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Effects of heavy strength training on performance determinants and performance in cycling and running

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Abstract

This thesis presents data from one large research project resulting in four research papers. The main aim of the project was to examine the effects of adding heavy strength training to female endurance athletes' normal endurance training on performance in cycling and running, factors affecting performance, and possible mechanisms behind changes in performance and performance determinants. The secondary aim of the thesis was to compare strength related adaptations after a strength-training program between endurance athletes maintaining their normal endurance training and untrained participants.

To answer the main research question, nineteen well-trained female duathletes ($\text{VO}_{2\text{max}}$ cycling: $54 \pm 3 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$, $\text{VO}_{2\text{max}}$ running: $53 \pm 3 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) were randomly assigned to either normal endurance training (E , $n = 8$) or normal endurance training combined with strength training ($E+S$, $n = 11$). To answer the second research question an additional group consisting of untrained females were recruited (S , $n = 10$). These participants performed the same strength training program as the athletes in $E+S$ but performed a maximal of one session of endurance training per week. The strength training program consisted of four lower body exercises (3 x 4-10 repetition maximum) twice a week for 11 weeks.

$E+S$ improved 40 min all-out cycling performance while E had no change. The improved performance in $E+S$ were related to improved cycling economy and improved fractional utilization of $\text{VO}_{2\text{max}}$, while $\text{VO}_{2\text{max}}$ remained unchanged. The main mechanisms behind improved cycling economy were increased muscle mass and muscle strength that probably made the athletes able to utilize the more economical type I fibers at higher power outputs after the intervention. A fiber type shift from type IIX to type IIA might also have contributed. Improved fractional utilization of $\text{VO}_{2\text{max}}$ was probably because of an increased muscle mass, which together with unchanged concentration of aerobic enzymes, made more mitochondria available for sharing a certain power output. Running economy, fractional utilization of $\text{VO}_{2\text{max}}$ in running, $\text{VO}_{2\text{max}}$ in running and hence 40 min all-out running performance did not change. $E+S$ improved 5 min all-out performance in both running and cycling tested after a prolonged period of submaximal work. No changes occurred in E . In cycling, the improved 5 min all-out performance after the intervention was related to improved cycling economy during the last 2 h of the prolonged cycling and to increased anaerobic capacity. No changes occurred in the prolonged running, so the improved 5 min all-out running performance were probably because of improved anaerobic capacity.

There were no differences in changes in the ability to develop force during low contraction velocities or in muscle hypertrophy between $E+S$ and S . However, S had a greater increase in maximal isokinetic knee extension torque at an angular velocity of $240^\circ\cdot\text{s}^{-1}$ and maximal squat jump height compared to $E+S$.

Sammendrag (Abstract in Norwegian)

I denne avhandlingen blir det presentert data fra et stort forskningsprosjekt som har resultert i fire forskningsartikler. Hovedmålsettingen for prosjektet var å undersøke effekten av legge inn en periode med tung styrketrening til kvinnelige utholdenhetsutøveres vanlige trening på prestasjonen og prestasjonsbestemmende faktorer i sykling og løping. I tillegg ble mekanismer bak potensielle endringer undersøkt. Et sekundært mål var å sammenligne typiske adaptasjoner til et styrketreningsprogram mellom utholdenhetsutøvere og utrente.

For å besvare hovedproblemstillingen ble nitten godt trente utøvere, aktive i både sykling og løping (VO_{2maks} sykling: $54 \pm 3 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$, VO_{2maks} løping: $53 \pm 3 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$), randomisert til å enten bare fortsette sin vanlige utholdenhets trening (E , $n = 8$) eller kombinere sin vanlige utholdenhets trening med styrketrening ($E+S$, $n = 11$). For å undersøke den sekundære problemstillingen ble i tillegg en gruppe utrente kvinner rekruttert (S , $n = 10$). De gjennomførte samme styrketreningsprogram som $E+S$, men med maksimalt en økt utholdenhets trening i uken. Styrketreningsprogrammet besto av fire øvelser for beina (3×4 -10 repetisjoner maksimum) og ble gjennomført to ganger i uken i elleve uker.

$E+S$ forbedret gjennomsnittlig effektutvikling i en 40 min prestasjonstest i sykling, mens det var ingen endring i E . Den forbedrede prestasjonen i $E+S$ var relatert til bedret sykkeløkonomi og forbedret utnyttingsgrad, mens det var ingen endring i VO_{2maks} . Den viktigste mekanismen bak bedret sykkeløkonomi var økt muskelmasse og muskelstyrke. Dette førte sannsynligvis til at utøverne kunne benytte de mer energiøkonomiske type I muskelfibrene på høyere effektutvikling etter intervensjonen. Det var en fibertypeovergang fra muskelfiber type IIA-X-IIX til type IIA som muligens også bidrog. Forbedret utnyttingsgrad etter styrketreningen skyldtes sannsynligvis økt muskelmasse, som sammen med uendret konsentrasjon av aerobe enzymer, førte til at et større volum av mitokondrier kunne dele en viss effektutvikling. Løpsøkonomi, utnyttingsgrad i løp, VO_{2maks} i løp og dermed løpsprestasjon i en 40 min prestasjonstest var uendret i begge grupper etter intervensjonen. $E+S$ bedret både gjennomsnittlig effektutvikling (sykkelprestasjon) og løpsdistanse i 5 min all-out prestasjonstester, gjennomført rett etter en langvarig submaksimal arbeidsperiode. Ingen endring skjedde i E . Bedret sykkelprestasjon var relatert til bedret sykkeløkonomi de siste 2 timene av det langvarige arbeidet og økt anaerob kapasitet etter intervensjonen. Ingen endringer skjedde under det langvarige submaksimale arbeidet i løp, og bedret løpsprestasjon skyldtes sannsynligvis økt anaerob kapasitet.

Det var ingen forskjeller endringer i evnen til å utvikle kraft ved langsomme forkortningshastigheter eller i muskelvekst mellom $E+S$ og S . På tross av dette hadde S en større forbedring i evnen til å utvikle dreiemoment i kneleddet ved en vinkelhastighet på $240^\circ\cdot\text{s}^{-1}$ og i maksimal hopp høyde i knebøyhopp.

List of papers

- I. Vikmoen O, Ellefsen S, Trøen Ø, Hollan I, Hanestadhaugen M, Raastad T, Rønnestad BR. Strength training improves cycling performance, fractional utilization of VO_{2max} and cycling economy in female cyclists. *Scand J Med Sci Sports* 2015: doi: 10.1111/sms.12468
- II. Vikmoen O, Raastad T, Seynnes O, Bergstrøm K, Ellefsen S, Rønnestad BR. Effect of heavy strength training on running performance in female endurance athletes. *Manuscript submitted to Eur J Appl Physiol.*
- III. Vikmoen O, Rønnestad BR, Ellefsen S, Raastad T. Heavy strength training improves running and cycling performance following prolonged submaximal work. *Manuscript.*
- IV. Vikmoen O, Raastad T, Ellefsen S, Rønnestad BR. The adaptation to strength training differs between endurance athletes and untrained individuals. *Manuscript.*

Abbreviations

ATP	Adenosine triphosphate
CAF	Capillaries around each fiber
CAFA	Capillaries related to fiber area
CMJ	Counter movement jump
COX4	Cytochrome c oxidase subunit IV
CS	Citrate synthase
CSA	Cross sectional area
<i>E</i>	Endurance training only group
<i>E+S</i>	Endurance training combined with strength training group
EMG	Electromyography
ES	Effect size
F_{PT}	Patellar tendon force
HADH	Hydroxyacyl-CoA dehydrogenase
HR	Heart rate
$[la^-]$	Lactate concentration
Leg_{LM}	Lean mass in the legs
MVC	Maximal isometric torque
MyHC	Myosin heavy chain
RFD	Rate of force development
qRT-PCR	Quantitative real-time PCR
<i>S</i>	Strength training only group
SJ	Squat jump
VO_2	Oxygen consumption
VO_{2max}	Maximal oxygen consumption
V_{max}	Peak running performance during the VO_{2max} test
W_{max}	Peak cycling performance during the VO_{2max} test

1 Introduction

A lot of effort and resources goes into investigating how to optimize training regimes for performance in various endurance sports like cycling and running. Traditionally and logically, cyclists and runners have focused their training on different forms of endurance training (e.g. long slow distance training and various forms of interval training) with little focus on strength training. However, during the last decades, the potential performance enhancing effects of adding different forms of strength training to endurance athletes' normal training has received increased attention by coaches, athletes and researchers [1, 2, 21, 26, 28, 33, 38, 40, 65, 77, 100, 103, 113, 115-117, 130, 133, 135, 142, 144, 146, 161, 165, 166, 198, 206, 213, 215, 217-220, 227, 228, 235, 240-243, 264, 265]. Indeed, improvements in different forms of performance tests have been reported in both running [21, 65, 113, 142, 198, 228, 235, 240, 242], cycling [2, 103, 213, 215, 217, 218] and other endurance sports like cross-country skiing [e.g. 117, 165] and rowing [130] after addition of strength training to athletes' normal training. However, the performance enhancing effect of added strength training is not always observed in running [33, 77, 113, 144, 185, 186, 227] or cycling [26, 38, 40, 133, 161, 206]. In addition, the practical experience among coaches and athletes is conflicting, and anecdotally many coaches and athletes are skeptical to include strength training in their training regime.

The mechanisms behind the reported improvements in cycling and running performance after strength training is still somewhat unclear. Since performance in endurance sports are mainly determined by an athlete's maximal oxygen consumption ($\text{VO}_{2\text{max}}$), work economy and fractional utilization of $\text{VO}_{2\text{max}}$ [24, 139], any effect of strength training should be through changes in one or more of these factors. Adding strength training to normal endurance training seem to have neither a positive nor a negative influence on $\text{VO}_{2\text{max}}$ [2, 65, 100, 113, 135, 161, 198, 213, 217, 218, 228, 240, 241]. Work economy on the other hand has been reported to be improved in both running [8, 21, 86, 100, 135, 188, 198, 225, 228, 235, 240, 249] and cycling [22, 166, 217, 241] after strength training. However, the results are equivocal both in running [65, 77, 144, 220, 243] and especially in cycling [2, 26, 133, 206, 213, 215, 218]. In addition, which physiological adaptations responsible for the strength training induced improvements in work economy reported in some studies are unclear. Effects of adding strength training to endurance athletes' normal endurance training on the fractional utilization of $\text{VO}_{2\text{max}}$ has not been investigated.

In addition to the main performance determinants, the final result in running and cycling competitions will also be decided by other abilities like the ability to generate a high power output over short periods of time. This will for example be important during a mass sprint or break away attempts. Anaerobic capacity and anaerobic power are important determinants of these abilities. Since strength training can increase muscle mass, increase glycolytic enzyme activity and augment intracellular fuel stores of adenosine triphosphate (ATP) and phosphocreatine [96], strength training could in theory improve anaerobic capacity.

Even though improved performance have been reported in both cyclists and runners after addition of strength training, the effect of strength training on performance in both cycling and running in the same athletes are unclear. To my best knowledge, this has only been investigated in one study which reported that duathletes increased time to exhaustion at VO_{2max} by 11% in cycling and 13% in running after adding strength training to their regular training for 10 weeks [113]. However, that study had no control group, and the results should be interpreted with caution.

Unfortunately, most previous research in this area are performed using male participants. This is especially true regarding cycling. In fact, to my best knowledge only one study have investigated the effects of adding heavy strength training to normal endurance training on cycling performance in female cyclists and this study reported no effect [40].

The purpose of this thesis was to investigate the effects of 11 weeks of heavy strength training on cycling and running performance and important determinants of endurance performance in female endurance athletes. An additional aim was to elucidate mechanisms behind possible changes in performance and to link the specific mechanisms to the respective performance determinants. One important aspect was to compare the effects of strength training on performance in both cycling and running in the same athletes.

1.1 What determines performance in running and cycling?

The physiological factors important for long-term endurance performance can be incorporated into a model (schematically presented in figure 1), which states that long-term endurance is mainly determined by the amount of metabolic energy produced during the competition and how efficient this energy can be translated into mechanical work [24, 60, 139]. The model in figure 1 is slightly modified from the model reported in Basset & Howley [24] and will serve

as a framework for evaluating the effects of strength training on cycling and running performance in the current thesis.

The amount of metabolic energy that can be produced during an event (referred to as energy availability in the model) is the sum of energy from aerobic and anaerobic sources. The aerobic energy production can be estimated from measures of the total amount of oxygen consumption (VO_2) during an endurance event [139]. In this thesis, this will be referred to as “performance VO_2 ”.

Since the contribution that comes from aerobic sources increases with work duration [91], the relative importance of performance VO_2 compared to anaerobic capacity for the energy availability and hence performance will largely be dependent on the duration of the endurance event. In typical endurance sport with competition times above 5 min, most of the energy comes from aerobic sources [91]. Therefore, performance VO_2 will be the most important factor determining energy availability during typical endurance competitions. However, in shorter competitions, the anaerobic contribution will be substantial and anaerobic capacity will be important. In addition, as described below (chapter 1.2.3), anaerobic capacity can be important in longer competition during periods of higher power outputs as during decisive parts in mass start races.

The average speed or power output a certain amount of metabolic energy can generate is decided by how efficient the aerobic energy can be used for mechanical work, referred to as “work economy”.

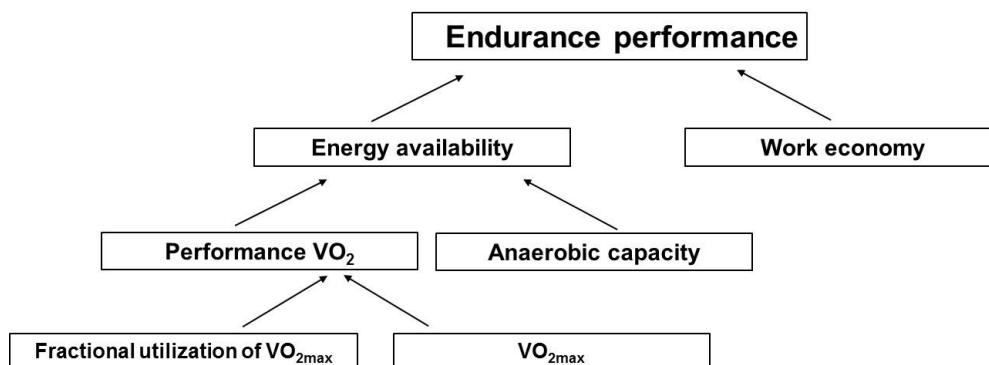


Figure 1. A model illustrating the interaction of important determinants for endurance performance. This model will serve as a framework for evaluating the effects of strength training on cycling and running performance in the current thesis. For details, see text. Modified from Basset and Howley [24].

1.1.1 Performance VO_2

Performance VO_2 is determined by VO_{2max} and the percentage of VO_{2max} that can be maintained for the duration of an endurance event [24, 60, 201]. In this thesis, this percentage will be referred to as the “fractional utilization of VO_{2max} ”.

1.1.1.1 VO_{2max}

VO_{2max} is defined as the highest rate at which oxygen can be taken up and utilized by the body during severe exercise [162]. VO_{2max} is determined by the maximal cardiac output and the maximal arterio-venous oxygen difference and is during whole body exercise mainly limited by the lungs, heart and blood’s ability to deliver oxygen to the working muscles [24, 162]. VO_{2max} is one of the major factors determining endurance performance. It sets the upper limit for the performance VO_2 and is important for the performance VO_2 at endurance events of different durations [139, 162]. Elite endurance athletes may have VO_{2max} values up to 100% greater than normal active individuals [162, 179].

VO_{2max} has also been reported to correlate well with different measures of endurance performance in heterogeneous groups of athletes [32, 59, 84, 199, 200]. However, in well-trained to elite endurance athletes with similar VO_{2max} and performance the relationship between VO_{2max} and performance is small [56, 62, 170]. In addition, VO_{2max} does not seem to be the parameter that discriminate between professional world class cyclists and highly trained sub-elite cyclists [171]. Therefore, it seems like a relatively high VO_{2max} is a prerequisite for elite endurance performance, but VO_{2max} is not the most important factor separating endurance athletes at a high performance level.

1.1.1.2 Fractional utilization of VO_{2max}

Depending on the training status, athletes can sustain a work intensity that demands metabolism equal to VO_{2max} for about 6 min, but with large inter-individual variation [36]. At this intensity, the fractional utilization can be said to be 100% even though the delayed rise in VO_2 at the onset of exercise [137, 179] will mean that the actual accumulated VO_2 will be lower than VO_{2max} . Events lasting longer have to be performed at an average pace that does not evoke VO_{2max} [139], and the fractional utilization of VO_{2max} will fall below 100%. In such events performance VO_2 is not only decided by VO_{2max} , but also the fractional utilization of VO_{2max} .

The fractional utilization of $\text{VO}_{2\text{max}}$ have been shown to be well associated with endurance performance [55, 59, 62, 66, 128, 178], and its relative importance increases as the competition time increases [67].

The duration and distance of the endurance event will of course be one of the main determinants of fractional utilization of $\text{VO}_{2\text{max}}$ [239]. However, fractional utilization of $\text{VO}_{2\text{max}}$ varies between athletes even when competition time is kept constant [62]. The fractional utilization of $\text{VO}_{2\text{max}}$ is thought to mainly be determined by mitochondrial content of the muscles and hence the oxidative capacity [60, 118, 129]. This is based on the theory that the amount of mitochondria available to share a certain VO_2 is a major determinant of the degree of muscle stress during exercise [60, 118, 139]. Naturally, the development of muscular fatigue is the reason why athletes must eventually stop exercising at a certain percentage of $\text{VO}_{2\text{max}}$, and resistance to fatigue should therefore be an important factor deciding the fractional utilization of $\text{VO}_{2\text{max}}$. Factors causing muscular fatigue will not be discussed in detail here. However, some disturbances of muscle cells homeostasis which cause fatigue and stimulate increased muscle glycogenolysis, glycolysis and lactate production are also important to drive mitochondrial respiration to produce ATP via oxidative phosphorylation [118]. Therefore, with a relative large number of mitochondria available to share a certain VO_2 , the need for respiration through each mitochondria will be relatively small. This means that the disturbances in the muscle cell homeostasis needed to stimulate oxidative phosphorylation will be small leading to small development of fatigue. On the other hand, if a small number of mitochondria is sharing the same VO_2 the disturbances in cell homeostasis would be larger and fatigue will develop at a faster rate. Furthermore, it has been reported that in cyclists with similar aerobic enzyme activity in the muscles and similar $\text{VO}_{2\text{max}}$, distributing the power output or VO_2 over a larger muscle mass is related to a larger performance VO_2 [60, 61]. The suggested mechanism is that the larger muscle mass will increase the total numbers of mitochondria sharing the VO_2 and thereby reduce the requirement for ATP synthesis from each mitochondria in the same way as described above. This illustrates that it is the numbers of mitochondria sharing the VO_2 that is important for fractional utilization of $\text{VO}_{2\text{max}}$ and not the concentration of mitochondria in the muscles per se [60].

Even though accumulation of lactate not necessarily is a cause of muscular fatigue [9, 93], measurements of lactate concentration in the blood is often used to indicate disturbances in

the muscle cells homeostasis [60]. Therefore, the VO_2 at the lactate threshold is often taken as an indirect measure of fractional utilization of $\text{VO}_{2\text{max}}$ [24, 25, 139, 201].

Muscle capillary density has also been suggested to affect fractional utilization of $\text{VO}_{2\text{max}}$ [60]. A high capillary density is thought to increase muscle perfusion, reduce diffusion distances from the blood to inner part of the muscles, and hence aid removal of metabolites from the muscle during exercise and thereby delay muscle fatigue [60].

1.1.2 Work economy

As described, the $\text{VO}_{2\text{max}}$ and the fractional utilization of $\text{VO}_{2\text{max}}$ will together decide the performance VO_2 , the main factor deciding energy availability in most endurance events. According to the model, the average speed or the mean power output that can be generated from a certain performance VO_2 and therefore the performance time is decided by how efficient the energy can be transformed into mechanical work. The efficiency of the human body is the ratio between the external mechanical work done and the total energy expended [24]. On a cycle ergometer, one can easily measure the external work and calculate efficiency. Measurements of external work and calculation of efficiency is not always possible and an alternative approach is to express the oxygen cost of moving a certain distance or the oxygen cost of keeping a certain speed. This is usually referred to as economy of movement or work economy [24]. The inter-individual variation in work economy can be about 30% in running [224] and about 15% in cycling [62, 64]. The work economy is therefore highly relevant for performance and may explain why athletes with similar $\text{VO}_{2\text{max}}$ can differ in performance. In fact, close associations between work economy and endurance performance are reported in both cycling [122] and running [56, 69, 200]. Furthermore, it has been suggested that exceptional work economy can explain why some elite endurance athletes can perform at a high level despite relatively low $\text{VO}_{2\text{max}}$ [81, 85, 168, 169].

Work economy can be influenced by several factors. Some of these factors can affect work economy quite different in cycling and running, while others should affect both cycling economy and running economy. In addition, the factors affecting running economy seems to be more complex than cycling economy, which also might explain the higher inter-individual variation in running economy [60].

Muscle fiber type composition can probably affect work economy in both cycling and running since it has been reported that type I fibers are more economical than type II fibers, and type IIA fibers are more economical than type IIX fibers [20, 233, 256]. In fact, a positive

relationship has been observed between proportion of type I muscle fibers and both cycling efficiency [23, 64, 104, 122, 191, 205] and cycling performance [122]. The proportion of type I fibers has also been suggested to be related to running economy [224], although most studies report quite low correlations between fiber type composition and running economy [127, 157]. One possible reason for this is the more complex nature of running compared to cycling making it more unlikely to reveal a significant relationship between running economy and individual factors affecting it.

Movement and pedaling kinematics and other biomechanical factors during the pedal stroke might affect cycling economy [50, 147], but running kinetics, running kinematics and other biomechanical factors are probably more important for running economy. Multiple biomechanical factors have been thought to affect running economy, including factors like leg length, leg mass distribution, vertical displacement during the running stride, stride frequency and stride length [224]. An important factor for running economy seems to be the stiffness of the muscles and tendons in the legs [14, 155, 224]. During the eccentric phase of the running stride mechanical energy is stored in the muscles, tendons and ligaments acting across joints [211]. A partly recovery of this stored elastic energy during the concentric phase reduces the energy expenditure of running [224]. It has been estimated that the VO_2 during running at a certain speed would increase by 30-40% without this contribution of elastic energy [225]. Studies have confirmed this association between running economy and the stiffness of the muscle-tendon units of the lower legs, indicating that compliant patellar tendons, but stiffer Achilles tendons, are beneficial for running economy [14].

1.1.3 Anaerobic capacity and other determinants of performance

The three factors discussed above will together decide the highest average speed a person can sustain during typical endurance competition and therefore the shortest possible time a person can finish a given distance. However, in most modern endurance sports, athletes often do not finish competitions in the shortest possible time. This is for example the case in most mass start races. In the finals in big championships races is often ran at speed lower than what the athletes are capable of, and the race is decided by a sprint in the end. This is even more prominent in road races in cycling. In addition, even though the largest part of the metabolic energy during most competitions in cycling and running is derived from aerobic sources, in the shortest races quite a large part can come from anaerobic sources [91]. Furthermore, both cycling and running races often consist of longer periods of quite low intensity and shorter decisive periods of high intensity, for example during break away attempts and while riding

up steep hills. During these parts the anaerobic energy contribution might be substantial. The abovementioned factors are therefore not the only factors deciding the outcome of a competition.

Therefore, the anaerobic capacity and anaerobic power are also important factors for performance in endurance events [47, 266]. Tests that reflect these abilities are the maximal workload (velocity or power output) during a $\text{VO}_{2\text{max}}$ test during running or cycling (V_{max} or W_{max} respectively), peak and mean power output during the Wingate test and the peak velocity during a maximal anaerobic running test (V_{max}) [140, 196, 198-200, 213]. In fact, performance in these tests has been well related to performance both in cycling and running [13, 18, 32, 37, 39, 101, 108, 170, 196, 199, 200].

1.2 Effects of adding strength training on performance determinants and performance in running and cycling

In trained runners, adding strength training to normal endurance training has been reported to improve performance [21, 65, 113, 142, 198, 228, 235, 240]. This appears to be the case both after heavy load strength training [21, 65, 113, 228, 240] and explosive/plyometric strength training [198, 235]. Paavolainen et al. [198] reported that elite cross country runners improved 5000 m time and V_{max} after replacing 32% of their normal endurance training with running specific explosive and plyometric strength training for 9 weeks. However, these results are controversial since other studies report no beneficial effects on running performance after addition of strength training [33, 77, 113, 144, 186, 227].

Improved performance after addition of strength training to normal endurance training has also been reported in cycling [2, 103, 113, 146, 213, 217, 218, 241]. Aagaard et al. [2] reported improved power output during a 45 min all-out test in young elite male cyclists after adding 16 weeks of heavy strength training to their normal training, whereas cyclists who continued their normal training had no change in performance. However, the results in cycling are also equivocal since many studies reports no effect of concurrent training compared to endurance training only [26, 38, 40, 133, 161, 206].

As described above, endurance performance is mainly determined by the performance VO_2 and work economy. In addition, other characteristics like anaerobic capacity and anaerobic power may be important for the outcome in relatively short competitions and under certain conditions in longer competitions. Therefore, the reported improvements in cycling and

running performance after strength training should be accompanied by changes in one or more of these factors.

1.2.1 Performance VO₂

1.2.1.1 VO_{2max}

Adding strength training to an endurance athletes' normal training does not seem to have any additional effect on VO_{2max} than endurance training alone [65, 100, 113, 135, 161, 198, 213, 217, 218, 228, 240, 241].

Strength training does not even seem to improve VO_{2max} in previously untrained individuals [51, 94, 96, 112, 119, 126, 148], although there are some exceptions [181]. However, the male participants in the latter study had a very low initial VO_{2maks} (below 40 ml·kg⁻¹·min⁻¹) which probably explained the improved VO_{2max}.

The lack of effect of strength training on VO_{2max} is not surprising considering the demands for energy expenditure during a strength training session. The average VO₂ consumed during a strength training session have been reported to be about 45% of VO_{2max} in non-endurance trained individuals [48]. This VO₂ was similar to walking/running at a treadmill at speeds between 5-8 km·h⁻¹ and gave an average heart rate of 110 beats·min⁻¹ [48]. This is hardly enough to lead to improvements in VO_{2max} perhaps with the exception in individuals with very low cardio-respiratory fitness.

Since strength training may increase body mass and muscle mass [82], it might be speculated that strength training in endurance athletes may have a negative impact on VO_{2max} related to body mass. It is therefore important to note that none of the studies cited above find a negative effect on VO_{2max} when endurance athletes add strength training to their normal endurance training.

1.2.1.2 Fractional utilization of VO_{2max} and the lactate threshold

Studies measuring fractional utilization of VO_{2max} after a strength training period in endurance athletes are lacking. However, the VO₂, velocity or power output at the lactate threshold is often used as indirect measurements of the fractional utilization of VO_{2max} [24, 201]. Unfortunately, because the lactate threshold reported in absolute values also is determined by the VO_{2max} and work economy [24], the lactate threshold should be expressed in percent of VO_{2max} to better represent the fractional utilization of VO_{2max}. The few studies measuring changes in this variable after addition of strength training in cyclists and runners

reports no change [188, 218, 240, 241]. However, in untrained individuals a 12% increase in VO_2 at the lactate threshold relative to $\text{VO}_{2\text{max}}$ has been reported after strength training [177]. However, the strength training program consisted of a circuit of 10 exercises with a relative high number of repetitions (15-20 RM), and the rest interval between exercises were low (30 s). Consequently, this training program may have led to some endurance adaptations in these untrained individuals.

Nevertheless, even not measured directly, there are indications of improved fractional utilization of $\text{VO}_{2\text{max}}$ in cyclists after addition of strength training [2, 213]. In these studies well-trained to elite cyclists increased average power output during relatively long all-out tests (40 and 45 min) after a period of concurrent training. Since $\text{VO}_{2\text{max}}$ and work economy did not change or changed to the same degree in the concurrent training and the control group, improved fractional utilization of $\text{VO}_{2\text{max}}$ is indicated. In fact, in the study by Aagaard et al. [2] the authors estimated that the average power output for the strength-training group during the test increased from 76 to 83% of the power output at 100% of $\text{VO}_{2\text{max}}$. Since the cycling economy did not change, the performance VO_2 probably increased and with no change in $\text{VO}_{2\text{max}}$ so did probably also the fractional utilization of $\text{VO}_{2\text{max}}$.

Regarding the velocity or power output at lactate threshold, most studies report no change after a period of concurrent training relative to control athletes in both trained runners [77, 198, 235, 240, 243] and trained cyclists [2, 133, 241]. However, there are two studies in both running [100, 185] and cycling [213, 218] that report improvements. Rønnestad et al. [218] reported that a group of elite male cyclist increased their power output at a blood lactate concentration of $4 \text{ mmol}\cdot\text{L}^{-1}$ after 25 weeks of heavy strength training. No changes in $\text{VO}_{2\text{max}}$ and cycling economy indicate that the fractional utilization of $\text{VO}_{2\text{max}}$ might have been improved, although the indirect measure of lactate threshold in percent of $\text{VO}_{2\text{max}}$ did not change.

1.2.2 Work economy

A large numbers of studies have reported improved running economy, ranging from 3-8%, when runners add strength training to their normal endurance training [86, 100, 135, 188, 198, 225, 228, 235, 240, 242, 243, 249]. This seems to apply both for runners on a recreational level [135], highly trained elite runners [225], and for both men [240] and women [249]. However, improved running economy is not a universal finding [65, 77, 186, 220]. Improved running economy seems to be one on the main mechanism behind the reported improvements

in running performance after concurrent training in endurance athletes. This is supported by the fact that the studies that do not report improved running economy after strength training also report no change in running performance [77, 113, 144, 186].

However, the effects of adding strength training to trained cyclists' normal training on cycling economy or cycling efficiency is more unclear since some studies reports improved cycling economy [22, 166, 241], whereas other studies reports no effect [2, 26, 133, 213, 215, 218]. Improved cycling economy after a strength training intervention has also been documented in untrained individuals [167] and non-cyclists [102]. After a closer scrutiny of these studies it looks like the cyclists in the studies which do not report improvements in work economy [2, 213, 215, 218], were at a higher performance level than the cyclists in the studies where improvements were observed [22, 166, 167, 241]. Therefore, it seems to be difficult to improve economy in highly trained cyclists just by adding strength training to their normal training. Perhaps cycling economy is already highly optimized in elite cyclists and therefore difficult to improve further.

In the studies mentioned so far, changes in cycling economy have been investigated with the participants in a non-fatigued state. However, mass start races in cycling can last up to 6-7 h, and the effects of strength training on cycling economy when the participants are fatigued is therefore highly interesting. Adding a heavy strength-training program for 12 weeks to well-trained cyclists normal training led to reduced VO_2 during the last hour of a 3 h submaximal cycling trial compared to cyclists only continuing their normal endurance training [217]. The improved cycling economy was accompanied by improved 5 min all-out performance tested directly after the prolonged cycling. Interestingly, when measured in the non-fatigued state, no change in cycling economy was observed [213].

Improved cycling economy after addition of strength training probably contributed to the improved cycling performance reported in some studies [241]. However, there are studies reporting improved cycling performance after addition of strength training without changes in cycling economy [2, 213, 215]. Improved cycling economy is therefore not the only mechanism behind improved cycling performance after added strength training. In addition, the physiological changes responsible for possible changes in cycling economy after strength training is quite unclear.

1.2.3 Anaerobic capacity and other determinants of performance

As described in chapter 1.1.3 many mass start competitions in both cycling and running is performed with quite variable intensity, and the final outcome of a race often is decided with an short period of all-out effort in the end. In addition, during quite short competitions, a quite large part of the metabolic energy will come from anaerobic sources, and anaerobic capacity will be important for the “energy availability” term in the model (figure 1). This will for example be the case in the middle distances in running and in certain cycling events (some track events and short prologues). Muscle mass is an important factor determining anaerobic capacity [19] and strength training could therefore have a positive impact on anaerobic capacity. However, the results from the literature regarding this is quite unclear. Sixteen week of heavy strength training did not improve 5 min all-out performance in young elite male cyclists. On the other hand, 5 min all-out performance was increased following 12 weeks of heavy strength training in well-trained male cyclists [217]. However, in the latter study performance was tested following 3 h submaximal cycling and the performance enhancements after the strength training protocol were related to reduced physiological stress during the submaximal cycling [217]. In running, strength training have been reported to increase time to exhaustion on maximal aerobic running speed (test duration around 5-6 min) [240]. W_{max} and V_{max} can be considered as measures of performance [114] and these measures has a quite large anaerobic component [114, 136]. Improved W_{max} and V_{max} have been reported after the addition of strength training to endurance athletes’ normal training [21, 65, 198, 213, 242] and after strength training in untrained individuals [167]. However, this is not a uniform finding [135, 187, 243]. The reasons for these divergent findings are unclear, but may be related to differences in the strength-training program performed.

Performance in the Wingate test in cycling and the V_{mart} test in running is largely dependent on anaerobic power and capacity [31, 184, 189, 199, 200, 250]. Strength training has been reported to increase peak power output in the Wingate tests in non-cyclists [29, 54] and well-trained cyclists [213, 218], as well as mean power output in both untrained individuals [29, 54] and trained cyclists [26]. Both addition of explosive and heavy strength training with endurance training have been reported to increase V_{mart} in runners [185, 186, 198]. On the other hand, 8 weeks of strength training failed to increase anaerobic capacity measured as accumulated O_2 deficit in previously untrained men, despite increased time to exhaustion on 120% of VO_{2max} [190].

1.3 Effects of performing concurrent training on basic adaptations to strength training

Based on the training principle of specificity, the muscular adaptations to training depends on the specific training performed. Strength training induces muscle fiber hypertrophy and neural adaptations leading to improved maximal strength [82]. Endurance training on the other hand increases muscle aerobic enzymes activity, intramuscular glycogen stores, capillary density, and leads to central circulatory adaptations [109, 136, 210] resulting in increased VO_{2max} and endurance performance [136]. However, endurance training has been associated with impaired maximal strength and reduced muscle fiber cross sectional area (CSA) [78, 148, 257, 258]. Therefore, performing concurrent endurance training together with strength training may hamper the typical adaptations to strength training. Since the pioneering study by Hickson [112], reporting that performing strength and endurance training in the same training program negatively affects the gain in maximal strength compared to strength training alone, this have been investigated quite extensively [6, 16, 17, 30, 49, 51, 68, 70-72, 89, 92, 94, 96, 101, 107, 112, 119, 126, 131, 138, 141, 148, 159, 160, 181, 182, 194, 195, 207, 209, 214, 222, 223, 231, 262]. Many of these studies have confirmed that concurrent training can lead to impaired strength gains [30, 49, 92, 112, 126, 131, 138], impaired muscle hypertrophy [30, 68, 141, 148, 207] and impaired neural adaptations [49, 101]. However, many studies contradict these findings and reports no negative effects on strength related adaptations after concurrent training compared to strength training only [6, 51, 96, 119, 181, 182, 195, 231]. Numerous variables are possible to manipulate in the design of these studies and the conflicting results are probably related to many different methodical aspects that vary between studies. These include training status of the study participants, modality and frequency of the strength training and endurance training, total numbers of training sessions, length of intervention, selection of dependent variables and how the two types of training are integrated. There is difficult to detect a pattern explaining which methodical features that are most important. However, the total amount of training sessions per week seems to have an effect. Although exceptions exists [68, 92, 96, 131], in most of the studies reporting attenuated strength gains, participants in the concurrent training groups performs training more than 5-6 days per week [30, 71, 94, 112, 148, 207]. In the studies that do not report impaired strength gains after concurrent training, the numbers of trainings days is usually lower [6, 51, 119, 181, 182, 195, 231]. In elderly individuals, impairments in strength adaptations appears to occur with only four training days per week [49, 141]. Selection of

dependent variables may also be important as the ability to develop high power outputs and force at high contractions velocities seems to be more impaired than the ability to develop force at lower velocities as during a 1RM lift [71, 94, 101, 126].

Typically, studies investigating the concurrent training effect include three groups; one group performing strength training only, one group performing endurance training only and one group performing both the strength training and the endurance training. This means that the increase in total training volume is considerable larger in the concurrent group compared to the strength training only group. However, endurance athletes that add strength training to their normal endurance training are often at a steady-state training level. Therefore, compared to untrained participants who add the same amount of strength training, the increase in training volume would be similar. Studies investigating if endurance athletes adding strength training to their normal endurance training have impaired adaptations to the strength training program compared to untrained individuals is sparse, and they yield conflicting results [126, 214]. In well-trained male cyclists attenuated increases in strength and muscle CSA compared to untrained participants after a strength training program have been reported [214]. However, in that study the amount of endurance training was very large (over 10 h per week). Recreational active runners running 1-3 h per week have been reported to have no impaired adaptations to strength training [126].

1.4 Aims of the study

This PhD-thesis consist of one large research project. The main aim of the project was to examine the effects of adding heavy strength training to female endurance athletes' normal endurance training on performance in cycling and running, factors affecting performance and possible mechanisms behind changes in performance and performance determinants. The secondary aim of the thesis was to compare strength related adaptations after a strength-training program between endurance athletes maintaining their normal endurance training and untrained participants. More specifically the aims were to investigate:

1. The effects of adding heavy strength training to well-trained female endurance athletes' normal endurance training on performance determinants and performance in running and cycling (papers I and II).
2. The effects of adding heavy strength training to well-trained female endurance athletes' normal endurance training on physiological responses during prolonged submaximal work and subsequent 5 min all-out performance in both cycling and running (paper III)
3. Mechanism behind possible changes in performance and performance determinants in cycling and running after the addition of heavy strength training to normal endurance training in well-trained female endurance athletes (papers I, II and III).
4. The strength related adaptations to a strength-training program in endurance athletes maintaining their normal endurance training compared to adaptations to the same strength-training program in untrained individuals (paper IV).

The hypotheses were:

1. The addition of strength training to the normal endurance training in well-trained female endurance athletes will improve cycling and running performance measured in a 40 min all-out test. The improved performance will be related to improved fractional utilization of VO_{2max} and work economy.
2. The addition of strength training to the normal endurance training in well-trained female endurance athletes will improve work economy during the last part of a prolonged submaximal work period in both cycling and running. This will make the athletes able to perform better in the 5 min all-out performance test in both cycling and running. The improved performance in the 5 min all-out test will also be related to improved anaerobic capacity.

3. Improved work economy in both running and cycling will be related to an increased proportion of type IIA muscle fibers at the expense of type IIX muscle fibers, and to the ability of stronger and larger type I muscle fibers to contribute to a larger proportion of the total work. In addition, improved running economy will be related to changes in patellar tendon stiffness.
4. Improved fractional utilization of VO_{2max} will be related to an increased muscle mass in the legs together with no change in concentration of aerobic enzymes, making more mitochondria available for sharing a certain workload.
5. Endurance athletes maintaining their normal endurance training will have smaller improvements in strength and muscle hypertrophy than previously untrained participants after the same strength-training program.

2 Methods

2.1 Ethical approval

The study was approved by the Local Ethics Committee at Lillehammer University College. Written informed consent was obtained from all athletes prior to inclusion, and the study was carried out in accordance with the Declaration of Helsinki.

2.2 Participants

This thesis presents data from one large study containing the total of 38 participants. To answer the main research questions regarding the effects of heavy strength training on cycling and running performance (paper I, II and III), 28 female endurance athletes active in both cycling and running were recruited. The athletes fulfilled at least two of Jeunekdrup et al.'s [134] training and race status descriptions of a well-trained athlete. To be eligible for inclusion the athletes should have performed at least an average of four weekly endurance training sessions and not performed any systematic strength training (in average less than one strength training sessions per week) for the last 12 months leading up to the study. The athletes were matched on VO_{2max} and randomly assigned to either adding heavy strength training to the ongoing endurance training ($E+S$, $n=14$) or endurance training only (E , $n=14$). During the study, three athletes in $E+S$ left the project for reasons unrelated to the project protocol: one because of an injury, one because of a prolonged period off illness during the last part of the intervention and one because of other medical reasons. In E , six athletes left the study for reasons unrelated to the project protocol (injuries from bicycle crash, pregnancy and lack of time). Therefore, the final numbers of athletes in $E+S$ and E were 11 and 8 respectively. A questionnaire regarding the menstrual cycle and use of oral contraceptives was filled out by 9 athletes in $E+S$ and 7 in E . Six of these 9 athletes in $E+S$ used oral contraceptives and the corresponding numbers in E were 4 out of 7.

To answer the research question regarding the effects of concurrent strength and endurance training on strength related adaptations (paper IV) a group of untrained females were recruited. These individuals made up a group (S , $n=10$) that performed the same strength training program as the athletes in $E+S$, but performed a maximal of one session of endurance like training per week. The changes in strength related adaptations to the strength-training program were compared between $E+S$ and S (paper IV).

The characteristics of the participants are displayed in table 1.

Table 1. Characteristics of the athletes adding strength training to their normal endurance training (*E+S*), the athletes continuing their normal endurance training (*E*), and the untrained individuals performing strength training only (*S*).

Group	N	Age (years)	Height (m)	Body mass (kg)	BMI (kg/m ²)
<i>E+S</i>	11	31.5 ± 8.0	1.69 ± 0.05	62.2 ± 5.2	21.7 ± 1.3
<i>E</i>	8	34.9 ± 7.5	1.70 ± 0.03	65.8 ± 8.2	22.8 ± 2.8
<i>S</i>	10	31.0 ± 9.9	1.72 ± 0.04	67.8 ± 13.5	22.8 ± 3.9

Values are mean ± SD.

2.3 Experimental overview

The intervention period for *E+S* and *E* was during the competition period from April to July. Because of logistical restraints the intervention period for *S* was done after the intervention for *E+S* and *E* was complete. Therefore, *S* performed the strength training intervention between August and December.

The strength and endurance tests performed in *E+S* and *E* before and after the intervention period were organized over six test-days as displayed in table 2. There were 2-7 days between the test days, and all tests for each participant were completed within 2-3 weeks. During post test, athletes in *E+S* maintained their strength training with one session per week until all testing were complete. During the 2-3 weeks leading up to the strength and endurance tests pre-intervention, both groups performed MR scans for determinations of *m. quadriceps femoris* CSA, DXA scans for determinations of lean mass in the legs (leg_{LM}), measurements of patella tendon mechanical and morphological properties and took a muscle biopsy from *m. vastus lateralis*. During post-testing the DXA and MR scan were performed before or in-between the physical tests. The measurements on the patella tendon and muscle biopsies were done in the week after the last physical test.

The *S* group performed only the strength tests, jumping tests and DXA scan. The tests were done in the same orders as in the other groups (table 2). In *S*, none of the strength tests were preceded by any endurance tests.

Table 2. Organization of the physical performance test performed by the participants before and after the intervention.

Testday	Test
1	Blood lactate profile and VO_{2max} in cycling followed by 1RM* tests
2	Blood lactate profile and VO_{2max} in running followed by SJ*, CMJ*, MVC* and isokinetic torque*
3	Wingate test followed by 40 min all-out test in cycling
4	40 min all-out test in running
5	Prolonged submaximal running test followed by 5-min all-out running test
6	Prolonged submaximal cycling test followed by 5-min all-out cycling test

* Test also performed by $\dot{V}O_{2max}$, Maximal oxygen consumption; RM, Repetition maximum; SJ, Squat jump; CMJ, Counter movement jump; MVC, Maximal isometric torque.

2.4 Training

2.4.1 Endurance training

Duration and intensity of the endurance training performed by $E+S$ and E was calculated based on heart rate (HR) recordings. Endurance training was divided into three HR zones: 1) 60%-82%, 2) 83%-87% and 3) 88%-100% of maximal HR. There were no significant differences between $E+S$ and E in the average weekly duration of the endurance training and the distribution of this training within the three intensity zones (table 3). Also, no significant difference was found between $E+S$ and E in total weekly training duration including endurance training, competitions, strength training, core stability training, and other alternative training forms ($E+S$: $7.0 \pm 1.4h$, E : $6.8 \pm 2.5h$, $p = 0.87$).

Table 3. Duration (in hours per week) of the training performed during the 11 week intervention period for athletes adding strength training to their normal endurance training ($E+S$,) and athletes performing normal endurance training only (E).

	HR zone 1 (h)	HR zone 2 (h)	HR zone 3 (h)	HR zone 1+2+3 (h)	Leg strength (h)	Other (h)	Total
$E+S$	3.2 ± 1.4	1.1 ± 0.6	0.8 ± 0.6	5.1 ± 1.1	1.5 ± 0.0	0.3 ± 1.0	7.0 ± 1.4
E	4.5 ± 1.8	1.1 ± 0.3	0.8 ± 0.5	6.3 ± 2.2	0.0 ± 0.0	0.5 ± 0.8	6.8 ± 2.5

Values are mean \pm SD.

