome proliferator activated receptor γ co-activator-1α; **TSC2** = tuberous sclerosis complex 2; ↑ indicates increase; ↓ indicates decrease.

Coffey & Hawley, Sports Med 2007
Atherton et al, FASEB 2005
Nader, Med Sci Sports Exerc 2006
Baar, Med Sci Sports Exerc 2006

SUMMARY

Positive and potential negative effects from resistance training on endurance performance

Positive effects

- Improved long-term endurance capacity (cycling, 10k run in trained runners)
- Improved economy (adaptive mechanism: ↑ RFD → prolonged relaxation phase?)
- Improved muscle blood flow ??
- Increased proportion of fatigue-resistant type II muscle fibers (↑ MHC IIA)
- Enhanced biogenesis of mitochondria ?

Aagaard & Andersen,
Scand J Med Sci Sports 2010

J Appl Physiol 111: 1335–1344, 2011.
First published August 11, 2011; doi:10.1152/japplphysiol.00086.2011.

Resistance exercise enhances the molecular signaling of mitochondrial biogenesis induced by endurance exercise in human skeletal muscle

Li Wang,[1] Henrik Mascher,[1,2]* Niklas Psilander,[1,2]* Eva Blomstrand,[1,2] and Kent Sahlin[1,2]
[1]*The Åstrand Laboratory of Work Physiology, GIH, the Swedish School of Sport and Health Sciences, and* [2]*Department of Physiology and Pharmacology, Karolinska Institutet, Stockholm, Sweden*

Submitted 21 January 2011; accepted in final form 9 August 2011

Wang L, Mascher H, Psilander N, Blomstrand E, Sahlin K. Resistance exercise enhances the molecular signaling of mitochondrial biogenesis induced by endurance exercise in human skeletal muscle. *J Appl Physiol* 111: 1335–1344, 2011. First published August 11, 2011; doi:10.1152/japplphysiol.00086.2011.—Combining endurance and strength training (concurrent training) may change the adaptation compared with single mode training. However, the site of interaction and the mechanisms are unclear. We have investigated the hypothesis that molecular signaling of mitochondrial biogenesis after endurance exercise is impaired by resistance exercise. Ten healthy subjects performed either only endurance exercise (E; 1-h cycling at ~65% of maximal oxygen uptake), or endurance exercise followed by resistance exercise (ER; 1-h cycling + 6 sets of leg press at 70–80% of 1 repetition maximum) in a randomized cross-over design. Muscle biopsies were obtained before and after exercise (1 and 3 h postcycling). The mRNA of genes related to mitochondrial biogenesis [(peroxisome proliferator-activated receptor-γ coactivator-1 (PGC-1)α, PGC-1-related coactivator (PRC)] related coactivator) and substrate regulation (pyruvate dehydrogenase kinase-4) increased after both E and ER, but the mRNA levels were about twofold higher after ER ($P < 0.01$). Phosphorylation of proteins involved in the signaling cascade of protein synthesis [mammalian target of rapamycin (mTOR), ribosomal S6 kinase 1, and eukaryotic elongation factor 2] was altered after ER but not after E. Moreover, ER induced a larger increase in mRNA of genes associated with positive mTOR signaling (cMyc and Rheb). Phosphorylation of AMP-activated protein kinase, acetyl-CoA carboxylase, and Akt increased similarly at 1 h postcycling ($P < 0.01$) after both types of exercise. Contrary to our hypothesis, the results demonstrate that ER, performed after E, amplifies the adaptive signaling response of mitochondrial biogenesis compared with single-mode endurance exercise. The mechanism may relate to a cross talk between signaling pathways mediated by mTOR. The results suggest that concurrent training may be beneficial for the adaptation of muscle oxidative capacity.

mitochondria; concurrent exercise; gene expression regulation; signal transduction; transcription factors/metabolism

performance give limited information of the type of adaptation and of the mechanisms involved. The results from long-term training studies may also be difficult to interpret due to various confounding factors (e.g., nutrition, initial training status, and differences in trainability between subjects). Measurement of the adaptive response in molecular signaling to acute exercise may provide a deeper understanding of the mechanisms underlying training adaptation and possible interactions between signaling pathways.