2.4.2 Strength training

The heavy strength training performed by *E+S* and *S* targeted leg muscles and was performed twice per week during the 11-week intervention period. Adherence to the strength training was high, with *E+S* athletes completing 21.4 ± 1.0 (range 19-22) and *S* participants completing 21.0 ± 0.8 (range 20-22) of the planned 22 strength-training sessions. The strength-training regimen was designed to improve cycling and running performance. Thus, strength-training exercises were performed using a range of motion from 90° knee flexion to almost full extension. In addition, since cyclists and runners work each leg alternately, one-legged exercises were chosen in two of the four exercises. The performed exercises were as follows: half squat in a Smith machine, one-legged leg press, standing one-legged hip flexion, and ankle plantar flexion (figure 2). The participants were instructed to perform the strength training with maximal acceleration and speed during the concentric phase (duration around 1 s), while the eccentric phase was performed more slowly (duration 2-3 s). At the start of each strength training session participants performed a 5-10 min warm-up at self-selected intensity on a cycle ergometer followed by 2-3 warm-up sets of half squats with gradually increasing load. For the first two weeks of the intervention, investigators supervised all training sessions. Thereafter, the follow-up frequency was kept at minimum once per week for the remainder of the intervention. During weeks one to three, participants trained with 10RM sets during the first session and 6RM sets during the second session. These alternating loads were adjusted to 8RM and 5RM during weeks four to six and were further adjusted to 6RM and 4RM during weeks seven to eleven. The numbers of repetitions were always the same as the prescribed RM load meaning that the sets were performed until failure. The participants were allowed assistance on the last repetition if necessary. The participants adjusted the absolute load as they got stronger to correspond to the prescribed RM load. The number of sets in each exercise was always three. During the warm up to every training session, the participants in both *E+S* and *S* eat a protein bar containing 15 grams of protein and 22 gram of carbohydrate (Squeezy recovery bar, Squeezy Sports Nutrition, Braunschweig, Germany).



Figure 2. Pictures of the strength training exercises performed in the study. A: Half-squat in a Smith-machine. B: One-legged legpress. C: One-legged hip flexion. D: Ankle plantar flexion.

2.5 Test procedures

2.5.1 Measurements of muscle mass and muscle size

CSA of *m. quadriceps femoris* was measured in *E+S* and *E* while the leg_{LM} was measured in all three groups. In group *E+S* and *E* the MRI scan were done right after the DXA scan measuring lean leg mass. The participants were instructed to refrain from physical activity for the 48 h leading up to the MRI and DXA scan and were instructed to refrain from ingesting any food or drinks for the last 2 h preceding the measurement.

2.5.1.1 CSA of *m. quadriceps femoris*

The participants lied on their back inside the MRI-machine (S-Scan, Esaote, Genova, Italy), and a coil was placed around the distal part of the upper thigh. Care was taken to assure similar positioning of the coil at pre and post. Twenty-three cross-sectional images were sampled starting at the proximal edge of the patella and moving up the thigh with 10 mm

between the images. Images were analyzed using the software OsiriX version 5.6 (Pixmeo, Geneva, Switzerland). Since the strength-training program mainly targeted the extensor muscles, *m. quadriceps femoris* were chosen for analyses. The 3-5 most proximal images (at the edge of the coil) had a low resolution and could not be used for analyses. Therefore, the CSA of the quadriceps muscles were measured using the four most proximal images with good resolution, and the average CSA of all four images was used for statistical analysis. For each participant, pre and post CSA were always calculated from the same images. The technician performing the MRI scan and the investigator performing image analyses were blinded for which group the participants belonged.

2.5.1.2 Lean mass in the legs

leg_{LM} was determined by DXA using a Lunar Prodigy densitometer (Prodigy Advance PA+302047, Lunar, San Francisco, CA, USA). Because of technical problems with some analyses, data from 2 participants in *S* had to be excluded. Therefore, the numbers of participants included in the leg_{LM} data in *S* are 8.

2.5.2 Tests of muscle strength and jumping performance

2.5.2.1 1 repetition maximum in one-legged leg-press and half squat

After a 10 min warm up at an easy and self-selected workload on a cycle ergometer in *S*, and 20 min after termination of the cycling VO_{2max} test in *E+S* and *E*, maximal strength in the legs was tested as 1RM in half-squat and one-legged leg press. Three to five days prior to the testing day, each participant was given a supervised familiarization session to learn proper lifting technique and find individual equipment settings. During this session, the load was gradually increased to allow estimation of a proper starting point for the 1RM testing.

The 1RM test started with a specific warm-up consisting of 3 sets with gradually increasing load (40, 75 and 85% of expected 1RM) and decreasing number of repetitions (10→6→3). The first attempt was performed with a load approximately 5% below the expected 1RM. If a lift was successful, the load was increased by approximately 5%. The test was terminated when the participants failed to lift the load in 2-3 attempts and the highest successful load lifted was noted as 1RM. Participants were given 3 min of rest between lifts.

2.5.2.2 Squat Jump (SJ) and Counter Movement Jump (CMJ)

After a 10 min warm up at an easy self-selected workload on a cycle ergometer in *S*, and 20 min after termination of the VO_{2max} in running in *E+S* and *E*, explosive strength was tested as

maximal jumping height in SJ and CMJ. These jumps were performed on a force plate (SG-9, Advanced Mechanical Technologies, Newton, MA, USA, sampling frequency of 1kHz). After 3-5 submaximal warm up jumps, the athletes performed 3 SJ and 3 CMJ with 2-3 min rest between each jump. The mean of the two highest SJ and CMJ were chosen for statistical analyses. During all jumps, the participants were instructed to keep their hands placed on their hips and aim for maximal jumping height. The SJ was performed from approximately 90 degrees knee angle. In this position, they paused for 3 s before the jump was performed. No downward movement was allowed prior to the jump, and the force curves were inspected to verify this. During the eccentric phase of the CMJ the participants were instructed to turn at a knee angle they felt was optimal for achieving maximal jumping height.

2.5.2.3 Maximal isometric and isokinetic torque in knee extension

Three to four minutes after the jumping tests maximal isometric torque (MVC) in knee extension and peak torque during a knee extension at $240^{\circ}\cdot\text{sec}^{-1}$ were tested in a dynamometer (Cybex 6000, Cybex International, Medway, USA). During these tests, the participants were seated with a 90° hip angle and were stabilized in this position using chest, hip and thigh straps. The input axis of the dynamometer was aligned with the participants' knee joint and the ankle was strapped to a lever arm. The participants held their arms in front of their chest during all tests. First, the participants performed three maximal knee extension against the lever arm with a 90° knee angle. The contractions lasted for 5 s and 1 min rest was given between each attempt. The participants were instructed to perform the muscle action as forcefully and quickly as possible. The attempt with the highest maximal torque was chosen for statistical analyses. Two min following the last MVC three maximal isokinetic knee extensions from 90° knee angle to full extension were performed against the lever arm at an angular velocity of $240^{\circ}\cdot\text{sec}^{-1}$. There was 1 min rest between each attempt and the attempt with the highest torque recorded during the contraction was chosen for statistical analyses. Strong verbal encouragement was given to the participants at all attempts.

2.5.3 Measurements of the mechanical and material properties of the patellar tendon

All the measurements of the mechanical and material properties of the patellar tendon were performed on the left leg. The measurements of patellar tendon mechanical properties, CSA and length were done as described in Helland et al. [110]. The procedure started with a 5 min warm-up on a stationary bike (Ergomedics 828, Monark, Varburg, Sweden). Then the athletes

were seated with a 90° angle in both knee and hip joint in a knee extension apparatus (Knee extension, Gym 2000, Geithus, Norway) instrumented with a force cell (U2A, Hottinger Baldwin Messtechnik GmbH, Darmstadt, Germany). To measure patellar tendon CSA, transversal scans were performed proximally, medially and distally along the tendon length using an B-mode ultrasound apparatus (HD11XE, Phillips, Bothell, WA, USA). Sagittal scanning was used to measure tendon length. A steel wire that was visible on the ultrasound image was attached to the ultrasound probe and, a pen was then used to mark the skin location of the patella apex and the osteotendinous junction.

To measure tendon force and elongation, the ultrasound probe was attached to the left knee with a custom made device. The athletes performed ramp contractions at a constant rate of 100 N·s⁻¹. This was achieved by following a drawn force curve on a monitor. After the knee extension test, maximal isometric knee flexion were performed. EMG data were recorded wirelessly (TeleMyo 2400 G2 telemetry Systems, Noraxon Inc., Scottsdale, AZ, USA) from the biceps femoris muscle during isometric knee extension and flexion to account for hamstring co-activation when calculating tendon force (see below). Tendon force (F_{PT}) was calculated as the force measured in the force cell, corrected for hamstring co-activation, internal and external moment arms as follows:

$$F_{PT} = ((F_q + F_h)M_e) / M_i$$

Where F_q is force measured by the force cell and F_h is estimated hamstrings co-activation force. M_i and M_e corresponds to internal and external moment arm respectively.

Tendon morphology data were analysed as previously described [110], using an image analysis software (ImageJ 1.45s, National Institute of Health, Austin, TE, USA). Tendon elongation data were analyzed using a video analysis software (Tracker Video Analysis and Modeling Tool, Open Source Physics, Douglas Brown, 2012). The patellar apex and the tibia plateau were digitally marked within a common coordinate system. The actual elongation of the tendon was calculated as the change in the distance between coordinates of these anatomical landmarks. To calculate tendon material and mechanical properties force-elongation curves were fitted with a 2nd degree polynomial. All the recordings used in the results had a fit of $R^2 = 0.92$ or higher. Stiffness was calculated as the slope of force – elongation curve between 90 and 100% of each athlete's maximal force. The Young's modulus was calculated by multiplying the stiffness values by the ratio between the patellar tendon resting length and mean CSA. Two sets of ultrasound data (from two *E+S* athletes)

had to be discarded because of an insufficient quality to enable analysis. Therefore, the number of athletes included in the data from tendon testing is 9 in *E+S* and 8 in *E*.

2.5.4. Running and cycling tests

The participants were instructed to refrain from intense exercise the day preceding testing, to prepare for the test as they would have done for a competition and to consume the same type of meal before each test. All cycling tests were performed on the same electromagnetically braked cycle ergometer (Lode Excalibur Sport, Lode B. V., Groningen, The Netherlands), which was adjusted according to each cyclist preference for seat height, horizontal distance between tip of seat and bottom bracket and handlebar position. During all cycling tests except the Wingate test, the ergometer was in a cadence-independent mode so that the power output was not affected by the cadence. The participants were allowed to choose their preferred cadence and used their own shoes and pedals. The running tests were performed on a motor driven treadmill (Woodway Desmo Evo, Waukesha, Wisconsin, USA). The inclination of the treadmill was set to 5.3% at all tests. All cycling and running tests were performed in similar environmental conditions (18-20 °C), and the participants were cooled with a fan during all cycling tests.

2.5.4.1 Blood lactate profile in cycling and running

The blood lactate profile tests consisted of 5 min work periods with progressively increasing workload. The workload at the first 5 min period was 50 W in cycling and 7 km·h⁻¹ running. In cycling, the power output was increased by 50 W to the second 5 min period and thereafter by 25 W for each 5 min period. In running, the velocity was increased by 1 km·h⁻¹ for each 5 min period. Blood were sampled from a finger-tip at the end of each 5 min bout and were analyzed for whole blood lactate concentration ([La⁻]) using a Lactate Pro LT-1710 analyzer (Arcray Inc., Kyoto, Japan). At the same time, rate of perceived exertion (RPE) was recorded using Borg's 6-20 scale [44]. The test was terminated when a [La⁻] of 4 mmol·L⁻¹ or higher was measured. VO₂ was measured during the last 3 min of each bout, and mean values were used for statistical analysis. VO₂ was measured (30 s sampling time) using a computerized metabolic system with mixing chamber (Oxycon Pro, Erich Jaeger, Hoechberg, Germany). The gas analyzer was calibrated with certified calibration gases of known concentrations before every test. The flow turbine (Triple V, Erich Jaeger, Hoechberg, Germany) was calibrated before every test with a 3 l, 5530 series, calibration syringe (Hans Rudolph, Kansas City, USA). From these continuous incremental tests, the power output (in cycling) and

running velocity at $3.5 \text{ mmol}\cdot\text{L}^{-1}$ [la^-] were calculated for each participant from the relationship between [la^-] and power output/velocity using linear regression between data points. Cycling economy was determined by the average VO_2 at a power output of 150 W and running economy by the average VO_2 at $10 \text{ km}\cdot\text{h}^{-1}$.

2.5.4.2 $\text{VO}_{2\text{max}}$ in cycling and running

After termination of the blood lactate profile tests, the participants cycled (after the cycle tests) or ran (after the running test) for 10 minutes at a freely chosen submaximal workload before completing another incremental test for determination of $\text{VO}_{2\text{max}}$. This test consisted of a continuous work period where the workload was increased every minute until exhaustion. In cycling, the test started at 100 W and the increases were 25 W. In running the test started at $8 \text{ km}\cdot\text{h}^{-1}$ and the increases were $1 \text{ km}\cdot\text{h}^{-1}$. $\text{VO}_{2\text{max}}$ was calculated as the average of the two highest 30 s VO_2 measurements. W_{max} and V_{max} were calculated as the mean power output/mean running velocity during the last 2 min of the incremental test. After the test blood [la^-] and HR_{peak} was noted. HR was measured using a Polar S610i heart rate monitor (Polar, Kempele, Finland).

2.5.4.3 Wingate test in cycling

The Wingate test was performed after a 10 min warm-up at a submaximal load on the Lode cycle ergometer (75-100 W), including three submaximal sprints during the last 2 min. The 30 s all-out test was preceded by pedaling at 60 rpm without braking resistance. Then, following a 3 s countdown, the braking resistance was applied to the flywheel and remained constant throughout the 30 s all-out test. Braking resistance was set to $0.67 \text{ Nm}\cdot\text{kg}^{-1}$ body mass. Peak and average power output during the 30 s was recorded. The participants remained seated throughout the test, and strong verbal encouragement was provided throughout. Participants were instructed to pedal as fast as possible from the start and not to conserve energy for the last part of the test.

2.5.4.4 40 min all-out performance tests

The 40 min all-out test in cycling was performed following 10 min cycling at a submaximal power output after the Wingate test. The 40 min all out test in running was performed after a 10 min warm up at submaximal workload. During the first 5 min of the tests, the power output/running velocity was set by the investigators. This individual selected workload was based on the lactate profile test and corresponded to the workload at $2.5 \text{ mmol}\cdot\text{L}^{-1}$ [la^-]. Thereafter, the participants were allowed to adjust the power output/running velocity

themselves. The participants were instructed to cycle with as high average power output as possible during the cycling test and to run as long as possible in the running test.

To estimate performance VO_2 and the fractional utilization of $\text{VO}_{2\text{max}}$ during the 40 min all-out tests, measurements of VO_2 were made during the last minute of every 5 min section of the tests. During this minute the participants were not allowed to adjust the workload. The average VO_2 during this minute was estimated to reflect VO_2 during the corresponding 5 min section. VO_2 was measured continuously during the last 5 min of the test as pilot testing showed that athletes performed numerous workload adjustments during this part of the test. Performance VO_2 was calculated as the average VO_2 of all 5 min sections, and fractional utilization of $\text{VO}_{2\text{max}}$ was calculated as performance VO_2 in percentage of $\text{VO}_{2\text{max}}$. The participants were allowed to drink water *ad libitum*.

2.5.4.5 Prolonged submaximal trials followed by 5 min all-out performance tests

The prolonged cycling lasted for 180 min on a power output corresponding to 44% of W_{max} (111 ± 9 W and 116 ± 8 W in *E+S* and *E* respectively). The prolonged running lasted for 90 min on a velocity corresponding to 60% of V_{max} (7.7 ± 0.4 km·h⁻¹ and 7.9 ± 0.3 km·h⁻¹ in *E+S* and *E* respectively). The same absolute workloads were used in the post-intervention test. VO_2 and HR were determined during 3 min periods every 30th min throughout the prolonged cycling and 3 min periods every 15th min throughout the prolonged running. RPE and $[\text{la}^-]$ were measured every 30th min during the prolonged cycling test and every 15th min during the prolonged running. The average values for each hour in cycling and each half hour in running were calculated and used for statistical analyses. During the prolonged trials the participants were allowed to consume water, and a sport drink containing 60 g·L⁻¹ carbohydrates, *ad libitum*. The amount of sport drink consumed were similar between groups, and from pre to post during both the prolonged cycling (across groups values were 1.24 ± 0.57 L and 1.26 ± 0.59 L at pre and post respectively) and the prolonged running (across groups values were 0.76 ± 0.27 L and 0.72 ± 0.24 L at pre and post respectively). After the conclusion of the prolonged trials, the participants got 3 min rest before 5 min all-out performance tests were performed. During the first minute of these tests the workload was set by the investigators. This individual selected workload was based on pilot work and corresponded to 85% of $W_{\text{max}}/V_{\text{max}}$. Thereafter the participants were allowed to adjust the workload themselves with the instruction to have the highest possible average power output in the cycling test and cover longest possible distance in the running test. The participants received feedback on instant workload and elapsed time, but not HR and average power

output/distance. At the post test, one athlete in *E+S* had to withdraw from the prolonged cycling trial due to pain in the hip. Therefore, the final numbers included in the statistical analysis of this test are 10 in *E+S* and 8 in *E*.

2.5.5 Muscle biopsy sampling

Muscle biopsies were sampled from *m. vastus lateralis* using the Bergström procedure and appropriate sized muscle samples were excised and selected for quantitative real-time PCR (qRT-PCR) analyses, homogenization/fractionation and later Western Blotting. Two samples were selected for immunohistochemical analyses, one was formalin fixed and one was mounted in Tissue-Tek. For details regarding biopsy handling, see appendix I.

2.5.6 Muscle biopsy analyses

2.5.6.1 Fiber type composition by immunohistochemistry

Details regarding the protocol for immunohistochemical analyses of muscle fiber type composition can be found in appendix I. Briefly, formalin-fixed muscle biopsies were paraffin-embedded and sectioned, whereupon transverse, serial sections were labelled for myosin heavy chain (MyHC) I (A4.840), MyHCIIA (*EPR5280*) and MyHCIIIX (*6H1*). Determination of muscle fiber composition was performed using Photoshop CS6 Extended (Adobe, San Jose, CA, USA). The investigator performing the image analyses were blinded as to which group the athlete belonged. Muscle fibers that were positive for both MyHCIIA and MyHCIIIX are referred to as muscle fiber type IIAX-IIX (see appendix I for details). Because of technical problems with some analyses, the number of individuals in the immunohistochemistry data is 8 in *E+S* and 8 in *E*.

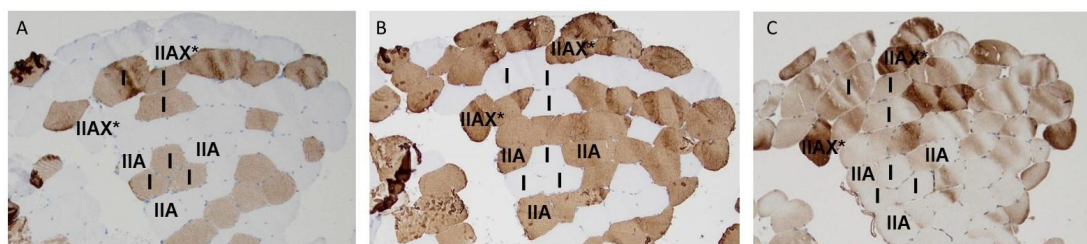


Figure 3. Representative labeling of myosin heavy chain (MyHC) in serial sections from muscle biopsies taken from one of the participants using antibodies towards A) MyHCII (A4.840), B) MyHCIIA (*EPR5280*) and C) MyHCIIIX (*6H1*). * Fibers positive for both antibodies towards MyHCIIA and MyHCIIIX are in the text referred to as type IIAX-IIX.

2.5.6.2 Western blotting analyses

For details regarding the Western Blotting analyses see appendix I. Briefly, ~50 mg of muscle tissue were homogenized and fractionated into cytosol, membrane, nuclear and cytoskeletal fractions. Membrane fractions (including the mitochondrial components) were analyzed. Proteins were separated on 4–12% SDS-PAGE gels in cold MES running under denaturing conditions. Thereafter, proteins were blotted onto a PDVF-membrane which were then blocked at room temperature for 2 h in a 5% fat-free skimmed milk and 0.05% TBS-T solution. Blocked membranes were incubated with antibodies against Citrate Synthase (CS), Hydroxyacyl-CoA dehydrogenase (HADH) and Cytochrome c oxidase subunit IV (COX4) overnight at 4°C, followed by incubation with secondary antibody at room temperature for 1 h. Bands were visualized using an HRP-detection system. All samples were run as duplicates and mean values were used for statistical analyses.

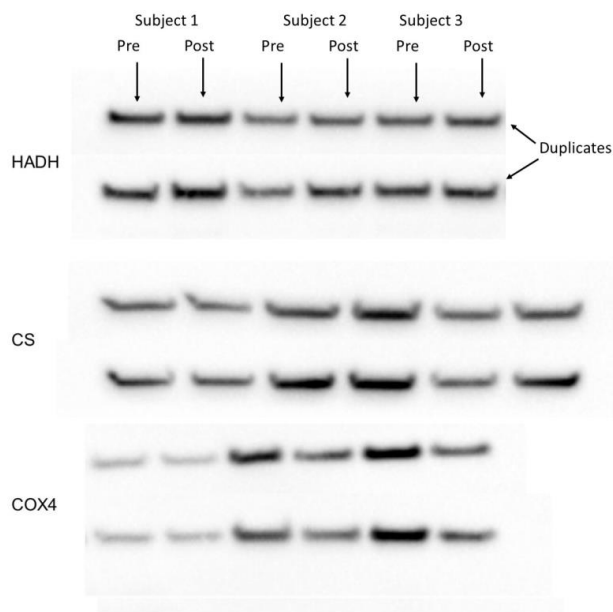


Figure 4. Representative Western Blot images for each of the measured proteins. Each lane represents three different individuals and two following bands are pre and post from the same individual. The two lanes for each protein are duplicate measurements. Note that the amount of protein inserted is the same in pre and post samples for each participant but not between different participants. Therefore, comparisons of bands between different participants is not relevant.

2.5.6.3 Capillarization and muscle fiber CSA measured by immunohistochemistry

Detailed description of these analyzes can be found in appendix I. Briefly, cross-sections 8 μm thick were cut using a microtome at $-20\text{ }^{\circ}\text{C}$. The muscle sections were blocked for 30 min with 1% BSA then incubated with antibodies against capillaries, MyHCII and dystrophin followed by incubation with appropriate secondary antibodies.

Muscle sections were visualized and pictures taken using a high-resolution camera mounted on a microscope. Fiber type distribution, fiber cross-sectional area and capillaries were identified using TEMA software (CheckVision, Hadsund, Denmark). Capillarization was expressed as capillaries around each fiber (CAF) and capillaries related to fiber area (CAFA), for type I and type II (IIA and IIX) fibers. Because of technical problems with some analyses, the number of athletes in the immunohistochemistry data is 8 in *E+S* and 5 in *E*.

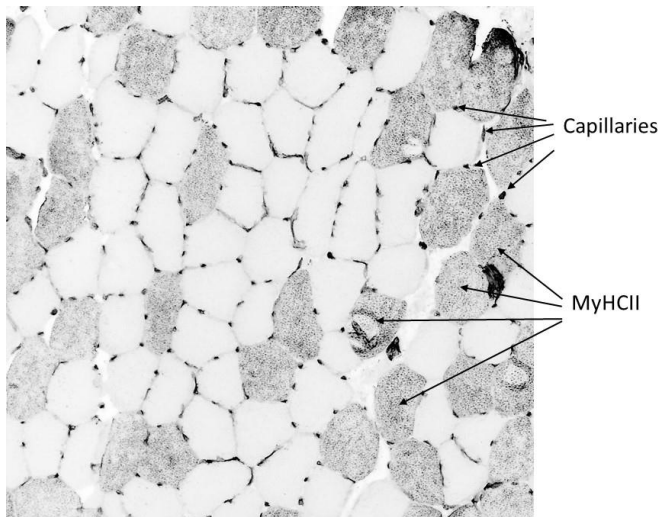


Figure 5. Representative labeling of capillaries and myosin heavy chain II (MyHCII) for classifications of capillaries related to fiber types in sections from muscle biopsies taken from one of the participants in the study.

2.5.6.4 Gene expression

Gene expression was assessed for genes involved in both aerobic and anaerobic energy metabolism. Details of primer design, RNA extraction, qPCR and evaluation of the stability of reference genes are described in appendix I. β_2 -microglobulin and ribosomal protein L32 were found to be the two most stable reference genes and were utilized for calculation of

normalisation factors using GeNorm, which were in turn utilized for calculation of target gene expression. All genes with associated primers are presented in table 4.

Table 4. Details of primers used for RT-qPCR.

Gene	Forward primer	Reverse Primer
CS	ACTGGCAAATCAGGAAGTGC	TCCTGAGTTGAGTGTGTTCCAG
SDHA	ATGCAGGCCTGGAGATAAAGTC	TGAGTCGCAGTCCCGATGTTC
SDHB	TGGCAGCGGTATAGAGAGAATG	GGATCTTGTCCCGATTTGAGC
SDHC	CGTGGCACTGGTATTGCTTTG	TCTTCAGGCCTTTTCTAGGTC
SDHD	TAGGAGGCCGAGCTCTGTTG	TCGGTCCTGAAGAAATGCTGAG
MDH1	GGAGAATTTGTCACGACTGTGC	TGGTCACAGATGGCTTTTGC
MDH2	TGCTGCTTGGGAAAAGGG	ATCTTCCCCTTCTTGATGGAG
MTCO1	TAATCGGAGGCTTTGGCAAC	GTTGTTTATGCGGGGAAACG
MTCO2	AACCACTTTCACCGCTACAC	ACGATGGGCATGAAACTGTG
MTCO3	TCGAGTCTCCCTTCACCATTTC	TTAGTTGGCGGATGAAGCAG
CYCS	ACACAGCCGCCAATAAGAAC	GTCTGCCCTTTCTTCTTCTTC
MyHCI	CAACGCAGAGTCGGTGAAG	TGTCCTCCTCCGTCTGGTAG
MyHCIIA	TGGAAGGAGAGGTTGAGAGTG	ACTCGCCTCTCATGTTTGC
MyHCIIIX	AGAAGCGCAATGTTGAAGC	TGCGGTCTTCTCAGTTTG
LDHA	ATTCAGCCCATTCCGTTAC	TTCCACTCCATACAGGCACAC
LDHB	CATGGATGGATTTTGGGGGAAC	AACACCTGCCACATTACAC
MCT1	TTGGAGTCATTGGAGGTCTTGG	CCAATGGTCGCCTCTTGTAG
MCT4	AGGCAAACCTCCTGGATGCG	AAAATCAGGGAGGAGGTGAGC
PFKM	TGACCTCCAGAAAGCAGGTAAG	AACCAGGCCACAATGTTC
GAPDH	AAGGCTGGGGCTCATTTG	ACGAACATGGGGGCATC
CPT2	AGCAGATGATGGTTGAGTGC	TCAAAGCCCTGGCCATTG
SLC25	GCATTGCAGGGATCTTCAACTG	ATATTTCCAGGAGGTGCAGTC

CS, Citrate Synthase; SDHA, Succinate dehydrogenase complex subunit A; SDHB, Succinate dehydrogenase complex subunit B; SDHC, Succinate dehydrogenase complex subunit C; SDHD, Succinate dehydrogenase complex subunit D; MDH1, Malate dehydrogenase 1; MDH2, Malate dehydrogenase 2; MTCO1, Cytochrome c oxidase subunit I; MTCO2, Cytochrome c oxidase subunit 2; MTCO3, Cytochrome c oxidase subunit 3; CYCS, cytochrome c somatic; MyHCI, Myosin heavy chain I; MyHCIIA, Myosin heavy chain IIA; MyHCIIIX, Myosin heavy chain IIX; LDHA, lactate dehydrogenase A; LDHB, lactate dehydrogenase B; MCT1, monocarboxylate transporter 1; MCT4, monocarboxylate transporter 4; PFKM, phosphofructokinase; GAPDH, glyceraldehyde 3-phosphate dehydrogenase; CPT2, carnitine palmitoyltransferase 2; SLC 25, carnitine/acylcarnitine translocase, member 20.

2.6 Statistics

All data in the text, figures and tables are presented as mean \pm standard deviation, unless otherwise stated. Prior to statistical testing, gene expression of aerobic enzymes data and protein data were log₂-transformed to maximize the likelihood of normal distribution. For data on muscle fiber composition, square-root arcsine-transformation was performed, representing the recommended transformation for proportional data [234].

To test for differences between $E+S$ and E at pre and post, and differences in changes from pre to post unpaired students t-test were used for all measurements except for evaluating responses during the prolonged trials. Within-group analyses (pre to post) were performed using paired t-tests.

To evaluate changes in responses during the prolonged trials within groups (pre to post) a two-way repeated measure analysis of variance (ANOVA) (time of intervention period and time during the prolonged trials as factors) with Sidek-Holm post hoc test were performed. To evaluate differences in changes in the responses during the prolonged trials between $E+S$ and E a two way repeated measures ANOVA (changes from pre to post in each group and time point during the prolonged trial as factors) with Sidek-Holm post hoc test were performed.

To test for differences between $E+S$ and S in strength related adaptations at pre and post, and differences in changes from pre to post, unpaired students t-tests were used and within-group analyses (pre to post) were performed using paired t-tests.

In addition, effect sizes (ES) for the key performance and physiological adaptations were calculated to compare the practical significance between the two groups included in the relevant analyses ($E+S$ vs E or $E+S$ vs S). Effects size were calculated as Cohen's d and the criteria to interpret the magnitude were the following: 0-0.2 = trivial, 0.2-0.6 = small, 0.6-1.2 = moderate, 1.2-2.0 = large and > 2 = very large [121].

Correlations analyses were done using the Pearson product-moment method and correlations coefficients were interpreted according to Hopkins et al. [121]; $r < 0.1$ trivial, 0.1-0.3 = small, 0.3-0.5 = moderate, 0.5-0.7 = large, 0.7-0.9 = very large, 0.9 = nearly perfect and 1.0 = perfect.

Analyses were performed in GraphPad Prism 6 (GraphPad Software Inc., California, USA) and Excel 2013 (Microsoft Corporation, Redmon, WA, USA). All analyses resulting in $p \leq 0.05$ were considered statistically significant and $p \leq 0.10$ are described as tendencies.

3 Results and discussion

3.1 Body mass, muscle strength and muscle hypertrophy

Body mass remained unchanged in *E+S* (from 62.4 ± 5.2 kg to 63.1 ± 5.6 kg) but was slightly reduced in *E* (from 65.6 ± 8.4 kg to 64.8 ± 8.0 kg, $p < 0.05$). The difference in change between groups were significant ($p < 0.05$). Because of the reduced body mass in *E* all VO_2 measurements are presented as body mass adjusted values. Since power output measured at a cycling ergometer does not correctly reflect the influence of body mass on outdoor cycling performance, especially during uphill cycling [13, 192], power output measurements are also reported as body mass adjusted values ($\text{W} \cdot \text{kg}^{-1}$). However, because running at a treadmill is influence by body mass to the same degree as outdoor running [183], no body mass adjustments are done on the reported running distances.

The mean 1RM in one-legged leg press and squat combined increased more in *E+S* than in *E* ($40.4 \pm 14.7\%$ vs. $4.5 \pm 5.3\%$ respectively, $p < 0.01$, figure 6), and the effect size analyses revealed a very large practical effect of *E+S* compared to *E* ($\text{ES} = 3.20$).

This increase in leg-strength is in the upper range compared to the previously observed 25 to 40% increase in 1RM in endurance athletes adding heavy strength training to their normal endurance training [22, 40, 100, 133, 135, 161, 188, 240]. Consequently, the strength training program used in the current study increased leg strength to a degree expected when endurance athletes add strength training to their normal endurance training. The results from the current study confirms previous results [e.g. 2, 65, 115, 116, 135, 206, 213, 218, 240, 241] that endurance athletes can achieve a quite large increase in muscular strength without increased body mass. This is important for runners and cyclists since increased body mass can negatively influence performance. It should be noted that the strength training probably had some effect on body mass in the present study since body mass was reduced in *E*, and the different in change in body mass between *E* and *E+S* were significant.

The results regarding the second research question; the effects of concurrent endurance training on typical adaptations to a strength training program (comparing strength related adaptations between *E+S* and *S*), are discussed in the last part of this thesis (chapter 3.10).

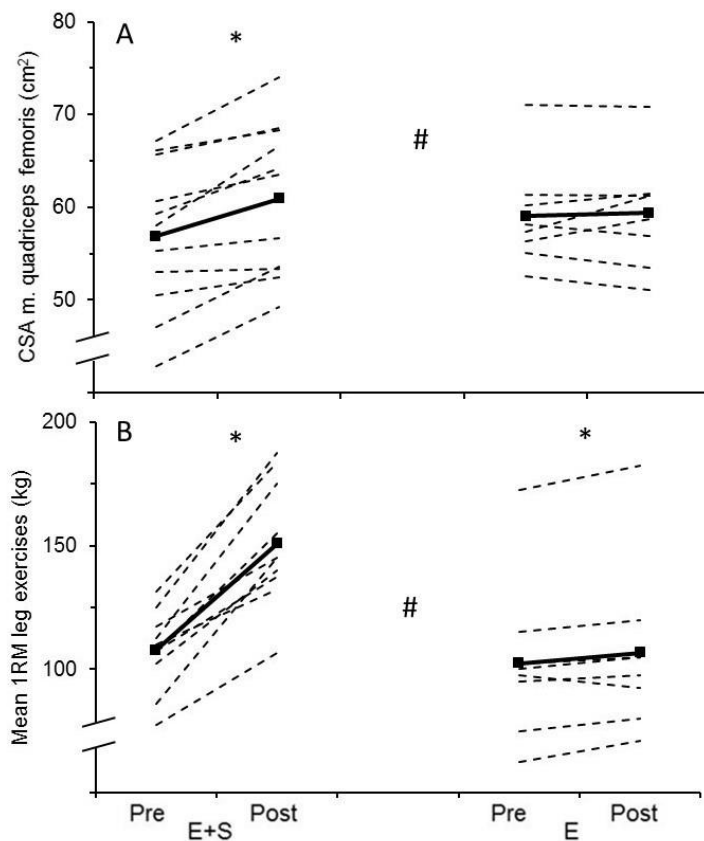


Figure 6. Individual values (dotted lines) and mean values (solid lines) before (Pre) and after (Post) the intervention period for athletes adding strength training to their normal endurance training (E+S), and athletes performing normal endurance training only (E). A: Cross sectional area (CSA) of the quadriceps femoris muscle. B: The mean 1 repetition maximum (1RM) in one-legged leg press and squat combined. * Different from Pre ($p < 0.05$), # the percent change from Pre is different between E+S and E ($p < 0.01$).

The CSA of *m. quadriceps femoris* increased in E+S with $7.4 \pm 5.3\%$ ($p < 0.01$, figure 6), whereas no change occurred in E. The change in CSA was larger in E+S than in E ($p < 0.01$) with a large practical effect of E+S compared to E ($ES = 1.57$).

In E+S, CSA of both type I and type II muscle fibers increased in *m. vastus lateralis* ($13.2 \pm 6.8\%$ and $30.8 \pm 19.6\%$, respectively, $p < 0.01$), while no change occurred in E (figure 7). Although this did not amount to a statistical difference between groups, E+S had a moderate practical effect on muscle fiber CSA compared to E ($ES = 0.83$).

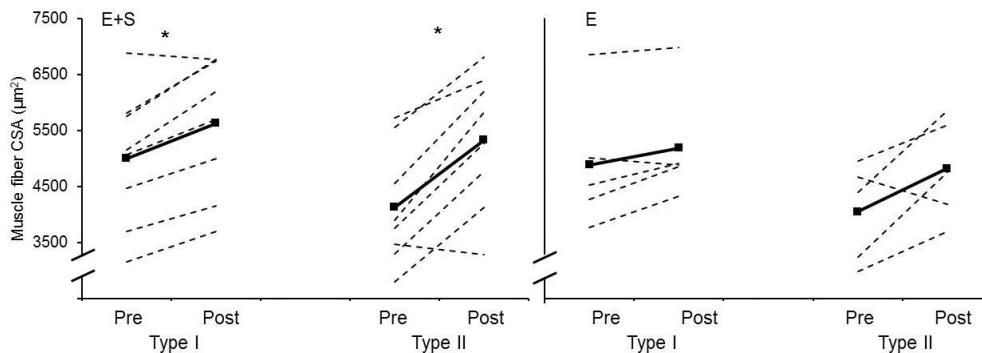


Figure 7. Individual values (dotted lines) and mean values (solid lines) before (Pre) and after (Post) the intervention period for athletes adding strength training to their normal endurance training (E+S, left panel), and athletes performing normal endurance training only (E, right panel). Muscle fiber cross sectional area (CSA) for both type I muscle fibers and type II muscle fibers * Different from Pre ($p < 0.01$).

These results clearly shows that the strength-training program used in the current study led to significant muscle hypertrophy. Therefore, some of the increased muscle strength observed was because of increased muscle CSA. The observed muscle hypertrophy is in agreement with increased muscle CSA or lean mass observed in endurance athletes adding similar strength training programs as used in the current study to their normal training [2, 165, 213, 218]. Increased muscle fiber CSA is not in accordance with a previous study in well-trained female cyclists [40] where no change in muscle fiber CSA was reported. A possible reason is that the strength training program in that study included only one exercise resulting in a low strength training volume [40]. Interestingly, there were no differences between the CSA of the type I and type II fibers in the current athletes, confirming the notion that in endurance athletes the type I fibers may be just as large [84, 232] or even larger [57] than the type II fibers.

3.2 Muscle fiber type composition

The proportion of fibers positive for both IIA and IIX MyHC were reduced from $9 \pm 7\%$ to 0% in E+S ($p < 0.01$) with a concomitant increase in fibers positive for type IIA only ($39 \pm 13\%$ to $51 \pm 10\%$, $p < 0.01$, figure 8). No changes in fiber type composition were observed in E.

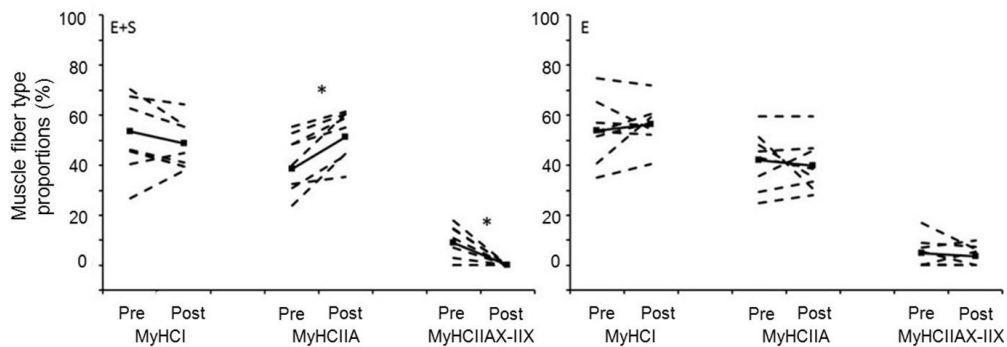


Figure 8. Individual values (dotted lines) and mean values (solid lines) before (Pre) and after (Post) the intervention period for athletes adding strength training to their normal endurance training (E+S, left panel), and athletes performing normal endurance training only (E, right panel). Immunohistochemistry determined muscle fiber proportions, presented as percentage of overall fiber abundance. * Different from Pre ($p < 0.01$).

The observed shift in fiber types from IIA-X-IIX towards pure type IIA fibers after a period of heavy strength training is in accordance with previous studies in untrained individuals [7, 12, 73, 148, 236-238]. In fact, in untrained individuals the decreased proportion of type IIX has been reported to occur already 2-4 weeks after initiation of heavy strength training [236, 237].

However, endurance training have also be reported to reduce the proportion of type IIX fibers [27, 124]. Therefore, it has been proposed that endurance athletes will have no additional effect of adding strength training to their normal training on muscle fiber type composition [40]. In fact, no change in the proportions of fiber type IIX was reported after 12 weeks of strength training in well-trained female cyclists [40]. Indeed, the cyclists in that study had small initial percentage of type IIX fibers (1.4%) and limited potential for type IIX to type IIA fiber transition. However, fiber type conversion from type IIX to type IIA after heavy strength training have been reported in elite male cyclists after 16 weeks of heavy strength training [2]. Together with the results from the current study this indicates that even though the initial proportions of type IIX and type IIA-X-IIX is low in endurance athletes (6 and 9 % respectively) compared to the 15-35% normally reported in untrained individuals [73, 236-238], addition of heavy strength training still have the potential to reduce this further. The lack of change in the E group shows that these athletes had stable fiber type composition, and that continuing normal endurance training does not alter muscle fiber type composition in well-trained endurance athletes.

3.3 Aerobic enzymes

To investigate possible effects of the three major pathways of aerobic metabolism we included a marker for the Krebs cycle (CS), oxidative phosphorylation (COX4) and the beta oxidation (HADH). The protein content of all three proteins per 50 mg of wet muscle did not change significantly in either group during the intervention period indicating that no changes in concentration of aerobic enzymes occurred (figure 9). In addition, it were small to none changes in expression of genes coding for aerobic enzymes during the study (figure 10).

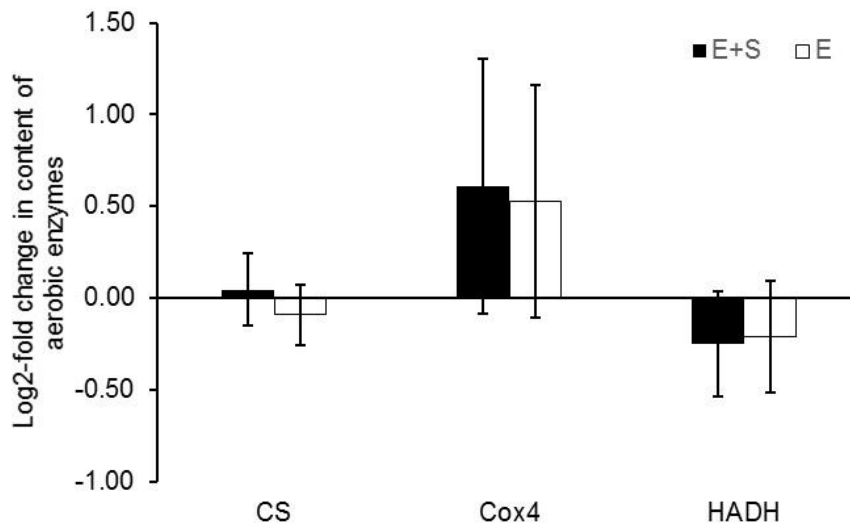


Figure 9. Log₂-fold change in protein levels of Citrate Synthase (CS), Cytochrome c oxidase subunit IV (COX4) and Hydroxyacyl-CoA dehydrogenase (HADH) during the intervention period for athletes adding strength training to their normal endurance training (E+S), and athletes performing normal endurance training only (E). Values are mean \pm CI.

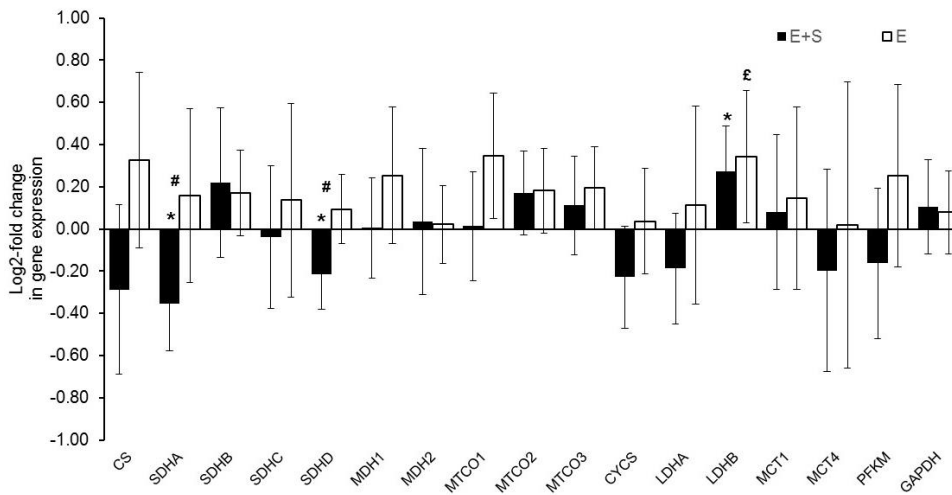


Figure 10. Log₂-fold change in GeNorm normalized mRNA levels of enzymes involved in aerobic and anaerobic metabolism during the intervention period for athletes adding strength training to their normal endurance training (E+S), and athletes performing normal endurance training only (E). * Different from Pre ($p < 0.05$), £ tendency to different from Pre ($p < 0.10$), # the percent change from Pre is different between E+S and E ($p < 0.05$). Values are mean \pm CI.