The knowledge of the molecular signaling involved in the muscle adaptive response to exercise has increased considerably during the last decade. The peroxisome proliferator-activated receptor-γ coactivator-1α (PGC-1α) has been recognized as the main transcriptional cofactor mediating mitochondrial biogenesis and improved oxidative capacity in skeletal muscle (reviewed by Ref. 19). The PGC-1-related coactivator (PRC) belongs to the PGC-1 family and has a similar role in mitochondrial biogenesis (2). The mRNA of PGC-1α and PRC shows an early robust increase after exercise (13, 14, 25, 27, 28, 37) and can, therefore, serve as early markers of the exercise-induced adaptive response of oxidative function. The mRNA of most mitochondrial enzymes has a slower response and increases after a delay period of 10–18 h after exercise (19). Several studies have, however, shown an early increase of mRNA of pyruvate dehydrogenase kinase (PDK)4, an enzyme that regulates carbohydrate oxidation and promotes lipid oxidation (27, 37). Three kinases [p38 mitogen-activated protein kinase (MAPK), AMP-activated protein kinase (AMPK), and calcium/calmodulin-dependent protein kinase II (CaMKII)] are particularly relevant to the exercise-induced regulation of PGC-1α expression and have an important role in mediating skeletal muscle adaptations to endurance training (reviewed by Refs. 6, 19). Resistance exercise, on the other hand, is known to stimulate the mammalian target of rapamycin (mTOR) signaling pathway, which stimu-

Wang, Sahlin et al, J Appl Physiol 111, 2011

J Appl Physiol 111: 1335–1344, 2011.
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"... Contrary to our hypothesis, the results demonstrate that RT, performed after ET, amplifies the adaptive signaling response of mitochondrial biogenesis compared with single-mode endurance exercise ..."

mitochondria; concurrent exercise; gene expression regulation; signal transduction; transcription factors/metabolism

Wang, Sahlin et al, J Appl Physiol 111, 2011

RESISTANCE TRAINING CAN BOOST MITOCHONDRIAL BIOGENESIS

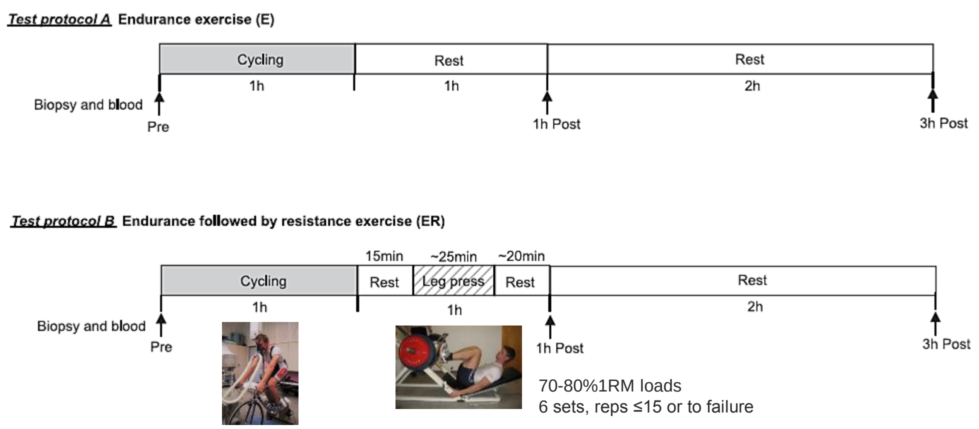
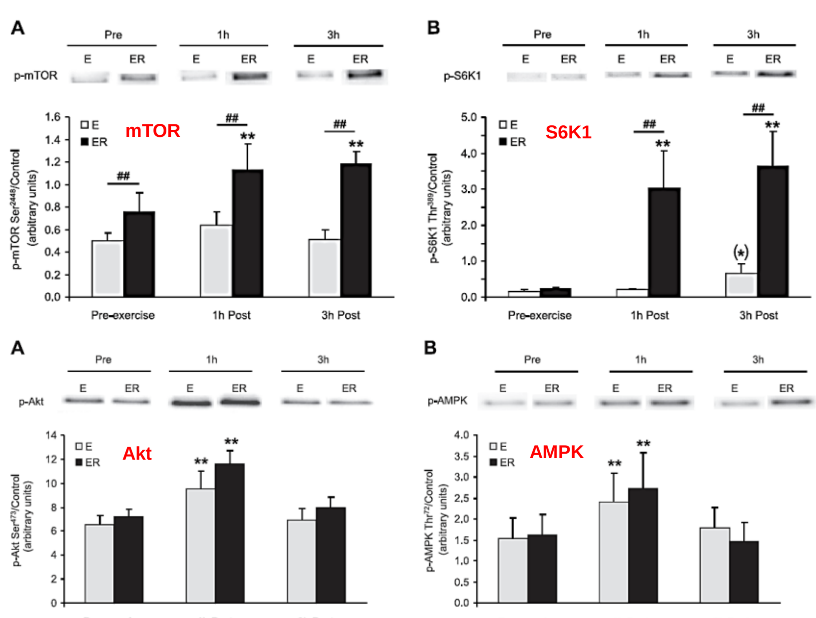