As confirmed in the current study, strength training may lead to quite large increases in muscle fibers CSA. It has been speculated that mitochondrial volume does not increase to the same degree after a strength training period, and that the increased muscle fiber CSA therefore will lead to a “dilution” of mitochondrial volume in the muscle cells [11, 173]. Indeed, numerous cross sectional studies reports lower mitochondrial density or lower activity and content of aerobic enzymes in strength-trained individuals compared to untrained [11, 173, 246]. However, most longitudinal training studies are in accordance with the results from the current study reporting unchanged content or activity of aerobic enzyme in previously untrained individuals [10, 30, 97, 254], although both reduced [53, 172, 173] and increased [203, 204, 244] content or activity of aerobic enzymes or mitochondrial function have been reported. This indicate that the absolute amount of mitochondria and aerobic enzymes usually increases to a similar degree as the muscle fiber CSA, leading to unchanged mitochondrial content. Wang et al [254] confirmed this in a study where 18 weeks of heavy strength training led to about 30% increase in muscle fiber CSA. However, the absolute volume of mitochondria increased to the same degree leading to no changes in the percent of the muscle fiber consisting of mitochondria [254].

However, these studies investigated the effects of strength training only and were performed with previously untrained participants and are therefore not directly comparable to the current study. Concurrent training in previously untrained participants seems to induce similar gains in aerobic enzyme activity compared to endurance training alone [30, 223], supporting the finding in the current study. However, Nelson et al. [195] demonstrated an increase in CS activity after 20 weeks of endurance training, but not after the same endurance training combined with strength training. In studies where endurance athletes add a period of strength training to their normal training [40, 113, 206], no change in the content or activity of the aerobic enzymes are reported indicating no negative effect of adding strength training on the concentration of aerobic enzymes in the muscle cells.

In fact, some studies indicate that strength training may actually enhance cell-signaling involved in mitochondrial biogenesis after endurance training in previously untrained individuals [253, 259]. A recent study involving recreationally active individuals reported that performing a strength training session 15 min after completing an endurance training session led to larger increase in mRNA levels of PGC-1 α than the endurance training session alone [253]. However, PGC-1 α exists in different isoforms with different roles in the signaling cascades leading to muscle adaptations after training. While PGC-1 α 1 is a marker of mitochondrial biogenesis, one isoform (PGC-1 α 4) seems to be more related to hypertrophy [221]. Unfortunately, since the primer pair used in the study by Wang et al (2012) were not able to separate the different isoforms of PGC-1 α [206] one cannot be sure if the increase in PGC-1 α mRNA was because of an increase in PGC-1 α 1 or some other isoform.

Nevertheless, all these studies taken together indicate that adding strength training to endurance athletes normal training does not negatively affect the content of aerobic enzymes per unit muscle. In fact, the unchanged concentration of aerobic enzymes together with increased muscle fiber CSA indicates that the total amount of mitochondria in the muscles has actually increased. As discussed below, this may have a positive impact on performance in endurance sports.

3.4 Capillarization

In *E+S* there were no changes in the numbers of capillaries around type I or type II fibers expressed as CAF or CAFA (figure 11). In *E* there were no changes in capillaries around type I fibers neither expressed as CAF or CAFA whereas in type II fibers a tendency to a reduction in both CAF ($-9.6 \pm 7.7\%$, $p = 0.06$) and CAFA ($-22.5 \pm 16.21\%$, $p = 0.09$) were observed.

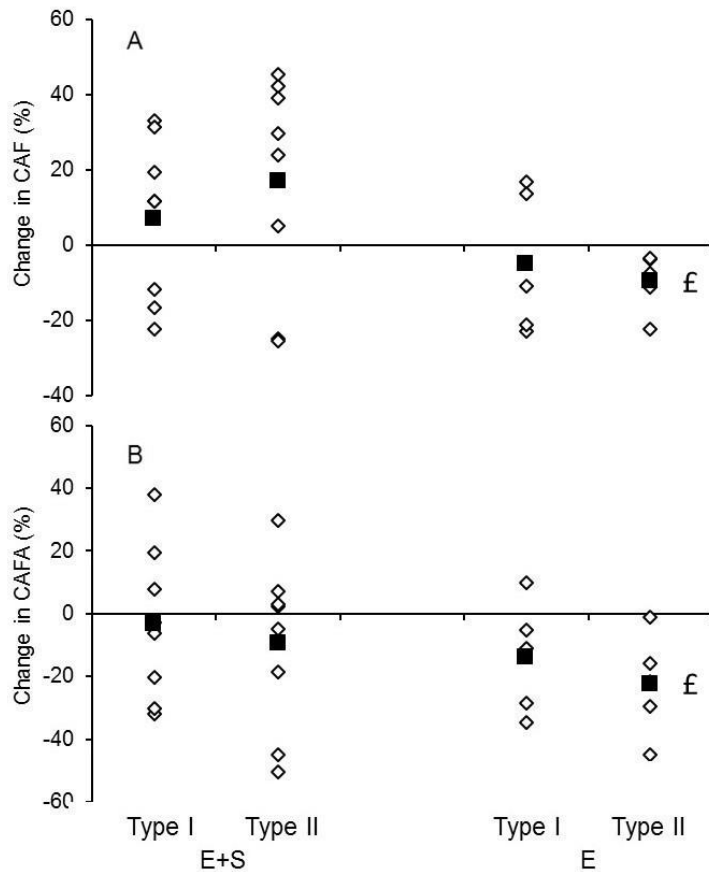


Figure 11. Individual values (open symbols) and mean values (solid squares) for athletes adding strength training to their normal endurance training (E+S), and athletes performing normal endurance training only (E). A: Percent change in capillaries around each muscle fiber (CAF) for both muscle fiber type I and muscle fiber type II. B: Percent change in capillaries related to fiber area (CAFA) for both muscle fiber type I and muscle fiber type II. £ Tendency to different from Pre ($p < 0.10$).

Anecdotally, some endurance athletes are afraid of including strength training in their training routine because of the fear of increased diffusion distances from the capillaries to the interior of muscle cells when the muscle cells grow bigger. In fact, cross-sectional studies have reported lower numbers of capillaries related to muscle fiber area in strength-trained athletes compared to untrained individuals [226, 247]. Since the numbers of capillaries around each fiber were similar, this was explained by larger muscle fiber CSA in the strength trained athletes [247].

However, despite significant muscle hypertrophy, addition of heavy strength training had no negative effect on CAFA in the current study indicating that the diffusion distances between blood and inner parts of muscle fibers did not change. The increased muscle fiber area was probably counteracted by a non-significant increase in CAF. Surprisingly, the only sign of change in capillarization was in *E*, where a tendency to both reduced CAF and CAFA in type II fibers were observed. However, because of the low numbers of athletes included in the analyses in *E* (n=5) these results should be interpreted with caution, but if anything these findings indicate that strength perhaps had a positive effect on capillary density.

In line with our finding, most intervention studies in previously untrained participants have reported no negative effect on CAFA despite increased muscle fiber area after strength training [30, 97, 105, 172, 180, 223, 254]. In most of these studies increased fiber area was accompanied by increased numbers of capillaries around each muscle fiber [97, 105, 180], although this increase was not always statistically significant [30, 105, 254]. This indicates that strength training resulting in muscle hypertrophy also will lead to capillary neof ormation and proliferation to such degree that no changes in CAFA occurs. The finding in the current study is also in agreement with results reported in elite male cyclists after 16 weeks of heavy strength training [2]. Based on all these studies, endurance athletes should not be afraid of reduced capillary density when they consider adding heavy strength training to their ongoing endurance training.

3.5 Mechanical and material properties of the patellar tendon

There were no significant changes in stiffness or Young's modulus of the patellar tendon in neither *E+S* nor *E*. The mean CSA of the patellar tendon increased by $5.2 \pm 3.6\%$ ($p < 0.05$) in *E+S* whereas no significant changes occurred in *E* (table 5).

Table 5 Stiffness, Young's modulus and mean cross section area (CSA) of the patellar tendon before (Pre) and after (Post) the intervention period for athletes adding strength training to their normal endurance training (E+S), and athletes performing normal endurance training only (E).

	<i>E+S</i>		<i>E</i>	
	Pre	Post	Pre	Post
Stiffness (N·mm ⁻¹)	2752 ± 402	2483 ± 733	2753 ± 947	2692 ± 697
Young's Modulus (MPa)	1038 ± 194	925 ± 162	1251 ± 296	1158 ± 273
Mean CSA (mm ²)	65.9 ± 7.1	69.2 ± 6.9*	60.3 ± 4.2	59.9 ± 4.4

Values are mean ± DS, * Different from Pre ($p < 0.05$).

The lack of change in the stiffness of the patellar tendon is in contrast to many studies reporting increased stiffness after periods of heavy strength training in previously untrained participants [145, 151, 153, 156, 208, 230]. The reason why the current study contradicts most other is unclear but may be related to methodological differences.

One possible reason can be that our study involved female participants whereas most previous studies are performed with males [145, 151, 153, 156, 208, 230]. In fact, female tendons have been reported to show a lower rate of new connective tissue formation and lower mechanical strength in response to mechanical loading [174].

The lack of changes in tendon stiffness may also be because of the training background of the athletes in the current study. Even though the participants in the current study were non-strength trained, they had a large base of endurance training including running. Runners have been reported to have stiffer muscle-tendon unit in the *m. vastus lateralis* than untrained [152], and it may therefore be speculated that additional stiffening of the patellar tendon does not occur after strength training in runners. In fact, no change in Achilles tendons stiffness was observed in highly trained runners after 8 weeks of isometric strength training [80]. In addition, the athletes in the current study also performed quite large amounts of concurrent endurance training that might lead to different adaptations in tendon stiffness compared to strength training only.

Differences in the strength-training program may also explain the lack of changes in the current study. Most of the previous studies reporting increased patellar tendon stiffness include heavy knee extension exercise [145, 153, 208, 230] or isometric muscle actions [151, 156]. In the current study, the exercises involved were more complex involving multiple joints that perhaps reduced the absolute mechanical loading on the patellar tendon compared to pure knee extension exercise. In addition, the athletes were instructed to perform the concentric phase of the exercises as fast as possible making the time under tension quite low.

The increased mean CSA of the patellar tendon is in line with some previous studies with untrained participants [145, 230]. However, this is not an universal finding [151, 153, 156, 208]. The reasons for the discrepancy in the literature is unclear, but it may be related to different methodologies used to measure tendon CSA and where on the patellar tendons measurements are performed. In fact, increased tendon hypertrophy after strength training has

been reported to be heterogeneous along the tendon length [230]. The results from our study is not in accordance with a similar study in male cyclists where no change in patellar tendon CSA occurred despite increases in a group of untrained male individuals performing the same strength training program [216]. The reason for this may be the large volume of endurance training performed (over 10 h per week) by the cyclist in the latter study [216].

Without changes in mechanical properties, the tendon hypertrophy measured here suggests that material properties may have also been altered. However, the unchanged Young's modulus does not support this. This may highlight the limitation of Young's modulus, which is based on finite tendon sections, to reflect whole tendon material properties.

3.6 40 min all-out performance

The mean power output during the 40 min all-out cycling test increased by $6.4 \pm 7.9\%$ in *E+S* ($p < 0.05$) with no change in *E* (figure 12). The difference in change between the groups was not significant, but the practical effect of *E+S* compared to *E* was moderate ($ES = 0.63$).

Running distance during the 40 min all-out test tended to increase in both groups during the intervention (*E+S*: $2.0 \pm 3.0\%$, $p = 0.06$, *E*: $2.1 \pm 2.8\%$, $p = 0.07$, figure 13), with no difference in change between the groups.

The improved cycling performance is in good agreement with findings of 6-8% improvement in 40 to 45 min all-out performance in well-trained to elite male cyclists after similar strength training interventions [2, 146, 213, 218]. Improvements have also been reported during time to exhaustion tests at 80 to 100% of VO_{2max} in trained male cyclists [113, 241]. However, there are studies reporting no effect on cycling performance after addition of strength training [26, 38, 40, 133, 161, 206]. In fact, in the only previous study involving female cyclists, 12 weeks of heavy strength training had no effect on power output during a 60 min cycling test [40]. A thorough scrutiny of all these studies reveals some possible explanation for the different findings. To positively affect performance it seems that the strength training should use high muscle loading intensity (4-10RM loads), a quite large volume of training and last for 8 weeks or longer [2, 146, 213, 218, 241]. The strength training performed in the studies showing no effect on cycling performance lacked one or more of these features [26, 40, 161, 206].

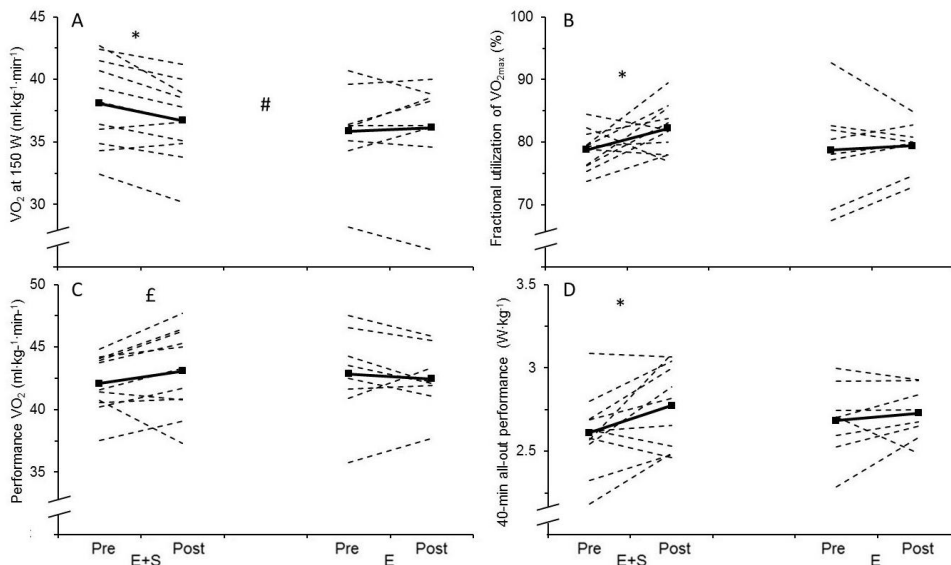


Figure 12. Individual values (dotted lines) and mean values (solid lines) before (Pre) and after (Post) the intervention period for athletes adding strength training to their normal endurance training (E+S), and athletes performing normal endurance training only (E). A: Oxygen consumption (VO_2) at 150 W during the blood lactate profile test in cycling. B: Fractional utilization of the maximal oxygen consumption (VO_{2max}) during the 40 min all-out test in cycling. C: Performance VO_2 during the 40 min all-out test in cycling. D: Body mass adjusted average power output during the 40 min all-out test in cycling. * Different from Pre ($p < 0.05$), £ tendency to different from Pre ($p < 0.10$), # the percent change from Pre is different between E+S and E ($p < 0.05$).

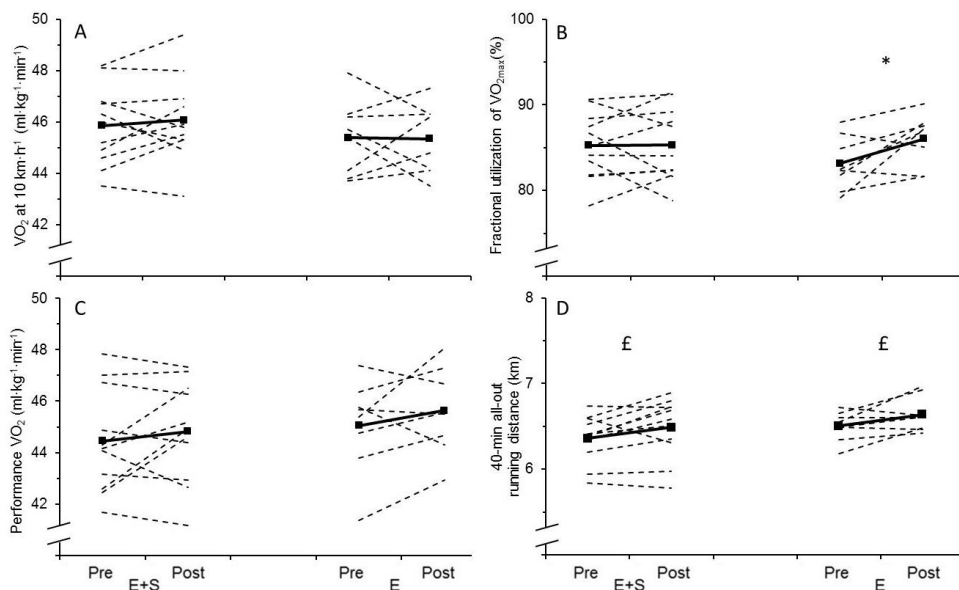


Figure 13. Individual values (dotted lines) and mean values (solid lines) before (Pre) and after (Post) the intervention period for athletes adding strength training to their normal endurance training (E+S), and athletes performing normal endurance training only (E). A: Oxygen consumption (VO_2) at 10 km·h⁻¹ during the blood lactate profile test in running. B: Fractional utilization of the maximal oxygen consumption (VO_{2max}) during the 40 min all-out test in running. C: Performance VO_2 during the 40 min all-out test in running. D: Running distance during the 40 min all-out test. * Different from Pre ($p < 0.05$), £ tendency to different from Pre ($p < 0.10$).

The lack of change in 40 min all-out performance in running is not in line with many previous studies in this area where improved running performance have been reported [21, 65, 113, 142, 198, 228, 235, 240, 243]. However, not all studies have found strength training to be beneficial for running performance [33, 77, 144, 186, 227]. Going through previous studies there seems to be some common features in the studies that reports improved running performance. To improve running performance, it seems that both explosive [198, 243], plyometric [21, 235] and heavy load strength training [21, 65, 228, 240] are effective, but the strength training should be performed at least two times per week. There are however some exceptions to this general trend [113, 144]. Since cycling performance seems to be improved only after strength training including heavy load, the strength training interventions that lead to improved performance somewhat differ between cycling and running. This observation also indicate that the mechanism behind possible improved performance after strength training may be somewhat different between cycling and running. Perhaps improved cycling performance is related to typical adaptations after prolonged periods of heavy strength

training like increased muscle mass and muscle fiber type transition from type IIX to type IIA. Improved running performance on the other hand might be more related to adaptations seen after both heavy strength training and explosive strength training, like changes in leg stiffness, rate of force development or other neuromuscular characteristics. The fact that the strength training intervention in the current only improved 40 min all-out performance in cycling further support the notion that the performance enhancing mechanisms after strength training may be different between cycling and running.

Therefore, the improved 40 min all-out cycling performance and the lack of effect on 40 min all-out running performance after strength training in the current study may be because the strength-training program led to changes important for improving cycling performance but not to adaptations important for improving running performance. In this regard it should be mentioned that the strength-training program utilized in the current study was originally developed for improving cycling performance [213] and has been proven good for this purpose [213, 215, 217, 218]. However, it has not been validated for running performance. The different effects on running and cycling performance in the current study may also be due to other methodological reasons related to the testing and training. The possible mechanisms behind the improved cycling performance and the lack of improved running performance will be discussed next.

As described in the introduction endurance performance in a 40 min all-out test is mainly determined by the performance $\dot{V}O_2$ and the work economy [24, 60]. Therefore, the improved cycling performance observed in the current study should be accompanied by an improvement in one or both of these factors. Likewise, the lack of effects on 40 min all-out running performance observed in the current study should also be accompanied by a lack of changes in these factors.

3.6.1 Performance $\dot{V}O_2$

In cycling, the performance $\dot{V}O_2$ during the 40 min all-out test tended to increase by $2.3 \pm 4.2\%$ ($p = 0.08$, figure 12) in $E+S$ with no change in E . In running, there was no change in performance $\dot{V}O_2$ in neither $E+S$ nor E (figure 13).

The tendency to increased performance $\dot{V}O_2$ in cycling in $E+S$ probably contributed to the improved 40 min all-out cycling performance as the change in cycling performance $\dot{V}O_2$ showed a large correlation with the change in mean power output during the 40 min all-out test ($r = 0.67$, $p < 0.01$). Performance $\dot{V}O_2$ depends on $\dot{V}O_{2max}$ and fractional utilization of

$\text{VO}_{2\text{max}}$ [24, 60, 139]. As $\text{VO}_{2\text{max}}$ did not increase (discussed below) the tendency to improved cycling performance VO_2 in $E+S$ was likely due to improved fractional utilization of $\text{VO}_{2\text{max}}$. In fact, a slight non-significant decrease in $\text{VO}_{2\text{max}}$ in cycling is probably the reason why increased cycling performance VO_2 in $E+S$ missed being significant.

3.6.1.1 $\text{VO}_{2\text{max}}$

Both cycling and running $\text{VO}_{2\text{max}}$ were unchanged in both groups during the intervention period (table 6).

The lack of effect of heavy strength training on $\text{VO}_{2\text{max}}$ in endurance athletes is not surprising and in accordance with the current literature [e.g. 65, 100, 198, 213, 218, 228, 240, 241]. The athletes in the current study were instructed to continue their normal endurance training, and with a good base of training from winter cycling, winter running and cross-country skiing, no changes in $\text{VO}_{2\text{max}}$ were expected.

3.6.1.2 Fractional utilization of $\text{VO}_{2\text{max}}$

The fractional utilization of $\text{VO}_{2\text{max}}$ during the 40 min all-out test in cycling increased in $E+S$ from $78.9 \pm 3.2\%$ to $82.2 \pm 3.9\%$ ($p < 0.05$, figure 12) whereas it remained unchanged in E . In running, there was no change in fractional utilization of $\text{VO}_{2\text{max}}$ in $E+S$, but fractional utilization of $\text{VO}_{2\text{max}}$ increased from 83.2 % to 86.0 % in E ($p < 0.05$, figure 13).

To the best of my knowledge, this is the first study investigating the effect of adding strength training to endurance athletes' normal training on fractional utilization of $\text{VO}_{2\text{max}}$. However, the VO_2 at the lactate threshold in percent of $\text{VO}_{2\text{max}}$ is reported to be closely linked to the fractional utilization of $\text{VO}_{2\text{max}}$ [24, 25, 60, 201] and is therefore often used as an indirect measure of fractional utilization of $\text{VO}_{2\text{max}}$ [24, 60, 139]. The few studies measuring changes in this variable after concurrent training in endurance athletes reports no change in neither cycling [218, 241] nor running [188, 240]. These results are therefore in accordance to our results in running, but not in cycling. One possible reason for the different result in previous studies in cyclists compared to the current study may be that the VO_2 at the lactate threshold in percent of $\text{VO}_{2\text{max}}$ is not a sensitive enough measure of fractional utilization of $\text{VO}_{2\text{max}}$. Dependent on how it is measured and defined, the lactate threshold usually ranges between 55-85 % of $\text{VO}_{2\text{max}}$ [76]. The fractional utilization of $\text{VO}_{2\text{max}}$ on the other hand will vary to a large degree depending on the duration of the competition [239, 267]. In fact, when estimating changes in VO_2 at the power output corresponding to $3.5 \text{ mmol}\cdot\text{L}^{-1}$ [la^-] in percent of $\text{VO}_{2\text{max}}$ in the current study both groups shows a similar increase of about 3 percentage points (data

not shown). Therefore, it might be possible that changes in fractional utilization of $\text{VO}_{2\text{max}}$ after a strength-training period cannot be detected when estimated as lactate threshold VO_2 in percent of $\text{VO}_{2\text{max}}$. In fact, data from a previous study [2] indicates that improvements in fractional utilization of $\text{VO}_{2\text{max}}$ might have occurred after heavy strength training. In that study, elite cyclists adding heavy strength training to their normal endurance training increased mean power output during a 45 min all-out test, while no changes. Since there were no changes in cycling economy or $\text{VO}_{2\text{max}}$, this indicate that fractional utilization of $\text{VO}_{2\text{max}}$ was improved and thus inducing improvement in the performance VO_2 . In fact, the authors estimated that the power output during the test had increased from 76% to 83% of the power output at $\text{VO}_{2\text{max}}$. Since cycling economy and $\text{VO}_{2\text{max}}$ did not change, the oxygen demand and hence fractional utilization of $\text{VO}_{2\text{max}}$ should also have increased.

The mechanisms behind increased fractional utilization of $\text{VO}_{2\text{max}}$ in cycling, and therefore the tendency to improved performance VO_2 in the present study are unclear, but it may be related to the increased CSA of the quadriceps muscles. In fact, there was a large correlation between change in performance VO_2 and the change in CSA of the quadriceps muscles ($r = 0.59$). The fractional utilization of $\text{VO}_{2\text{max}}$ is mainly determined by the amount of aerobic enzymes and mitochondria sharing a certain workload and VO_2 [60, 118, 129, 139]. It has been reported that cyclists that are able to spread the power output over a larger amount of their muscle mass has larger fractional utilization of $\text{VO}_{2\text{max}}$ during a 60 min all-out test [60, 62]. The suggested explanation is that a larger amount of active muscle mass is accompanied with a larger amount of activated mitochondria. The increased muscle CSA in the current study probably led to more muscle mass available to share a certain power output, and because the concentration of aerobic enzymes in *m. vastus lateralis* was unchanged, the total amount aerobic enzymes (mitochondria) available for sharing the workload should be increased.

Any mechanism that will increase power output (performance) and performance VO_2 without improved $\text{VO}_{2\text{max}}$ will improve fractional utilization of $\text{VO}_{2\text{max}}$. For example, a frequently proposed mechanism for improved endurance performance after heavy strength training is an increase in rate of force development (RFD) [1, 4, 116, 117, 197, 241]. Increased RFD may reduce the time required to reach the force needed to sustain a certain power output and may thus allow for a prolonged relaxation phase in each pedal stroke, and consequently facilitate better blood flow to exercising muscles [1, 219]. If increased RFD improves performance, thereby allowing cycling at a higher mean power output, this will increase oxygen demand

and therefore also performance VO_2 . As long as $\text{VO}_{2\text{max}}$ remains unchanged, this will lead to an increase in fractional utilization of $\text{VO}_{2\text{max}}$. Unfortunately, pedaling characteristic and RFD were not measured in the current study, but it is possible that some of the improvements in fractional utilization of $\text{VO}_{2\text{max}}$ were due to an increased RFD and earlier peak force during the pedal stroke. In fact, a recent study demonstrated that elite cyclists exhibited earlier peak torque during pedal strokes following a strength-training program similar to the one used in the current study. Indeed, this trait was closely related to a concomitant improvement in mean power output during a 40 min all-out test [218].

Power output or velocity at the lactate threshold is often included in models describing the factors important for endurance performance instead of the fractional utilization of $\text{VO}_{2\text{max}}$ [201]. This is unfortunate because the power output at the lactate threshold will be affected by the VO_2 at the lactate threshold (again decided $\text{VO}_{2\text{max}}$ and % $\text{VO}_{2\text{max}}$ at the lactate threshold) by and work economy [25]. Therefore, the power output at the lactate threshold is decided by the same factors as performance itself. In the current study, power output at $3.5 \text{ mmol}\cdot\text{L}^{-1} [\text{la}^-]$ tended to increase by $7.6 \pm 12.0\%$ ($p = 0.06$) in $E+S$ with no change in E (table 6). There were no changes in the velocity at $3.5 \text{ mmol}\cdot\text{L}^{-1} [\text{la}^-]$ any group in running. In previous studies there are reports of both improved power output/velocity at a certain blood $[\text{la}^-]$ after strength training in endurance athletes [146, 213, 218], but there are also reports of no change [2, 40, 133, 241].

In running, adding heavy strength training to normal endurance training had no positive effect on fractional utilization of $\text{VO}_{2\text{max}}$ (figure 13). To my best knowledge, this is the first study directly measuring fractional utilization of $\text{VO}_{2\text{max}}$ in running after addition of heavy strength training in endurance athletes. As mentioned above, the few studies reporting changes in VO_2 at the lactate threshold in percent of $\text{VO}_{2\text{max}}$ in running, reports no effect after addition of heavy strength training [188, 240].

Surprisingly, there was a slight increase in fractional utilization of $\text{VO}_{2\text{max}}$ in E over the course of the intervention. However, this was likely due to a combination of two factors; a small but non-significant reduction in $\text{VO}_{2\text{max}}$, largely due to one athlete exhibiting a large reduction, and a small but non-significant increase in performance VO_2 .

Table 6. Data from the maximal oxygen consumption (VO_{2max}) test in both cycling and running, power output/velocity at 3.5 mmol·L⁻¹ blood [La⁻], and data from Wingate tests before (Pre) and after (Post) the intervention period for athletes adding strength training to their normal endurance training (E+S), and athletes performing normal endurance training only (E).

	E+S		E		Cohen's D
	Pre	Post	Pre	Post	
VO_{2max} cycling (ml·kg ⁻¹ ·min ⁻¹)	53.5 ± 3.6	52.5 ± 4.2	54.6 ± 3.4	53.5 ± 1.8	-
W_{max} (W·kg ⁻¹)	4.0 ± 0.3	4.2 ± 0.3	4.0 ± 0.4	4.2 ± 0.2	0.21
HR_{peak} cycling (beats·min ⁻¹)	188 ± 9	186 ± 8	182 ± 8	182 ± 6	-
RPE cycling (Borg scale)	19.5 ± 0.5	19.8 ± 0.4	19.4 ± 0.7	19.3 ± 0.6	-
[La ⁻] _{peak} cycling (mmol·l ⁻¹)	10.8 ± 3.2	10.4 ± 2.9	9.8 ± 2.4	9.7 ± 2.3	-
Power output at 3.5 mmol·L ⁻¹ blood [La ⁻] (W·kg ⁻¹)	2.5 ± 0.3	2.7 ± 0.3£	2.7 ± 0.2	2.8 ± 0.2	-
VO_{2max} running (ml·kg ⁻¹ ·min ⁻¹)	52.2 ± 2.3	52.7 ± 3.3	54.2 ± 2.9	53.1 ± 1.9	-
V_{max} (km·h ⁻¹)	12.8 ± 0.7	13.0 ± 0.9*	13.1 ± 0.5	13.3 ± 0.6	0.22
HR_{peak} running (beats·min ⁻¹)	193 ± 9	192 ± 9	189 ± 8	187 ± 7	-
RPE running (Borg scale)	19 ± 1	20 ± 1	19 ± 1	19 ± 1	-
[La ⁻] _{peak} running (mmol·l ⁻¹)	9.7 ± 3.0	8.1 ± 3.8	8.9 ± 2.2	7.7 ± 1.8	-
Running velocity at 3.5 mmol·L ⁻¹ blood [La ⁻] (km·h ⁻¹)	9.6 ± 0.7	9.7 ± 0.6	9.8 ± 0.5	10.0 ± 0.5	-
Peak power Wingate (W·kg ⁻¹)	17.0 ± 2.0	19.1 ± 2.5*	17.7 ± 1.4	18.7 ± 1.7	0.49
Mean power Wingate (W·kg ⁻¹)	8.1 ± 0.7	8.4 ± 0.6*	8.1 ± 0.5	8.1 ± 0.6	0.83

Values are mean ± SD. *Larger than Pre ($p < 0.05$), £ tendency to different from Pre ($p < 0.10$). VO_{2max} , maximal oxygen consumption; W_{max} , mean power output during the last 2 min of the VO_{2max} test, HR_{peak} : peak heart rate; [La⁻]_{peak}, peak blood lactate concentration; RPE, rate of perceived exertion; V_{max} , mean running velocity during the last 2 min of the VO_{2max} test.

The reasons for the different effects of the heavy strength training program used in the current study on fractional utilization of VO_{2max} in cycling and running is unclear and difficult to explain, but some speculations are possible. During running at flat or at slightly elevated inclinations, as used in the current study, the plantar flexor muscle have been reported to be more active than the knee extensors [35, 58, 88], while the monoarticular muscles in the thigh are probably more important muscles during cycling [34, 35, 74, 125].

As discussed, the probable mechanisms behind improved fractional utilization of $\text{VO}_{2\text{max}}$ in cycling after the strength training intervention were related to changes located in the quadriceps musculature (increased CSA with unchanged concentration of aerobic enzymes). Since measurements were not done on the plantar flexors, it is unknown whether similar changes occurred there. Interestingly, both the *m. soleus* and *m. gastrocnemius lateralis* have been reported to have higher SDH activity than *m. vastus lateralis* in recreationally active individuals [99]. Based on this it may be speculated that the changes in aerobic enzymes in the plantar flexor muscles after a strength training intervention may differ from that observed in *m. vastus lateralis*. In addition, it has been reported that *m. soleus* have a larger percentage of type I muscle fibers [99]. This, together with the fact that the plantar flexors were subjected to lower strength training volume than the knee extensors in the current study, might have led to a smaller increase in muscle CSA in the plantar flexors than what was observed in the knee extensors.

3.6.2 Work economy

During the blood lactate profile test in cycling, *E+S* reduced the VO_2 at a power output of 150 W with $3.5 \pm 3.1\%$ ($p < 0.01$) whereas there was no change in *E* (figure 12). The reduction in VO_2 was larger in *E+S* than in *E* ($p < 0.05$), and the ES analysis revealed a moderate practical effect ($\text{ES} = 1.0$). No changes in cycling economy were observed at lower power outputs. There was no change in VO_2 measured at $10 \text{ km}\cdot\text{h}^{-1}$ during the blood lactate profile test in running in *E+S* or *E* (figure 13).

As cycling economy is an important determinant of cycling performance, the improved cycling economy in *E+S* probably explains some of the improved power output during the 40 min all-out test. This is supported by a moderate relationship between change in VO_2 at 150 W and change in mean power output during the 40 min all-out test ($r = -0.47$). Improved cycling economy are in accordance with studies in both untrained individuals [167], master athletes [166] and moderately to well-trained cyclists [22, 241]. However, studies involving highly trained cyclists do not report any improved cycling economy after addition of strength training [2, 26, 213, 218]. This indicates that addition of strength training may not be enough to positively affect cycling economy in elite cyclists. Perhaps the cycling economy in these cyclists is already highly optimized and therefore is difficult to improve further, especially in the quite short time period typically used in strength training interventions studies. The athletes in the current study were well-trained recreational active athletes, so the improved cycling economy is in accordance with previous results. However, this is not in accordance

with the only previous study involving female cyclists where no changes in cycling economy were reported after heavy strength training [40]. The most probable reason for this discrepancy is that the latter study only included one strength-exercise twice a week making the training volume quite low compared to studies reporting improved cycling economy.

The precise mechanism for improved cycling economy after heavy strength training is not completely understood. Barret-O'Keefe et al. [22] found a reduced pulmonary VO_2 together with a corresponding reduced leg VO_2 after 8 weeks heavy strength training. This indicates that the mechanism may lie inside the trained muscles. Increased quadriceps muscle CSA and increased muscle strength probably contributed to improved cycling economy as indicated by a large correlation ($r = -0.54$) between change in CSA of the quadriceps muscle and improved cycling economy. Similar correlations have also been reported between cycling efficiency and MVC in knee extensors [166, 241]. When the maximal muscle strength increases, the force levels required to ride at a certain power output is reduced relatively to maximal force. This implies that the economical type I muscle fibers can account for a larger proportion of a certain absolute power output [113, 219], as follows from the size principle of motor unit recruitment [111]. Type I fibers have been reported to be more efficient than type II fibers [20, 233], and although not an universal finding [120, 202], cycling efficiency has been related to proportions of type I fibers in the active muscles [23, 64, 104, 122, 175, 176, 191, 205]. In addition, increasing type II fiber recruitment by glycogen depletion [150], neuromuscular blockage [149] and prior fatigue [263] of type I muscle fibers elevates VO_2 during submaximal exercise. Therefore, greater reliance on type I fibers could improve cycling economy. This is further supported by the observation that the improved cycling economy in the current study was only observed at the highest common power output during the blood lactate profile test. Again, because of the size principle, type II fibers should only contribute to the power output at high power outputs, while at low power outputs type I muscle fibers would dominate. Therefore, at lower power outputs the possibility for improving work economy from an increased use of type I fibers would be limited. In fact, during 60 min cycling at 43% of $\text{VO}_{2\text{max}}$ in active individuals, glycogen depletion was observed in 89% of type I fibers and only 17% of type IIA fibers indicating limited recruitment of the type II muscle fibers at this intensity [251]. However, after a similar trial at 61% of $\text{VO}_{2\text{max}}$ glycogen depletion was observed in 98% of type I fibers and 68% of type IIA fibers [251]. In the current study, the VO_2 at 150 W corresponded to $71 \pm 5\%$ of and $70 \pm 5\%$

VO_{2max} at pre and post respectively for *E+S*, while the corresponding numbers for *E* were 66 ± 7 and $68 \pm 8\%$.

The increase in the proportion of type IIA muscle fibers at the expense of type IIX muscle fibers might also have been a contributor to improved cycling economy [1, 100, 219]. Since some studies have reported that type IIA fibers is more economical than type IIX fibers *in vitro* [20, 46, 233, 256], this fiber type shift could in theory improve work economy. Our data might indicate that this fiber type conversions contributed to improved cycling economy, as the *E+S* group displayed an increased proportion of type IIA fibers with a concomitant reduction in the proportion of type IIX fibers. However, there was no correlation between the reduced proportion of fiber type IIX and changes in cycling economy. This mechanism should only be evident at power output high enough to recruit type IIX fibers and 150 W (about 70% of VO_{2max} se above) is probably a borderline intensity regarding recruitment of type IIX fibers. In fact, 20 min of cycling at 75% of VO_{2max} has been reported to lead to no significant glycogen depletion in both type IIX and type IIA fibers in moderately trained individuals [252]. In addition, fiber type transitions between type IIA and type IIX are not always accompanied with changes in work economy. Despite a large shift from type IIA to IIX fibers there were no changes in work economy (tested at 74% of VO_{2max}) in well-trained endurance athletes after cessation of training for 84 days [63]. In fact, the *in vitro* differences in efficiency between type IIX and type IIA muscle fibers seem to be quite small at physiological temperatures [164]. Based on these considerations the fiber type shift from type IIX to IIA might not have been important for the observed improvements measured in cycling economy at 150 W. However, during the 40 min all-out test the recruitment of type IIX fibers were probably substantial, especially during the last part, and this fiber type transition might therefore have improved cycling economy during the 40 min all-out test. Although this is highly speculative, it is supported by a large correlation ($r = -0.63$) between change in type IIX fiber proportion and change in power output during the 40 min all-out test.

Many previous studies report improved running economy ranging from 3-8% after the addition of both explosive/plyometric [198, 225, 235, 242, 249] and heavy strength training [8, 21, 86, 100, 135, 188, 228, 240]. However, some studies are in agreement with the current study reporting no improvements in running economy after strength training [33, 65, 77, 186, 220]. In two of this studies [77, 186], the lack of improved running economy might be

explained by the fact the strength training program only consisted of one training session for the legs per week making the strength training volume quite low.

Improved running economy seems to be the main mechanism behind the improved running performance reported in other studies [21, 198, 228, 240] since the studies that do not report improved performance also report no changes in running economy [77, 144, 186]. The lack of change in 40 min all-out running performance in the current study follows this line with no change in running economy.

The reason for the different findings in the current study compared to most previous studies regarding running economy and hence performance is unclear. Importantly, in the current study all running tests were performed at 5.3% inclination. This inclination resulted in a quite low running velocity compared to some other studies, which may explain the different findings. Indeed, changes in running economy after strength training have previously been found to be related to running velocity [225, 249]. However, improvements in running economy after strength training have also been reported at similar velocities [86, 242, 249] and at the same inclination [116] used in the current study. Therefore, the inclination used is probably not the explanation why no changes in running economy and 40 min all-out performance were observed.

To my best knowledge, this is the first study investigating the effects of a strength-training program on work economy in both cycling and running in the same athletes. The reason for the different effects on cycling economy and running economy is unclear. Logically, it might be because the mechanisms suggested to improve cycling economy in the current study may not be as important for running economy, and that the current strength training program may not have induced adaptations important for improving running economy.

Even though there is an association between the proportion of type I muscle fibers and cycling economy, this association is not as clear regarding running economy [45, 127, 157, 260]. The probable reason for this weak link between fiber type composition and running economy is because of the more complex nature of factors affecting running economy [60]. The suggested mechanisms behind improved cycling economy were related to changes measured in the quadriceps musculature (increased muscle CSA and perhaps fiber type transition). Since these changes not necessarily occurred in the plantar flexors (see discussion above) this may explain the lack of change in running economy after the addition of heavy strength training. On the other hand, it may also be speculated that increased CSA of the plantar

flexors did occur this would not positively affected running economy. A large mass in the lower legs has shown to increase the cost of running [158, 168], and increased muscle CSA in the lower legs may therefore have both a positive and a negative effect on running economy. If increased CSA and muscle strength of the lower legs muscles occurred and led to changes in muscle fiber type usage positive for running economy this may have been offset by an increased weight of the lower legs.

In running, the stretch shortening cycle in each stride enables the possibility to store and recoil elastic energy, whereas cycling mainly consist of concentric muscle work [35, 74]. Consequently, the possibility to take advantage of stored elastic energy is negligible in cycling. The stiffness of the series elastic component, mainly tendons, can affect both the utilization of this elastic energy and the muscle contraction mechanics during the running stride [14]. In fact, the stiffness of the Achilles and patellar tendon have been associated with running economy [14] and running performance [154, 155]. However, these studies no not agree if a stiffer or more compliant patellar tendon is beneficial for performance [154, 155]. As described above, strength training have the potential to affect the stiffness and energy storage capacities in tendons. In fact, one of the most frequent proposed mechanisms behind improved running economy after strength training reported in many studies are changes in the stiffness of lower limbs muscles and tendons [8, 21, 100, 225, 240, 249]. Perhaps the lack of changes in patellar tendons stiffness in the current study explain why no changes in running economy occurred.

The observation that the strength-training program in the current study was more successful in improving cycling performance than running performance might, as speculated above, be related to the fact that the strength-training program had a higher training volume for the thigh muscle than the calf muscles. However, it should be noted that improved running performance has been reported after inclusion of only one strength training exercise for the plantar flexors [65], and after a strength training program only including heavy squat as an exercise [240]. Even though an optimal strength-training program for running performance maybe should have included more exercises for the calf muscles and perhaps some explosive/plyometric exercises, the program should, based on previous studies, have been sufficient to induce improvements in running performance.

3.7 Anaerobic capacity and other determinants of endurance performance

As described in section 1.1.3 the actual results in running and cycling competitions will in addition to the three main determinants of endurance performance also be affected by other factors like anaerobic performance and the ability to generate high power output for a short period of time [15]. Performances that reflect this ability is V_{\max}/W_{\max} , and peak and mean power during the Wingate test. In the current study, W_{\max} was unchanged in both groups during the intervention period. V_{\max} increased in $E+S$ by $1.7 \pm 2.8\%$ ($p = 0.05$, table 5) with no change in E . However, no difference was found between the two groups and the practical effect of $E+S$ was small ($ES = 0.22$).

Peak power in the Wingate test increased in $E+S$ by $12.7 \pm 12.6\%$ ($p < 0.01$) with no change in E (table 6). There was however no significant difference in the change between groups, and the effect size analysis revealed a small practical effect of $E+S$ compared to E ($ES = 0.49$). $E+S$ also increased mean power output during the Wingate test by $3.4 \pm 4.3\%$ ($p < 0.05$), whereas there was no change in E (table 6). There was a tendency to a larger increase in $E+S$ ($p = 0.07$), with a moderate practical effect of $E+S$ compared to E ($ES = 0.83$).

Increased V_{\max} after heavy strength training in trained runners are in agreement with existing literature [185, 228, 242], though not conclusively [100]. $VO_{2\max}$ is an important factor determining V_{\max} , but V_{\max} is also determined by anaerobic capacity [114, 136]. Since there was no change in $VO_{2\max}$ the main reason for improved V_{\max} is probably improved anaerobic capacity. Muscle mass is an important factor determining anaerobic capacity [19], and as indicated by the increased quadriceps muscle CSA and increase muscle fiber CSA in *m. vastus lateralis* the strength training program clearly increased muscle mass in the legs.

The lack of change in W_{\max} in $E+S$ is surprising since the factors determining V_{\max} and W_{\max} is similar, and therefore the improved anaerobic capacity should also improved W_{\max} . These results are also in conflict with previous studies in well-trained to elite male cyclists where improved W_{\max} has been reported after performing a similar strength training intervention as in the current study [213, 218]. I have no good explanation for the lack of improved W_{\max} despite increased V_{\max} . Perhaps the test was not sensitive enough to detect small changes in W_{\max} . However, since the non-significant increase was even larger in E than in $E+S$ this is unlikely.

The finding of improved peak power output in the Wingate test after combining heavy strength training and endurance training was expected since similar findings have been reported in both untrained individuals [29, 54] and cyclists of various performance levels [206, 213, 218]. Because peak power often occur during the first 5 s of an all-out sprint, the size of the involved muscle mass and maximal leg strength is one of the main determinants of peak power output [132, 250]. The increase in peak power during the Wingate test in the *E+S* group may therefore be explained by the increase in muscle mass and leg strength. The improved mean power output during the Wingate test is in accordance with a study showing that 9 weeks of explosive strength training prevented a decrease in mean power output during a modified Wingate test in male cyclists [26], and in studies with untrained individuals after periods of heavy strength training [29, 54]. Most part of the energy used during the Wingate test comes from the glycolysis pathway [31], and since strength training have the potential to increase glycolytic enzyme activity and augment intracellular fuel stores of ATP and phosphocreatine [96] this is a likely mechanism for the increased mean power during the Wingate test.