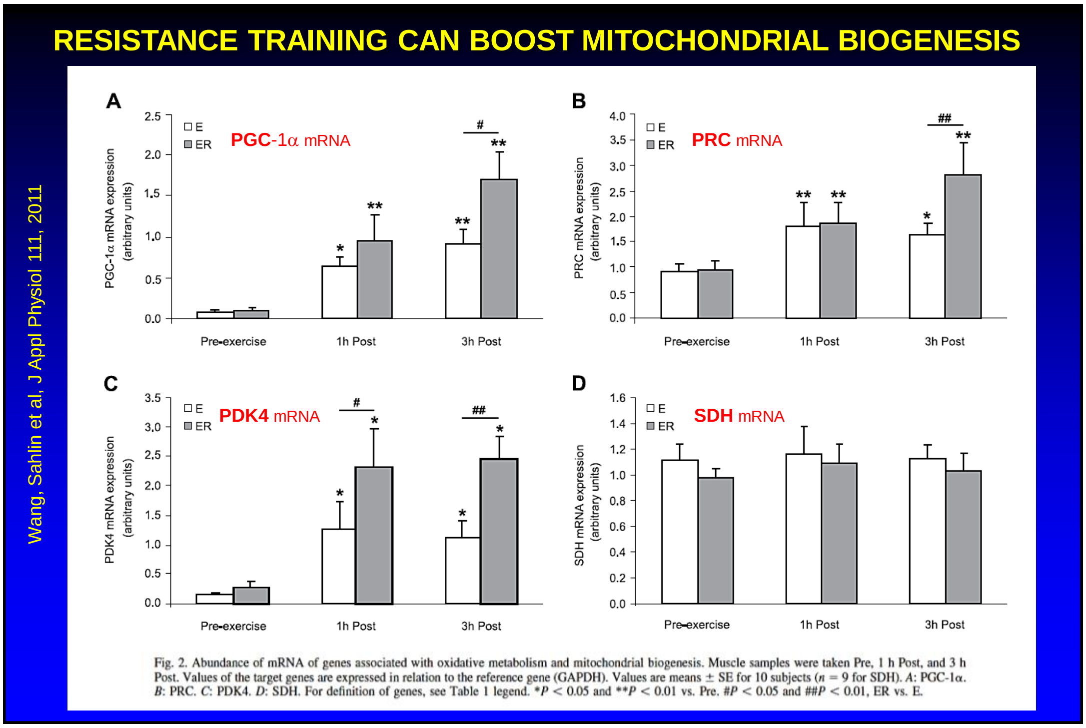
Fig. 1. Schematic of the experimental protocol. Endurance exercise (E) was performed as cycling at 65% of maximal oxygen uptake ($\dot{V}O_{2max}$), and resistance exercise (ER) as leg press at 70–80% of 1 repetition maximum with 15 repetitions or until failure. Pre, preexercise; Post, postexercise.