3.8 Physiological responses during the prolonged tests

Figure 14 and table 7 shows the responses during the prolonged trials. After the intervention *E+S* had reduced VO_2 during the last two hours of the prolonged cycling compared to pre ($3.5 \pm 4.6\%$ and $3.3 \pm 4.4\%$ during the second and third hour respectively, $p < 0.05$). No changes occurred in *E*. The changes during the last two hours were different between the groups ($p < 0.05$). In addition, the effect size analysis revealed a large practical effect of *E+S* compared to *E* during the last hour of the test ($\text{ES} = 1.2$). There were no changes in VO_2 for neither *E+S* nor *E* during the prolonged running. Compared to pre, *E+S* had a lower HR throughout the prolonged cycling ($p < 0.05$) whereas *E* had a lower HR during the first hour only ($p < 0.05$). There was a moderate practical effect of *E+S* compared to *E* during the last hour of the trial ($\text{ES} = 1.12$). Both *E+S* and *E* had a lower HR during the entire prolonged running trial after the intervention period ($p < 0.05$). There was no difference in changes between the groups.

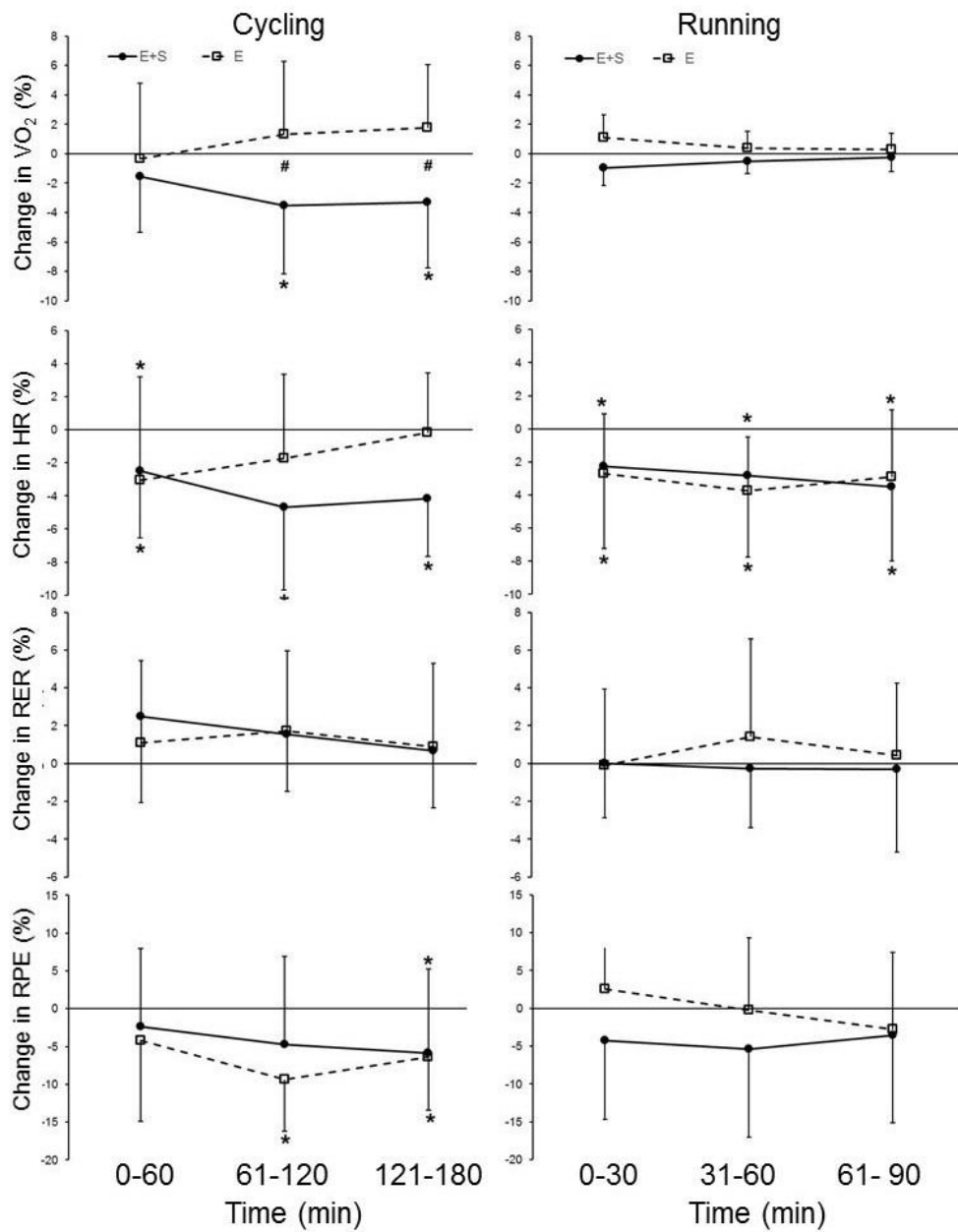


Figure 14. Percent change in responses during the prolonged trials in cycling (left panels) and running (right panels) for athletes adding strength training to their normal endurance training (E+S), and athletes performing normal endurance training only (E). Values are mean \pm SD. * Different from Pre ($p < 0.05$), # the percent change from Pre is different between E+S and E ($p < 0.05$).

Table 7. Responses during the prolonged trials in cycling and running for athletes adding strength training to their normal endurance training (E+S), and athletes performing normal endurance training only (E).

		E+S				E				
	Test section	First section	Middle section	Last section	First section	Middle section	Last section	First section	Middle section	Last section
VO ₂ (ml·kg ⁻¹ ·min ⁻¹)	Cycling	Pre	30.5 ± 2.9	31.3 ± 3.0	31.9 ± 2.9	30.1 ± 3.2	30.5 ± 3.4	31.0 ± 3.1		
		Post	30.0 ± 2.5	30.2 ± 2.9#	30.9 ± 3.2#	29.9 ± 2.4	30.8 ± 2.9	31.5 ± 3.0		
	Running	Pre	37.3 ± 1.8	37.7 ± 1.8	37.7 ± 1.8	37.0 ± 2.1	37.3 ± 2.0	37.3 ± 1.8		
		Post	37.0 ± 2.2	37.5 ± 2.0	37.6 ± 1.9	37.4 ± 2.0	37.4 ± 1.5	37.4 ± 1.4		
HR (beats·min ⁻¹)	Cycling	Pre	134 ± 12	138 ± 14	143 ± 14	129 ± 11	130 ± 9	135 ± 7		
		Post	131 ± 12*	131 ± 14*	137 ± 13*	125 ± 9*	128 ± 10	135 ± 9		
	Running	Pre	158 ± 12	163 ± 13	165 ± 13	152 ± 11	157 ± 11	158 ± 11		
		Post	154 ± 11*	158 ± 10*	159 ± 11*	148 ± 13*	151 ± 11*	153 ± 11*		
RER	Cycling	Pre	0.85 ± 0.03	0.84 ± 0.03	0.82 ± 0.03	0.87 ± 0.03	0.84 ± 0.03	0.81 ± 0.04		
		Post	0.87 ± 0.04	0.85 ± 0.03	0.82 ± 0.03	0.88 ± 0.03	0.85 ± 0.03	0.82 ± 0.03		
	Running	Pre	0.90 ± 0.02	0.89 ± 0.02	0.88 ± 0.02	0.90 ± 0.02	0.87 ± 0.03	0.86 ± 0.03		
		Post	0.91 ± 0.03	0.88 ± 0.03	0.86 ± 0.03	0.90 ± 0.02	0.88 ± 0.03	0.86 ± 0.03		
RPE (Borg scale)	Cycling	Pre	11 ± 1	12 ± 1	13 ± 1	11 ± 2	12 ± 2	13 ± 2		
		Post	11 ± 1	12 ± 1	12 ± 1*	10 ± 2	11 ± 1*	12 ± 1*		
	Running	Pre	12 ± 1	13 ± 1	13 ± 1	11 ± 2	12 ± 1	13 ± 1		
		Post	11 ± 1	12 ± 1	13 ± 1	11 ± 1	12 ± 1	13 ± 1		
Cadence (rev·min ⁻¹)	Cycling	Pre	84 ± 8	83 ± 10	83 ± 10	83 ± 10	81 ± 12	80 ± 13		
		Post	85 ± 9	83 ± 8	83 ± 9	81 ± 11	81 ± 12	80 ± 14		
	Running	Pre	-	-	-	-	-	-		
		Post	-	-	-	-	-	-		

Values are mean ± SD. * Different than Pre (p < 0.05), # the percent change from Pre is different between E+S and E (p < 0.05).

The reduced VO_2 in $E+S$ during the last two hours of the prolonged cycling after the strength training intervention is in accordance with previous observations in well-trained cyclists [217]. Therefore, even though no change in cycling economy at this low power output was observed during the first hour, cycling economy was clearly improved when the athletes started to get fatigued. This is highly important in cycling where many races include prolonged submaximal intensities for several hours.

As for VO_2 , the effect on HR was more pronounced during the last two hours. The reduced HR was probably because of the reduced VO_2 and hence reduced energy cost. In fact, the reduced HR mirrored the changes in VO_2 and a large correlation ($r = 0.59$) between change in VO_2 and change in HR during the last hour was observed.

The mechanisms behind improved cycling economy and reduced HR during the last two hours of the trial is somewhat unclear. Like the superior changes in cycling economy at the highest power outputs during the blood lactate test, the improved cycling economy during the last two hours may come from altered recruitment of type II fibers. A possible explanation might be that increased muscle strength and muscle mass delayed recruitment of type II muscle fibers thereby improving economy during the last part [217]. As it was seen for the lower power outputs during the blood lactate profile test, there were no improvements in cycling economy at this quite low power output during the first hour of the prolonged cycling. This was probably because of limited recruitment of type II fibers at this intensity already before the intervention. However, as the duration of the work increases and muscle fibers starts to get fatigued, additional motor units needs to be recruited to sustain the power output [95, 251]. The suggested mechanisms is therefore that the increased strength and muscle CSA allowed $E+S$ to use the more economical type I muscle fibers for a longer duration of the trial after the intervention, leading to improved cycling economy during the last part. In fact, reduced EMG activity in *m. vastus lateralis* has been reported during the last hour of a two hour long prolonged cycling trial following 5 weeks of strength training in well-trained triathletes. This may indicate delayed recruitments of type II fibers [106]. Further support for this mechanism is given by a study showing that exercise-induced glycogen depletion of type I fibers, leading to greater recruitment of type II fibers, elevates VO_2 during submaximal cycling [150].

Above it was speculated that the fiber type transition from type IIX-IIX to type IIA in $E+S$ might have contributed to the improved cycling economy at 150 W during the lactate profile

test, but that the workload perhaps was too low for this to be an important mechanism. There was also no correlation between the change in the proportions of type IIAX-IIIX and change in cycling economy during the last hour of the prolonged cycling. Again, this was probably because the relatively low power output did not recruit any type IIAX-IIIX fibers during the trial even before the intervention [251].

Other possible explanations for improved cycling economy during the last two hours of the prolonged cycling test could have been a change in substrate utilization towards larger carbohydrate utilization [191] or a reduction in cadence [83, 102]. However, there were no changes in RER or cadence during the prolonged cycling making these explanations unlikely. In addition, the lack of changes in the content of aerobic enzymes, including HADH and no change in mRNA levels of important genes coding for proteins involved in transportation and oxidation of fatty acids support no changes in substrate utilization.

In contrast to cycling, no changes occurred in VO_2 during the prolonged running. This is surprising since the proposed mechanisms for the reduced VO_2 during prolonged cycling in theory also could reduce VO_2 during the prolonged running. However, some methodological differences might explain the different finding between cycling and running. The prolonged running was only half as long as the prolonged cycling and was performed at a higher relative workload (60% vs 44% of V_{\max} and W_{\max} , respectively). Because the reduced VO_2 during the cycling trial was seen during the last two hours, it may be speculated that the prolonged running was too short. However, running races do seldom last as long as cycling races, and the shorter duration was therefore chosen for the prolonged running. To compensate for the shorter duration, the prolonged running was performed at a higher relative intensity than the prolonged cycling. This may have led to a quite high recruitment of type II motor units from the start, and the potential for reduced VO_2 due to postponed activation of type II fibers during the last part of the trial may therefore have been limited. In fact, in a glycogen breakdown study it was estimated that a large proportion of type IIA fibers were recruited already from the start at a power output corresponding to 61% of $\text{VO}_{2\max}$ [251].

3.9 Performance during the 5 min all-out tests

The mean power output during the 5 min all-out cycling test increased by $7.0 \pm 4.5\%$ in $E+S$ ($p < 0.05$) with no change in E (figure 15). The difference between the groups was not statistically significant, but the practical effect of $E+S$ compared to E was moderate ($ES = 0.62$). $E+S$ increased running distance in the 5 min all-out running test by $4.7 \pm 6.0\%$ ($p < 0.05$) with no change in E (figure 15). The increase in running distance was larger in $E+S$ than in E ($p = 0.05$), and the practical effect of $E+S$ compared to E was moderate ($ES = 0.95$).

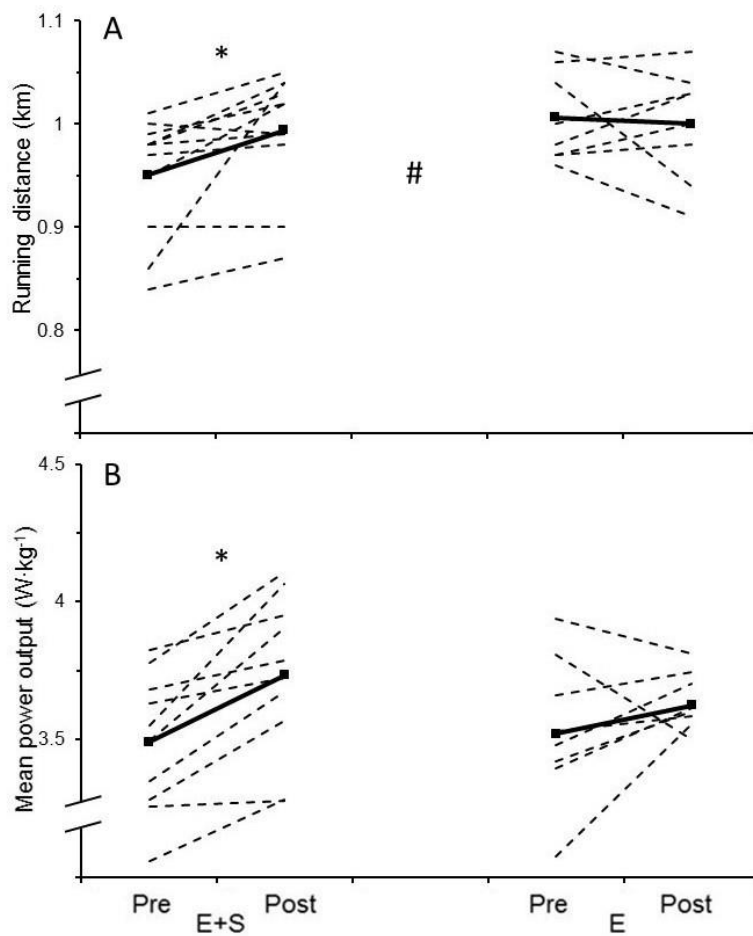


Figure 15. Individual values (dotted lines) and mean values (solid lines) before (Pre) and after (Post) the intervention period for athletes adding strength training to their normal endurance training ($E+S$), and athletes performing normal endurance training only (E). A: Running distance during the 5 min all-out running test. B: Mean power output during the 5 min all-out cycling test. * Different from Pre ($p < 0.05$), # the percent change from Pre is different between $E+S$ and E ($p = 0.05$).

The improved 5 min all-out cycling performance is in accordance with a similar study in male cyclists, which reported increased 5 min all-out performance following prolonged cycling after adding strength training to normal endurance training [217]. However, this is the first study reporting improved running performance after this kind of test setup.

Since the performance tests were performed right after the prolonged trials, changes in the physiological responses to the submaximal exercise were expected to affect performance. The reduced VO_2 and HR observed during the last two hours of the cycling trial might have reduced physiological strain and induced less fatigue making the athletes in *E+S* capable of producing higher mean power output during the final 5 min all-out test. Furthermore, reduced VO_2 in *E+S* means that the total energy consumption during the prolonged cycling trial was lower after the intervention and with no change in substrate utilization the total carbohydrate utilization was reduced. Therefore, some of the improved cycling performance in *E+S* may be due to a smaller reduction in glycogen stores during the prolonged trial. The importance of less physiological strain during the submaximal exercise is supported by the fact that 5 min all-out performance, tested in the rested state, was unchanged after 16 weeks of strength training in elite cyclists [2].

However, the superior change in performance in the 5 min all-out running test in *E+S* cannot be explained by changes in physiological responses during prolonged running. Therefore, the effect of strength training on 5 min all-out running performance has to be through other mechanisms. During a 5 min all-out trial a large part of the energy is produced from anaerobic metabolism [91]. As discussed above, the strength training intervention probably improved anaerobic capacity in *E+S*. Consequently, some of the improved 5 min-all out running performance in *E+S* was probably caused by improved anaerobic capacity. This is supported by a large correlation between changes in V_{\max} and changes in 5 min-all out performance ($r = 0.53$). In addition, running performance has been reported to correlate well with measurements of anaerobic performance [47, 123, 200]. Further support for the importance of anaerobic capacity as an mechanisms behind the improved 5 min all-out running performance is given by the fact that there was no effect of strength training on the 40 min all-out test where the anaerobic energy contribution to the performance VO_2 is small [91].

Anaerobic capacity will also affect performance in a 5 min all-out test in cycling, and increased anaerobic capacity probably also contributed to the improved cycling performance.

Support for this is given a large correlation between change in 5 min all-out cycling performance and change in W_{\max} ($r = 0.54$).

When data from both groups were included, there was a large correlation between change in 5 min all-out performance and change in IIX-IIX fiber proportion in both cycling ($r = -0.54$) and running ($r = -0.50$). However, when only $E+S$ was included in the analysis the correlation got very large in cycling ($r = -0.73$) but disappeared in running ($r = -0.28$). This indicates that the fiber type shift from type IIX-IIX toward type IIA contributed to the improved cycling performance to a larger degree than the improved running performance. The type IIA fibers is less fatigable than the type IIX fibers [256], and a fiber type transition should therefore improve performance. However, a correlation between two variables does not necessary mean a causal relationship [98]. Perhaps the athletes with a large reduction in fiber type IIX-IIX proportions had a large respond to the strength training and that other adaptations after the strength training actually were responsible for the improved performance. In fact, there was a large negative correlation ($r = -0.65$) between the change in Leg_{LM} and change in the proportion of type IIX-IIX fibers.

To sum up, adding 11 weeks of strength training improved 40 min-all out cycling performance in well-trained female duathletes. The main reason for this performance improvement was improved cycling economy and improved fractional utilization of $VO_{2\max}$ with unchanged $VO_{2\max}$. The main mechanisms behind improved cycling economy were probably increased muscle CSA and muscle strength leading to larger contribution of type I fibers. The main mechanisms behind improved fractional utilization of $VO_{2\max}$ were probably increased muscle CSA with unchanged concentration of mitochondrial enzymes making more mitochondria available for sharing a certain power output. Further support for the role of increased CSA behind these improvements is given by the fact that there was a very large correlation between change in mean power output during the 40 min all-out test and change in quadriceps muscle CSA ($r = 0.73$). In addition, in the previous studies measuring changes in both performance and including measurements of muscle growth there is a clear pattern that the studies that reports improved performance also reports muscle hypertrophy [2, 213, 218], while studies reporting no effect on performance report no muscle growth [26, 40, 206]. The strength-training program did not lead to changes in $VO_{2\max}$, work economy and fractional utilization of $VO_{2\max}$ and hence 40 min all-out performance in running.

The strength training program improved 5 min all-out performance tested after prolonged periods of submaximal work in both cycling and running. In cycling this was probably related to improved cycling economy during the last part of the submaximal cycling leading to less fatigue development before the 5 min all-out performance test. Improved anaerobic capacity probably also contributed. Improved anaerobic capacity seemed to be the main mechanism behind improved 5 min all-out running performance since no changes in the physiological responses to prolonged running occurred. The mechanisms behind improved cycling performance in the current study is summed up in figure 16.

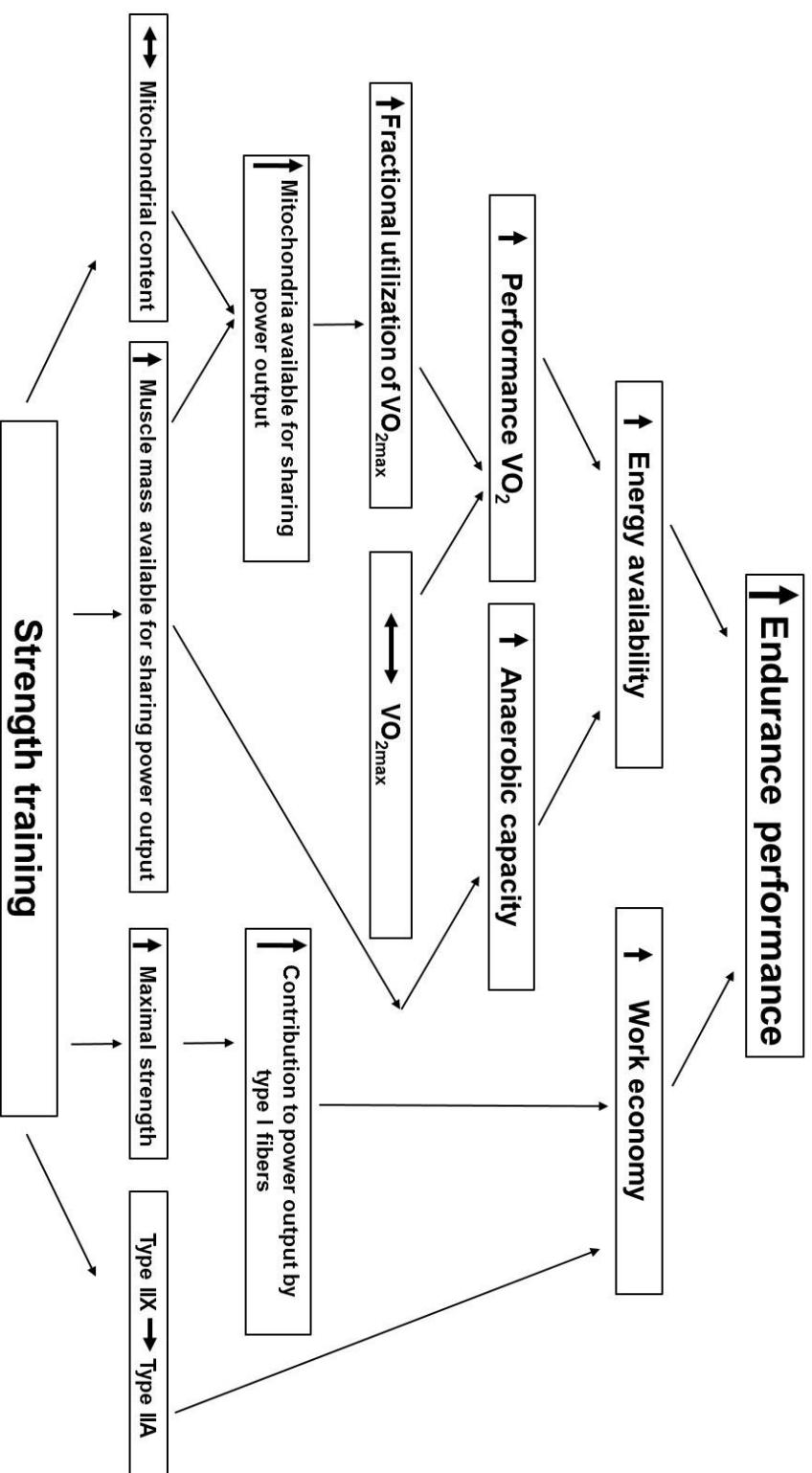


Figure 16. Proposed mechanisms behind how adding strength training improved 40 min all-out cycling performance and 5 min all-out performance in the current study. The long thin arrows between boxes represent proposed links between each factor while the thick arrows inside boxes represents proposed effects of adding strength training on each factor.

3.10 Effects of performing concurrent strength and endurance training on typical adaptations to strength training

3.10.1 Leg_{LM} and the ability to develop force at low contraction velocities

The percent increase in the load used at 6RM from week 2 to week 11 was similar in both groups ($E+S$: $38.9 \pm 11.0\%$, $p < 0.01$, S : $40.0 \pm 11.2\%$, $p < 0.01$, figure 17). Both $E+S$ and S increased 1RM in one-legged leg press ($E+S$: $38.6 \pm 19.0\%$, S : $42.2 \pm 17.4\%$, $p < 0.01$), MVC ($E+S$: $12.1 \pm 10.8\%$, S : $8.1 \pm 9.6\%$, $p < 0.05$) and leg_{LM} ($E+S$: $3.1 \pm 4.0\%$, S : $3.3 \pm 3.3\%$, $p < 0.05$, figure 18) to the same degree.

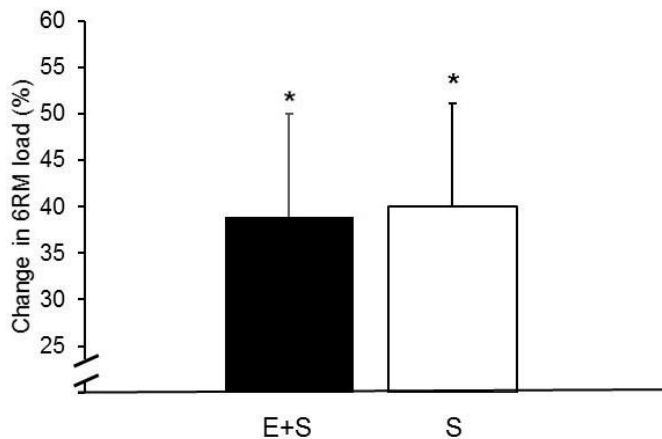


Figure 17. Percent change in 6 repetition maximum (6RM) load from training week 2 to training week 11 during the intervention period for athletes adding strength training to their normal endurance training ($E+S$), and previously untrained participants performing strength training only (S). * Different from Pre ($p < 0.01$). Values are mean \pm SD.

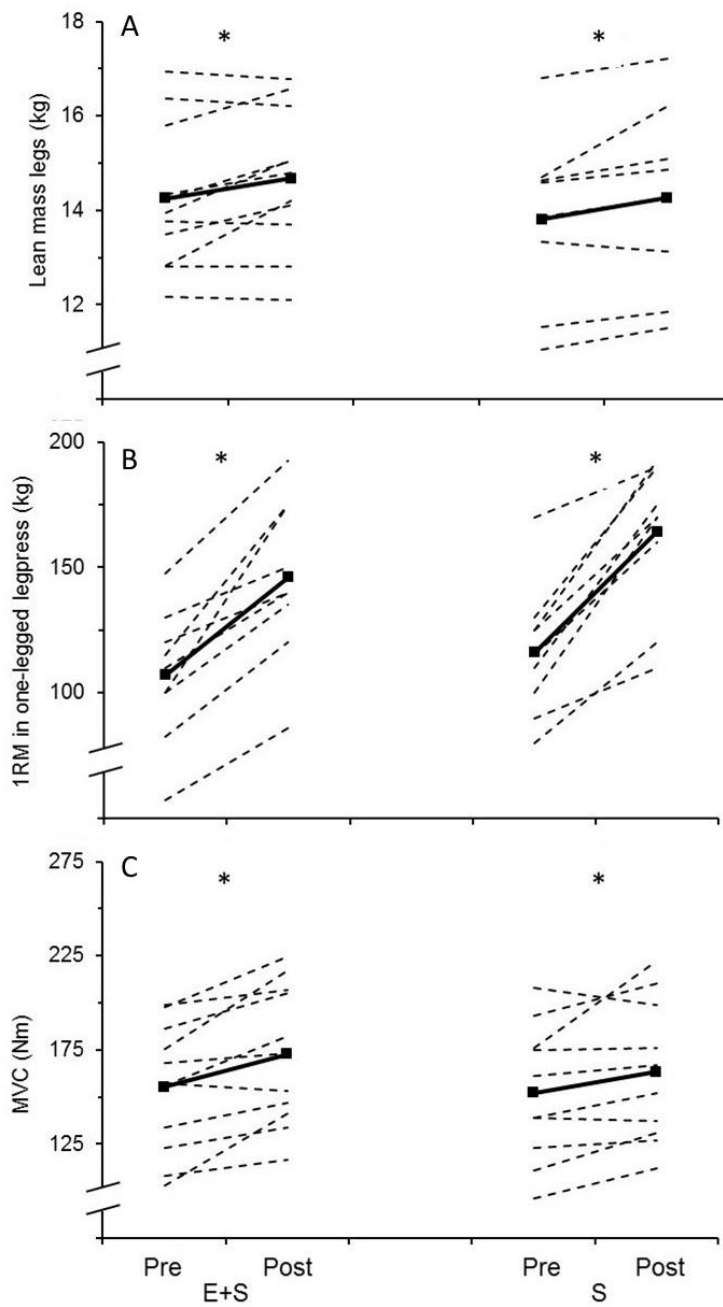


Figure 18. Individual values (dotted lines) and mean values (solid lines) before (Pre) and after (Post) the intervention period for athletes adding strength training to their normal endurance training (E+S), and previously untrained individuals performing strength training only (S). A: Lean mass in the legs. B: 1 repetition maximum (1RM) in one-legged legpress. C: Maximal isometric torque in knee extension (MVC). * Different from Pre ($p < 0.05$).

These findings indicate that the concurrent endurance training performed by *E+S* had no negative effect on the increase in the ability to develop force at low contraction velocities. This differs from many studies reporting that concurrent training interferes with increases in maximal strength after a strength training period [30, 49, 92, 112, 126, 131, 138]. However, there are also many studies reporting no negative effect of concurrent endurance training on changes in maximal strength at low contraction velocities [6, 51, 119, 181, 182, 195, 231]. The reason for these conflicting results is unclear but probably relates to numerous methodological differences between these studies since there are many variables that can be manipulated regarding both the strength and endurance training and how they are integrated.

The classical concurrent training studies normally includes a strength training group, an endurance training group and a concurrent training group. The concurrent training group typically performs the same amount of endurance training as the endurance training group and the same amount of strength training as the strength training group. Therefore, the total training volume will be considerable larger in the concurrent groups, and for untrained individuals this is often an unusual and quite large increase in total training volume. Among these concurrent training studies, it is difficult to find a common feature in the studies that reports attenuated changes in strength developments after concurrent training. However, although exceptions exists [68, 92, 96, 131], there is a trend that the studies reporting impaired gains in maximal strength include more total trainings sessions and therefore more endurance training sessions per week [30, 112, 126, 138, 148], than studies that do not report any attenuated adaptations [119, 181, 182, 231]. Therefore, the total amount of endurance training performed in the concurrent groups is probably an important factor deciding if any impaired strength training adaptations occurs. In fact, in a recent study it was reported that combining strength training with one endurance training session per week had no negative effects on strength gains in recreationally strength-trained men. However, increasing the numbers of endurance training session to three negatively affected the strength gains [138].

The design of the current study is different from most previous concurrent training studies in some important aspects. In this study, we investigated if well-trained endurance athletes who maintained a steady level of endurance training would have different adaptations to a strength training intervention than previously untrained individuals. Consequently, the actual increase in training volume were not different between the groups. In a previous study with similar design [214], well-trained male cyclists had reduced increases in 1RM after 12 weeks of strength training compared to a group of recreational active individuals only performing

strength training. The reasons for the different findings compared to the current study probably relates to the amount of endurance training performed. In the current study, the athletes in *E+S* performed about 5 h of endurance training per week, whereas in the study by Ronnestad et al. [214] the cyclists performed endurance training for about 10 h per week. In recreationally active runners, 1-3 h of weekly endurance training did not lead to any impaired strength adaptations compared to a group only performing strength training [126].

The lack of attenuated gains in maximal strength after concurrent training in the current study is probably explained by the lack of attenuated muscle hypertrophy. Many studies reports that endurance athletes who add heavy strength training to their normal training do not increase body mass and argues that this indicate lack of muscle hypertrophy [115, 116, 240, 241]. In addition, no changes in muscle fiber area have been reported in endurance athletes after periods of strength training [2, 40, 113]. However, muscle fiber area may vary considerable in the same muscle [163], and recent studies including hypertrophy measured at the whole muscle levels reports increased muscle CSA [165] or increased leg_{LM} [2, 218] in endurance athletes performing strength training. However, the reported increase in CSA of about 5% [165] is quite low compared to what is expected in normal active individuals after periods of heavy strength training [e.g. 79, 82, 255]. Unfortunately, these studies did not include a control group performing the same strength training intervention as the endurance athletes, and it is difficult to assess the exact impact of the endurance training. The lack of impairments of muscle hypertrophy in the current study is in contrast to impaired muscle growth reported in the highly trained cyclists in the above-mention study by Ronnestad et al. [214]. Again, the difference between these studies is probably explained by the different amount of endurance training performed. The “classic” concurrent training studies also yield conflicting results regarding effects on muscle hypertrophy. Some report attenuated hypertrophy after concurrent training [30, 68, 141, 148, 207] whereas others do not [181, 182]. The reason behind these conflicting results are probable the same as discussed above regarding the different findings on maximal strength.

The reason for no attenuated effect of the strength training intervention on muscle hypertrophy in the current study may be related to the nutrition of the participants. There was no difference between *E+S* and *S* in total energy, carbohydrate or fat intake either in absolute values or normalized to body mass (table 8). However, *E+S* had higher protein intake than *S* both in absolute values and normalized to body mass ($p < 0.05$).

Table 8. Energy and macro-nutrient intake for athletes adding strength training to their normal endurance training (*E+S*), and previously untrained individuals performing strength training only (*S*).

Nutrient	<i>E+S</i>	<i>S</i>
Energy intake (kJ·day ⁻¹)	8901 ± 2119	7752 ± 811
Energy intake (kJ ·kg ⁻¹ · day ⁻¹)	141 ± 24	123 ± 32
Carbohydrate (g·day ⁻¹)	218 ± 93	232 ± 35
Carbohydrate (g ·kg ⁻¹ · day ⁻¹)	3.5 ± 1.5	3.6 ± 0.9
Protein (g·day ⁻¹)	104 ± 26*	67 ± 26
Protein (g ·kg ⁻¹ · day ⁻¹)	1.7 ± 0.4*	1.2 ± 0.3
Fat (g·day ⁻¹)	80 ± 26	75 ± 11
Fat (g ·kg ⁻¹ · day ⁻¹)	1.3 ± 0.3	1.2 ± 0.3

Values are mean ± SD. *Larger than *S* ($p < 0.05$).

Even though both groups had protein intakes that were within ACSM's recommendations for endurance and strength trained athletes [212], the daily protein intake was significantly higher in *E+S*. Perhaps the higher protein intake in *E+S* compensated for the concurrent training making the gains in leg_{LM} and maximal strength similar between the groups. Indeed, increasing protein intake in individuals already consuming the recommended amount of proteins may augment responses to strength training [52]. On the other hand, since the total energy intake was similar between the groups despite *E+S* performing 5 h of endurance training per week the energy balance probably was more positive in *S*. Furthermore, the carbohydrate intake in *E+S* was lower than the ACSM's recommendations for endurance athletes. A positive energy balance is important for optimal hypertrophy [90, 212]. However, some underestimation of energy intake might occur and the unchanged body mass in both groups suggests that both groups were in positive energy balance. Overall, the nutrition of the participants in the current study probably does not explain why no interference effect occurred.

3.10.2 The ability to develop forces at high contraction velocities, SJ and CMJ

S had a larger increase in peak torque during knee extension at $240^{\circ}\cdot\text{sec}^{-1}$ ($14.9 \pm 7.1\%$, $p < 0.01$) than *E+S* ($8.2 \pm 4.8\%$, $p < 0.01$), and the practical effect of the percent change in *S* compared to *E+S* was moderate ($ES = 1.11$). Before the intervention period *E+S* performed better than *S* in both SJ (*E+S*: 24.3 ± 6.0 cm, *S*: 18.9 ± 3.2 cm) and CMJ (*E+S*: 25.6 ± 4.2 cm, *S*: 21.0 ± 3.6 cm, figure 19). *E+S* increased SJ and CMJ height by $7.8 \pm 6.1\%$ and $5.9 \pm 6.4\%$ respectively ($p < 0.05$) while the corresponding numbers in *S* were $14.1 \pm 7.1\%$ and $11.0 \pm 7.6\%$ ($p < 0.05$, figure 19). The increase in SJ was larger in *S* than in *E+S*. The effect size analyses revealed a moderate practical effect in favor of *S* in both SJ ($ES = 0.95$) and CMJ ($ES = 0.73$).

The finding of attenuated increase in peak torque at high velocities and jumping performance after concurrent training is in accordance with previous “classical” concurrent training studies [71, 94, 126, 209]. Dudley & Djamil [71] reported attenuated strength gains in maximal knee extension torque at angular velocities between 140 and $240^{\circ}\cdot\text{s}^{-1}$ in untrained individuals performing concurrent training compared to individuals performing strength training only. Also in accordance with our results, the changes in peak torque were similar between the groups at lower angular velocities.

Increases in jumping performance are a common finding after a period of heavy strength training in untrained individuals [e.g. 248, 261]. However, in endurance athletes many studies reports no improvements in jumping performance after addition of strength training [86, 100, 165, 188], supporting our finding of a smaller improvement in jumping height in *E+S* compared to *S*. Attenuated increase in jumping performance has also been reported in well-trained male cyclists after addition of strength training compared to individuals only performing strength training [214].

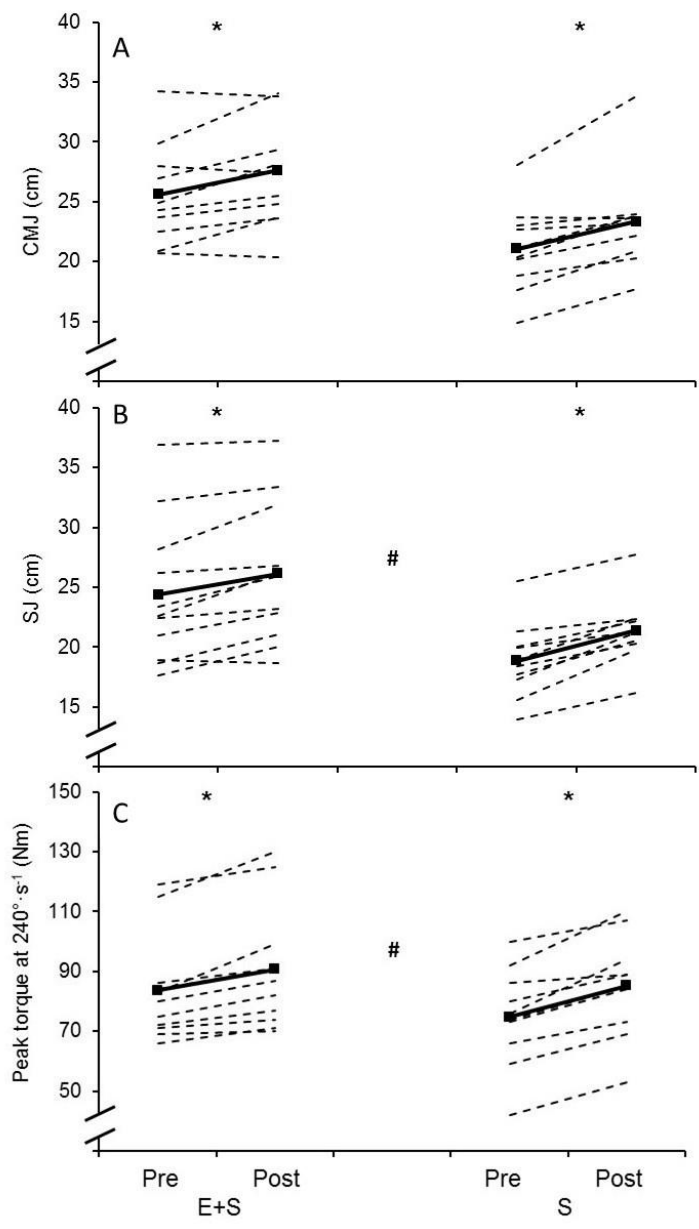


Figure 19. Individual values (dotted lines) and mean values (solid lines) before (Pre) and after (Post) the intervention period for athletes adding strength training to their normal endurance training (E+S), and, previously untrained individuals performing strength training only (S). A: Counter movement jump (CMJ). B: Squat jump (SJ). C: Peak torque in isokinetic knee extension at an angular velocity of 240 °·s⁻¹. * Different from Pre (p < 0.05), # the percent change from Pre is different between E+S and S (p < 0.05).

One factor regarding the results from the SJ and CMJ are worth mentioning. The *E+S* group jumped higher than *S* before the intervention period, and it may therefore be argued that the superior gains in SJ in *S* were because they were at a lower performance level. However, none of the participants in neither group had performed any jump training or explosive training during the last year leading up to the study. In addition, all other strength measurements were similar between the groups at baseline. The superior jumping performance in *E+S* before the intervention was probably because of better motor skills and non-significant lower body mass. Therefore, the larger improvement in *S* is most likely not because of their lower jumping performance before the start of the intervention.

Taken together, the findings from the current study support the notion that concurrent training impairs power related adaptations more than the ability to produce high forces during low contraction velocities. The reason for this is unclear, and the measurements and design in the current study is not suited to answer this question. However, some speculations are possible. The ability to produce high power output and force rapidly is in addition to maximal muscular strength dependent on percent of type II muscle fibers [79], rapid neural activation of the muscles [82, 209] and muscle fascicle length [41]. Häkkinen et al. [101] reported attenuated adaptations in RFD during isometric knee extension in untrained men after 21 weeks of concurrent training compared to participants performing strength training only. The strength training only group also increased iEMG in *m. vastus lateralis* muscle during the first 500 ms of the isometric contraction with no changes in the concurrent group. The authors therefore suggested that the lack of improved RFD after the concurrent training was due to lack of improved rapid voluntary neural activation [101].

There appear to be small to none differences in adaptations in fiber type composition after strength training only and concurrent training [148, 207]. In addition, the concurrent studies reporting impaired hypertrophy in muscle fibers indicate that this happens predominately in the type I fibers [30, 148]. Therefore, concurrent training does not seem to induce adaptations in muscle fiber type composition compared to strength training only that should have a larger negative impact on maximal power and rapid force production compared to force productions at low contraction velocities.

Long muscle fascicles with a large numbers of serially arranged sarcomeres is advantageous for developing forces at high contraction velocities compared to shorter muscle fascicles [41]. Strength training usually leads to increased anatomical muscle CSA and increased pennation

angles [3, 42, 43, 75, 87, 229]. These adaptations will have opposite effects on fascicle length. Therefore, fascicle length either remains unchanged [143] or increases slightly [42, 87, 229] after strength training. Research on the effects of endurance training on fascicle length is sparse [193]. However, long-distance runners have been reported to have shorter fascicles than sprinters and untrained individuals [5], and a recent study indicates that endurance running leads to increased pennation angle and shorter fascicles in *m. gastrocnemius* [193]. On the other hand, running training did not affect fascicle length in *m. vastus lateralis* [193] and 10 weeks of cycle training did not affect pennation angle in *m. vastus lateralis* or muscle CSA, indicating no changes in fascicle length [75]. Therefore it is unknown whether different adaptations in muscle architecture after concurrent training compared to strength training only can explain why concurrent training seems to impair adaptations in the ability to develop power. To the best of my knowledge, no concurrent training study have investigated this.

A limitation in the current study is that there may be differences in genotype and/or phenotype between endurance athletes and untrained individuals that might affect strength training adaptations. For example, the fiber type distribution between the groups might have been different. Endurance athletes have been reported to have larger proportions of type I fibers than untrained [245], and endurance training is reported to lead to fiber type shift from type IIX to type IIA [27, 124]. Since the hypertrophic response to strength training may differ between fiber types [82, 105] this could affect strength training adaptations. Another potential limitation of the current study is the fact that *E+S* performed their strength tests 20 min after a VO_{2max} test while *S* performed the strength test in a non fatigued state. Fatigue after the VO_{2max} test might have affected performance in the strength test. However, this was similar at both pre and post, and should therefore not affect the relative changes in strength from pre to post in *E+S* compared to *S*. Nevertheless, these limitations need to be kept in mind when the results are interpreted.

4 Conclusions

The conclusions of the current study are:

1. Adding 11 weeks of heavy strength training to female endurance athletes' normal training improved 40 min all-out cycling performance. The improved performance was related to improved cycling economy and improved fractional utilization of VO_{2max} . The heavy strength training had no effect on 40 min all-out running performance, running economy, or fractional utilization of VO_{2max} in running. VO_{2max} did not change in neither cycling nor running. The strength training program was effective in increasing anaerobic performance and other factors that can be decisive in endurance competitions as indicated by improved V_{max} and increased mean and peak power output during the Wingate test.
2. Adding 11 weeks of heavy strength training to female endurance athletes' normal training led to lower VO_2 during the last two hours of a 3 h submaximal cycling trial compared to pre intervention. The reduced VO_2 was accompanied by reduced HR. The heavy strength training had no effect on physiological variables during 1.5 h of submaximal running. Performance following the prolonged trials measured in a 5 min all out test was improved in both cycling and running after addition of strength training. The improved cycling performance was probably related to the improved cycling economy during the last part of the prolonged trial, leaving the athletes less fatigued before starting the 5 min all-out test. Improved anaerobic capacity probably contributed to the improved performance in both cycling and running. A fiber type transition from type IIAX-IIX to type IIX fibers was related to the improved performance in both cycling and running.
3. The improved cycling economy both at 150 W during the blood lactate profile test and during the last 2 h of the prolonged trial after strength training were related to increased muscle CSA and muscle strength. This probably led to less contribution of type II fibers at higher power outputs during the blood lactate test and delayed recruitment of type II fibers during prolonged cycling. The improved cycling economy may also been related to the muscle fiber type transition from type IIAX-IIX towards type IIA.
4. The probable reason for increased fractional utilization of VO_{2max} was increased muscle mass together with unchanged concentration of aerobic enzymes. This

probably led to a larger number of aerobic enzymes available for contributing to a certain power output.