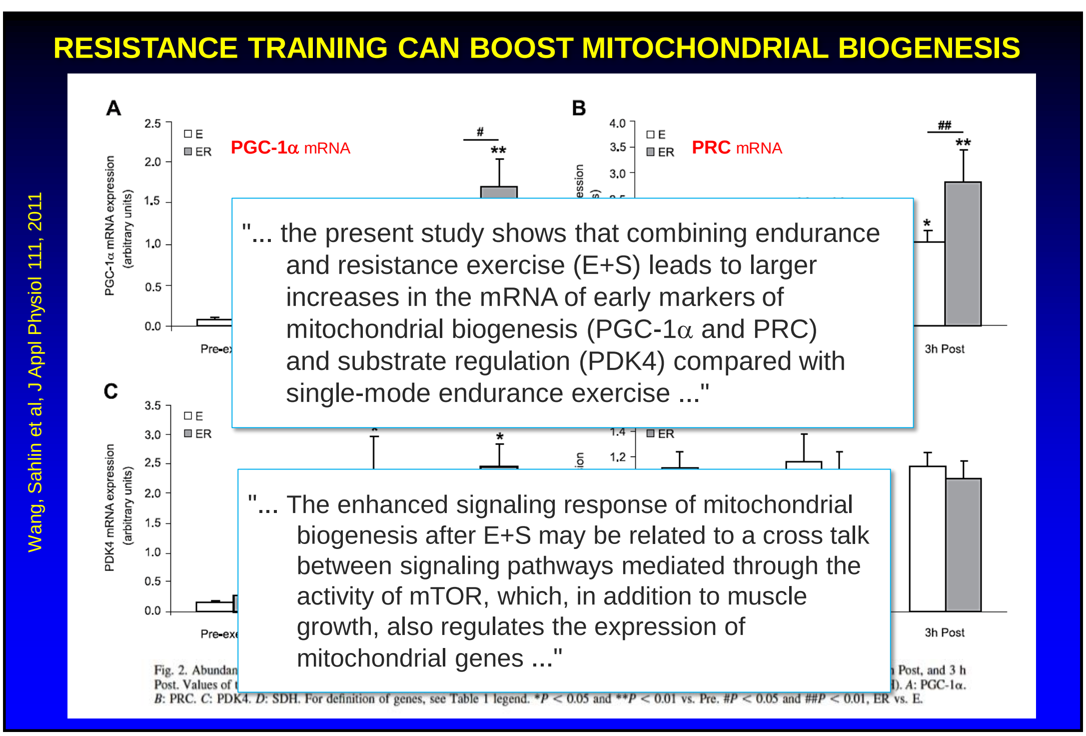
Wang, Sahlin et al, J Appl Physiol 111, 2011

RESISTANCE TRAINING CAN BOOST MITOCHONDRIAL BIOGENESIS

Wang, Sahlin et al, J Appl Physiol 111, 2011




RESISTANCE TRAINING CAN BOOST MITOCHONDRIAL BIOGENESIS
Wang, Sahlin et al, J Appl Physiol 111, 2011
A
PGC-1α mRNA
PGC-1α mRNA expression (arbitrary units)
E
ER
Pre-exercise
1h Post
3h Post
B
PRC mRNA
PRC mRNA expression (arbitrary units)
C
PDK4 mRNA
PDK4 mRNA expression (arbitrary units)
D
SDH mRNA
SDH mRNA expression (arbitrary units)
Fig. 2. Abundance of mRNA of genes associated with oxidative metabolism and mitochondrial biogenesis. Muscle samples were taken Pre, 1 h Post, and 3 h Post. Values of the target genes are expressed in relation to the reference gene (GAPDH). Values are means ± SE for 10 subjects (n = 9 for SDH). A: PGC-1α. B: PRC. C: PDK4. D: SDH. For definition of genes, see Table 1 legend. *P < 0.05 and **P < 0.01 vs. Pre. #P < 0.05 and ##P < 0.01, ER vs. E.


RESISTANCE TRAINING CAN BOOST MITOCHONDRIAL BIOGENESIS
Wang, Sahlin et al, J Appl Physiol 111, 2011
"... the present study shows that combining endurance and resistance exercise (E+S) leads to larger increases in the mRNA of early markers of mitochondrial biogenesis (PGC-1α and PRC) and substrate regulation (PDK4) compared with single-mode endurance exercise ..."
"... The enhanced signaling response of mitochondrial biogenesis after E+S may be related to a cross talk between signaling pathways mediated through the activity of mTOR, which, in addition to muscle growth, also regulates the expression of mitochondrial genes ..."