5. Endurance athletes had similar improvements as untrained individuals in the ability to develop force at low contraction velocities measured as 1RM in one-legged leg press and MVC, and similar increase in leg_{LM} after an 11 weeks strength-training program. However, the endurance athletes had smaller improvements in the ability to develop knee extension torque at an angular velocity of $240^\circ \cdot \text{s}^{-1}$ and SJ height. This indicates that concurrent endurance training attenuated increases in the ability to develop high power.

5 Perspectives and directives for future research

Based on the results from the current thesis, female runners and cyclists can be recommended to include heavy strength training in their training programs for maximal gains in performance. Since both the improved cycling economy and the increased fractional utilization of VO_{2max} in cycling were related to increased muscle CSA, it seems imperative that strength training programs for improving cycling performance are design and performed in a way that ensures muscle growth. With the results from the current study, the strength training program used has been proven effective for improving cycling performance in both female athletes as well as well-trained [213, 217] and elite male cyclists. Consequently, the program seems to be effective for improving cycling performance

However, the lack of effect of this strength-training program on 40 min all-out running performance and the classical determinants on endurance performance are intriguing. As many previous studies reports improved running performance and running economy this indicate that the strength-training program used is not optimal for improving running performance, or that strength training may not be as beneficial for running performance when tested at a slight inclination. A focus in future studies should therefore be to elucidate what kind of strength training program that is optimal for runners. This program should maybe include more focus on the planar flexors and perhaps include some explosive and plyometric exercises. Since no previous study have investigated the effects of strength training on uphill running performance, this may also be an interesting topic for future studies.

In the current study, tendon properties were measured in the patellar tendon. Because of logistical restrains, measurements on the Achilles tendon was not possible. Since there seems to be differences in the optimal stiffness of the patellar and Achilles tendon for running economy and performance [14, 155], future studies should also include measurements on the Achilles tendon.

The findings of the current study also indicate that the mechanisms behind changes in cycling and running performance after strength training are different. A further elucidation of these differences would be an interesting topic for future studies. With better understanding of the mechanisms through which strength training affects performance in cycling and running, strength training programs can be better designed to be optimal for either runners, cyclists or athletes active in both sports.

In the current study, improved cycling economy was related to improved strength and muscle mass. Consequently, it was speculated that some of the improved exercise economy during submaximal cycling was attributed to a larger contribution of type I fibers at the same absolute workload. However, measurements of muscle fiber activation during the tests were not done. This mechanisms can be further explored in future studies, perhaps by using glycogen depletion or EMG to more directly asses changes in the contribution from different fiber types after a strength training intervention.

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Errata

Where in thesis	Original text/Error	Corrected text
Table of content, heading 2.5.2	Tests of muscle strength and explosive power	Tests of muscle strength and jumping performance
Page 28, heading 2.5.2	Tests of muscle strength and explosive power	Tests of muscle strength and jumping performance
Page 29, line 7	angel	angle
Page 29, line 10	angel	angle
Appendix I	There was an error with the references in appendix I. The numbers used to designate these references does not correspond to the correct reference numbers in the reference list after the main thesis.	Reference numbers in appendix I has been changed to correspond to a separate reference list that has been added to appendix I.

Papers I-IV

Paper I

Vikmoen O, Ellefsen S, Trøen Ø, Hollan I, Hanestadhaugen M, Raastad T, Rønnestad BR.
Strength training improves cycling performance, fractional utilization of $\text{VO}_{2\text{max}}$ and cycling
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Strength training improves cycling performance, fractional utilization of VO_{2max} and cycling economy in female cyclists

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The purpose of this study was to investigate the effect of adding heavy strength training to well-trained female cyclists' normal endurance training on cycling performance. Nineteen female cyclists were randomly assigned to 11 weeks of either normal endurance training combined with heavy strength training (E+S, $n = 11$) or to normal endurance training only (E, $n = 8$). E+S increased one repetition maximum in one-legged leg press and quadriceps muscle cross-sectional area (CSA) more than E ($P < 0.05$), and improved mean power output in a 40-min all-out trial, fractional utilization of VO_{2max} and cycling economy ($P < 0.05$). The proportion of type IIX-

IIX muscle fibers in m. vastus lateralis was reduced in E+S with a concomitant increase in type IIA fibers ($P < 0.05$). No changes occurred in E. The individual changes in performance during the 40-min all-out trial was correlated with both change in IIX-IIX fiber proportion ($r = -0.63$) and change in muscle CSA ($r = 0.73$). In conclusion, adding heavy strength training improved cycling performance, increased fractional utilization of VO_{2max} , and improved cycling economy. The main mechanisms behind these improvements seemed to be increased quadriceps muscle CSA and fiber type shifts from type IIX-IIX toward type IIA.

During the last decades, improved cycling performance after adding strength training to endurance training has been observed in several studies (Hickson et al., 1988; Rønnestad et al., 2010, 2011, 2014; Sunde et al., 2010; Aagaard et al., 2011). However, other studies do not report beneficial effects (Bishop et al., 1999; Bastiaans et al., 2001; Levin et al., 2009). Differences in methodology and specific training paradigms probably explain these conflicting results. To improve cycling performance, it seems vital that the strength training is performed with sufficiently high loads, typically between 10 to 4 repetition maximum (RM), and that the training volume is sufficiently high and performed for 8 weeks or longer (Aagaard & Andersen, 2010; Rønnestad & Mujika, 2014). The mechanisms behind the observed improvements in cycling performance are still somewhat unclear. Since endurance performance is mainly determined by the amount of oxygen that can be consumed during a given event (the performance VO_2) and the movement economy (Bassett & Howley, 2000), it seems reasonable to assume that strength training improves one or both of these factors.

Improved cycling economy has been observed after the addition of heavy strength training in trained male cyclists and master athletes (Sunde et al., 2010; Barrett-O'Keefe et al., 2012; Louis et al., 2012). The precise mechanism behind this improvement remains uncertain. An increase in the proportion of type IIA at the expense of type IIX muscle fibers has been reported in elite cyclists after 16 weeks of heavy strength training (Aagaard et al., 2011), and in untrained subjects after periods of heavy strength training (Staron et al., 1994; Kraemer et al., 1995; Andersen & Aagaard, 2000). Because type IIA fibers are more economical and less fatigable than type IIX fibers (Westerblad et al., 2010), this change in muscle fiber distribution may contribute to improved cycling economy and performance after heavy strength training (Aagaard & Andersen, 2010; Rønnestad & Mujika, 2014). However, improved cycling economy cannot be the sole mechanism behind the ergogenic effects as numerous studies report improved performance without changes in cycling economy (Rønnestad et al., 2010, 2014; Aagaard et al., 2011).

The performance VO_2 is determined by the maximal oxygen consumption (VO_{2max}) and the percent of VO_{2max} an athlete can sustain for the duration of the race (the fractional utilization of VO_{2max}) (Basset & Howley, 2000). Addition of strength training has neither a

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positive nor a negative effect on VO_{2max} (e.g. Hickson et al., 1988; Bishop et al., 1999; Aagaard et al., 2011). To our knowledge, no study has directly examined the effects of adding heavy strength training to normal endurance training on performance VO_2 or fractional utilization of VO_{2max} .

Fractional utilization of VO_{2max} is mainly determined by the amount of aerobic enzymes or mitochondria in the active muscles that contribute to a certain power output (Holloszy & Coyle, 1984; Coyle, 1995). Theoretically, the fractional utilization of VO_{2max} can therefore be improved by increasing the content and activity of the aerobic enzymes in the active muscles or increasing the amount of engaged/activated muscles at a certain power output (with no change in the content and activity of the aerobic enzymes). Since strength training can increase muscle mass and muscle cross-sectional area (CSA), this can theoretically increase the amount of mitochondria sharing the power output and therefore improve fractional utilization of VO_{2max} .

Most previous research on the effects of strength training on cycling performance has focused on male cyclists. In fact, to our knowledge, only one study has investigated the effects of adding heavy strength training to normal endurance training on cycling performance in female cyclists, and this study reported no effect on mean power output during a 60-min all-out cycling trial (Bishop et al., 1999). However, the strength training in that study included only one exercise that resulted in a low training volume, which might explain its lack of effect on performance. Therefore, it remains unknown whether or not addition of a larger volume of strength training to usual endurance training has an effect on cycling performance in female cyclists.

The main purpose of this study was to investigate the effects of 11 weeks of heavy strength training on 40-min all-out performance in trained female cyclists. We also wanted to investigate the effects on important determinants of cycling performance with a special focus on fractional utilization of VO_{2max} and cycling economy. To elucidate mechanisms behind any changes in performance and performance determinants, we measured changes in muscle strength, muscle CSA, fiber type composition and content, and expression of aerobic enzymes.

Methods

Ethical approval

The study was approved by the Local Ethics Committee at Lillehammer University College. Written informed consent was

obtained from all participants prior to inclusion, and the study was carried out in accordance with the Declaration of Helsinki.

Participants

Twenty-eight female cyclists that fulfilled at least two of Jeukendrup et al.'s (2000) training and race status descriptions of a well-trained cyclist were recruited to this study. Inclusion criteria were that the cyclist had trained on average four sessions or more per week with endurance training, and had not performed any systematic strength training for the last 12 months leading up to the study. The cyclists were matched for VO_{2max} , then randomly assigned to either adding heavy strength training to the ongoing endurance training (E+S, $n = 14$) or endurance training only (E, $n = 14$). During the study, three cyclists in E+S left the project, all for reasons unrelated to the project protocol: one because of medial tibial stress syndrome, one because of a prolonged period of illness during the last part of the intervention and one because of other medical reasons. In E, six cyclists left the study for reasons unrelated to the project protocol (injuries from bicycle crash, pregnancy, and lack of time). Therefore, the final numbers of cyclists in E+S and E were 11 and 8, respectively (Table 1). A questionnaire regarding the menstrual cycle and use of oral contraceptives was filled out by nine cyclist in E+S and seven in E. Six of these nine cyclist in E+S used oral contraceptives and the corresponding numbers in E were 4 out of 7.

Experimental overview

The strength-training program for the E+S group lasted for 11 weeks (during the competition period from April to July) and consisted of two strength-training sessions per week. Testing before and after the intervention period was performed over four test days. During pretests, muscle biopsies were sampled from m. vastus lateralis at day 1, while at day 2, CSA of the thigh muscles was measured by magnetic resonance imaging (MRI). Day 3 consisted of an incremental cycle tests for determination of blood lactate profile followed by a VO_{2max} test and a test of 1RM in one-legged leg press. Day 4 consisted of a Wingate test and a 40-min all-out test. There were 3–7 days between test days. At post, the only difference in test order was that muscle biopsies were taken on the last test day.

Training

Duration and intensity of endurance training was calculated based on heart rate (HR) recordings. Endurance training was divided into three HR zones: (1) 60–82%; (2) 83–87%; and (3) 88–100% of maximal HR. There were no significant differences between groups in the average weekly duration of the endurance training and the distribution of this training within the three intensity zones (Table 2). Also, no significant difference was found between E+S and E in total weekly training duration including endurance training, competitions, strength training, core stability training, and other alternative training forms (E+S: 7.0 ± 1.4 h, E: 6.8 ± 2.5 h, $P = 0.87$).

Table 1. Characteristics of the cyclists adding strength training to their normal endurance training (E+S) and cyclists performing normal endurance training only (E)

Group	<i>n</i>	Age (years)	Height (m)	Body mass (kg)	BMI (kg/m ²)
E+S	11	31.5 ± 8.0	1.69 ± 0.05	62.2 ± 5.2	21.7 ± 1.3
E	8	34.9 ± 7.5	1.70 ± 0.03	65.8 ± 8.2	22.8 ± 2.8

Values are mean ± SD.

Strength training and cycling performance

Table 2. Duration (in hours per week) of the training performed during the 11-week intervention period for cyclists adding strength training to their normal endurance training (E+S) and cyclists performing normal endurance training only (E)

	HR zone 1 (h)	HR zone 2 (h)	HR zone 3 (h)	HR zone 1 + 2 + 3 (h)	Leg strength (h)	Other (h)	Total
E+S	3.2 ± 1.4	1.1 ± 0.6	0.8 ± 0.6	5.1 ± 1.1	1.5 ± 0.0	0.3 ± 1.0	7.0 ± 1.4
E	4.5 ± 1.8	1.1 ± 0.3	0.8 ± 0.5	6.3 ± 2.2	0.0 ± 0.0	0.5 ± 0.8	6.8 ± 2.5

Values are mean ± SD.

Endurance training was divided into three heart rate (HR) zones: HR zone (1) 60–82%, HR zone; (2) 83–87%, and HR zone 3 (3) 88–100% of maximal HR.

The heavy strength training performed by E+S targeted leg muscles and was performed twice per week during the 11-week intervention period. Adherence to strength training was high, with E+S cyclists completing 21.4 ± 1.0 (range 19–22) of the planned 22 strength-training sessions. The strength-training regimen was designed to improve cycling performance by using cycling-specific exercises. Thus, strength-training exercises were performed using a range of motion from 90° knee flexion to almost full extension. In addition, since cyclists work each leg alternately during cycling, one-legged exercises were chosen in two of the four exercises. The performed exercises were as follows: half squat in a smith machine, one-legged leg press, standing one-legged hip flexion, and ankle plantar flexion. The cyclist were instructed to perform the strength training with maximal acceleration and speed during the concentric phase (duration around 1 s), while the eccentric and noncycling-specific phase was performed more slowly (duration 2–3 s). At the start of each strength-training session, cyclists performed a 5–10-min warm-up at self-selected intensity on a cycle ergometer, followed by 2–3 warm-up sets of half squats with gradually increasing load. For the first 2 weeks of the intervention, investigators supervised all training sessions. Thereafter, the follow-up frequency was kept at minimum once per week for the remainder of the intervention. During weeks one to three, cyclists trained with 10RM sets during the first session and 6RM sets during the second session. These alternating loads were adjusted to 8RM and 5RM during weeks 4–6, and were further adjusted to 6RM and 4RM during weeks 7–11. The numbers of repetitions was always the same as the prescribed RM load meaning that the sets were performed until failure. The cyclists were allowed assistance on the last repetition if necessary. The cyclists adjusted the absolute load as they got stronger to correspond to the prescribed RM load. The number of sets in each exercise was always three.

Strength and cycling performance tests

The cyclists were instructed to refrain from intense exercise the day preceding testing, to prepare for the test as they would have done for a competition, and to consume the same type of meal before each test. All cycling tests were performed on the same electromagnetically braked cycle ergometer (Lode Excalibur Sport, Lode B. V., Groningen, the Netherlands), which was adjusted according to each cyclist preference for seat height, horizontal distance between tip of seat and bottom bracket, and handlebar position. Cyclists were allowed to choose their preferred cadence during all cycling tests and they used their own shoes and pedals. All cycling tests were performed in similar environmental conditions (18–20 °C), and the cyclists were cooled with a fan during all cycling tests.

Blood lactate profile

The test was not preceded by a warm-up, and started with 5-min cycling at 50 W, which was subsequently increased every 5 min. The first increase was 50 W; thereafter, power output was

increased by 25 W every 5 min. Blood was sampled from a fingertip at the end of each 5-min bout and analyzed for whole blood [La⁻] using a Lactate Pro LT-1710 analyzer (Arcray Inc., Kyoto, Japan). At the same time, rate of perceived exertion was recorded. The test was terminated when a [La⁻] of 4 mmol/L or higher was measured. VO₂ was measured during the last 3 min of each bout, and mean values were used for statistical analysis. VO₂ was measured (30-s sampling time) using a computerized metabolic system with mixing chamber (Oxycon Pro, Erich Jaeger, Hoechberg, Germany). The gas analyzer was calibrated with certified calibration gases of known concentrations before every test. The flow turbine (Triple V, Erich Jaeger, Hoechberg, Germany) was calibrated before every test with a 3 L, 5530 series, calibration syringe (Hans Rudolph, Kansas City, Missouri, USA). From this continuous incremental cycling test, the power output at 3.5 mmol/L [La⁻] was calculated for each cyclist from the relationship between [La⁻] and power output using linear regression between data points. Work economy was determined by the average VO₂ at a power output of 150 W.

VO_{2max} and W_{max}

After termination of the blood lactate profile test, the cyclists cycled for 10 min at a submaximal power output (between 75 and 100 W) before completing another incremental cycling test for determination of VO_{2max}. This test has been described elsewhere (Rønnestad et al., 2010). Briefly, the test was initiated with 1 min of cycling at a power output of 100 W, which was subsequently increased by 25 W every minute until exhaustion. VO_{2max} was calculated as the average of the two highest 30-s VO₂ measurements. W_{max} was calculated as the mean power output during the last 2 min of the incremental test. After the test, blood [La⁻] and HR_{peak} was noted. HR was measured using a Polar S610i HR monitor (Polar, Kempele, Finland).

1RM tests

Approximately 20 min after termination of the VO_{2max} test, maximal strength in the legs was tested as 1RM in one-legged leg press. Prior to the testing day, each cyclist was given a supervised familiarization session to learn proper lifting technique and find individual equipment settings. During this session, the load was gradually increased to allow estimation of a proper starting point for the 1RM testing.

The 1RM test started with a specific warm-up, consisting of three sets with gradually increasing load (40%, 75%, and 85% of expected 1RM) and decreasing number of repetitions (10→6→3). The first attempt was performed with a load approximately 5% below the expected 1RM. If a lift was successful, the load was increased by approximately 5%. The test was terminated when the cyclists failed to lift the load in 2–3 attempts and the highest successful load lifted was noted as 1RM. Cyclists were given three minutes of rest between lifts.

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Wingate test

The Wingate test was performed after a 10-min warm-up at a submaximal load on the Lode cycle ergometer (75–100 W), including three submaximal sprints during the last 2 min. The 30-s all-out test started while pedaling at 60 rpm without braking resistance. Then, following a 3-s countdown, the braking resistance was applied to the flywheel and remained constant throughout the 30-s all-out test. Braking resistance was set to 0.67 Nm/kg body mass. Peak and average power output during the 30 s was recorded. Cyclists remained seated throughout the test, and strong verbal encouragement was provided throughout. Cyclists were instructed to pedal as fast as possible from the start and not to conserve energy for the last part of the test.

40-min all-out trial

After the Wingate test, the cyclists cycled at a submaximal power output (between 75 and 100 W) for 10 min before the start of the 40-min all-out trial. During the first 5 min of the test, the power output was set by the investigators. This individual selected power output was based on the lactate profile test and corresponded to the power output at 2.5 mmol/L [La]. Thereafter, the control unit controlling the power output was given to the cyclists, which were allowed to adjust the power output themselves, with instructions to cycle at as high average power output as possible. During this test, the ergometer was in a cadence-independent mode (constant-Watt production), so that the power output was not affected by the cadence.

To estimate performance VO_2 and the fractional utilization of $\text{VO}_{2\text{max}}$, measurements of VO_2 were made during the last minute of every 5-min section of the test. During this minute the cyclists were not allowed to adjust the power output. The average VO_2 during this minute was estimated to reflect VO_2 during the corresponding 5-min section. VO_2 was measured continuously during the last 5 min of the test, as pilot testing showed that cyclists performed numerous power output adjustments during this part of the test. The mouthpiece used to measure VO_2 was inserted into the mouth 30 s before the start of every measurement by the cyclists themselves. This process took 1–3 s and was practiced by the cyclists during the warm-up and at previous tests. Performance VO_2 was calculated as the average VO_2 of all 5-min sections, and fractional utilization of $\text{VO}_{2\text{max}}$ was calculated as performance VO_2 in percentage of $\text{VO}_{2\text{max}}$. The cyclists were allowed to occasionally stand in the pedals during the trial and to drink water *ad libitum*.

CSA of m. quadriceps femoris

MRI was used to measure quadriceps muscle CSA. The cyclists lied on their back inside the MRI machine (S-Scan, Esaote, Genova, Italy) and a coil was placed around the distal part of the upper thigh. Care was taken to assure similar positioning of the coil at pre and post. Twenty-three cross-sectional images were sampled starting at the proximal edge of the patella and moving up the thigh with 10 mm between the images. Images were analyzed using the software OsiriX version 5.6 (Pixmeo, Geneva, Switzerland). Since the strength-training program mainly targeted the extensor muscles, m. quadriceps femoris were chosen for analyses. The 3–5 most proximal images (at the edge of the coil) had a low resolution and could not be used for analyses. Therefore, the CSA of the quadriceps muscles was measured using the four most proximal images with good resolution and the average CSA of all four images was used for statistical analysis. For each cyclist, pre and post CSA was always calculated from the same images. The technician performing the MRI scan and the investigator performing image analyses were blinded for which group the cyclists belonged to. The cyclists were instructed to refrain from physical

activity for the 48 h leading up to the MRI scan and were instructed to refrain from ingesting any food or drinks for the last 2 h.

Muscle biopsy sampling

Muscle biopsies were sampled from m. vastus lateralis using the Bergström procedure (Ellefsen et al., 2014b). Cyclists were instructed to refrain from physical activity during the last 24 h before biopsy sampling. During each biopsy sampling event, two separate muscle biopsies were retrieved and pooled in a Petri dish filled with sterile physiological saline. An appropriately sized muscle sample was immediately excised and selected for quantitative real-time PCR (qRT-PCR) analyses (average wet weight \pm SD: 38 ± 7 mg), whereupon a similarly sized sample was selected for immunohistochemical analyses (average wet weight \pm SD: 34 ± 13 mg). A sample of about 50 mg (average wet weight \pm SD: 51 ± 5 mg) was selected for homogenization/fractionation and later Western blotting. Pre- and post-biopsies were sampled at the same time of day for each particular cyclist. Biopsies for immunohistochemical analyses were immediately immersed in 10% buffered formaldehyde solution (Chemi-Teknik AS, Oslo, Norway), wherein they were left to fixate for 3–4 days before further preparation. Biopsies for Western blotting analyses were snap-frozen in isopentane, cooled with dry ice, before storage at -80°C until extraction. Biopsies for qRT-PCR analyses were immediately immersed in RNAlater® (Ambion, Foster City, CA, USA) and were treated according to manufacturer's protocol before storage at -80°C until RNA extraction.

Muscle biopsy analyses

Immunohistochemistry

Formalin-fixed muscle biopsies were processed using a Shandon Excelsior ES (Thermo Scientific, Hanover Park, Illinois USA), before it was paraffin-embedded and sectioned, whereupon transverse, serial sections were immunolabeled for myosin heavy chain I (MyHC1) (*A4.840*), MyHCIIA (*EPR5280*), and MyHCIIIX (*6H1*), as previously described (Ellefsen et al., 2014b). The detection system used for determination of muscle fiber types was EnVision™ Flex+ (Dako, Glostrup, Denmark) by using the immunostainer, Autostainer Link 48 (Dako). Determination of muscle fiber composition was based on analysis of a minimum of 200 fibers, performed using Photoshop CS6 Extended (Adobe, San Jose, California, USA). The investigator performing image analyses were blinded for which group the cyclists belonged. Because of technical problems with some analyses, the number of cyclists in the immunohistochemistry data is 8 in E+S and 8 in E.

For fibers that labeled for the IIX antibody, a particular issue became evident during analyses. All these fibers were found to co-label for the IIA-antibody, but not vice versa. This means one of two things: (a) either all IIX-positive fibers are IIX hybrids or (b) the IIA-antibody recognizes the IIX antigen in addition to recognizing the IIA antigen. In this study, we have chosen to refer to all these fibers type as IIX-IIX fibers.

Gene expression

Changes in gene expression were measured for proteins involved in aerobic energy metabolism. All genes included in the array with associated primers are listed in the Supporting Information. All results from gene expression are available as Supporting Information.

Primers design, RNA extraction, evaluation of expression stability of reference genes, and qPCR were performed as previously described (Ellefsen et al., 2014a,b). β_2 -microglobulin and ribo-

somal protein L32 was evaluated to be the two most stable references genes and used for calculation of normalization factors by GeNorm, which were then utilized for calculation of target gene expression.

Protein immunoblot

For Western blotting analyses, ~50 mg of muscle tissue was homogenized and fractionated into cytosol, membrane, nuclear, and cytoskeletal fractions using ProteoExtract Subcellular Proteo Extraction Kit (Cat. no. 539790, Calbiochem, San Diego, CA, USA; EMD Biosciences GmbH, Schwalbach, Germany), performed according to the manufacturer's protocol. Protein concentrations were assessed with a commercial kit (BioRad DC protein micro plate assay, nos. 0113, 0114, 0115; Bio-Rad Laboratories, Inc., Hercules, California, USA), a filter photometer (Expert 96; ASYS Hitech Cambridge, UK), and the provided software (Kim Version 5.45.0.1; Daniel Kittrich). Membrane fractions (including the mitochondrial components) were analyzed by the Western blotting technique. Equal amounts of protein from pre- and post-biopsies (7–12 µg) were loaded into wells and separated on 4–12% SDS-PAGE gels for 35–45 min at 200 V in cold MES running buffer (NuPAGE MES SDS running buffer; Invitrogen, Inc., Carlsbad, California, USA) under denaturing conditions. Thereafter, proteins were blotted onto a PDVF-membrane (immunoblot, cat. no. 162–0177; Bio-Rad Laboratories, Inc.), at 30 V for 90 min in cold transfer buffer (NuPAGE transfer buffer, cat. no. NP0006-1; Life Technologies, Inc., Calsbad, CA, USA). Membranes were blocked at room temperature for 2 h in a 5% fat-free skimmed milk and 0.05% TBS-T solution [TBS, cat. no. 170–6435 (Bio-Rad Laboratories, Inc.); Tween 20, cat. no. 437082Q (VWR International, Radnor, Pennsylvania, USA); skimmed milk, cat. no. 1.15363 (Merck KGaA, Darmstadt, Germany)]. Blocked membranes were incubated with antibodies against Citrate Synthase (CS, rabbit anti-Citrate Synthase, cat. no. Ab96699, diluted 1:1000; Abcam Plc, Cambridge, UK), Hydroxyacyl-CoA dehydrogenase (HADH, rabbit anti-HADH cat. no. Ab15088, diluted 1:8000; Abcam Plc) and cytochrome c oxidase subunit IV (COX 4, mouse anti-COX4, cat. no. Ab14744, diluted 1:1000; Abcam Plc) overnight at 4 °C, followed by incubation with secondary antibody (COX4: goat anti-mouse, cat. no. 31430, diluted 1:30000; Thermo Fisher Scientific, Inc., Hanover Park, Illinois, USA, CS and HADH: Anti rabbit cat. No. 70745 diluted 1:3000; Cell Signaling Technology, Inc., Danvers, MA, USA) at room temperature for 1 h. All antibodies were diluted in a 1% fat-free skimmed milk and 0.05% TBS-T solution. Between stages, membranes were washed in 0.05% TBS-T solution. Bands were visualized using an HRP-detection system (Super SignalWest Dura Extended Duration Substrate, cat. no. 34076; Thermo Fisher Scientific, Inc., Waltham, Massachusetts, USA). Chemiluminescence was measured using a CCD image sensor (Image Station 2000R or Image Station 4000R; Eastman Kodak, Inc., Rochester, New York, USA), and band intensities were calculated with Carestream molecular imaging software (Carestream Health, Inc., Rochester, New York, USA). All samples were run as duplicates and mean values were used for statistical analyses.

Statistical analyses

All data in text, figures, and tables are presented as mean ± standard deviation, unless otherwise stated. Prior to statistical testing, gene expression of aerobic enzymes data and protein data were log₂-transformed. This was done to maximize the likelihood of normal distribution. For data on muscle fiber composition, square-root arcsine-transformation was performed, representing the recommended transformation for proportional (Sokal & Rohlf, 2012).

To test for differences between groups at pre, post as well as differences in changes from pre to post, unpaired Student's *t*-test

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were used. Within-group analyses were performed using paired *t*-tests. Effect sizes (ES) were calculated for key performance and physiological adaptations to elucidate on the practical significance of strength training. ES were calculated as Cohen's *d* and the criteria interpret the magnitude were the following: 0–0.2 = trivial, 0.2–0.6 = small, 0.6–1.2 = moderate, 1.2–2.0 = large and > 2 = very large (Hopkins et al., 2009).

Correlation analyses were done using the Pearson product-moment method.

Analyses were performed in Excel 2013 (Microsoft Corporation, Redmond, Washington, USA) and in GraphPad Prism 6 (GraphPad Software Inc., California, USA). All analyses resulting in $P \leq 0.05$ were considered statistically significant. P -values ≤ 0.10 were considered as tendencies.

Results

There were no significant differences between E+S and E in any of the measured variables at baseline.

Body mass, maximal strength and muscle CSA

Body mass remained unchanged in E+S (from 62.4 ± 5.2 kg to 63.1 ± 5.6 kg) but was slightly reduced in E (from 65.6 ± 8.4 kg to 64.8 ± 8.0 kg, $P = 0.03$). The change in body mass was different between the groups ($P = 0.01$).

E+S increased 1RM in one-legged leg press with $38.6 \pm 19.0\%$ ($P = 0.0005$) while no statistically significant changes occurred in E ($5.6 \pm 9.0\%$, $P = 0.15$, Fig. 1). The change in 1RM was larger in E+S than in E ($P = 0.004$), with a large practical effect of E+S compared with E (ES = 2.20).

The CSA of m. quadriceps femoris increased in E+S with $7.4 \pm 5.3\%$ ($P = 0.0004$, Fig. 1) while no change occurred in E ($0.6 \pm 3.5\%$, $P = 0.98$). The change in CSA was larger in E+S than in E ($P = 0.006$) with a large practical effect of E+S compared with E (ES = 1.57).

Because of the change in body mass in E, VO_2 and power output measurements are presented as body mass-adjusted values.

Muscle fiber type composition

Fiber type composition determined by immunohistochemistry is displayed in Fig. 2. Proportions of fibers positive for both IIA and IIX MyHC were reduced from $9 \pm 7\%$ to 0% in E+S ($P = 0.005$) with a concomitant increase in fibers positive for type IIA only ($39 \pm 13\%$ to $51 \pm 10\%$, $P = 0.002$). No changes in fiber type composition were observed in E.

Aerobic enzymes

The protein content of CS, COX, and HADH per 50 mg of muscle did not change significantly in any of the groups during the intervention period (Fig. 3).

$\text{VO}_{2\text{max}}$, W_{max} , and Wingate test

$\text{VO}_{2\text{max}}$ and W_{max} were unchanged in both groups during the intervention period ($\text{VO}_{2\text{max}}$: E+S $-1.1 \pm 2.6\%$ and E

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$-1.0 \pm 2.4\%$, W_{max} : E+S $3.9 \pm 6.7\%$ and E $5.7 \pm 10.3\%$, Table 3). Peak power in the 30-s Wingate test increased in E+S by $12.7 \pm 12.6\%$ ($P = 0.004$) with no statistically significant change in E ($6.2 \pm 14.2\%$, $P = 0.27$, Table 3). There was however no significant difference in the change between groups, but the ES analysis revealed a small practical effect of E+S compared with E

(ES = 0.49). E+S also increased mean power output during the Wingate test by $3.4 \pm 4.3\%$ ($P = 0.02$) while there were no change in E ($-0.2 \pm 3.4\%$, $P = 0.85$, Table 3). There was a tendency to a larger increase in E+S ($P = 0.07$), with a moderate practical effect of E+S compared with E (ES = 0.83). The rate of fatigue (percent power decline) during the 30-s all-out test did not change in E+S (pre: $82.7 \pm 11.8\%$, post: $82.6 \pm 8.4\%$) or in E (pre: $81.6 \pm 8.1\%$, post: $78.6 \pm 8.3\%$).

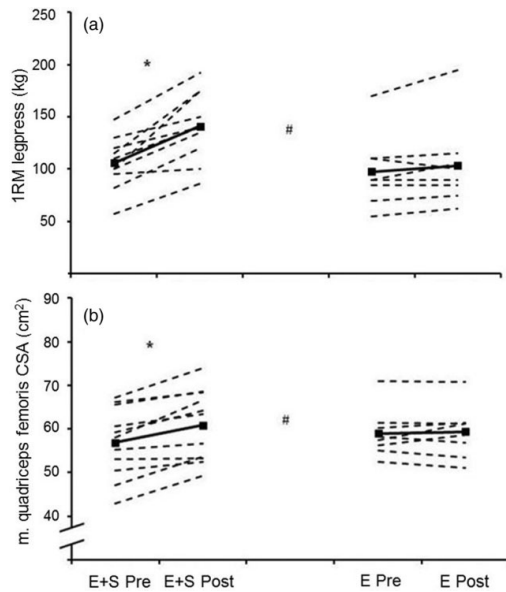


Fig. 1. Individual values (dotted lines) and mean values (solid lines) before (pre) and after (post) the intervention period for cyclists adding strength training to their normal endurance training (E+S) and cyclists performing normal endurance training only (E). (a) 1 repetition maximum (RM) in one-legged leg press. (b) Cross-sectional area (CSA) of m. quadriceps femoris. *Different than pre ($P < 0.05$), #the percent change from pre is different in E+S than in E ($P < 0.05$).

Power output at 3.5 mmol/L [$l a^{-1}$] and cycling economy

Power output at 3.5 mmol/L [$l a^{-1}$] tended to increase by $7.6 \pm 12.0\%$ ($P = 0.06$) in E+S with no significant change in E ($4.1 \pm 8.4\%$, $P = 0.23$, Fig. 4). There were no difference in change between the groups and the practical effect was small (ES = 0.34).

During the blood lactate profile test, E+S decreased the VO_2 at a power output of 150 W (corresponding to $93 \pm 13\%$ of the power at 3.5 mmol/L [$l a^{-1}$] at pre) by $3.5 \pm 3.1\%$ ($P = 0.004$) while there were no change in E ($0.7 \pm 4.8\%$, $P = 0.85$, Fig. 4). The change in VO_2 was larger in E+S than in E ($P = 0.022$) and the ES analysis revealed a moderate practical effect (ES = 1.0). The VO_2 at 150 W for E+S corresponded to $71 \pm 5\%$ of and $70 \pm 5\%$ VO_{2max} at pre and post respectively, and the corresponding numbers for E was 66 ± 7 and $68 \pm 8\%$. There were no changes from pre to post, and no difference between groups. The change in VO_2 at 150 W showed a significant negative correlation with the change in CSA of the quadriceps muscles ($r = -0.54$, $P = 0.02$, Fig. 5) meaning an increased CSA of the quadriceps muscles was associated with improved cycling economy.

40-min all-out test

The mean power output during the 40-min all-out trial increased by $6.4 \pm 7.9\%$ in E+S ($P = 0.002$), with no

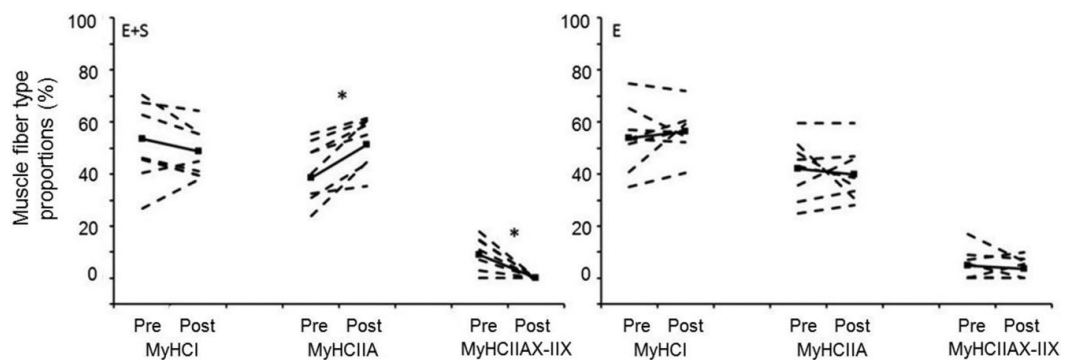


Fig. 2. Individual values (dotted lines) and mean values (solid lines) before (pre) and after (post) the intervention period for cyclists adding strength training to their normal endurance training (E+S) and cyclists performing normal endurance training only (E). Immunohistochemistry determined muscle fiber proportions, presented as percentage of overall fiber abundance. *Different from pre ($P < 0.05$).

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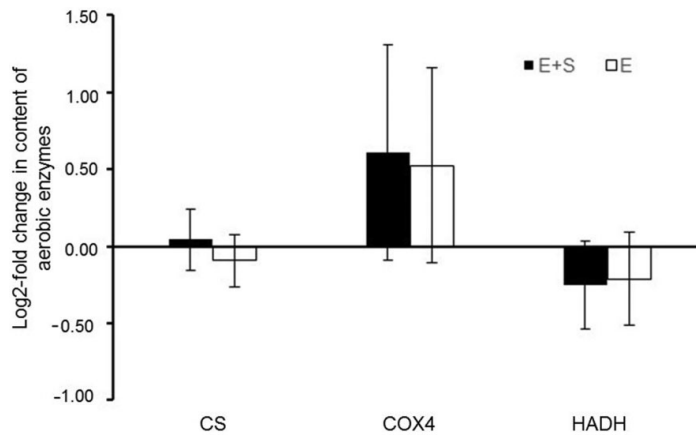


Fig. 3. Log2-fold change in protein levels of citrate synthase (CS), cytochrome c oxidase subunit IV (Cox4), and hydroxyacyl-CoA dehydrogenase (HADH) during the intervention period for cyclists adding strength training to their normal endurance training (E+S) and cyclists performing normal endurance training only (E). Values are mean \pm CI.

Table 3. Data from the maximal oxygen consumption (VO_{2max}) and Wingate tests before (pre) and after (post) the intervention period for cyclists adding strength training to their normal endurance training (E+S) and cyclists performing normal endurance training only (E)

	E+S		E		Cohen's <i>d</i>
	Pre	Post	Pre	Post	
W_{max} (W/kg)	4.0 \pm 0.3	4.2 \pm 0.3	4.0 \pm 0.4	4.2 \pm 0.2	0.21
VO_{2max} (mL/kg/min)	53.5 \pm 3.6	52.5 \pm 4.2	54.6 \pm 3.4	53.5 \pm 1.8	0.05
HR_{peak} (beats/min)	188 \pm 9	186 \pm 8	182 \pm 8	182 \pm 6	–
RPE	19.5 \pm 0.5	19.8 \pm 0.4	19.4 \pm 0.7	19.3 \pm 0.6	–
$[la]_{peak}$ (mmol/L)	10.8 \pm 3.2	10.4 \pm 2.9	9.8 \pm 2.4	9.7 \pm 2.3	–
Peak power Wingate (W/kg)	17.0 \pm 2.0	19.1 \pm 2.5*	17.7 \pm 1.4	18.7 \pm 1.7	0.49
Mean power Wingate (W/kg)	8.1 \pm 0.7	8.4 \pm 0.6*	8.1 \pm 0.5	8.1 \pm 0.6	0.83

Values are mean \pm SD.

*Larger than pre ($P < 0.05$).

HR_{peak} , peak heart rate; $[la]_{peak}$, peak blood lactate concentration; RPE, rate of perceived exertion.

change in E ($2.0 \pm 6.1\%$, $P = 0.44$, Fig. 4). There was no difference between groups, but there was a moderate practical effect of E+S compared with E ($ES = 0.63$). There were significant correlations between the change in mean power output during the 40-min all-out test and change in the proportion of IIX fibers ($r = -0.63$, $P = 0.009$) and change in CSA ($r = 0.73$, $P < 0.0004$, Fig. 5), respectively. The change in 40-min all-out performance also correlated with change in power output at 3.5 mmol/L $[la]$ ($r = 0.63$, $P = 0.004$) and change in cycling economy ($r = -0.47$, $P = 0.04$), but not with change in VO_{2max} ($r = 0.234$, $P = 0.33$).

The performance VO_2 during the 40-min all-out trial tended to increase by $2.3 \pm 4.2\%$ in E+S ($P = 0.08$), with no change in E ($-0.6 \pm 4.2\%$, Fig. 6). The change in performance VO_2 showed a significant correlation with the change in mean power output during the 40-min all-out trial ($r = 0.67$, $P = 0.002$) and the change in CSA of the quadriceps muscles ($r = 0.59$, $P = 0.01$, Fig. 5).

The fractional utilization of VO_{2max} during the 40-min all-out trial increased in E+S from $78.9 \pm 3.2\%$ to $82.2 \pm 3.9\%$ ($P = 0.05$, Fig. 6), with no change in E (from $78.7 \pm 8.0\%$ to $79.4 \pm 3.4\%$).

Discussion

In the present study, we investigated the effects of adding heavy strength training to the regular endurance training of well-trained female cyclists on cycling performance, muscle strength, muscle CSA, muscle fiber type composition, and muscle concentration as well as expression of aerobic enzymes. This is the first study to show that addition of heavy strength training to female cyclists' normal endurance training improves 40-min all-out performance and cycling economy. Another novel finding was that heavy strength training improved the fractional utilization of VO_{2max} during a 40-min all-out trial in endurance athletes, which has not been previously

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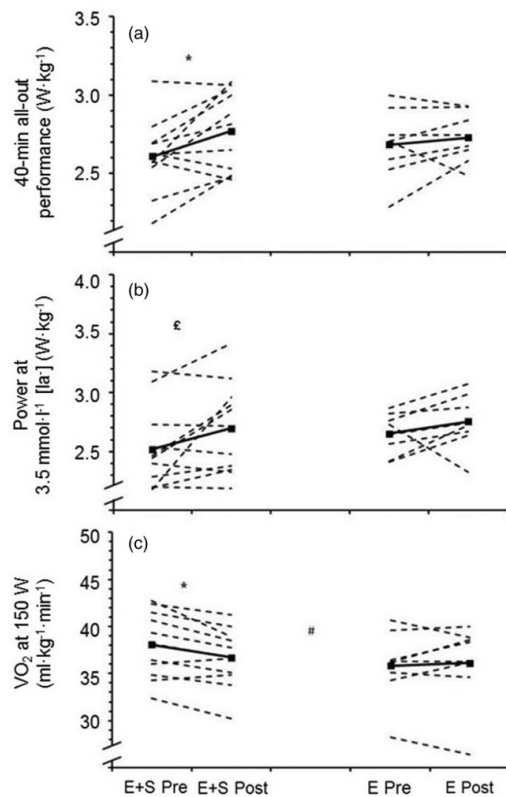


Fig. 4. Individual values (dotted lines) and mean values (solid lines) before (pre) and after (post) the intervention period for cyclists adding strength training to their normal endurance training (E+S) and cyclists performing normal endurance training only (E). (a) Body mass-adjusted mean power output during the 40-min all-out trial. (b) Body mass-adjusted power output at 3.5 mmol/L [La] calculated during the blood lactate profile test. (c) Body mass-adjusted oxygen consumption (VO_2) at 150 W. *Different from pre ($P < 0.05$), # the percent change from pre is different in E+S than in E ($P < 0.05$), £ tendency to be different from pre ($P < 0.1$).

demonstrated. These improvements were associated with both increased CSA of the quadriceps muscles and reduced proportion of type IIAX-II muscle fibers. Cyclists that continued with their normal endurance training only did not exhibit changes in any of the measurements.

40-min all-out trial

The 6.4% improvement in performance is in agreement with findings of 6–8% improvement in 40- to 45-min all-out performance in well-trained to elite male cyclists after similar strength-training interventions (Rønnestad et al., 2010, 2014; Aagaard et al., 2011). Improvements after heavy strength training have also been reported

during time to exhaustion tests at 80% to 100% of $\text{VO}_{2\text{max}}$ in trained male cyclists (Hickson et al., 1988; Sunde et al., 2010). However, there are also studies reporting no effect on cycling performance after the addition of heavy strength training (Bishop et al., 1999; Bastiaans et al., 2001; Levin et al., 2009). In fact, in the only previous study involving female cyclists, 12 weeks of heavy strength training had no effect on power output during a 60-min cycling test (Bishop et al., 1999). However, the low strength training volume used in that study (only one exercise was performed) may explain the lack of improved cycling performance.

Long-term cycling performance is mainly determined by the performance VO_2 and the cycling economy (Coyle, 1995; Bassett & Howley, 2000). Therefore, the improved performance observed in the current study should be accompanied by an improvement in one or both of these factors.

Performance VO_2

The tendency to increased performance VO_2 in E+S probably contributed to the improved performance in the 40-min all-out trial as indicated by the relationship between change in performance VO_2 and change in 40-min all-out performance ($r = 0.67$). Because performance VO_2 depends on $\text{VO}_{2\text{max}}$ and fractional utilization of $\text{VO}_{2\text{max}}$ (Coyle, 1995; Bassett & Howley, 2000), and because $\text{VO}_{2\text{max}}$ did not increase in E+S, the tendency to improved performance VO_2 and the concomitantly improved 40-min all-out performance was likely due to the improved fractional utilization of $\text{VO}_{2\text{max}}$.