However, in a subsequent longitudinal study there was no measurable effect on mitochondrial biogenesis following 8 wks concurrent S+E training...

Scand J Med Sci Sports 2015: 25: e353–e359
doi: 10.1111/sms.12338

SCANDINAVIAN JOURNAL OF MEDICINE & SCIENCE IN SPORTS

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

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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

Accepted for publication 10 September 2014

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.

Psilander, Sahlin et al, Scand J Med Sci Sports 2015

SUMMARY

Positive and potential negative effects from resistance training on endurance performance

Positive effects

- Improved long-term endurance capacity (cycling, 10k run in trained runners)
- Improved economy (adaptive mechanism: ↑ RFD → prolonged relaxation phase?)
- Improved muscle blood flow ??
- Increased proportion of fatigue-resistant type II muscle fibers (↑ MHC IIA)
- ↑ biogenesis of mitochondria (in novice subjects: yes, longitudinally:maybe not)
- Enhanced sprint and acceleration capacity

Aagaard & Andersen,
Scand J Med Sci Sports 2010

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- Enhanced sprint and acceleration capacity

Potential negative effects

- Body weight may increase (however only rarely seen with concurrent SE training)
- Impaired capillarization (↓ cap mm^{-2})? No, not observed with SE training
- Requires additional training ressources (time, energy, restitution)

! Aerobic capacity (VO_{2max}) is not impaired!

Aagaard & Andersen, Scand J Med Sci Sports 2010

SUMMARY
Potential negative effects from endurance training on maximal strength and power performance

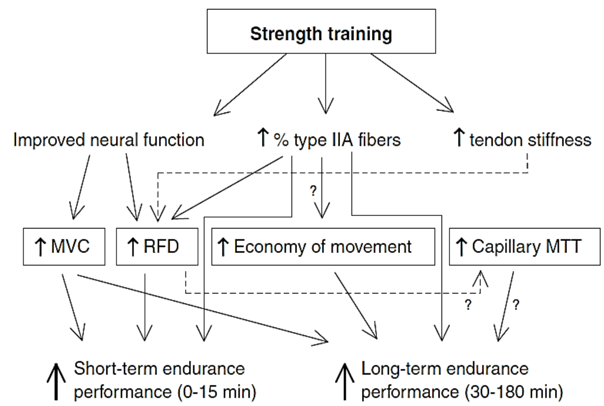
Potential negative effects

- Reductions in maximal muscle strength
- Reductions in explosive strength (RFD)
- Impaired gains in muscle size, RFD and power with concurrent strength training (well-trained to highly trained individuals) compared to strength training alone

Rønnestad et al, Eur J Appl Physiol 112, 2012
Häkkinen et al, Eur J Appl Physiol 89, 2003

Effects of resistance training on endurance capacity in Top Level Athletes

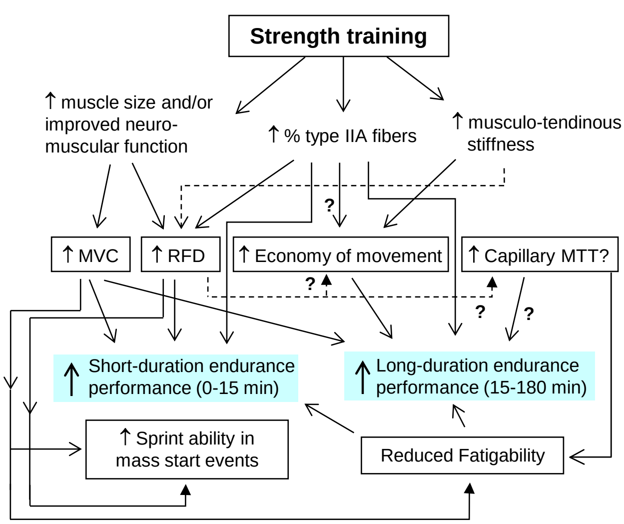
OVERALL CONLUSIONs



Aagaard & Andersen,
Scand J Med Sci Sports 2010

Effects of resistance training on endurance capacity in Top Level Athletes

OVERALL CONLUSIONs



Aagaard & Raastad.
Strength training for
endurance performance, 2012

Effects of resistance training on endurance capacity in Top Level Athletes

OVERALL CONLUSIONs

Short-term (≤10 min) endurance capacity* can be improved by means of concurrent endurance (E) and strength (S) training in both previously untrained individuals as well as in well-trained and highly trained (top level) endurance athletes