To our best knowledge, this is the first study to investigate effects of concurrent training on fractional utilization of $\text{VO}_{2\text{max}}$ and performance VO_2 in endurance athletes. However, a few studies have measured effects of concurrent training on VO_2 at the lactate threshold in percent of $\text{VO}_{2\text{max}}$, an indirect measure of fractional utilization of $\text{VO}_{2\text{max}}$, none of which has reported an ergogenic effect in cycling (Sunde et al., 2010; Rønnestad et al., 2014). The reason for the different results in the current study compared with previous studies is unclear, but one obvious reason is the different methods used to estimate fractional utilization of $\text{VO}_{2\text{max}}$. In fact, when estimating changes in VO_2 at the power output corresponding to 3.5 mmol/L [La] in percent of $\text{VO}_{2\text{max}}$ in the current data set, both groups showed a similar increase of about three percentage points (results not shown).

Even though not measured directly, data from a study by Aagaard et al. (2011) indicate that improvements in fractional utilization of $\text{VO}_{2\text{max}}$ occurred after heavy strength training. They found concurrent training to increase mean power output during a 45-min all-out trial without changes in cycling economy and $\text{VO}_{2\text{max}}$, suggesting improvement of fractional utilization of $\text{VO}_{2\text{max}}$ and thus performance VO_2 . In fact, the authors estimated

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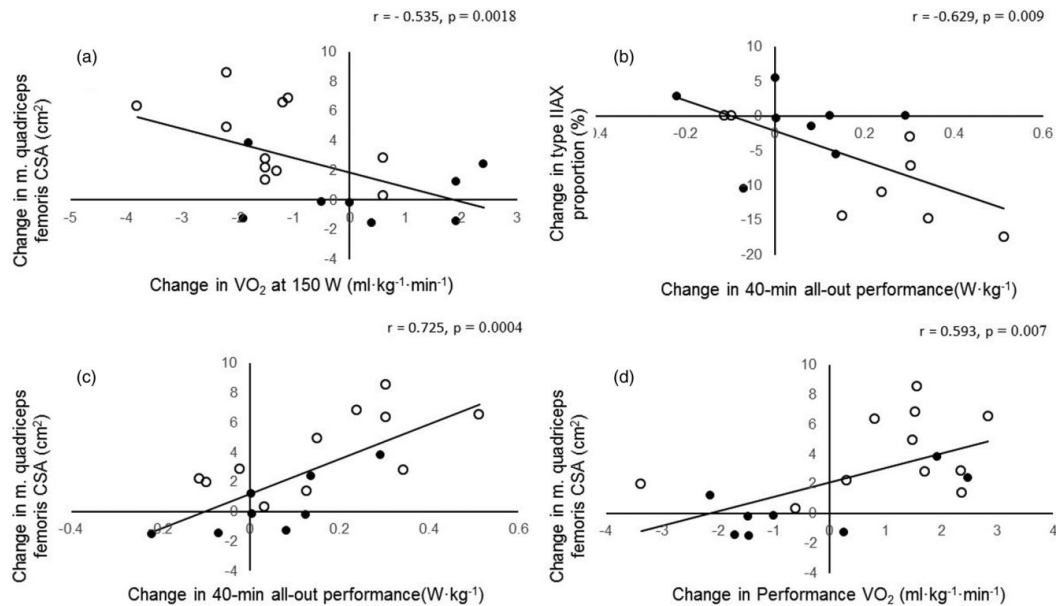


Fig. 5. (a) Correlation between change in m. quadriceps femoris cross-sectional area (CSA) and change in oxygen consumption (VO_2) at 150 W. (b) Correlation between changes in type IIX-IIX proportions and changes in mean power output during the 40-min all-out trial. (c) Correlation between change in m. quadriceps femoris CSA and change in mean power output during the 40-min all-out trial. (d) Correlation between change in m. quadriceps femoris CSA and change in performance VO_2 during the 40-min all-out trial. Open circles represents cyclists in E+S while solid circles represents cyclists in E.

that the power output during the test had increased from 76% to 83% of the power output at VO_{2max} .

The mechanisms behind the increased fractional utilization of VO_{2max} and hence the tendency for improved performance VO_2 is unclear, but may relate to the increased CSA in the quadriceps muscles. This is supported by the observed relationship between changes in CSA and performance VO_2 ($r = 0.59$). It has been reported that cyclists that uses a larger amount of their muscle mass have larger fractional utilization of VO_{2max} (Coyle, 1995). The reason for this is probably that a larger amount of mitochondria is activated and the fractional utilization of VO_{2max} is mainly determined by the amount of aerobic enzymes and mitochondria sharing a certain amount of VO_2 (Holloszy & Coyle, 1984; Coyle, 1995). The increased muscle CSA in the current study probably allowed for more muscle mass to be activated, and since the concentration of aerobic enzymes in m. vastus lateralis was unchanged, the total amount of aerobic enzymes available for sharing a certain power output should be increased. This may allow the increased power output to be sustained with unchanged relative load experienced by the mitochondria, thereby improving fractional utilization of VO_{2max} .

Although the method for measuring fractional utilization of VO_{2max} and performance VO_2 in the current study is not validated, we are confident that our measurement

of performance VO_2 is accurate and valid. To minimize the subjects' discomfort and increase the external validity, the VO_2 was only measured during the last minute of every 5 min section during the 40-min all-out trial. The mean VO_2 during this minute was assumed to reflect the VO_2 for the entire 5-min period. Large changes in power output will of course threaten this assumption. However, based on pilot work, we noticed that the athletes did not change their power output often with the exception of the last 5 min of the 40-min all-out trial. Therefore, VO_2 was measured during the last 5 min and the average consumption was taken as VO_2 during this period.

Cycling economy

The improved cycling economy observed in E+S probably explains some of the improved power output during the 40-min all-out trial. This is supported by the relationship between change in VO_2 at 150 W and change in mean power output during the 40-min all-out trial ($r = -0.47$). The finding of improved cycling economy is in agreement with results from other studies in master athletes (Louis et al., 2012) and trained cyclists (Sunde et al., 2010; Barrett-O'Keefe et al., 2012). However, the addition of heavy strength training does not seem to improve cycling economy in highly trained to elite

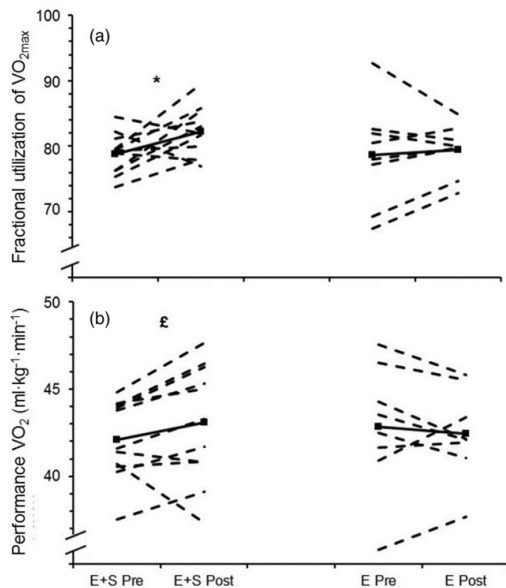


Fig. 6. Individual values (dotted lines) and mean values (solid lines) before (pre) and after (post) the intervention period for cyclists adding strength training to their normal endurance training (E+S) and cyclists performing normal endurance training only (E). (a) The fractional utilization of VO_{2max} during the 40-min all-out trial. (b) The performance VO_2 during the 40-min all-out trial. *Different from pre ($P < 0.05$), £ tendency to be different from pre ($P < 0.1$).

cyclists (Rønnestad et al., 2010, 2014; Aagaard et al., 2011).

The precise mechanism for improved cycling economy after heavy strength training is not completely understood. Barrett-O'Keefe et al. (2012) found a reduced pulmonary VO_2 and a corresponding reduced leg VO_2 after 8 weeks of heavy strength training. This indicates that the mechanism may lie inside the trained muscles. One suggestion is that an increase in the proportion of type IIA muscle fibers at the expense of type IIX muscle fibers might be a contributor to improved cycling economy (Aagaard & Andersen, 2010; Rønnestad & Mujika, 2014). Since the type IIA fibers have been suggested to be more economical than the type IIX fibers (Westerblad et al., 2010), this would in theory improve work economy. Our data support a role for such fiber type conversions behind the improved cycling economy, as the E+S group displayed an increase in proportions of type IIA fibers with a concomitant reduction in proportions of type IIX fibers. However, no correlation between fiber type conversion and changes in cycling economy was observed.

In line with the discussion on more efficient muscle fibers, an increase in the quadriceps muscle CSA and increased muscle strength may also contribute to

improved cycling economy. This is supported by the relationship between change in CSA of the quadriceps muscle and improved cycling economy ($r = -0.54$). When the maximal muscle strength increases, the force levels required to ride at a certain power output is reduced relatively to maximal force. This implies that the economical type I and type IIA muscle fibers can account for a larger proportion of a certain absolute power output (Hickson et al., 1988; Rønnestad & Mujika, 2014), as follows from the size principle of motor unit recruitment. Indeed, cycling economy has been related to proportions of type I fibers in the active muscles (Coyle et al., 1992; Mogensen et al., 2006), although this is not a universal finding (Hopker et al., 2013). The combined effect of the aforementioned mechanisms of increased proportions of type IIA fibers at the expense of type IIX fibers and increased quadriceps CSA should also have an extra effect on both 40-min all-out performance and cycling economy since the total muscle CSA covered by the fatigue resistant and more economical IIA fibers will be substantially increased.

W_{max} and power output in the Wingate test

Cycling races are often decided in a sprint or in a few minutes of all-out performance at the end of the race. Furthermore, the ability to close gaps or break away from the pack can also be important for success. Therefore, the ability to generate high power output for a short period of time is an important factor for cycling performance (Atkinson et al., 2003). In the present study, none of the groups had a significant increase in W_{max} . The lack of an effect of heavy strength training on W_{max} is in conflict with previous studies from our group in well-trained to elite male cyclists performing the same strength-training program as in the current study (Rønnestad et al., 2010, 2014). The reasons for this discrepancy are unclear. One explanation may be that this study included female athletes while the aforementioned studies examined male cyclists. However, a lack of effect of strength training on W_{max} has also been reported in male cyclists after explosive type strength training (Bastiaans et al., 2001) and after a short period (6 weeks) of heavy strength training (Levin et al., 2009). In addition, increased W_{max} in double poling have been reported in female cross-country skiers after concurrent training (Hoff et al., 1999), making this explanation unlikely. VO_{2max} is an important factor determining W_{max} , but W_{max} also has a large anaerobic component (Jones & Carter, 2000). The lack of change in W_{max} can therefore indicate a lack of change in anaerobic power and/or capacity. However, our other indicator of anaerobic abilities, the Wingate test, showed improved anaerobic performance.

The finding of improved Wingate peak power output after combining heavy strength training and endurance training was expected since similar findings have been

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reported in both untrained subjects (Chromiak et al., 2004) and cyclists of various performance levels (Rønnestad et al., 2010, 2014). Because peak power often occurs during the first 5 s of an all-out sprint, the size of the involved muscle mass and maximal leg strength is one of the main determinates of peak power output (Izquierdo et al., 2004; Van Praagh, 2007). The increase in peak power during the Wingate test in the E+S group might therefore be explained by the increase in quadriceps CSA and leg strength.

The strength-training intervention also increased the mean power output during the Wingate test, which was not seen in the control cyclists. This is in accordance with a study showing that 9 weeks of explosive strength training prevented a decrease in mean power output during a modified Wingate test in male cyclists (Bastiaans et al., 2001), and in studies with untrained individuals after periods of heavy strength training (Chromiak et al., 2004). The dominant energy source during the Wingate test is anaerobic lactic acid metabolism (Beneke et al., 2002). Since strength training has the potential to increase glycolytic enzyme activity and augment intracellular fuel stores of ATP and phosphocreatine (Gravelle & Blessing, 2000), this is a likely mechanism for the increased mean power during the Wingate test.

Strength, muscle CSA, and muscle fiber type composition

The observed 39% increase in leg strength (1RM in leg press) is in accordance with the previously observed 28% to 40% increase in 1RM in endurance athletes adding 8–12 weeks of heavy strength training to their normal endurance training (e.g., Bishop et al., 1999; Levin et al., 2009; Barrett-O'Keefe et al., 2012). Likewise, the 7.4% increase in quadriceps CSA seen in the present study is in accordance with previous reports of increases in lean mass of the legs or increases in CSA of thigh muscles after similar strength-training programs in well-trained and elite male cyclists (Rønnestad et al., 2010, 2014). Some of the improved leg strength seen in E+S in the present study is likely due to the increased quadriceps CSA.

The 11 weeks of strength training reduced the proportion of type IIAX-IIIX fibers and increased the proportion of type IIA fibers to such a degree that no fibers were positive for the type IIX antibody after the intervention. This finding is in accordance with a previous study in elite male cyclists (Aagaard et al., 2011) and previous studies in untrained subjects (Staron et al., 1994; Kraemer et al., 1995; Andersen & Aagaard, 2000).

Aerobic enzymes

There were no changes in the concentration of aerobic enzymes in *m. vastus lateralis* during the course of the study in neither E+S nor E. To investigate possible

effects on the three major pathways of aerobic metabolism, we measured changes in CS, COX4, and HADH as markers for the Krebs cycle, oxidative phosphorylation, and beta-oxidation, respectively. The findings of no effect of concurrent training compared with endurance training alone are not surprising considering data from the majority of previous studies. For example, in untrained subjects, concurrent training has led to similar gains in CS activity when compared with endurance training alone (Sale et al., 1990; Bell et al., 2000). Similarly, studies in endurance athletes adding a period of strength training to their normal training have reported change in the content or activity of the aerobic enzymes (Hickson et al., 1988; Bishop et al., 1999).

The results from the present study demonstrated that adding heavy strength training to endurance training in well-trained female cyclists leads to improvements in cycling performance and cycling economy. This is the first study to show that concurrent training improves fractional utilization of VO_{2max} . The main mechanisms behind these improvements seem to be increased muscle CSA and fiber type shifts from type IIX toward type IIA in the main propulsive muscles.

Perspectives

Based on the results from the current study, female cyclists can be recommended to include heavy strength training in their training programs for maximal gains in cycling performance. The present finding is in accordance with previous findings in moderately trained to elite male cyclists (Sunde et al., 2010; Rønnestad et al., 2010, 2014; Aagaard et al., 2011). This is however not in accordance with the only previous study in female cyclists (Bishop et al., 1999). However, the strength training volume in that study may have been too low to enhance cycling performance. These results also confirm that heavy strength training has the potential to improve cycling economy as previously reported in master cyclists (Louis et al., 2012) and trained cyclists (Sunde et al., 2010; Barrett-O'Keefe et al., 2012). In addition, the present study shows that strength training may improve fractional utilization of VO_{2max} and that this can be an important mechanism behind improved cycling performance. Finally, this study indicates that muscle fiber type transition from type IIAX-IIIX to type IIA and increased muscle CSA are possible mechanisms behind the present observations of improved cycling economy and fractional utilization of VO_{2max} , hence representing the two main factors for the improvement in cycling performance induced by concurrent strength training.

Key words: Concurrent training, cycling performance, work economy, muscle fiber type composition, aerobic power.

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Supporting information

Additional Supporting Information may be found in the online version of this article at the publisher's web-site:

Fig. S1. Log₂-fold change in gene expression of protein involved in aerobic metabolism during the intervention period for cyclists adding strength training to their normal endurance training (E+S) and cyclists performing normal endurance training only (E). Values are mean ± CI

Table S1. Details of primers used for RT-qPCR

Paper II

Vikmoen O, Raastad T, Seynnes O, Bergstrøm B, Ellefsen S, Rønnestad BR. Effect of heavy strength training on running performance in female endurance athletes. *Manuscript submitted to Eur J Appl Physiol.*

**Effect of heavy strength training on running performance in female
endurance athletes**

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Short title: Strength training and running performance

Abstract

Purpose: To investigate the effects of adding strength training to normal endurance training on running performance and running economy in well-trained female athletes. **Methods:** Nineteen female endurance athletes ($\text{VO}_{2\text{max}}$: $53 \pm 3 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) were randomly assigned to either normal endurance training (E , $n=8$) or normal endurance training combined with strength training ($E+S$, $n=11$). The strength training consisted of four leg exercises [3 x 4-10 repetition maximum (RM)], twice a week for 11 weeks. **Results:** $E+S$ increased 1RM in leg exercises ($40 \pm 15\%$) and maximal jumping height in counter movement jump ($6 \pm 6\%$) and squat jump ($9 \pm 7\%$). This was accompanied by increased muscle fiber cross sectional area of both fiber type I ($13 \pm 7\%$) and fiber type II ($31 \pm 20\%$) in *m. vastus lateralis* ($p < 0.05$), with no change in capillary density or the stiffness of the patellar tendon. Both $E+S$ and E had a tendency to increased running distance during a 40 min all-out test ($E+S$: $2.0 \pm 3.0\%$, E : $2.1 \pm 2.8\%$, $p < 0.10$). Neither $E+S$ nor E changed running economy, fractional utilization of $\text{VO}_{2\text{max}}$ or $\text{VO}_{2\text{max}}$. However, peak running velocity achieved during the $\text{VO}_{2\text{max}}$ test (V_{max}) increased in $E+S$ by $1.7 \pm 2.8\%$ with no change in E . **Conclusion:** Adding heavy strength training to endurance training did not affect 40 min all-out running performance or running economy compared to endurance training only. However, the small improvement in V_{max} may be important, especially for the result in competitions ending in a mass sprint.

Key words

Concurrent training; muscle strength; running economy; patellar tendon; tendon properties; capillarization

Abbreviations

CAF	Capillaries around each fiber
CAFA	Capillaries related to fiber area

CMJ	Counter movement jump
CSA	Cross sectional area
E	Endurance training only group
E+S	Endurance training combined with strength training group
ES	Effect size
F _{PT}	Patellar tendon force
HR	Heart rate
la ⁻	lactate
l ₀	Patellar tendon resting length
l _{max}	Patellar tendon maximal length
RM	Repetition maximum
RPE	Rate of perceived exertion
SJ	Squat jump
V _{max}	Peak running performance during the VO _{2max} test
VO ₂	Oxygen consumption
VO _{2max}	Maximal oxygen consumption

Introduction

An increasing number of studies report improved running performance after different strength training protocols (Barnes et al. 2013; Damasceno et al. 2015; Hickson et al. 1988; Paavolainen et al. 1999; Sedano et al. 2013; Storen et al. 2008). However, the literature is far from conclusive, and some studies report no beneficial effect of strength training on endurance performance (Bertuzzi et al. 2013; Ferrauti et al. 2010; Kelly et al. 2008; Roschel et al. 2015).

Running performance is mainly determined by the maximal oxygen consumption (VO_{2max}), fractional utilization of VO_{2max} and running economy (Bassett and Howley 2000). Addition of strength training has neither a negative nor a positive effect on VO_{2max} (e.g. Millet et al. 2002;

Sedano et al. 2013; Storen et al. 2008). The effect of combining strength and endurance training on fractional utilization of $\text{VO}_{2\text{max}}$ has not been directly investigated, but the indirect measure of VO_2 at the lactate threshold, expressed as percent of $\text{VO}_{2\text{max}}$, seems to be unchanged (Millet et al. 2002; Storen et al. 2008). Running economy on the other hand seems to be positively affected by strength training (e.g. Guglielmo et al. 2009; Johnston et al. 1997; Millet et al. 2002; Sedano et al. 2013; Storen et al. 2008). An improved running performance following strength training is therefore suggested to be mainly related to improved running economy (Sedano et al. 2013; Storen et al. 2008).

One of the most frequent proposed mechanisms behind improved running economy after strength training is changes in the stiffness of lower limbs muscles and tendons (Guglielmo et al. 2009; Spurr et al. 2003; Storen et al. 2008). During the first part of the contact phase in the running stride, elastic energy is stored in the muscles, tendons and ligaments acting across joints (Roberts and Azizi 2011). A partial return of this stored energy during the second part of the contact phase limits the muscle energy expenditure and amplifies the mechanical output of the muscle-tendon complex (Roberts and Azizi 2011). Hence, the stiffness of series elastic component, mainly tendons, can affect both the utilization of this elastic energy and the muscle contraction mechanics during the running stride. In fact, stiffer Achilles tendons have been associated with better running economy (Arampatzis et al. 2006). Intriguingly, more compliant patellar tendons were associated with better running economy (Arampatzis et al. 2006), whereas heavy strength training has been shown to increase patellar tendon stiffness (Reeves et al. 2003; Seynnes et al. 2009). A more compliant patellar tendon may indeed allow the muscle to operate at mechanically efficient lengths and velocities during the contact phase (Arampatzis et al. 2006). However, for a given tendon stiffness a stronger muscle would enable larger energy storage. Consequently, heavy strength training might induce changes in muscle and tendon properties with both potential beneficial and negative effects on running

economy. It is therefore important to gain insight into the effects of strength training on patellar tendon mechanical properties, and if possible effects induces changes in running economy. However, to our best knowledge, no studies to date have investigated this.

Even though strength training may induce improved running performance through improved running economy it will also normally increase cross sectional area (CSA) of the muscle fibers (Folland and Williams 2007). Therefore, it can be speculated that strength training can increase diffusion distances from the capillaries to the interior of muscle cells, which will be negative for performance. Anecdotally, some endurance athletes use this as an argument against strength training. In untrained individuals there are reports of increased or unchanged numbers of capillaries around each muscle fiber (Bell et al. 2000; Hather et al. 1991) and no change capillaries per fiber area (Hather et al. 1991) after strength training. However, as performing endurance training concurrently with strength training may blunt the hypertrophic response (e.g. Kraemer et al. 1995), and endurance trained athletes have larger numbers of capillaries than untrained (Brodal et al. 1977; Ingjer and Brodal 1978) these findings may not apply for endurance athletes. Consequently, there is a need to look closer into the effects of combined strength and endurance training on capillarization and fiber CSA in well-trained endurance athletes.

The purpose of this study was to investigate the effects of 11 weeks of heavy strength training on running performance during a 40 min all-out test and running economy in well-trained female endurance athletes. Furthermore, we wanted to assess the effects of the strength training on the mechanical properties of the patellar tendon to elucidate whether this could be related to changes in running performance and running economy. To investigate if strength training would have any effect on capillarization in endurance athletes we measured muscle fiber CSA and capillarization in *m. vastus lateralis*.

We hypothesized that the addition of heavy strength training would result in improved 40 min all-out performance and improved running economy and that these changes would be related to changes in mechanical properties of the patellar tendon, together with no negative effect on capillarization.

Methods

Ethical approval

The study was approved by the Local Ethics Committee at Lillehammer University College. Written informed consent was obtained from all athletes prior to inclusion, and the study was carried out in accordance with the Declaration of Helsinki.

Participants

Twenty-eight female endurance athletes active in both cycling and running and that fulfilled at least two of Jeunkedrup et al.'s (2000) training and race status descriptions of a well-trained endurance athlete were recruited to this study. None of the athletes had performed systematic strength training for the last 12 months leading up to the study. The athletes were matched on VO_{2max} and randomly assigned to either adding heavy strength training to the ongoing endurance training ($E+S$, $n=14$) or endurance training only (E , $n=14$). During the study, three athletes in $E+S$ left the project for reasons unrelated to the project protocol: one because of an injury, one because of a prolonged period off illness during the last part of the intervention and one because of other medical reasons. In E , six athletes left the study for reasons unrelated to the project protocol (injuries in training, pregnancy and lack of time). Therefore, the final numbers of athletes in $E+S$ and E were 11 and 8, respectively.

Experimental overview

This study is part of a larger study investigating the effects of heavy strength training on various aspects of endurance performance. Some of the basic characteristics as anthropometrics and endurance training have been reported previously (Vikmoen et al. 2015).

The strength training program for the *E+S* group consisted of two strength training sessions per week and lasted for 11 weeks (during the competition period from April to July). Testing before and after the intervention period was performed over four test-days. During pre-tests, day one was utilized to sample muscle biopsies from the right *m. vastus lateralis*, and measure the mechanical properties of the left patellar tendon. At day two 1RM in one-legged leg press and half squat was measured. Day 3 consisted of an incremental running test for determination of blood lactate profile, a VO_{2max} test and testing of maximal squat jump (SJ) and counter movement jump (CMJ) height. Day 4 consisted of a 40 min all-out running test. There were 3-7 days between test days. In general, post-tests were performed in the same order as pre-tests. However, muscle biopsies and patellar tendon measurements were moved to the last test day.

Training

Endurance training duration and intensity were calculated based on heart rate (HR) recordings. Endurance training was divided into three HR zones: 1) 60%-82%, 2) 83%-87%, and 3) 88%-100% of maximal HR. The endurance training performed has been previously reported (Vikmoen et al. 2015). In brief, there were no significant differences between groups in the mean weekly duration of the endurance training and the distribution of this training within the three intensity zones (across groups values were: zone 1: 3.7 ± 1.6 h, zone 2: 1.1 ± 0.5 h, zone 3: 0.8 ± 0.5).

The heavy strength training sessions for the *E+S* groups targeted leg muscles and were performed twice per week during the 11-week intervention period. Adherence to the strength

training was high, with *E+S* athletes completing 21.4 ± 1.0 (range 19-22) of the planned 22 strength-training sessions. The strength-training program was performed as reported in Vikmoen et al. (2015). Briefly, each strength training session consisted of four leg exercises: half squat in a smith machine, leg press with one leg at a time, standing one-legged hip flexion, and ankle plantar flexion. Three sets were performed *per* exercise. An investigator supervised the athletes at all workouts during the first two weeks and at least one workout per week thereafter. During weeks one to three, athletes trained with 10RM sets at the first session and 6RM sets at the second session. These alternating loads were adjusted to 8RM and 5RM during weeks four to six, and was further adjusted to 6RM and 4RM during weeks seven to eleven. The athletes were encouraged to increase their RM loads continually throughout the intervention period and they were allowed assistance on the last repetition.

Strength, jumping and running tests

The athletes were instructed to refrain from intense exercise the day preceding testing, to prepare for the tests as they would have done for a competition, and to consume the same type of meal before each test. Running tests was performed on a motor driven treadmill (Woodway Desmo Evo, Waukesha, Wisconsin, USA). The inclination of the treadmill was set to 5.3% at all tests. All testing were performed under similar environmental conditions (18-20°C).

1RM tests

1RM was tested in one-legged leg press and half squat and the mean value from these two exercises were used for statistical analyses. Prior to the testing day, each athlete was given a supervised familiarization session to learn proper lifting technique and find individual equipment settings. During this session, the load was gradually increased to allow estimation of a proper starting point for the 1RM testing.

The 1RM tests in both exercises was performed as previously described (Vikmoen et al. 2015). Briefly, testing started with a specific warm-up, consisting of three sets with gradually increasing load (40, 75 and 85% of expected 1RM) and decreasing number of repetitions (10→6→3). The first attempt was performed with a load approximately 5% below the expected 1RM. If a lift was successful, the load was increased by approximately 5%. The test was terminated when the athletes failed to lift the load in 2-3 attempts and the highest successful load lifted was noted as 1RM. Athletes were given 3 min rest between lifts.

Blood lactate profile

The blood lactate profile tests started with 5 min running at 7 km·h⁻¹, which was subsequently increased every 5 min by 1 km·h⁻¹. Between consecutive 5 min bouts there was a 1 min break, wherein blood was sampled from a finger-tip and analyzed for whole blood lactate concentration ([La⁻]) using a Lactate Pro LT-1710 analyzer (Arcray Inc., Kyoto, Japan), and the rate of perceived exertion (RPE) was recorded. The test was terminated when a [La⁻] of 4 mmol·L⁻¹ or higher was measured. VO₂ and HR were measured during the last 3 min of each bout, and mean values were used for statistical analysis. VO₂ was measured (30 s sampling time) using a computerized metabolic system with mixing chamber (Oxycon Pro, Erich Jaeger, Hoechberg, Germany). The gas analyzers were calibrated with certified calibration gases of known concentrations before every test. The flow turbine (Triple V, Erich Jaeger, Hoechberg, Germany) was calibrated before every test with a 3 l, 5530 series, calibration syringe (Hans Rudolph, Kansas City, USA). HR was recorded using a Polar S610i heart rate monitor (Polar, Kempele, Finland). From this incremental running test, the running velocity at 3.5 mmol·L⁻¹ [La⁻] was calculated for each athlete from the relationship between [La⁻] and running velocity using linear regression between data points. Running economy was determined by the mean VO₂ at a running velocity of 10 km·h⁻¹.

VO_{2max}

After termination of the blood lactate profile test the athletes ran for 10 min at a freely chosen submaximal workload. The VO_{2max} test was then initiated with 1 min running at 8 km·h⁻¹ and the speed was increased by 1 km·h⁻¹ every minute until exhaustion. The athletes received strong verbal encouragement to run for as long as possible. VO₂ was measured continuously, and VO_{2max} was calculated as the mean of the two highest 30 s VO₂ measurements. Peak running performance during the test (V_{max}) was calculated as the mean running velocity during the last 2 min of the incremental test. After the test blood [La⁻], HR_{peak} and RPE was noted.

SJ and CMJ

Twenty min after termination of the VO_{2max} test, explosive strength was tested as maximal jumping height in SJ and CMJ. These jumps were performed on a force plate (SG-9, Advanced Mechanical Technologies, Newton, MA, USA, sampling frequency of 1kHz). After 3-5 submaximal warm up jumps, the athletes performed three SJ and three CMJ with 2-3 min rest between jumps. The highest SJ and CMJ were utilized for statistical analyses. During all jumps the athletes were instructed to keep their hands placed on their hips and aim for maximal jumping height. The SJ was performed from ~90 degrees knee angle. In this position, they paused for 3 s before jumping. No downward movement was allowed prior to the jump and the force curves were inspected to verify this. During the eccentric phase of the CMJ the athletes were instructed to turn at a knee angle they felt was optimal for achieving maximal jumping height.

40 min all-out test

Prior to the 40 min all-out test, athletes performed 10 min warm up at self-selected submaximal velocities, containing three submaximal sprints performed during the last 2 min.

During the first 5 min of the test, the investigators set the velocity. This individual selected velocity was based on the lactate profile test and corresponded to the velocity at 2.5 mmol·L⁻¹ [la⁻]. Thereafter, running velocity were controlled by the athletes themselves, with instructions to run as long as possible during the 40 min. Measurements of VO₂ was made during the last minute of every 5 min section, to allow estimation of performance VO₂ and fractional utilization of VO_{2max}. During this minute, athletes were not allowed to adjust the running velocity. The mean VO₂ during this minute was estimated to reflect the mean VO₂ during the corresponding 5 min section. During the last 5 min of the test, VO₂ was measured continuously as pilot testing showed that athletes performed numerous velocity adjustments during this part of the test. Performance VO₂ was calculated as the mean VO₂ of all 5 min sections, and fractional utilization of VO_{2max} was calculated as performance VO₂ in percentage of VO_{2max}. During the test, the athletes were allowed to drink water *ad libitum*.

Measurements of the mechanical and material properties of the patellar tendon

All the measurements of the mechanical and material properties of the patellar tendon were performed on the left leg and were done as previously described (Helland et al. 2013). Briefly, the athletes were seated with a 90° angle in both knee and hip joint in a knee extension apparatus (Knee extension, Gym 2000, Geithus, Norway) instrumented with a force cell (U2A, Hottinger Baldwin Messtechnik GmbH, Darmstadt, Germany). To measure patellar tendon CSA, transversal scans were performed proximally, medially and distally along the tendon length using an B-mode ultrasound apparatus (HD11XE, Phillips, Bothell, WA, USA). Sagittal scanning was used to measure tendon length. To measure tendon force and elongation the ultrasound probe was attached to the left knee with a custom-made device. The athletes performed ramp contractions at a constant rate of 100 N·s⁻¹. To correct for hamstring co-activation when calculating tendon force (see below), a maximal isometric knee flexion were performed after the knee extension test. In addition, EMG data were recorded (TeleMyo 2400

G2 telemetry Systems, Noraxon Inc., Scottsdale, AZ, USA) from the biceps femoris muscle during isometric knee extension and flexion. Patellar tendon force (F_{PT}) was calculated as the force measured in the force cell, corrected for hamstring co-activation, internal and external moment arms as follows:

$$F_{PT} = ((F_q + F_h)M_e) / M_i$$

Where F_q is force measured by the force cell, F_h is estimated hamstrings co-activation force, M_i and M_e corresponds to internal and external moment arm respectively.

Tendon morphology data were analysed as previously described (Helland et al. 2013), using an image analysis software (ImageJ 1.45s, National Institute of Health, Austin, TE, USA).

Tendon elongation data were analyzed using a video analysis software (Tracker Video Analysis and Modeling Tool, Open Source Physics, Douglas Brown, 2012). The patellar apex and the tibia plateau were digitally marked within a common coordinate system. The actual elongation of the tendon was calculated as the change in the distance between coordinates of these anatomical landmarks. To calculate tendon material and mechanical properties force-elongation curves were fitted with a 2nd degree polynomial. All the recordings used in the results had a fit of $R^2 = 0,92$ or higher. Stiffness was calculated as the slope of the force – elongation curve, between 90 and 100% of each athlete's maximal force. The Young's modulus was calculated by multiplying the stiffness values by the ratio between the patellar tendon resting length (l_0) and mean CSA. Patellar tendon l_0 and maximal length (l_{max}) was used to calculate the patellar tendon strain. Two sets of ultrasound data (from two *E+S* athletes) had to be discarded because of an insufficient quality to enable analysis. Therefore, the number of athletes included in the data from tendon testing is 9 in *E+S* and 8 in *E*.

Muscle biopsy sampling

Muscle biopsies were sampled from *m. vastus lateralis* using the Bergström procedure.

Athletes were instructed to refrain from physical activity for the last 24h leading up to biopsy sampling. During each biopsy sampling-event, two separate muscle biopsies were retrieved and pooled in a petri dish filled with sterile physiological salt water. An appropriately sized muscle sample (mean wet weight: 29 ± 8 mg) was selected for immunohistochemical analyses and mounted in Tissue-Tek (Sakura Finetek USA, Inc., Torrance, CA, USA) and quickly frozen in isopentane cooled on liquid nitrogen. Muscle samples were stored at -80°C until later analyses.

Immunohistochemistry

Cross-sections $8\ \mu\text{m}$ thick were cut using a microtome at -20°C (CM3050; Leica Microsystems GmbH, Wetzlar, Germany) and mounted on microscope slides (Superfrost Plus; Thermo Fisher Scientific, Inc., Waltham, MA, USA). The sections were then air-dried and stored at -80°C . Prior to antibody labelling, muscle sections were blocked in a solution containing 1% BSA (cat. no. A4503; Sigma-Aldrich Corp., St Louis, MO, USA) and 0.05% PBS-T solution (cat. no. 524650; Calbiochem, EMD Biosciences, Inc., San Diego, CA, USA) for 30 min. Then they were incubated overnight at 4°C with antibodies against the capillary marker CD31 (1:200; clone JC70A, M0823; Dako A/S, Glostrup, Denmark), followed by incubation with appropriate secondary antibodies (Alexa Fluor, cat. no. A11005).

Following staining, muscle sections were visualized and pictures taken using a high-resolution camera (DP72; Olympus Corp., Tokyo, Japan) mounted on a microscope (BX61; Olympus Corp.) with a fluorescence light source (X-Cite 120PCQ; EXFO Photonic Solutions Inc., Mississauga, Ontario, Canada).

These muscle sections were then incubated for 1 hour at room temperature with antibodies against myosin heavy chain type II (1:1000; SC71; gift from Professor S. Schiaffino) and

dystrophin (1:1000; cat. no. ab15277; Abcam Plc), followed by incubation with appropriate secondary antibodies (Alexa Fluor, cat. no. A11005 or A11001; Invitrogen, Inc.).

Muscle sections were then covered with a coverslip and glued with ProLong Gold Antifade Reagent with DAPI (cat.no. P36935; Invitrogen Molecular Probes, Eugene, OR, USA) and left to dry overnight at room temperature. Muscle sections were again visualized and new pictures were taken at the exactly same location in the section as the CD31 picture. Between all stages, sections were washed for 3×5 min using a 0.05% PBS-T solution.

Fiber type distribution, fiber cross-sectional area and capillaries were identified using TEMA software (CheckVision, Hadsund, Denmark). Capillarisation was expressed as capillaries around each fiber (CAF) and capillaries related to fiber area (CAFA), for type I and type II (IIA and IIX) fibers. Because of technical problems with some analyses, the number of athletes in the immunohistochemistry data is 8 in *E+S* and 5 in *E*.

Statistical analyses

All data in the text, figures and tables are presented as mean \pm standard deviation, unless otherwise stated. To test for differences between groups at pre, post and differences in changes from pre to post, unpaired students t-test were used. Within-group analyses were performed using paired t-tests. Effect sizes (ES) were calculated for key performance and physiological adaptations to elucidate on the practical significance of strength training. ES were calculated as Cohen's *d* and the criteria to interpret the magnitude were the following: 0-0.2 = trivial, 0.2-0.6 = small, 0.6-1.2 = moderate, 1.2-2.0 = large and > 2 = very large (Hopkins et al. 2009).

Correlations analyses were done using the Pearson product-moment method. Analyses was performed in Excel 2013 (Microsoft Corporation, Redmon, WA, USA). Analyses resulting in $p \leq 0.05$ were considered statistically significant. $P \leq 0.10$ were considered as tendencies.

Results

There were no significant differences between *E+S* and *E* in any of the measured variables at baseline.

Body mass, maximal leg strength and muscle fiber area

Body mass remained unchanged in *E+S* (from 62.4 ± 5.2 kg to 63.1 ± 5.6 kg) but was slightly reduced in *E* (from 65.6 ± 8.4 kg to 64.8 ± 8.0 kg, $p < 0.05$). The change in body mass was different between the groups ($p < 0.05$).

1RM in the leg exercises increased $40.4 \pm 14.7\%$ in *E+S* ($p < 0.01$, Fig. 1) and $4.5 \pm 5.3\%$ ($p < 0.05$) in *E*. The change in 1RM was larger in *E+S* than in *E* ($p < 0.01$), and effect size analyses revealed a very large practical effect of *E+S* compared to *E* ($ES = 3.20$).

In *E+S*, CSA of both type I and type II muscle fibers increased in *m. vastus lateralis* ($13.2 \pm 6.8\%$ and $30.8 \pm 19.6\%$, respectively, $p < 0.01$), with no changes occurring in *E* (Fig. 2).

Although this did not amount to a statistical difference between groups, *E+S* had a moderate practical effect on muscle fiber CSA compared to *E* ($ES = 0.83$).

Insert Fig. 1 around here

Insert Fig. 2 around here

Capillarization

In *E+S* there were no changes in capillarization around type I or type II fibers expressed as CAF or CAFA (Fig. 3). In *E* there were no changes in capillarization in type I fibers neither expressed as CAF or CAFA, whereas in type II fibers a tendency to a reduction in both CAF ($-9.6 \pm 7.7\%$, $p = 0.06$) and CAFA ($-22.5 \pm 16.21\%$, $p = 0.09$) were observed.

Insert Fig.3 around here

SJ and CMJ

$E+S$ increased SJ and CMJ height by $8.9 \pm 6.8\%$ and $5.9 \pm 6.0\%$ respectively ($p < 0.05$) while no changes occurred in E (Fig. 1). The change in SJ was larger in $E+S$ than in E ($p < 0.05$).

The effect size analyses revealed a moderate practical effect in favor of $E+S$ in both SJ (ES = 1.06) and CMJ (ES = 0.65).

Mechanical and material properties of the patellar tendon

There were no significant changes in stiffness or Young's modulus of the patellar tendon in neither $E+S$ nor E (Table 1). The mean CSA of the patellar tendon increased by $5.2 \pm 3.6\%$ in $E+S$ while no significant changes occurred in E (Table 1).

Insert table1 around here

VO_{2max} and V_{max}

VO_{2max} was unchanged in both groups during the intervention period (Table 2). However, V_{max} increased in $E+S$ by $1.7 \pm 2.8\%$ ($p = 0.05$) with no change in E . However, no difference was found between the two groups and the practical effect of $E+S$ was small (ES = 0.22).

Insert Table 2 around here

Running economy and running velocity at 3.5 mmol·L⁻¹ [la⁻¹]

There were no changes in running economy measured at 10 km·h⁻¹ during the blood lactate profile test (Fig. 4) or running velocity at 3.5 mmol·L⁻¹ [la⁻¹] (Fig. 4) in neither of the groups.

40 min all-out test

Running distance during the 40 min all-out test tended to increase in both groups during the intervention ($E+S$: $2.0 \pm 3.0\%$, $p = 0.06$, E : $2.1 \pm 2.8\%$, $p = 0.07$, Fig. 4), with no difference in changes between the groups ($p = 0.92$). The performance VO₂ during the 40 min all-out test

did not change in either of the groups (Fig. 4). Fractional utilization of $\text{VO}_{2\text{max}}$ did not change in $E+S$ (from 85.3 ± 3.9 to 85.3 ± 4.3 , Fig. 4), but increased in E , going from $83.2 \pm 3.1\%$ to $86.0 \pm 3.0\%$ ($p < 0.05$).

Before the intervention the performance in the 40 min all-out test correlated with velocity at $3.5 \text{ mmol}\cdot\text{L}^{-1} [\text{la}^-]$, $\text{VO}_{2\text{max}}$ and V_{max} ($r = 0.65$, $r = 0.58$, $r = 0.79$, respectively), but not with running economy ($r = -0.24$). No significant correlations between changes in these variables and changes in 40 min all-out running distance were observed.

Insert fig. 4 around here

Discussion

The main results from the current study were that adding heavy strength training to well-trained female athletes' normal endurance training did not affect the mechanical properties of the patellar tendon or running economy. Furthermore, there was no effect on running performance during a 40 min all-out running test. However, strength training improved maximal running performance measured as V_{max} and had no negative effect on capillary density despite increase in muscle fiber CSA and muscle strength.

Maximal strength and muscle fiber cross sectional area

The strength-training program used in the current study was effective in increasing maximal leg strength as showed by the increase in 1RM in the leg exercises. This is in accordance with previously observed increases in 1RM in endurance athletes adding heavy strength training to their normal endurance training (e.g. Hickson et al. 1988; Johnston et al. 1997; Storen et al. 2008). The results from the current study confirms previous results (Rønnestad et al. 2010; Storen et al. 2008) that a quite large increase in muscular strength can be achieved without an increased body mass. This is important for runners since increased body mass can negatively influence running performance. In spite of this, the improved strength seemed to be at least

partially dependent on increases in muscle mass, as evident from the increase in muscle fiber CSA. The present muscle hypertrophy is in agreement with other studies using similar strength training protocols in endurance athletes (Aagaard et al. 2011; Ronnestad et al. 2010; Taipale et al. 2010). Interestingly, there were no difference in the CSA of the type I and type II fibers in the current athletes, confirming the notion that in endurance athletes the type I fibers may be just as large (Sjogaard 1984) or even larger (Costill et al. 1976) than the type II fibers.

SJ and CMJ

The current strength training protocol was also effective in increasing leg muscle power, as evident from the increased SJ and CMJ performance. This is in line with previous reports of effects of heavy strength training on jumping ability in untrained participants (Tricoli et al. 2005; Wilson et al. 1993). However, previous data from endurance athletes are more unclear, as some studies report improved jumping performance (Ronnestad et al. 2008; Taipale et al. 2010) whereas others do not (Francesca et al. 2012; Guglielmo et al. 2009). The current study indicate that quite large improvements in jumping ability and explosive strength can be achieved with heavy strength training despite concurrently performing endurance training.

Capillarization

Eleven weeks of heavy strength training did not affect capillarization expressed as either CAF or CAFA, despite leading to significant muscle fiber hypertrophy. Importantly, this suggest that the potentially negative effect of increased muscle fiber CSA on diffusion distances between blood and inner parts of muscle fibers was counteracted by a non-significant increase in CAF. Surprisingly, the only sign of change in capillarization was in *E*, where a tendency to both reduced CAF and CAFA in the type II fibers was observed. However, because of the low numbers of athletes included in the analyses in *E* (n=5) these results should be interpreted

with caution. In line with our finding, previous studies in untrained participants have reported either no change or a slight increase in CAF (Bell et al. 2000; Hather et al. 1991) and no change in CAFA (Hather et al. 1991) after a period of heavy strength training. Our finding is also in agreement with results reported in elite male cyclists after 16 weeks of heavy strength training (Aagaard et al. 2011). Therefore, it seems like endurance athletes should not be afraid of reduced capillarization when they consider adding heavy strength training to their ongoing endurance training.

Mechanical properties of the patellar tendon

The lack of changes in mechanical properties of the patellar tendon following heavy strength training is in contrast to most studies, typically reporting increased patellar tendon stiffness, at least in previously untrained participants (Kongsgaard et al. 2007; Kubo et al. 2001; Kubo et al. 2006a; Seynnes et al. 2009). A possible reason for the different results can be that our study involved female participants while most previous studies involves males (Kongsgaard et al. 2007; Kubo et al. 2001; Kubo et al. 2006a; Seynnes et al. 2009). In fact, female tendons have been reported to show a lower rate of new connective tissue formation in response to mechanical loading (Magnusson et al. 2007). Differences in the strength training protocol may also explain the lack of changes in the current study. Indeed, most previous studies reporting increased patellar tendon stiffness following strength training have included heavy knee extension exercise (Kongsgaard et al. 2007; Kubo et al. 2006a; Seynnes et al. 2009) or isometric muscle actions (Kubo et al. 2006b). In the current study, the exercises involved were more complex involving multiple joints that perhaps reduced the absolute mechanical loading on the patellar tendon compared to a pure knee extension exercise. In addition, the athletes were instructed to perform the concentric phase of the exercises as fast as possible making the time under tension quite low.

In contrast to the lack of effect of strength training on patellar tendon properties, it led to increases in its CSA. In line with these findings, some studies on the effect of strength training, yet not all (Reeves et al. 2003), reports an increase in patellar tendon CSA (Kongsgaard et al. 2007; Seynnes et al. 2009). Without changes in mechanical properties, the tendon hypertrophy measured here suggests that material properties may also have been altered. The lack of change in Young's modulus following training may highlight the limitation of this parameter based on finite tendon sections to reflect whole tendon material properties. Interpreting the mechanisms driving tendon hypertrophy extends beyond the scope of the present article. One could speculate that increasing tendon CSA may shield this tissue against damage caused by excessive and/or unusual stresses. Taken together, the present measurements indicate that resistance training triggers an adaptive response in the patellar tendon of female runners, without affecting the mechanical properties of this tissue.

VO_{2max}, fractional utilization of VO_{2max}, running velocity at 3.5 mmol·L⁻¹ blood [La⁻], and running economy

No effect on VO_{2max} after strength training is not surprising and in accordance with the current literature (e.g. Millet et al. 2002; Saunders et al. 2006; Sedano et al. 2013). Fractional utilization of VO_{2max} measured during the 40 min all-out test did not change in *E+S* during the course of the study. To our knowledge, this is the first study directly measuring fractional utilization of VO_{2max} in running after addition of heavy strength training in endurance athletes. However, VO₂ at lactate threshold in percentage of VO_{2max}, is often taken as an indirect measure of fractional utilization of VO_{2max} (Bassett and Howley 2000). The few studies measuring this variable in running reports no effect after addition of heavy strength training (Millet et al. 2002; Storen et al. 2008). Notably, there was a slight increase in fractional utilization of VO_{2max} in *E* over the course of the intervention. This was likely due to a combination of two factors; a small but non-significant reduction in VO_{2max}, largely due to

one athlete exhibiting a large reduction, and a small but non-significant increase in performance VO_2 .

Surprisingly, we found no effect of heavy strength training on running economy, contrasting the majority of previous studies, typically reporting improvements from 3-8% (Guglielmo et al. 2009; Johnston et al. 1997; Sedano et al. 2013; Storen et al. 2008). However, some studies supports the lack of an effect of strength training on running economy (Damasceno et al. 2015; Ferrauti et al. 2010; Mikkola et al. 2007; Roschel et al. 2015). In two of these studies (Ferrauti et al. 2010; Mikkola et al. 2007) the lack of improved running economy might be because the strength training program only consisted of one session for the legs per week.

Supporting the lack of an effect on $\text{VO}_{2\text{max}}$, fractional utilization of $\text{VO}_{2\text{max}}$ and running economy, strength training had no effect on running velocity at 3.5 $\text{mmol}\cdot\text{L}^{-1}$ blood $[\text{La}^-]$. The latter is in accordance with most parts of the current literature which reports no change in velocity at a certain blood $[\text{La}^-]$ or ventilatory threshold after adding various forms of strength training to runners normal training (Ferrauti et al. 2010; Millet et al. 2002; Storen et al. 2008), although exceptions exists (Guglielmo et al. 2009). This is quite surprising considering that improved running economy in theory should affect the running speed at a certain lactate threshold (Bassett and Howley 2000).

Running performance

The lack of changes in 40 min all-out performance is not in line with many of the studies in this area where improved running performance have been reported (Barnes et al. 2013; Damasceno et al. 2015; Hickson et al. 1988; Sedano et al. 2013; Storen et al. 2008). However, this is in line with the present lack of changes in the important performance determining factors like $\text{VO}_{2\text{max}}$, running economy and fractional utilization of $\text{VO}_{2\text{max}}$. Since strength training does not affect $\text{VO}_{2\text{max}}$ and the fractional utilization of $\text{VO}_{2\text{max}}$, the mechanism for the

previous observations of improved running performance seems to be improved running economy (Sedano et al. 2013; Storen et al. 2008). However, not all studies have found strength training to be beneficial for running performance (Ferrauti et al. 2010; Kelly et al. 2008; Mikkola et al. 2007; Roschel et al. 2015), and are in accordance with the current study. Interestingly, these studies do also report no improvements in running economy. Therefore, the lack of improved running performance in the current study is probably because of no changes in running economy.

The reason for the different findings in the current study compared to the majority of previous studies regarding running economy and hence performance is unclear. However, it may relate to some methodological differences. In the current study, all running tests were performed at 5.3% inclination. This inclination resulted in a quite low running velocity compared to some other studies. Indeed, changes in running economy after strength training have previously been found to be related to running velocity (Saunders et al. 2006). However, improvements in running economy after strength training have also been reported at similar velocities (Francesca et al. 2012; Taipale et al. 2010) and at the same inclination (Hoff and Helgerud 2003) used in the current study. Therefore, the inclination used is probably not the only explanation why no changes in running economy and performance were observed.

One of the most frequent proposed mechanism for the possible ergogenic effect of strength training on running economy is changes in stiffness of the muscle or tendons in the legs (Guglielmo et al. 2009; Storen et al. 2008). Despite of this, no previous study has investigated the effect of heavy strength training on patellar tendon mechanical properties measured together with changes in running economy. In the current study, the unchanged tendon stiffness and the increased strength suggest that more elastic energy may be stored in the patellar tendon during the stance phase, amplifying muscular power output and efficiency. However, the lack of changes in running economy do not support this hypothesis, and

conclusion cannot be drawn regarding the influence of patellar tendon properties in the present study.

V_{\max} have been shown to correlate well with running performance in distances from 1500 m up to the marathon (Hill and Rowell 1996; Noakes et al. 1990). In fact, V_{\max} was reported to be the best laboratory measure to predict performance in various running distances (Noakes et al. 1990) and can actually be considered a measure of running performance (Hill and Rowell 1996). The ~ 2% improvement in V_{\max} observed in $E+S$ with no change in E was small, but above the ~ 1% threshold suggested as a meaningful improvement in athletic performance (Hopkins 2004). Increased V_{\max} after heavy strength training in trained runners are in agreement with existing literature (Sedano et al. 2013; Taipale et al. 2010), though not conclusively (Guglielmo et al. 2009). $VO_{2\max}$ is an important factor determining V_{\max} , but V_{\max} is also determined by anaerobic capacity (Hill and Rowell 1996). Since there were no changes in $VO_{2\max}$ the main reason for improved V_{\max} is probably improved anaerobic capacity. Muscle mass is an important factor determining anaerobic capacity (Bangsbo et al. 1993), and with increased muscle fiber area in $E+S$ it is reasonable to assume that the muscle mass in the legs also increased. Furthermore, strength training can also contribute to increased anaerobic capacity by increased glycolytic enzyme activity and augmented intracellular stores of ATP and phosphocreatine (Gravelle 2000). Improved anaerobic capacity will also be an important factor in the decisive part of some running competitions for example during sprint finishes.

Conclusion

In contrast to our hypothesis, adding heavy strength training to endurance training in well-trained female endurance athletes did not affect running performance measured as running distance during a 40 min all-out test. The lack of effect on performance was probably because

the strength training intervention did not improve running economy or changed the mechanical properties of the patellar tendon. However, the improved V_{\max} indicates improved anaerobic capacity that can be important in certain part of running competitions. In addition, strength training had no negative effect capillary density.

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Conflict of interest

None declared.

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Figure legends

Fig. 1 Individual values (dotted lines) and mean values (solid lines) before (Pre) and after (Post) the intervention period for athletes adding strength training to their normal endurance training (*E+S*) and athletes performing normal endurance training only (*E*). a: Squat jump (SJ) height. b: Counter movement jump (CMJ). c: Mean 1 repetition maximum (1RM) in half-squat and one-legged leg press (leg exercises). * Different from pre ($p < 0.05$), # the percent change from pre to post is different in *E+S* than in *E* ($p < 0.05$)

Fig. 2 Individual values (dotted lines) and mean values (solid lines) before (Pre) and after (Post) the intervention period for athletes adding strength training to their normal endurance training (*E+S*, left panel) and athletes performing normal endurance training only (*E*, right

panel). Muscle fiber cross sectional area (CSA) for both type I muscle fibers and type II muscle fibers * Different from pre ($p < 0.05$)

Fig. 3 Individual values (open symbols) and mean values (solid squares) for athletes adding strength training to their normal endurance training ($E+S$) and athletes performing normal endurance training only (E). a: Percent change in capillaries around each muscle fiber (CAF) for both muscle fiber type I and muscle fiber type II for $E+S$ and E . b: Percent change in capillaries related to fiber area (CAFA) for both muscle fiber type I and muscle fiber type II for $E+S$ and E . £ tendency to different from pre ($p < 0.1$)

Fig. 4 Individual values (dotted lines) and mean values (solid lines) before (Pre) and after (Post) the intervention period for athletes adding strength training to their normal endurance training ($E+S$) and athletes performing normal endurance training only (E). a: Body mass adjusted oxygen consumption at $10 \text{ km}\cdot\text{h}^{-1}$. b: Running velocity at $3.5 \text{ mmol}\cdot\text{L}^{-1} [\text{la}^-]$ calculated during the blood lactate profile test. c: The fractional utilization of $\text{VO}_{2\text{max}}$ during the 40 min all-out test. d: Running distance during the 40 min all-out test. * Different from pre ($p < 0.05$), £ tendency to different from pre ($p < 0.1$)

Table 1 Stiffness, Young's modulus and mean cross section area (CSA) of the patellar tendon before (Pre) and after (Post) the intervention period for athletes adding strength training to their normal endurance training (*E+S*) and athletes performing normal endurance training only (*E*).

	<i>E+S</i>		<i>E</i>	
	Pre	Post	Pre	Post
Stiffness (N·mm ⁻¹)	2752 ± 402	2483 ± 733	2753 ± 947	2692 ± 697
Young's Modulus (MPa)	1038 ± 194	925 ± 162	1251 ± 296	1158 ± 273
Mean CSA (mm ²)	65.9 ± 7.1	69.2 ± 6.9*	60.3 ± 4.2	59.9 ± 4.4

Values are mean ± DS, * Different from pre (p < 0.05)

Table 2 Data from the maximal oxygen consumption (VO_{2max}) test before (Pre) and after (Post) the intervention period for athletes adding strength training to their normal endurance training (*E+S*) and athletes performing normal endurance training only (*E*).

	<i>E+S</i>		<i>E</i>	
	Pre	Post	Pre	Post
VO _{2max} (ml·kg ⁻¹ ·min ⁻¹)	52.2 ± 2.3	52.7 ± 3.3	54.2 ± 2.9	53.1 ± 1.9
V _{max} (km·h ⁻¹)	12.8 ± 0.7	13.0 ± 0.9*	13.1 ± 0.5	13.3 ± 0.6
HR _{peak} (beats·min ⁻¹)	193 ± 9	192 ± 9	189 ± 8	187 ± 7
RPE	19 ± 1	20 ± 1	19 ± 1	19 ± 1
[La ⁻¹] _{peak} (mmol·l ⁻¹)	9.7 ± 3.0	8.1 ± 3.8	8.9 ± 2.2	7.7 ± 1.8

Values are mean ± DS, * Different from pre (p < 0.05)

Figure 1

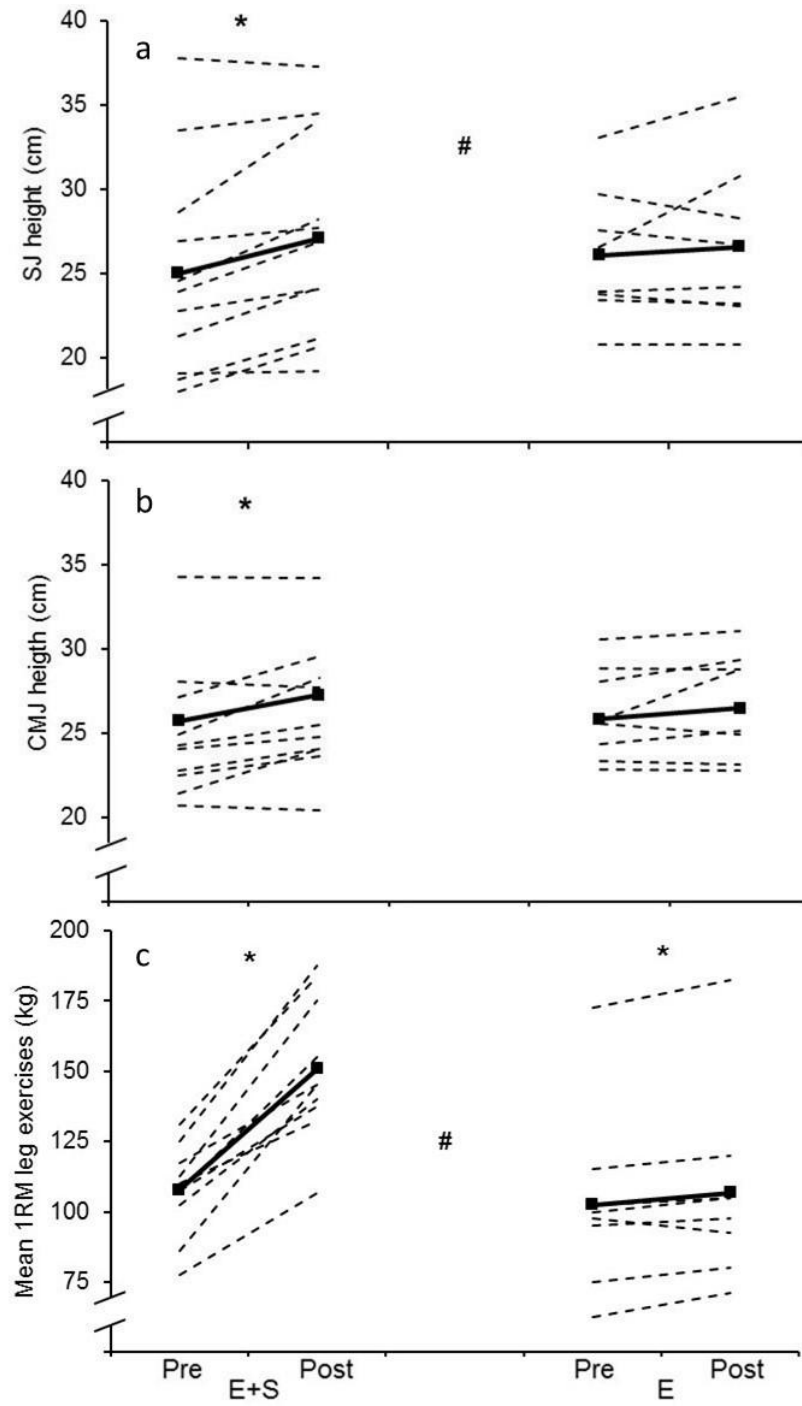


Figure 2

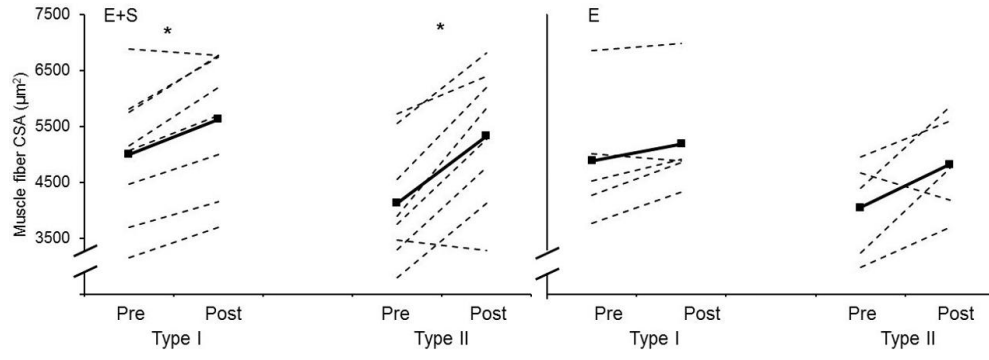


Figure 3

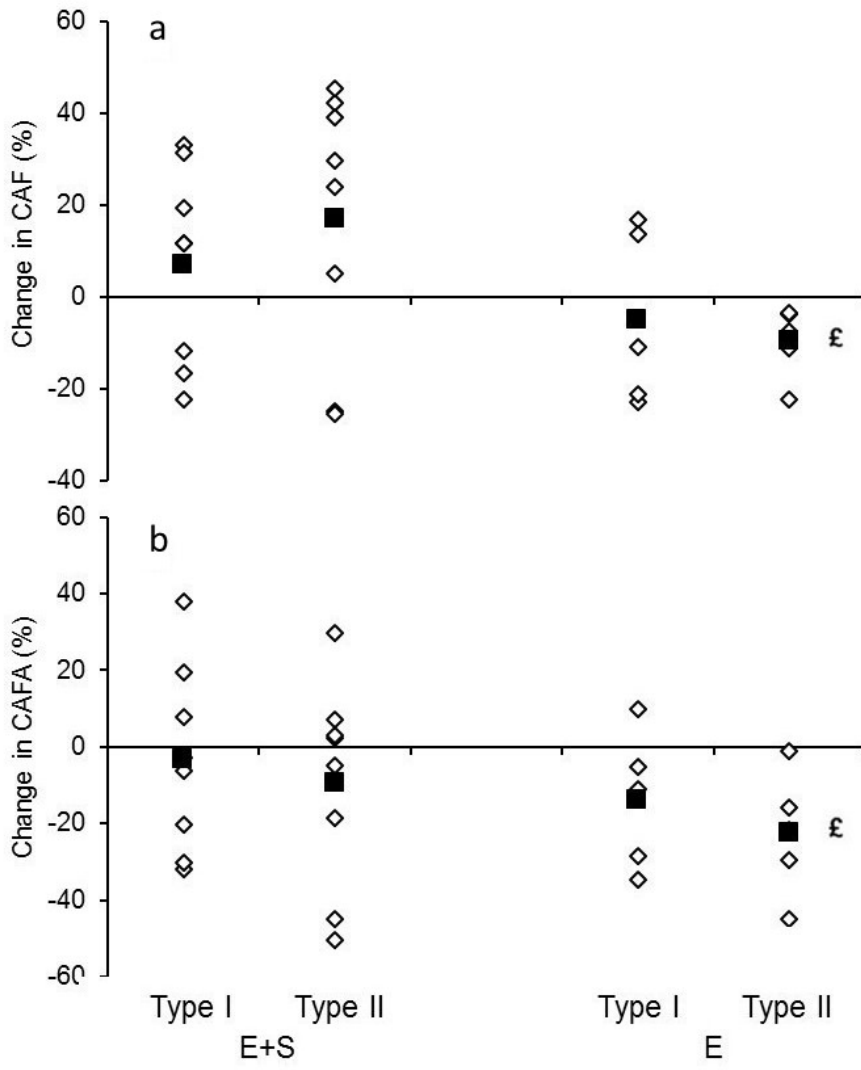
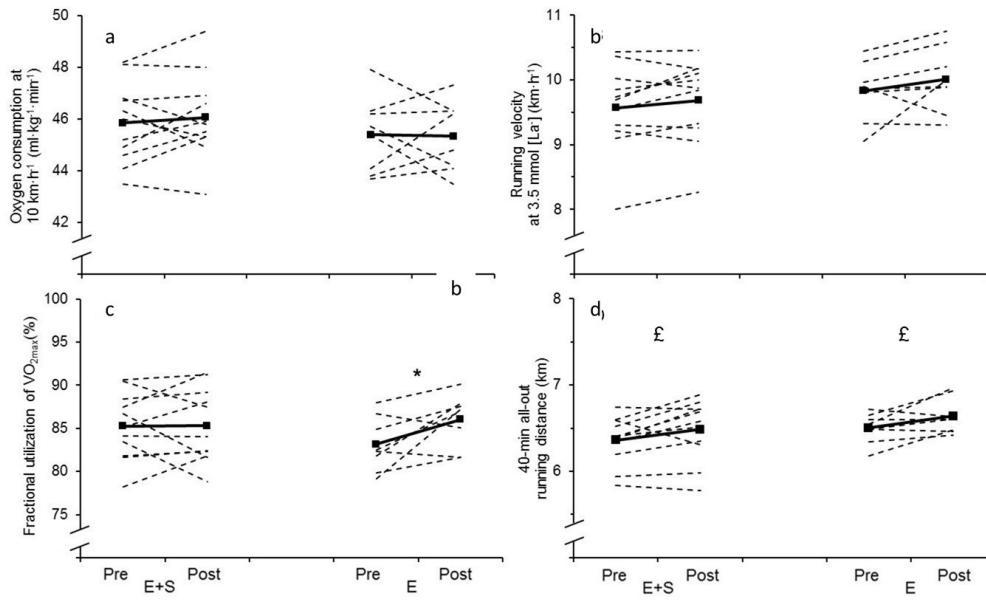


Figure 4



Paper III

Vikmoen O, Rønnestad BR, Ellefsen S, Raastad T. Heavy strength training improves running and cycling performance following prolonged submaximal work. *Manuscript*.

1 **Heavy strength training improves running and cycling performance**
2 **following prolonged submaximal work**

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8 Running head: Strength training and endurance performance

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13

14 **Abstract**

15 The purpose of the current study was to investigate the effects of adding heavy strength
16 training to female duathletes' normal endurance training on both cycling and running
17 performance. Nineteen well-trained female duathletes (VO_{2max} cycling: 54 ± 3 ml·kg⁻¹·min⁻¹,
18 VO_{2max} running: 53 ± 3 ml·kg⁻¹·min⁻¹) were randomly assigned to either normal endurance
19 training (*E*, n=8) or normal endurance training combined with strength training (*E+S*, n=11).
20 The strength training consisted of four lower body exercises [3x4-10 repetition maximum
21 (RM)] twice a week for 11 weeks. Running and cycling performance were assessed using 5
22 min all-out tests, performed immediately after prolonged periods of submaximal work (3 h
23 cycling or 1.5 h running). *E+S* increased 1RM in half-squat and lean mass in legs more than
24 *E*. Performance during the 5 min all-out test increased in both cycling and running in *E+S*
25 whereas no changes occurred in *E*. The changes in running performance were different
26 between groups. *E+S* reduced oxygen consumption and heart rate during the final 2 h of
27 prolonged cycling. No changes occurred during the prolonged running in any group. In
28 conclusion, adding strength training to normal endurance training in well-trained female
29 duathletes improved both running and cycling performance when tested immediately after
30 prolonged submaximal work.

31 **Key words:** Concurrent training; prolonged cycling; prolonged running; cycling economy;
32 running economy

33 **Introduction**

34 During the last decade, increased attention has been given the effects of adding strength
35 training to endurance athletes' normal training on running and cycling performance (e.g.
36 Aagaard et al. 2011; Paavolainen et al. 1999; Ronnestad et al. 2015; Sedano et al. 2013).
37 Improvements in performance have been reported in both running (Damasceno et al. 2015;

38 Paavolainen et al. 1999; Sedano et al. 2013; Storen et al. 2008) and cycling (Aagaard et al.
39 2011; Koninckx et al. 2010; Ronnestad et al. 2010a; Ronnestad et al. 2015). However, the
40 literature is far from conclusive, and numerous studies does not report such improvements in
41 neither running (Ferrauti et al. 2010; Roschel et al. 2015) nor cycling (Bastiaans et al. 2001;
42 Bishop et al. 1999; Levin et al. 2009). Some methodological differences may explain these
43 equivocal findings. To positively affect cycling performance, it seems that the strength
44 training regime needs to involve heavy loads, typically between 10 to 4 repetition maximum
45 (RM) (Aagaard et al. 2011; Koninckx et al. 2010; Ronnestad et al. 2010a; Ronnestad et al.
46 2015). To improve running performance on the other hand, both explosive, plyometric and
47 heavy strength training seems effective (Damasceno et al. 2015; Paavolainen et al. 1999;
48 Sedano et al. 2013). To our best knowledge, only one study have investigated the effect of
49 strength training on performance in both cycling and running in the same athletes. This study
50 reported increased time to exhaustion at VO_{2max} in both cycling and running (Hickson et al.
51 1988). However, the study did not include a control group, and therefore the results should be
52 interpreted with caution.

53 The observation that somewhat different strength training regimes affect performance in
54 cycling and running indicates that some of the performance enhancing mechanisms may differ
55 between these sports. Suggested mechanisms by which strength training can improve cycling
56 and running performance include changes in the nervous system, changes in tendon stiffness,
57 changes in movement mechanics and changes in muscular characteristics such as increased
58 muscle strength, muscle mass and improved anaerobic capacity (Ronnestad & Mujika 2014;
59 Saunders et al. 2006). Some of these factors may be important for performance in both
60 running and cycling, whereas other mechanisms may affect performance differently in these
61 sports. For example, in running the stretch shortening cycle in each stride enables the
62 possibility to store and recoil elastic energy, whereas in cycling the possibilities to take

63 advantage of stored elastic energy is negligible. Consequently, a factor such as muscle-tendon
64 stiffness may play a role for running performance, but likely not for cycling performance. On
65 the other hand, a factor like improved anaerobic capacity should affect performance to the
66 same degree in both running and cycling.

67 Road races in cycling often consist of a long initial period of cycling at a moderate intensity,
68 followed by an all-out performance at the end. Even though running competitions are ran at a
69 more even pace, they are also often decided with an all-out effort in the end. During such
70 efforts a quite large proportion of the energy demand will come from anaerobic sources
71 (Gastin 2001). Therefore, performance during a relatively short test will in addition to VO_{2max}
72 and other aerobic parameters also be largely influenced by anaerobic capacity. Muscle mass is
73 an important determinant of anaerobic capacity (Bangsbo et al. 1993). We have previously
74 reported increased CSA of *m. quadriceps femoris* after 11 weeks of heavy strength training in
75 female endurance athletes together with increased mean and peak power during the Wingate
76 test (Vikmoen et al 2015) and increased peak running velocity during a VO_{2max} test (Vikmoen
77 et al submitted). This indicates improved anaerobic capacity in the same athletes included in
78 the current study. Therefore, performance in a quite short performance test should be
79 positively affected by this strength-training regime. In addition to increased muscle CSA,
80 changes in protein levels and expression of associated genes that are involved in the anaerobic
81 processes might contribute to increased anaerobic performance.

82 Performance in an all-out effort at the end of long competitions should also be affected by the
83 fatigue developed during the competition. In Ronnestad et al. (2011) such performance was
84 simulated by 3 h of submaximal cycling followed by a 5 min all-out test. Power output during
85 the 5 min all-out test was improved following 12 weeks of heavy strength training in well-
86 trained male cyclists. This was related to improved cycling economy and physiological strain
87 during the final hour of the submaximal trial, leaving the strength-trained athletes less

88 fatigued before the 5 min all-out test (Rønnestad et al. 2011). However, no previous study has
89 assessed effects of heavy strength training on all-out performance following a prolonged
90 submaximal work or physiological responses during prolonged submaximal running.

91 The primary purpose of this study was to investigate the effects of 11 weeks of heavy strength
92 training on 5 min all-out performance after separate trials of prolonged submaximal work in
93 both running and cycling and on physiological responses during the prolonged work. We
94 especially wanted to identify performance enhancing mechanisms after strength training
95 which acts similarly and differently on cycling and running performance.

96 We hypothesized that the addition of heavy strength training would result in improved 5 min
97 all-out performance in both cycling and running. Furthermore, we hypothesized that changes
98 in 5 min all-out performance would be related to improved work economy during the
99 prolonged trials and to changes related to anaerobic capacity as increased muscle mass and
100 changes in expression of genes that are involved in anaerobic processes. We also anticipated
101 that some of the underlying mechanisms for improved work economy would differ between
102 running and cycling.

103 **Methods**

104 **Ethical approval**

105 The study was approved by the Local Ethics Committee at Lillehammer University College.
106 Written informed consent was obtained from all athletes prior to inclusion, and the study was
107 carried out in accordance with the Declaration of Helsinki.

108 **Participants**

109 Twenty-eight female duathletes that fulfilled at least two of Jeukendrup et al.'s (Jeukendrup et
110 al. 2000) training and race status descriptions of a well-trained athlete were recruited to this

111 study. None of the athletes had performed systematic strength training for the last 12 months
112 leading up to the study. The athletes were matched on VO_{2max} and randomly assigned to either
113 adding heavy strength training to the ongoing endurance training ($E+S$, $n=14$) or endurance
114 training only (E , $n=14$). During the study, three athletes in $E+S$ left the project for reasons
115 unrelated to the project protocol: one because of an injury, one because of a prolonged period
116 off illness during the last part of the intervention and one because of other medical reasons. In
117 E , six athletes left the study for reasons unrelated to the project protocol (injuries from bicycle
118 crash, pregnancy and lack of time). Therefore, the final numbers of cyclists in $E+S$ and E
119 were 11 and 8 respectively

120 **Experimental overview**

121 This study is part of a larger study investigating the effects of heavy strength training on
122 various aspects of cycling and running performance, some of which has been previously
123 reported (Vikmoen et al., 2015; Vikmoen et al submitted). Whenever data from these studies
124 are utilized for correlation purposes or otherwise, it will be specified clearly. The strength
125 training program for the $E+S$ group consisted of two strength training sessions per week and
126 lasted for 11 weeks (during the competition period from April to July). The testing before and
127 after the intervention period was organized in five test-days. During pre-tests, test day 1
128 consisted of biopsy sampling from *m. vastus lateralis* for determination of muscle fiber type
129 composition and mRNA expression of genes related to fat and anaerobic metabolism. Test
130 day 2 consisted of a VO_{2max} test in cycling followed by 1RM test in half squat. Test day 3
131 consisted of a VO_{2max} test in running. Test day 4 consisted of a prolonged submaximal
132 running trial followed by a 5 min all-out test. Test day 5 consisted of a prolonged submaximal
133 cycle trial followed by a 5 min all-out test. There were 3-7 days between the test days. After
134 the intervention period, the only difference in test order was that muscle biopsies were taken
135 on the last test day.

136 **Training**

137 Endurance training duration and intensity were calculated based on heart rate (HR)
138 recordings. Endurance training was divided into three HR zones: 1) 60%-82%, 2) 83%-87%,
139 and 3) 88%-100% of maximal HR. For detailed information on endurance-training
140 characteristics, see Vikmoen et al. (2015). Briefly, there were no significant differences
141 between groups in their average weekly endurance training duration or distribution between
142 intensity zones.

143 The heavy strength training for the *E+S* groups targeted leg muscles and were performed
144 twice per week during the 11-week intervention period. Adherence to the strength training
145 was high, with *E+S* athletes completing 21.4 ± 1.0 (range 19-22) of the planned 22 strength-
146 training sessions. The strength-training program was performed as reported in Vikmoen et al.
147 (2015). Briefly, each strength training session consisted of four leg exercises: half squat in a
148 smith machine, leg press with one leg at a time, standing one-legged hip flexion, and ankle
149 plantar flexion. Three sets were performed *per* exercise. An investigator supervised the
150 athletes at all workouts during the first two weeks and at least one workout per week
151 thereafter. During weeks 1-3, athletes trained with 10RM sets at the first session and 6RM
152 sets at the second session. These alternating loads were adjusted to 8RM and 5RM during
153 weeks 4-6, and was further adjusted to 6RM and 4RM during weeks 7-11. The athletes were
154 encouraged to increase their RM loads continually throughout the intervention period and
155 they were allowed assistance on the last repetition.

156 **Physical performance tests**

157 The athletes were instructed to refrain from intense exercise the day preceding testing, to
158 prepare for the tests as they would have done for a competition, and to consume the same type
159 of meal before each test. All cycling tests was performed on a electromagnetically braked

160 cycle ergometer (Lode Excalibur Sport, Lode B. V., Groningen, The Netherlands), which was
161 adjusted according to each athlete preference for seat height, horizontal distance between tip
162 of seat and bottom bracket, and handlebar position. During all cycling test the ergometer was
163 in a cadence-independent mode (constant watt-production) so the power output was not
164 affected by the cyclists` chosen cadence. The running tests was performed on a motor driven
165 treadmill (Woodway Desmo Evo, Waukesha, Wisconsin, USA). The inclination of the
166 treadmill was set to 5.3% at all tests. All testing were performed under similar environmental
167 conditions (18-20°C)

168 VO_{2max} in cycling

169 The cycling VO_{2max} test protocol utilized in the present study and its results has been
170 described elsewhere (Vikmoen et al., 2015). Briefly, the test was initiated with 1 min cycling
171 at a power output of 100 W that was subsequently increased by 25 W every minute until
172 exhaustion. VO_2 was measured (30 s sampling time) using a computerized metabolic system
173 with mixing chamber (Oxycon Pro, Erich Jaeger, Hoechberg, Germany). The gas analyzers
174 were calibrated with certified calibration gases of known concentrations before every test. The
175 flow turbine (Triple V, Erich Jaeger, Hoechberg, Germany) was calibrated before every test
176 with a 3 l, 5530 series, calibration syringe (Hans Rudolph, Kansas City, USA). VO_{2max} was
177 calculated as the average of the two highest 30 s VO_2 measurements. Peak cycling
178 performance during the test (W_{max}) was calculated as the mean power output during the last 2
179 min of the incremental test. After the test blood $[La^-]$ and HR_{peak} was noted. $[La^-]$ were
180 analyzed in whole blood with a Lactate Pro LT-1710 analyzer (Arcray Inc., Kyoto, Japan).
181 RPE was recorded using the Borg scale (Borg, 1982). HR was measured using a Polar S610i
182 heart rate monitor (Polar, Kempele, Finland).

183 *Prolonged submaximal cycling followed by a 5 min all-out cycling test*

184 The prolonged cycling lasted for 180 min on a power output corresponding to 44% of W_{\max}
185 (111 ± 9 W and 116 ± 8 W in *E+S* and *E* respectively). The same absolute power output was
186 utilized post-intervention. VO_2 and HR were determined during 3 min periods every 30th min
187 throughout the prolonged cycling and RPE and [$l \cdot a^{-1}$] were measured every 30th min. Average
188 values for each hour were calculated and used for statistical analyses. Athletes were allowed
189 to occasionally stand in the pedals during the prolonged cycling, but not during the 3 min
190 periods of measurements and not during the final 5 min all-out test. Athletes were allowed to
191 consume water and a sport drink containing 60 g/L carbohydrates, ad libitum, in order to
192 maintain fluid balance and mimic race conditions. The amount of sport drink consumed were
193 similar between groups and from pre to post (across groups values were 1.24 ± 0.57 L and
194 1.26 ± 0.59 L, respectively) After conclusion of the prolonged cycling, athletes were allowed
195 three minutes rest before a 5 min all-out test for determination of cycling performance was
196 performed. During the first minute of the test the power output was set by the investigators.
197 This individual selected power output was based on pilot work and corresponded to 85% of
198 W_{\max} . Thereafter the control unit for the power output was put next to the ergometer and the
199 athletes were allowed to adjust the power output themselves with the instruction to cycle at
200 the highest average power output as possible. The participant received feedback regarding
201 power output and elapsed time, but not HR or cadence. Performance was measured as the
202 mean power output during the 5 min all-out test. At the post-test, one athlete in *E+S* had to
203 withdraw during the prolonged test due to pain in the hip. Therefore, the final numbers
204 included in the statistical analysis of these tests are 10 in *E+S* and 8 in *E*.

205 *VO_{2max} in running*

206 The $VO_{2\max}$ test protocol utilized in this study and its results has been described elsewhere
207 (Vikmoen et al., submitted). Briefly, the test was initiated with 1 min running at $8 \text{ km} \cdot \text{h}^{-1}$ that
208 was subsequently increased by $1 \text{ km} \cdot \text{h}^{-1}$ every minute until exhaustion. $VO_{2\max}$ was calculated

209 as the average of the two highest 30 s VO₂ measurements. Peak running performance during
210 the test (V_{max}) was calculated as the mean running velocity during the last 2 min of the
211 incremental test.

212 *Prolonged submaximal running followed by a 5 min all-out running test*

213 The prolonged running lasted for 90 min at a speed corresponding to 60% of V_{max} (7.7 ± 0.4
214 km·h⁻¹ and 7.9 ± 0.3 km·h⁻¹ in *E+S* and *E* respectively). The same absolute speed was used in
215 the post-intervention trial. VO₂ and HR were measured during 3 min periods every 15th min
216 throughout the prolonged running and RPE and [la⁻] were measured every 15th min. Average
217 values for each 30 min period were calculated and used for statistical analyses. The athletes
218 were allowed to consume water and a sport drink containing 60 g·L⁻¹ carbohydrates, ad
219 libitum, in order to maintain fluid balance. The amount of sport drink consumed were similar
220 between groups and from pre to post (across groups values were 0.76 ± 0.27 L and 0.72 ±
221 0.24 L, respectively). After conclusion of the prolonged running, the athletes were allowed
222 three minutes rest before a 5 min all-out test was performed for determination of running
223 performance. During the first minute of the test the speed was set by the investigators. This
224 individual selected speed was based on pilot work and corresponded to 85% of V_{max}.
225 Thereafter the athletes were allowed to adjust the speed themselves with the instruction to run
226 as fast as possible. The athletes received feedback on speed and elapsed time, but not HR or
227 distance. Performance was measured as the distance covered during the 5 min all-out test.

228 *1RM tests*

229 Approximately 20 min after termination of the cycling VO_{2max} test maximal strength in the
230 legs was tested as 1RM in half squat. The 1RM protocol used has been described elsewhere
231 (Vikmoen et al., 2015). Briefly, the 1RM test started with a specific warm-up, consisting of
232 three sets with gradually increasing load (40, 75 and 85% of expected 1RM) and decreasing

233 number of repetitions (10→6→3). The first attempt was performed with a load approximately
234 5% below the expected 1RM. If a lift was successful, the load was increased by
235 approximately 5%. The test was terminated when the athletes failed to lift the load in 2-3
236 attempts and the highest successful load lifted was noted as 1RM. Athletes were given 3 min
237 rest between lifts.

238 **Lean mass in the legs**

239 Lean mass in the legs (Leg_{LM}) was determined by dual-energy x-ray absorptiometry using a
240 Lunar Prodigy densiometer (Prodigy Advance PA+302047, Lunar, San Francisco, CA, USA).
241 The athletes were instructed to refrain from training for the 24 h leading up to the
242 measurement. They were also instructed to not ingest any food or liquid for the 3 h preceding
243 the measurement.

244 **Muscle biopsy sampling**

245 Muscle biopsies were sampled from *m. vastus lateralis* using the Bergström procedure and
246 treated as previously described (Vikmoen et al 2015). An appropriately sized muscle sample
247 was excised and selected for quantitative real-time PCR (qRT-PCR) analyses (average wet
248 weight ± SD: 38 ± 7 mg), and a similarly sized sample was selected for immunohistochemical
249 analyses (average wet weight ± SD: 34 ± 13 mg). Pre- and post-biopsies were sampled at the
250 same time of day for each particular athlete. Biopsies for qRT-PCR analyses were immersed
251 immediately in RNAlater® and treated according to manufacturers' protocol before storage at
252 -80 °C (Ambion, Foster City, CA, USA). Biopsies for immunohistochemical analyses were
253 formaldehyde fixated (Chemi-teknik AS, Oslo, Norway).

254 **Muscle biopsy analyses**

255 *Immunohistochemistry*

256 Protocols for immunohistochemical analyses of muscle fiber type composition and
257 accompanying results have been presented elsewhere (Vikmoen et al., 2015). Briefly,
258 formalin-fixed muscle biopsies were paraffin-embedded and sectioned, whereupon transverse,
259 serial sections were labelled for MyHCI (*A4.840*), MyHCIIA (*EPR5280*) and MyHCIIIX
260 (*6HI*). Determination of muscle fiber composition was performed using Photoshop CS6
261 Extended (Adobe, San Jose, CA, USA). The investigator performing the image analyses were
262 blinded as to which group the athlete belonged. Muscle fibers that were positive for both
263 MyHCIIA and MyHCIIIX are referred to as muscle fiber type IIAX-IIX (Vikmoen et al.,
264 2015). Because of technical problems with some analyses, the number of individuals in the
265 immunohistochemistry data is 8 in *E+S* and 8 in *E*.

266 *Gene expression*

267 Gene expression was assessed for genes involved in fatty acid oxidation and anaerobic energy
268 metabolism. Primer design, RNA extraction, quantitative PCR (qPCR), and evaluation of the
269 stability of reference genes was performed as previously described (Ellefsen et al. 2014). β_2 -
270 microglobulin and ribosomal protein L32 were found to be the two most stable references
271 genes and were utilized for calculation of normalisation factors using GeNorm, which were in
272 turn utilized for calculation of target gene expression. All genes with associated primers are
273 presented in table 1.

274 *Insert table 1 around here*

275 **Statistics**

276 All data in the text, figures and tables are presented as mean \pm standard deviation, unless
277 otherwise stated. Prior to statistical testing, gene expression were log₂-transformed to
278 maximize the likelihood of normal distribution.

279 To test for differences between groups at pre and post, and differences in changes from pre to
280 post, unpaired students t-test were used Within-group analyses were performed using paired t-
281 tests except for evaluating responses during the prolonged trials.

282 To evaluate changes in responses during the prolonged trials within groups (pre to post) a
283 two-way repeated measure analysis of variance (ANOVA) (time of intervention period and
284 time during the prolonged trials as factors) with Sidek-Holm post hoc test were performed. To
285 evaluate differences in changes in the responses during the prolonged trials between the
286 groups a two way repeated measures ANOVA (changes from pre to post in each group and
287 time point during the prolonged trial as factors) with Sidek-Holm post hoc test were
288 performed.

289 In addition, effect sizes for the key performance and physiological adaptations were
290 calculated to compare the practical significance between the two groups. Effects size were
291 calculated as Cohen's d and the criteria to interpret the magnitude were the following: 0-0.2 =
292 trivial, 0.2-0.6 = small, 0.6-1.2 = moderate, 1.2-2.0 = large and > 2 = very large (Hopkins et
293 al. 2009).

294 Correlations analyses were done using the Pearson product-moment method and correlations
295 coefficients were interpreted according to Hopkins et al. (2009); $r < 0.1$ trivial, 0.1-0.3 =
296 small, 0.3-0.5 = moderate, 0.5-0.7 = large, 0.7-0.9 = very large, 0.9 = nearly perfect and 1.0 =
297 perfect.

298 Analyses was performed in GraphPad Prism 6 (GraphPad Software Inc., California, USA) and
299 Excel 2013 (Microsoft Corporation, Redmon, WA, USA). All analyses resulting in $p \leq 0.05$
300 were considered statistically significant and $p \leq 0.10$ are described as tendencies.

301 **Results**

302 There were no significant differences between $E+S$ and E at pre-intervention in any of the
303 measured variables.

304 **Body mass, maximal strength and leg_{LM}**

305 Body mass remained unchanged in $E+S$ (pre: 62.4 ± 5.2 kg, post: 63.1 ± 5.6 kg), but was
306 slightly reduced in E (from 65.6 ± 8.4 kg to 64.8 ± 8.0 kg $p < 0.05$). The change in body mass
307 was different between groups ($p < 0.05$).

308 $E+S$ increased 1RM in half-squat with $45 \pm 22\%$ ($p < 0.01$) while no changes occurred in E (3
309 $\pm 10\%$, $p = 0.52$, figure 1). The change in 1RM was larger in $E+S$ than in E ($p < 0.01$) and the
310 ES analysis revealed a very large practical effect of $E+S$ compared to E ($ES = 2.4$).

311 Leg_{LM} increased in $E+S$ with $3.1 \pm 4.0\%$ ($p < 0.05$) while it decreased in E with $2.2 \pm 2.1\%$ (p
312 < 0.05 , figure 1). The change in leg_{LM} was larger in $E+S$ than in E ($p < 0.01$) with a large
313 practical effect of $E+S$ compared to E ($ES = 1.69$).

314 Because of the reduced body mass in E all VO_2 measurements are presented as body mass
315 adjusted values. Since power output measured using cycling ergometers does not correctly
316 reflect the influence of body mass on outdoor cycling performance, especially during uphill
317 cycling (Anton et al. 2007), power outputs measurements are reported as body mass adjusted
318 values ($W \cdot kg^{-1}$). However, running at a treadmill is influence by body mass to the same
319 degree as outdoor running (McMiken & Daniels 1976) so no body mass adjustments are done
320 on the reported running distances.

321 *Insert figure 1 around here*

322 **Muscle fiber type composition**

323 Effect of the present intervention on fiber type composition has been previously reported
324 (Vikmoen et al., 2015). In brief, there was a reduction in the proportions of fibers positive for

325 both IIA and IIX MyHC from $9 \pm 7\%$ to 0% in $E+S$ ($p < 0.01$) with a concomitant increase in
326 type IIA fibers proportions from $39 \pm 13\%$ to $51 \pm 10\%$ ($p < 0.01$).