* Time to exhaustion at VO_{2max}

Furthermore, concurrent endurance and heavy-resistance strength training can increase running speed and power output at VO_{2max} (V_{max} and W_{max}) and improve time to exhaustion at V_{max} and W_{max}

Concurrent E+S training training (using either explosive or heavy strength training) can improve running performance (↑ economy) and also lead to enhanced cycling performance

Effects of resistance training on endurance capacity in Top Level Athletes

OVERALL CONLUSIONs

Long-term (30-180 min) endurance capacity* can be improved by means of concurrent E+S training in both previously untrained individuals as well as in well-trained and highly trained (top level) endurance athletes

* Distance covered in 45-60 min (time trial testing), duration of 5-k run or 40-k cycling, sprint performance at the end of 180 min cycling

Adaptive mechanisms involve: conversion of fast-twitch type IIX fibers into more fatigue-resistant type IIA fibers, ↑ maximal muscle strength (MVC), ↑ rapid force characteristics (RFD)

and may likely also involve: improvements in neuromuscular function (↑ neural drive) and improved musculo-tendinous stiffness...

!! ALSO in top level endurance athletes !!

Scand J Med Sci Sports 2010: 20 (Suppl. 2): 39–47
doi: 10.1111/j.1600-0838.2010.01197.x

SCANDINAVIAN JOURNAL OF
MEDICINE & SCIENCE
IN SPORTS

Review

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

P. Aagaard[1], J. L. Andersen[2]

[1]Institute of Sports Science and Clinical Biomechanics, University of Southern Denmark, Odense, Denmark, [2]Institute of Sports Medicine Copenhagen, University of Copenhagen, Copenhagen, Denmark
Corresponding author: Prof. Per Aagaard, PhD, Institute of Sports Science and Clinical Biomechanics, University of Southern Denmark, Campusvej 55, DK-5230 Odense, Denmark. Tel: +45 6550 3876, Fax: +45 6550 3480, E-mail: paagaard@health.sdu.dk

Accepted for publication 21 January 2010

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.

Aagaard & Andersen,
Scand J Med Sci Sports 2010

Scand J Med Sci Sports 2014: 24: 603–612
doi: 10.1111/sms.12104

SCANDINAVIAN JOURNAL OF
MEDICINE & SCIENCE
IN SPORTS

Review

Optimizing strength training for running and cycling endurance performance: A review

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[1]Section for Sport Science, Lillehammer University College, Lillehammer, Norway, [2]Department of Physiology, Faculty of Medicine and Odontology, University of the Basque Country, Leioa, Basque Country, Spain, [3]School of Kinesiology and Health Research Center, Faculty of Medicine, Finis Terrae University, Santiago, Chile
Corresponding author: Bent R. Rønnestad, PhD, Section for Sport Science, Lillehammer University College, PB. 952, 2604 Lillehammer, Norway. Tel: +47 61288193, Fax: +47 61288200, E-mail: bent.ronnestad@hil.no

Accepted for publication 5 July 2013

Here we report on the effect of combining endurance training with heavy or explosive strength training on endurance performance in endurance-trained runners and cyclists. Running economy is improved by performing combined endurance training with either heavy or explosive strength training. However, heavy strength training is recommended for improving cycling economy. Equivocal findings exist regarding the effects on power output or velocity at the lactate threshold. Concurrent endurance and heavy strength training can increase running speed and power output at VO_{2max} (V_{max} and W_{max}, respectively) or time to exhaustion at V_{max} and W_{max}. Combining endurance training with either explosive or heavy strength training can improve running performance, while there is most compelling evidence of an additive effect on cycling performance when heavy strength training is used. It is suggested that the improved endurance performance may relate to delayed activation of less efficient type II fibers, improved neuromuscular efficiency, conversion of fast-twitch type IIX fibers into more fatigue-resistant type IIA fibers, or improved musculo-tendinous stiffness.

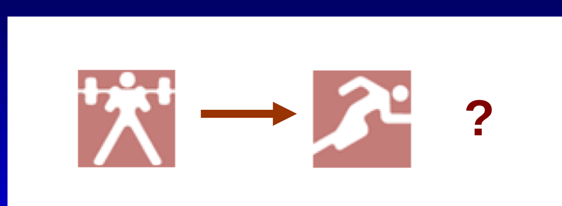
Rønnestad & Mujika,
Scand J Med Sci Sports 2014

Acknowledgements

Jesper L. Andersen
Benny Larsson
Charlotte Suetta
Ulrik Frandsen
Niels Ørtenblad
Morten Bennekou
Michael Kjær
Peter Magnusson
Anders Jørgensen
Peter Krustrup

Institute of Sports Science and Clinical Biomechanics, University of Southern Denmark;
Institute of Sports Medicine Copenhagen, University of Copenhagen

If performed in the same training session:
What is the optimal order of concurrent S and E exercise?



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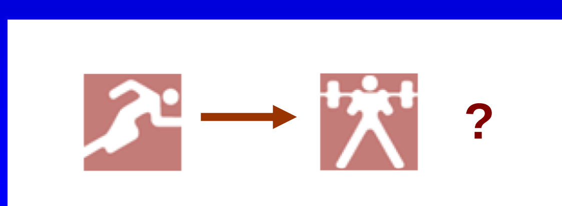
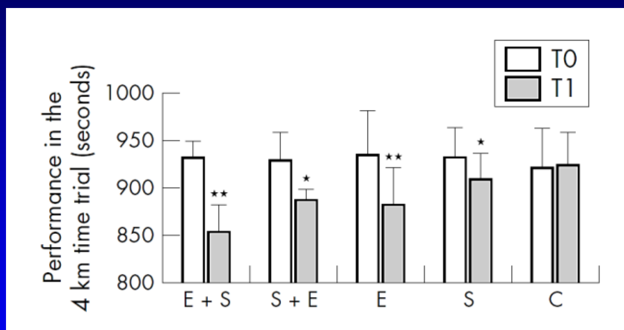


Order of concurrent training?

4 km maximal running



Pairwise comparisons

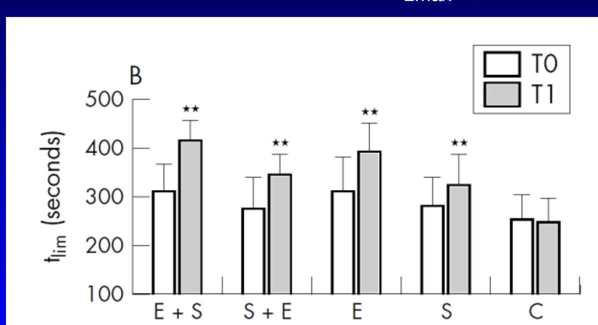
	S + E	E	S	C
E + S	**	*	**	**
S + E		§	*	**
E			**	**
S				§

Young male sports students (21 yrs, SD 1.3 yrs)
12 wks of training, 2 sessions per week

Chtara, Millet et al, Br J Sports Med 2005

Order of concurrent training?

Time to exhaustion at VO_{2max} speed



Pairwise comparisons

	S + E	E	S	C
E + S	**	*	**	**
S + E		*	**	**
E			**	**
S				**

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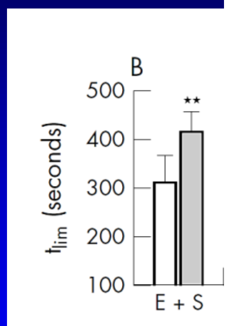

What is already known on the topic

Recent studies have shown that adding strength training to endurance training improves both aerobic capacity and endurance performance. However, the effects on endurance performance of the order in which the two types of training are performed in the same session have not been studied.

What this study adds

Endurance training followed by strength training produced greater improvements in endurance performance and aerobic capacity than the reverse order or if the training methods were performed separately.

:omparisons

E	S	C
*	**	**
*	**	**
E	**	**
	S	**

Young male sports st
12 wks of training, 2 sessions per week

Chtara, Millet et al, Br J Sports Med 2005

If performed in the same training session:
E exercise before S exercise!
(when the aim is to improve running performance)