327 **Gene expression**

328 Of the nine genes investigated, only mRNA levels for CPT2 and LDHB increased 1.8 ± 0.5
329 fold and 1.2 ± 0.3 fold respectively in $E+S$ ($p < 0.05$), and LDHB tended to increase 1.3 ± 0.4
330 fold in E ($p = 0.07$, figure 2). The remainder of the genes did not change expression in
331 response to the intervention (figure 2).

332 *Insert figure 2 around here*

333 **VO_{2max} and W_{max}/V_{max}**

334 The effect of the intervention used in this study on VO_{2max} and W_{max}/V_{max} has been previously
335 described (Vikmoen et al., 2015; Vikmoen et al., submitted). In brief, VO_{2max} in both cycling
336 and running and W_{max} were unchanged in both groups during the intervention period.
337 However, V_{max} increased in $E+S$ by $1.7 \pm 2.8\%$ ($p = 0.05$) with no change in E .

338 **Responses during the prolonged trials**

339 The physiological responses during the prolonged trials are displayed in figure 3 and table 2.
340 After the intervention $E+S$ reduced VO₂ during the last two hours of the prolonged cycling
341 trial ($p < 0.05$) with no changes in E . The changes during the last two hours was different
342 between the groups ($p < 0.05$). In addition, the effect size analysis revealed a large practical
343 effect of $E+S$ compared to E during the last hour of the trial ($ES = 1.2$). There were no
344 changes in VO₂ for neither $E+S$ nor E during the prolonged running.

345 $E+S$ had a reduced HR throughout the prolonged cycling after the intervention ($p < 0.05$)
346 while E had a reduced HR during the first hour only ($p < 0.05$). There was a moderate
347 practical effect of $E+S$ compared to E during the last hour of the trial ($ES = 1.12$). The

348 correlation between changes in VO_2 and HR during the last hour of the prolonged cycling was
349 large ($r = 0.59$). Both $E+S$ and E had a reduced HR during the entire prolonged running trial
350 after the intervention period ($p < 0.05$). There was no difference in changes between the
351 groups.

352 RPE was lower during the last hour of prolonged cycling for $E+S$ and lower during the last
353 two hours for E ($p < 0.05$). However, there were no differences in changes between the
354 groups. RPE did not change during the prolonged running. There were no changes in RER in
355 neither of the groups during the prolonged trial in both cycling and running. In cycling,
356 cadence did not change in either group during the intervention.

357 *Insert figure 3 around here*

358 *Insert table 2 around here*

359 **5 min all-out tests**

360 After the intervention the mean power output during the 5 min all-out cycling test increased
361 by $7.0 \pm 4.5\%$ ($p < 0.05$) in $E+S$ with no change in E ($3.3 \pm 7.1\%$, $p = 0.27$ figure 4). The
362 difference between the groups was not statistically significant, but the practical effect of $E+S$
363 compared to E was moderate ($ES = 0.62$). $E+S$ increased running distance in the 5 min all-out
364 running test by $4.7 \pm 6.0\%$ ($p < 0.05$) with no change in E ($-0.6 \pm 5.0\%$, figure 4). The
365 increase in running distance was larger in $E+S$ than in E ($p = 0.05$), and the practical effect of
366 $E+S$ compared to E was moderate ($ES = 0.95$). Correlation analyses revealed a large
367 correlation between change in all-out cycling performance and W_{\max} ($r = 0.54$, $p < 0.05$) and
368 between all-out running performance and V_{\max} ($r = 0.53$, $p < 0.05$). There was a large
369 correlation between change in all-out performance and change in IIX fibers in cycling (r
370 $= -0.54$, $p < 0.05$, figure 5) and in running ($r = -0.50$, $p < 0.05$, figure 5) when data from both
371 groups were included. When only $E+S$ athletes were included the correlation got very large in

372 cycling ($r = -0.73$, $p = 0.065$, figure 5) but disappeared in running ($r = -0.28$, $p = 0.547$, figure
373 5). The correlation between the percent change in running distance and mean power output in
374 cycling was moderate but not statistically significant ($r=0.40$, $p = 0.10$).

375 *Insert figure 4 around here*

376 *Insert figure 5 around here*

377 **Discussion**

378 The main finding of the present study is that addition of heavy strength training to the regular
379 endurance training of female duathletes improved both running and cycling performance
380 measured as 5 min all-out performance tested immediately after prolonged submaximal work.
381 In addition, VO_2 and HR were reduced during the last two hours of a 3 h prolonged cycling
382 trial after the addition of heavy strength training, whereas no effects of added strength training
383 were observed on physiological responses during prolonged submaximal running.

384 **Strength, leg_{LM} and muscle fiber type composition**

385 The observed increase in 1RM in half-squat and leg_{LM} is in accordance to previously observed
386 improvements in endurance athletes adding 8-12 weeks of heavy strength training (e.g.
387 Aagaard et al. 2011; Bishop et al. 1999; Ronnestad et al. 2010a; Ronnestad et al. 2015; Storen
388 et al. 2008). The results lend further support to the notion that a substantial increase in
389 strength can be achieved with little or no change in body mass (Ronnestad et al. 2010a;
390 Ronnestad et al. 2015; Storen et al. 2008; Sunde et al. 2010). Increased body mass is usually
391 undesirable for performance in cycling and running and therefore a concern among endurance
392 athletes considering adding strength training. The increase in leg_{LM} reported in the current
393 study indicates that at least some of the improved strength was due to muscle hypertrophy. In
394 addition, we observed a fiber type shift from type IIX towards type IIA fibers (Vikmoen
395 et al., 2015), a common adaptation to strength training among both untrained and endurance

396 trained individuals (Aagaard et al. 2011; Staron et al. 1994). The increased leg_{LM} and fiber
397 type shift shows that the strength training program was effective in inducing adaptations at the
398 muscular level.

399 **Physiological responses during the prolonged trials**

400 As previously observed in well-trained male cyclists (Rønnestad et al. 2011), $E+S$ reduced
401 VO_2 during the last two hours of the prolonged cycling after the strength training intervention.
402 Therefore, even though no change in cycling economy was observed during the first hour,
403 cycling economy was clearly improved when the athletes started to get fatigued. This is
404 highly important in cycling where many races include prolonged submaximal intensities for
405 several hours. Improved cycling economy have also been reported in untrained individuals
406 (Loveless et al. 2005) and trained male cyclists (Sunde et al. 2010) after strength training
407 interventions when measured in a non-fatigued state. However, this seems not to be the case
408 in highly-trained to elite cyclists (Aagaard et al. 2011; Rønnestad et al. 2010a; Rønnestad et
409 al. 2015). The results from the current study and the study by Rønnestad et al. (2011) indicate
410 that after a strength training intervention cycling economy should also be tested when the
411 athletes are somewhat fatigued.

412 HR was reduced throughout the prolonged cycling trial after the intervention period in $E+S$
413 and as for VO_2 the effect was more pronounced during the last two hours. Consequently, the
414 reduced HR was probably because of the reduced VO_2 and hence reduced energy cost. In fact,
415 the reduced HR mirrored the changes in VO_2 and a large correlation between change in VO_2
416 and change in HR during the last hour was observed ($r = 0.59$).

417 The mechanisms behind improved cycling economy during the last two hours of the trial is
418 somewhat unclear. One explanation might be delayed recruitment of type II muscle fibers
419 brought on by increased muscle strength and muscle mass (Rønnestad et al. 2011). When the

420 maximal muscle strength increases and the absolute power output and cadence remains the
421 same the level of force developed in each pedal trust is reduced relatively to the maximal
422 force. Given the size principle of motor unit recruitment this implies that the more economical
423 type I muscle fibers can account for a larger proportion of the same absolute power output
424 (Hickson et al. 1988; Ronnestad & Mujika 2014). This may also explain the lack of changes
425 in cycling economy during the first hour where the relative low power output should mainly
426 recruit type I muscle fibers, thereby leaving little potential for improvements. In fact, it has
427 previously been reported that after exercise for 60 min at an intensity requiring 43% of
428 VO_{2max} , glycogen breakdown mainly occurred in the type I muscle fibers (Vollestad & Blom
429 1985), indicating limited recruitment of type II fibers. However, as the duration of the work
430 increases and muscle fibers starts to get fatigued, additional motor units needs to be recruited
431 to sustain the power output (Gollnick et al. 1973; Vollestad & Blom 1985). The suggested
432 mechanisms is therefore that the strength training allowed *E+S* to use the more economical
433 type I muscle fibers for a longer duration of the trial after the intervention, leading to
434 improved cycling economy during the last part. Supporting this, 5 weeks of strength training
435 has been shown to reduce EMG activity in *m. vastus lateralis* during the last hour of a two
436 hour prolonged cycling trial in well-trained triathletes (Hauswirth et al. 2010).

437 The fiber type transition from type IIAX-IIX to type IIA in *E+S* might also contribute to the
438 improved cycling economy since it has been suggested that type IIA fibers are more
439 economical than the type IIX fibers (Westerblad et al. 2010). However, there was no
440 correlation between the changes in the proportions of type IIAX-IIX and changes in economy
441 during the last hour of the prolonged cycling. This may be because the relatively low power
442 output did not recruit any type IIX fibers during the trial even before the intervention.

443 Other possible explanations for improved cycling economy during the last two hours of the
444 prolonged cycling trial could have been changes in substrate utilization towards larger

445 carbohydrate utilization (Mogensen et al. 2006) or reduction in cadence (Foss & Hallen
446 2004). However, there were no changes in RER or cadence during the prolonged cycling
447 making these explanations unlikely. In fact, based on the increased mRNA levels of CPT2, a
448 protein involved in fatty acid oxidation in the mitochondria an increased utilization of fat as
449 an energy substrate might have been expected. However, in Vikmoen et al. (2015), we did not
450 find changes in the content of the beta-oxidation enzyme hydroxyacyl-CoA dehydrogenase
451 (HADH) in the very same biopsy material, supporting the notion that rates of fatty acid
452 oxidation did not change.

453 In contrast to cycling, no changes occurred in VO_2 during the prolonged running. This is
454 surprising since the proposed mechanisms for the reduced VO_2 during cycling in theory also
455 could reduce VO_2 during the prolonged running. However, some methodological differences
456 might explain the different finding between cycling and running. The prolonged running was
457 only half as long as the prolonged cycling and was performed at a higher relative workload
458 (60% vs 44% of V_{max} and W_{max} , respectively). Because the reduced VO_2 during the cycling
459 trial was seen during the last two hours, it may be speculated that the prolonged running were
460 too short. However, running races do seldom last as long as cycling races, and the shorter
461 duration was therefore chosen for the prolonged running. To compensate for the shorter
462 duration, the prolonged running was performed at a higher relative intensity than the
463 prolonged cycling. This may have led to a quite high recruitment of type II motor units from
464 the start, and the potential for reduced VO_2 during the last part of the trial may therefore have
465 been limited. In fact, in a glycogen breakdown study it was estimated that a large proportion
466 of type IIA fibers were recruited already from the start at a power output corresponding to
467 61% of $\text{VO}_{2\text{max}}$ (Vollestad & Blom 1985).

468 No changes in running economy after addition of strength training is in conflict with results
469 from previous studies where improved running economy ranging from 3-8% have been

470 reported (e.g. Paavolainen et al. 1999; Sedano et al. 2013; Storen et al. 2008). Some
471 methodological differences might explain this discrepancy. Running economy was tested with
472 an inclination of 5.3% in our study, and in combination with the relative low workload, the
473 velocity during the prolonged running was quite low compared to previous studies. In fact, the
474 improvements in running economy after strength training have been reported to be dependent
475 on running velocity (Saunders et al. 2006). The lack of effect on running economy may also
476 be because the strength-training program used did not induce any changes in patellar tendon
477 stiffness (Vikmoen et al submitted). Changes in muscle-tendon stiffness is a frequently
478 proposed mechanism behind improved running economy after strength training (Saunders et
479 al. 2006; Storen et al. 2008).

480 **Performance during the 5 min all-out tests**

481 The improved cycling performance observed in the 5 min all-out test is in accordance with a
482 similar study in male cyclists, who found increased 5 min all-out performance following
483 prolonged cycling after adding strength training to their normal endurance training
484 (Rønnestad et al. 2011). A novel finding in the current study is that 5 min all-out running
485 performance after a prolonged submaximal trial also seems to be affected to the same degree
486 as in cycling. Improved running and cycling performance after strength training is in
487 accordance with previous studies in cycling (Aagaard et al. 2011; Koninckx et al. 2010;
488 Rønnestad et al. 2010a; Rønnestad et al. 2010b; Rønnestad et al. 2015; Sunde et al. 2010)
489 Vikmoen et al 2015) and running (Damasceno et al. 2015; Paavolainen et al. 1999; Sedano et
490 al. 2013; Storen et al. 2008) when performance is measured in a more traditional way.
491 However, other studies contradicts these findings both in in cycling (Bastiaans et al. 2001;
492 Bishop et al. 1999; Levin et al. 2009) and running (Ferrauti et al. 2010; Roschel et al. 2015).
493 Some methodological differences may explain these equivocal findings. To positively affect
494 cycling performance, it seems that the strength training regime needs to involve heavy

495 training load (4-10RM), rather large volumes of training and last for 8 weeks or longer
496 (Aagaard et al. 2011; Koninckx et al. 2010; Ronnestad et al. 2010a; Ronnestad et al. 2015).
497 On the other hand, both explosive, plyometric and heavy strength training seems effective in
498 improving running performance (Damasceno et al. 2015; Paavolainen et al. 1999; Sedano et
499 al. 2013; Storen et al. 2008).

500 Together these observations indicate that the mechanisms behind changes in running and
501 cycling performance after strength training may be somewhat different. Whereas
502 improvements in both cycling and running performance may be related to typical adaptations
503 to prolonged periods of heavy strength training such as increased muscle mass and fiber type
504 transitions from type IIX to type IIA, improvements in running performance may also rely on
505 adaptations such as changes in leg stiffness, rate of force development and other
506 neuromuscular characteristics. Therefore, mechanisms behind the improved performance in
507 cycling and running in the current study might be different. This is supported by the fact that
508 the correlation between changes in running and cycling performance ($r = 0.40$) were not
509 statistically significant.

510 Since the performance tests were performed right after the prolonged trials, changes in the
511 physiological responses to the submaximal exercise was expected to affect performance. We
512 suggest that the reduced VO_2 and HR observed during the last two hours of the cycling trial,
513 indicating reduced physiological strain and less fatigue, made the athletes in *E+S* capable of
514 producing higher mean power output during the final 5 min all-out test. Furthermore, reduced
515 VO_2 in *E+S* means that the total energy consumption during the prolonged cycling trial was
516 lower after the intervention and with no change in substrate utilization the total carbohydrate
517 utilization was reduced. Therefore, some of the improved cycling performance in *E+S* may be
518 due to a better conservation of glycogen stores during the prolonged trial. The importance of
519 less physiological strain during the submaximal exercise is indirectly supported by the fact

520 that 5 min all-out performance, tested in the rested state, was unchanged after 16 weeks of
521 strength training in elite cyclists (Aagaard et al. 2011).

522 Based on the present data, the positive effect of strength training on performance in the 5 min
523 all-out running test cannot be explained by changes in physiological responses during
524 prolonged running. Therefore, the effect of strength training on running performance have to
525 be through other mechanisms. During a 5 min all-out test a substantial part of the energy is
526 derived from anaerobic metabolism (Gastin 2001) and therefore, increased anaerobic capacity
527 might be a mechanism behind improved running performance. In fact, running performance
528 has been reported to correlate well with measurements of anaerobic performance (e.g.
529 Bulbulian et al. 1986; Houmard et al. 1991). $E+S$ improved V_{\max} , and anaerobic capacity is an
530 important determinant for V_{\max} (Jones & Carter 2000). Consequently, we suggest that some of
531 the improved 5 min-all out running performance in $E+S$ was caused by improved anaerobic
532 capacity. This is supported by the large correlation between changes in V_{\max} and changes in 5
533 min-all out performance. Increased anaerobic capacity can be achieved through increases in
534 muscle mass (Bangsbo et al. 1993), or through increasing amount of anaerobic enzymes.
535 Whereas $E+S$ displayed increased muscle mass, small to none changes were found in mRNA
536 expression of genes coding for important proteins in anaerobic metabolic pathways,
537 suggesting that increased muscle mass played the more important role.

538 Arguably, such improvements in anaerobic capacity should also affect performance in the 5
539 min all-out test in cycling, even though the addition of strength training was not found to
540 affect W_{\max} (Vikmoen et al. 2015). Support for this is given by the large correlation between
541 change in cycling 5 min all-out performance and change in W_{\max} . Increased muscle mass in
542 the legs is, as previously mentioned, a likely contributor to improved anaerobic capacity and
543 power output during short performance tests, and there was a very large correlation between

544 leg_{LM} and absolute average power output during the 5 min all-out test before the intervention
545 ($r = 0.71$, data not shown).

546 There were large correlations between the reduction in muscle fiber type IIAX-IIX
547 proportions and changes in 5 min all-out performance in both cycling and running. The type
548 IIA fibers is less fatigable than the type IIX fibers (Westerblad et al. 2010), and a fiber type
549 transition could therefore improve performance. However, a correlation between two
550 variables does not necessary mean a cause and effect relationship (Greenfield et al. 1998).
551 Perhaps the athletes with a large reduction in fiber type IIAX-IIX proportions had a large
552 response to the strength training and that other adaptations to the strength training actually
553 were responsible for the improved performance. Indeed, there was a large negative correlation
554 ($r = -0.65$, data not shown) between the change in leg_{LM} and change in the proportion of type
555 IIAX-IIX fibers. Notably, when only $E+S$ was included the correlation between 5 min all-out
556 performance and IIAX-IIX fiber transitions got very large in cycling and disappeared in
557 running. This indicate that the possible performance enhancing effects from fiber type shift
558 from type IIAX-IIX toward type IIA was more important in cycling than in running.

559 The improved performance cannot be explained by changes in VO_{2max} since VO_{2max} did not
560 change in neither cycling nor running. The lack of effect of strength training on VO_{2max} is not
561 surprising and in accordance with the current literature (e.g. Aagaard et al. 2011; Ronnestad et
562 al. 2015; Storen et al. 2008). Importantly, we expected no change in VO_{2max} in the current
563 study, as athletes were instructed to continue their normal endurance training, having a good
564 base of training from their winter training consisting of running, cycling and cross-country
565 skiing.

566 This is the first study to demonstrate that adding heavy strength training to endurance training
567 leads to improvements in both cycling and running performance in the same athletes.

568 Performance was tested as 5 min all-out performance, measured immediately after prolonged
569 periods of submaximal work. The improved cycling performance was probably related to
570 reduced physiological strain during the submaximal trial. There were no changes in the
571 physiological responses to prolonged running. Therefore, improved running performance was
572 more likely related to other mechanisms like changes in anaerobic capacity, which probably
573 also contributed to improved cycling performance. A fiber type shift from type IIAX-IIX
574 towards type IIA in the main propulsive muscles also seemed to contribute to the improved
575 performance, especially in cycling.

576 **Perspectives**

577 Based on the results of the current study, both runners and cyclists should include heavy
578 strength training in their training programs for maximal gains in performance. This seems to
579 be particularly important for performance during late phases of long-lasting competitions. The
580 present finding of improved cycling performance following a prolonged submaximal effort is
581 in accordance to previous results in male cyclists (Rønnestad et al. 2011) and with previous
582 studies testing performance in a more traditional way (e.g. Aagaard et al. 2011; Barnes et al.
583 2013; Paavolainen et al. 1999; Rønnestad et al. 2010a). The improved cycling performance
584 was related to better cycling economy and therefore less physiological strain during the last
585 part of the prolonged cycling. This is the first study reporting improved running performance
586 following a prolonged submaximal effort. However, the improved running performance did
587 not seem to be related to any physiological changes during the prolonged trial, indicating that
588 other mechanisms were responsible for the improved running performance. We suggest that
589 improved anaerobic capacity and a muscle fiber type transition from type IIAX-IIX to type
590 IIA were related to both the improved running and cycling performance.

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741

742 **Figure legends**

743 Figure 1: Individual values (dotted lines) and mean values (solid lines) before (Pre) and after
744 (Post) the intervention period for athletes adding strength training to their normal endurance
745 training (*E+S*) and athletes performing normal endurance training only (*E*). A: Lean mass in
746 the legs. B: 1 repetition maximum (RM) in squat. * Different than pre ($p < 0.05$), # the
747 percent change from pre is different in *E+S* than in *E* ($p < 0.05$).

748 Figure 2: Log₂-fold change in mRNA expression for genes involved in fat transport and
749 anaerobic metabolism during the intervention period for athletes adding strength training to

750 their normal endurance training (*E+S*) and athletes performing normal endurance training
751 only (*E*). * Different than pre ($p < 0.05$) £ tendency to different from pre ($p < 0.10$). Values
752 are mean \pm CI

753 Figure 3: Percent change in responses during the prolonged trials in cycling (left panels) and
754 running (right panels) for athletes adding strength training to their normal endurance training
755 (*E+S*) and athletes performing normal endurance training only (*E*). Values are mean \pm SD. *
756 Different than pre ($p < 0.05$), # the percent change from pre is different in *E+S* than in *E* ($p <$
757 0.05).

758 Figure 4: Individual values (dotted lines) and mean values (solid lines) before (Pre) and after
759 (Post) the intervention period for athletes adding strength training to their normal endurance
760 training (*E+S*) and athletes performing normal endurance training only (*E*). A: Running
761 distance during the 5 min all-out running test. B: Mean power output during the 5 min all-out
762 cycling test. * Different than pre ($p < 0.05$), # the percent change from pre is different in *E+S*
763 than in *E* ($p = 0.05$).

764 Figure 5: A: Correlation between changes in type IIAX-IIIX proportions and changes in mean
765 power output during the 5 min all-out cycling test. The inserted panel shows the correlation
766 when only the athletes adding strength training to their normal endurance training are
767 included. B: Correlation between changes in type IIAX-IIIX proportions and changes in
768 running distance during the 5 min all-out running test. The inserted panel shows the
769 correlation when only the athletes adding strength training to their normal endurance training
770 are included.

771

772

773 Table 1. Details of primers used for RT-qPCR

Gene	Forward primer	Reverse Primer
LDHA	ATTCAGCCCGATTCCGTTAC	TTCCACTCCATACAGGCACAC
LDHB	CATGGATGGATTTTGGGGGAAC	AACACCTGCCACATTCACAC
MCT1	TTGGAGTCATTGGAGGTCTTGG	CCAATGGTCGCCTCTTGTAG
MCT4	AGGCAAACCTCCTGGATGCG	AAAATCAGGGAGGAGGTGAGC
PFKM	TGACCTCCAGAAAGCAGGTAAG	AACCAGGCCCCACAATGTTC
GAPDH	AAGGCTGGGGCTCATTG	ACGAACATGGGGGCATC
CPT2	AGCAGATGATGGTTGAGTGC	TCAAAGCCCTGGCCCATTG
SLC25	GCATTGCAGGGATCTTCAACTG	ATATTTCCCAGGAGGTGCAGTC

774 LDHA, lactate dehydrogenase A; LDHB, lactate dehydrogenase B; MCT1, monocarboxylate
775 transporter 1; MCT4, monocarboxylate transporter 4; PFKM, phosphofructokinase; GAPDH,
776 glyceraldehyde 3-phosphate dehydrogenase; CPT2, carnitine palmitoyltransferase 2; SLC 25,
777 carnitine/acylcarnitine translocase, member 20.
778

Table 2. Responses during the prolonged trials in cycling and running for athletes adding strength training to their normal endurance training (*E+S*) and athletes performing normal endurance training only (*E*).

		<i>E+S</i>				<i>E</i>			
	Test section	First section	Middle section	Last section	First section	Middle section	Last section	First section	Last section
$\dot{V}O_2$ (ml·kg ⁻¹ ·min ⁻¹)	Cycling	Pre	30.5 ± 2.9	31.3 ± 3.0	31.9 ± 2.9	30.1 ± 3.2	30.5 ± 3.4	31.0 ± 3.1	31.0 ± 3.1
		Post	30.0 ± 2.5	30.2 ± 2.9*#	30.9 ± 3.2*#	29.9 ± 2.4	30.8 ± 2.9	31.5 ± 3.0	31.5 ± 3.0
	Running	Pre	37.3 ± 1.8	37.7 ± 1.8	37.7 ± 1.8	37.0 ± 2.1	37.3 ± 2.0	37.3 ± 1.8	37.3 ± 1.8
		Post	37.0 ± 2.2	37.5 ± 2.0	37.6 ± 1.9	37.4 ± 2.0	37.4 ± 1.5	37.4 ± 1.4	37.4 ± 1.4
HR (beats·min ⁻¹)	Cycling	Pre	134 ± 12	138 ± 14	143 ± 14	129 ± 11	130 ± 9	135 ± 7	135 ± 7
		Post	131 ± 12*	131 ± 14*	137 ± 13*	125 ± 9*	128 ± 10	135 ± 9	135 ± 9
	Running	Pre	158 ± 12	163 ± 13	165 ± 13	152 ± 11	157 ± 11	158 ± 11	158 ± 11
		Post	154 ± 11*	158 ± 10*	159 ± 11*	148 ± 13*	151 ± 11*	153 ± 11*	153 ± 11*
RER	Cycling	Pre	0.85 ± 0.03	0.84 ± 0.03	0.82 ± 0.03	0.87 ± 0.03	0.84 ± 0.03	0.81 ± 0.04	0.81 ± 0.04
		Post	0.87 ± 0.04	0.85 ± 0.03	0.82 ± 0.03	0.88 ± 0.03	0.85 ± 0.03	0.82 ± 0.03	0.82 ± 0.03
	Running	Pre	0.90 ± 0.02	0.89 ± 0.02	0.88 ± 0.02	0.90 ± 0.02	0.87 ± 0.03	0.86 ± 0.03	0.86 ± 0.03
		Post	0.91 ± 0.03	0.88 ± 0.03	0.86 ± 0.03	0.90 ± 0.02	0.88 ± 0.03	0.86 ± 0.03	0.86 ± 0.03
RPE (Borg scale)	Cycling	Pre	11 ± 1	12 ± 1	13 ± 1	11 ± 2	12 ± 2	13 ± 2	13 ± 2
		Post	11 ± 1	12 ± 1	12 ± 1*	10 ± 2	11 ± 1*	12 ± 1*	12 ± 1*
	Running	Pre	12 ± 1	13 ± 1	13 ± 1	11 ± 2	12 ± 1	13 ± 1	13 ± 1
		Post	11 ± 1	12 ± 1	13 ± 1	11 ± 1	12 ± 1	13 ± 1	13 ± 1
Cadence (rev·min ⁻¹)	Cycling	Pre	84 ± 8	83 ± 10	83 ± 10	83 ± 10	81 ± 12	80 ± 13	80 ± 13
		Post	85 ± 9	83 ± 8	83 ± 9	81 ± 11	81 ± 12	80 ± 14	80 ± 14
	Running	Pre	-	-	-	-	-	-	-
		Post	-	-	-	-	-	-	-

Values are mean ± SD. * Different than pre ($p < 0.05$), # the change from pre to post is different in *E+S* than in *E* ($p < 0.05$).

Figure 1

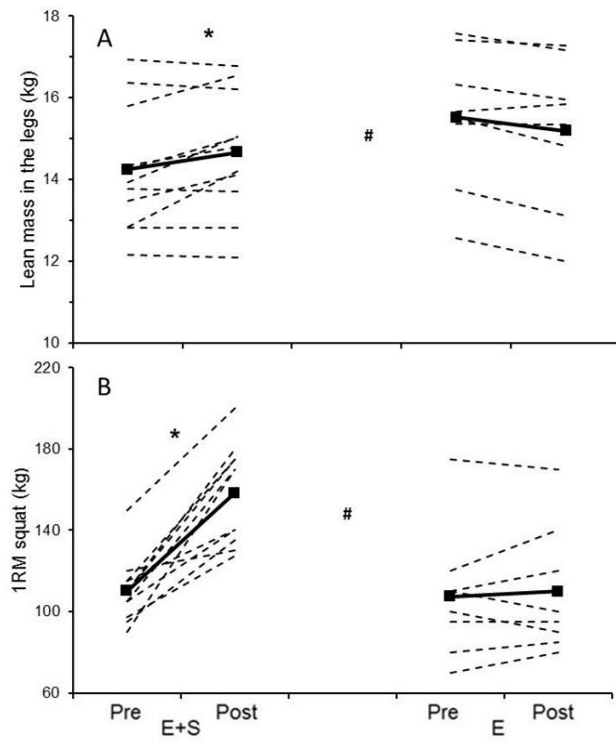


Figure 2

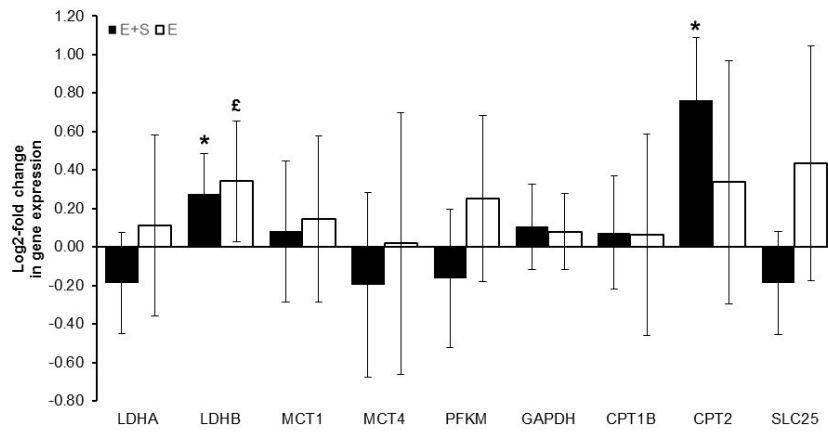


Figure 3

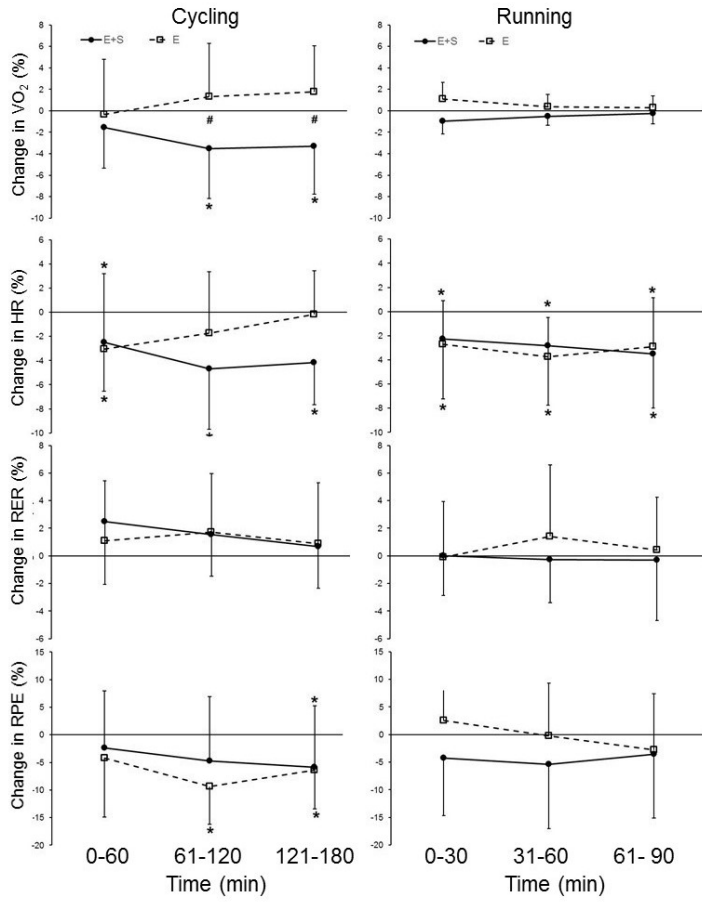


Figure 4

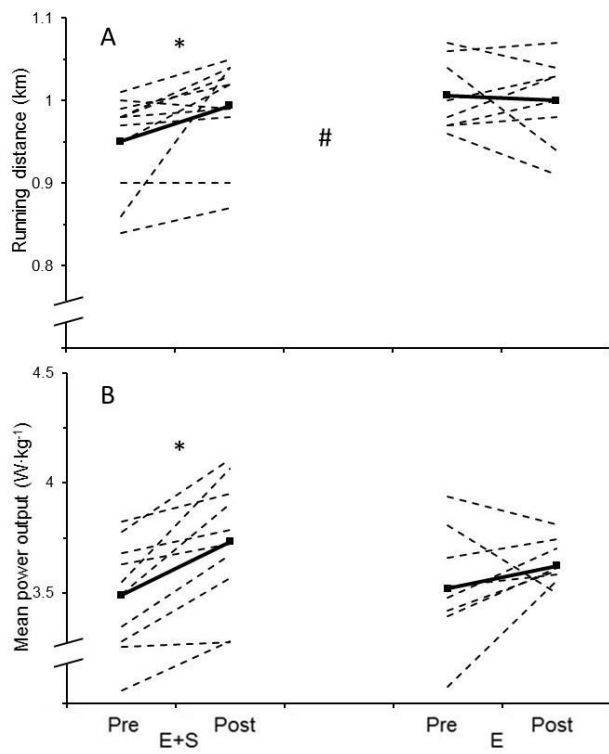
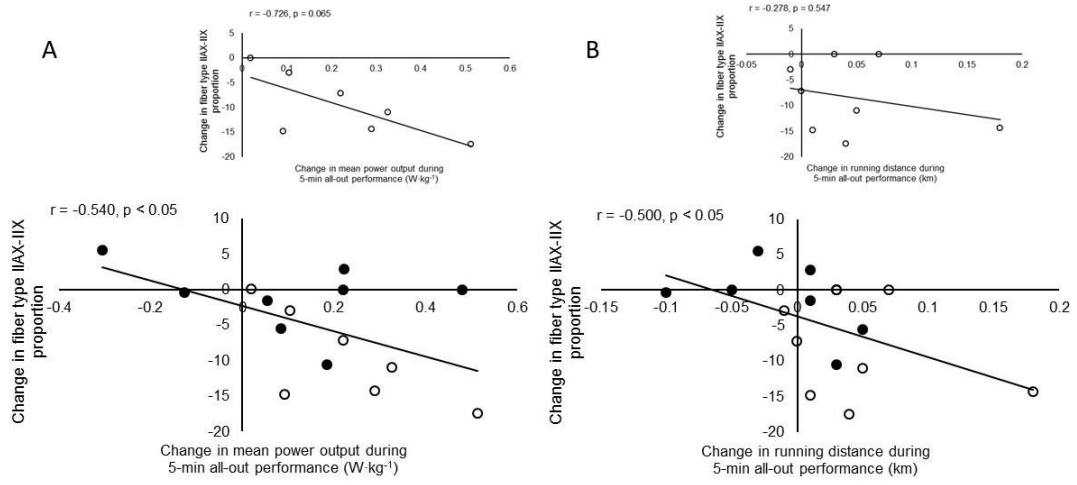


Figure 5



Paper IV

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1 **The adaptation to strength training differs between endurance athletes and**
2 **untrained individuals**

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17 Short title: Strength training in endurance athletes

18 **Abstract**

19 The purpose of this study was to investigate whether endurance athletes, maintaining their
20 normal endurance training, would experience attenuated adaptations to strength training
21 compared to untrained individuals. Eleven non-strength trained female endurance athletes
22 (*E+S*) added 11 weeks of strength training to their normal endurance training (5.1 ± 1.1 h per
23 week), and 10 untrained women (*S*) performed the same strength training without any
24 endurance training. The strength training consisted of four leg exercises [3 x 4-10 repetition
25 maximum (RM)], twice a week for 11 weeks. Both groups increased 1RM in one-legged leg
26 press (*E+S*: $39 \pm 19\%$, *S*: $42 \pm 17\%$, $p < 0.01$), maximal isometric torque in knee extension
27 (*E+S*: $12 \pm 11\%$, *S*: $8 \pm 10\%$, $p < 0.05$) and lean mass in the legs (*E+S*: $3 \pm 4\%$, *S*: $3 \pm 3\%$, p
28 < 0.05) to the same degree. However, *S* had a larger improvement than *E+S* ($p < 0.05$) in peak
29 torque in knee extension at an angular velocity of $240^\circ \cdot \text{sec}^{-1}$ (*E+S*: $8 \pm 5\%$, *S*: $15 \pm 7\%$, $p <$
30 0.01) and maximal squat jump height (*E+S*: $8 \pm 6\%$, *S*: $14 \pm 7\%$, $p < 0.05$). This study do not
31 support any impaired adaptation in the ability to develop force at low contraction velocities or
32 any impaired hypertrophy with concurrent training. However, this study support the notion
33 that concurrent training can attenuate the strength training induced changes in the ability to
34 develop force at high muscular contraction velocities.

35

36 **Key words**

37 Concurrent training; muscle strength; squat jump, counter movement jump, muscle
38 hypertrophy

39

40 **Introduction**

41 Since the pioneering study by Hickson (1980), showing that performing strength and
42 endurance training in the same training program (concurrent training) can attenuate increases
43 in maximal strength compared to strength training alone, a large numbers of investigations
44 have been conducted to elucidate further on the effects of concurrent training. Many of these
45 studies have confirmed that concurrent training can lead to impaired strength gains (Bell et al.
46 2000; Cadore et al. 2010; Gergley 2009; Hunter et al. 1987; Izquierdo et al. 2005; Jones et al.
47 2013), impaired muscle hypertrophy (Bell et al. 2000; de Souza et al. 2007; Gergley 2009;
48 Putman et al. 2004) and reduced neural adaptations (Cadore et al. 2010; Hakkinen et al. 2003)
49 after a strength training program. However, other studies report no negative effects on
50 strength related adaptations after concurrent training compared to strength training only
51 (Abernethy and Quigley 1993; Cantrell et al. 2014; Gravelle 2000; Holviala et al. 2012;
52 McCarthy et al. 1995; McCarthy et al. 2002; Nelson et al. 1990; Shaw et al. 2009). The
53 conflicting results are probably related to many different methodological aspects like training
54 status of the study participants, modality and frequency of both the strength and endurance
55 training, total numbers of training sessions, length of intervention, the way the two type of
56 training are integrated and selection of depended variable. Interestingly, it has been reported
57 that improvements in peak force at high contraction velocities (Dudley and Djamil 1985),
58 jumping performance (Glowacki et al. 2004; Hunter et al. 1987) and maximal rate of force
59 development (RFD) (Hakkinen et al. 2003) can be attenuated after concurrent training
60 compared to strength training only, despite of similar increases in isometric force or 1RM.
61 This indicates that improvements in the ability to produce high power seem to be more
62 attenuated by concurrent training than changes in the ability to produce high forces during
63 slow shortening velocities.

64 To the contrary, an increasing numbers of studies reporting that endurance athletes actually
65 can improve performance in endurance competitions by adding heavy strength training to
66 their normal endurance training are emerging (e.g. Aagaard et al. 2011; Ronnestad et al. 2015;
67 Sedano et al. 2013), and anecdotally the use of strength training among endurance athletes are
68 increasing. However, the typical studies investigating the concurrent training effect include
69 three groups of similarly trained individuals; one group performing strength training only, one
70 group performing endurance training only and one group performing both the strength
71 training and the endurance training. This means that the increase in total training volume is
72 much larger in the concurrent group compared to the other groups. However, endurance
73 athletes that add strength training to their normal endurance training are often at a tolerable
74 and steady state training level, and compared to untrained individuals who add a the same
75 strength training program, the change in training volume would be similar. To the best of our
76 knowledge, only two studies have compared the effects of adding heavy strength training
77 between already endurance trained participants maintaining their normal endurance training
78 and untrained participants, and they yield conflicting results (Hunter et al. 1987; Ronnestad et
79 al. 2012). Hunter et al. (1987) reported similar increases in 1RM squat and CMJ in runners
80 adding strength training to their normal endurance training compared to a group of untrained
81 that added the same strength training. However, a group of highly trained male cyclists had
82 smaller gains in 1RM in leg exercises, thigh muscle CSA, maximal RFD and SJ height when
83 adding strength training compared to untrained participants adding the same strength training
84 protocol (Ronnestad et al. 2012). The probable reason for the different results is that the study
85 by Ronnestad et al. (2012) included competitive cyclists performing about 10 h of endurance
86 training per week, whereas the study by Hunter et al. (1987) included recreational active
87 runners running for only 1-3 h per week. Consequently, there is need for more studies

88 comparing the adaptations to strength training between non-strength trained endurance
89 athletes and previously untrained individuals performing the same strength training.

90 The purpose of this study was to compare the effects of 11 weeks of strength training on lean
91 mass in the legs (leg_{LM}), 1RM, isometric torque, maximal torque at high contraction
92 velocities, and jumping performance between well-trained female endurance athletes
93 maintaining normal endurance training and previously untrained participants.

94 **Methods**

95 **Ethical approval**

96 The study was approved by the Local Ethics Committee at Lillehammer University College.
97 Written informed consent was obtained from all participants prior to inclusion, and the study
98 was carried out in accordance with the Declaration of Helsinki.

99 **Participants**

100 Fourteen well-trained female endurance athletes that was active in both cycling and running,
101 [classified according to Jeukendrup et al. (2000)], and ten untrained female participants were
102 recruited to this study. None of the participants had performed systematic strength training for
103 the last 12 months leading up to the study. During the study, three of the endurance athletes
104 left the project for reasons unrelated to the project protocol: one because of an injury, one
105 because of a prolonged period off illness during the last part of the intervention and one
106 because of other medical reasons.

107 **Experimental overview**

108 This study is part of a larger study investigating the effects of heavy strength training on
109 various aspects of cycling and running performance, and the effects of concurrent training on

110 adaptations to strength training. Some of the results on the effects of heavy strength training
111 on endurance performance have previously been reported (Vikmoen et al. 2015).
112 The endurance athletes added heavy strength training to their normal endurance training for
113 11 weeks ($E+S$, $n = 11$). The endurance training mainly consisted of cycling and running at an
114 average of 5.1 ± 1.1 h per week (for details see Vikmoen et al. (2015)). The untrained
115 participants performed exactly the same strength-training program but performed at most one
116 endurance training session per week in addition to the strength training (S , $n=10$). The
117 characteristics of the participants are displayed in table 1.

118 *Insert table 1 around here*

119 The strength training program consisted of two strength-training sessions per week and lasted
120 for 11 weeks. The testing before and after the intervention period was organized in three test-
121 days, which was similar before and after the intervention. At the first test-day, a dual-energy
122 x-ray absorptiometry (DXA) scan was performed to determine leg_{LM} . Day two consisted of a
123 1RM test in one-legged leg press. In $E+S$ this was performed twenty minutes after a VO_{2max}
124 test in cycling. At test day three, maximal jump height in counter movement jump (CMJ) and
125 squat jump (SJ) was measured followed by test of maximal isometric torque (MVC) in knee
126 extension and a test of peak torque during a isokinetic knee extension at $240 \text{ }^\circ \cdot \text{sec}^{-1}$. In $E+S$
127 these test were done twenty minutes after a VO_{2max} test in running.

128 **Training**

129 The heavy strength training targeted leg muscles and was performed twice per week during
130 the 11-week intervention period. Adherence to the strength training was high, with $E+S$
131 athletes completing 21.4 ± 1.0 (range 19-22) and S participants completing 21.0 ± 0.8 (range
132 20-22) of the planned 22 strength-training sessions. The strength training program was
133 designed as described in Vikmoen et al (2015). Briefly, each training session consisted of half

134 squat in a smith machine, leg press with one leg at a time, standing one-legged hip flexion,
135 and ankle plantar flexion. An investigator supervised all participants at all workouts during
136 the first two weeks and at least one workout per week thereafter. During the first three weeks,
137 the participants trained with 10RM sets at the first session and 6RM sets at the second session
138 of the week. These alternating loads were adjusted to 8RM and 5RM during week four to six,
139 and was further adjusted to 6RM and 4RM during week seven to eleven. The number of sets
140 in each exercise was always three. During the warm up to every training session, the
141 participants eat a protein bar containing 15 grams of protein and 22 gram of carbohydrate
142 (Squeezy recovery bar, Squeezy Sports Nutrition, Braunschweig, Germany).

143 **Testing**

144 *Lean mass in the legs*

145 leg_{LM} was determined by DXA using a Lunar Prodigy densiometer (Prodigy Advance
146 PA+302047, Lunar, San Fransisco, CA, USA). The participants were instructed to refrain
147 from training for the 24 h leading up to the measurement, and not to ingest any food or liquid
148 for the 2 h preceding the measurement. Because of technical problems with some analyses
149 data from 2 participants in *S* had to be excluded. Therefore, the numbers of participants
150 included in the leg_{LM} data are 11 in *E+S* and 8 in *S*.

151 *1RM in one-legged legpress*

152 Prior to the testing day, each participant was given a supervised familiarization session to
153 learn proper lifting technique and find individual equipment settings. During this session, the
154 load was gradually increased to allow estimation of a proper starting point for the 1RM
155 testing.

156 After a 10 min warm-up on a cycle ergometer, the 1RM test started with a specific warm-up,
157 consisting of three sets with gradually increasing load (40, 75 and 85% of expected 1RM) and
158 decreasing number of repetitions (10→6→3). The first attempt was performed with a load
159 approximately 5% below the expected 1RM. If a lift was successful, the load was increased
160 by approximately 5%. The test was terminated when the participants failed to lift the load in
161 2-3 attempts and the highest successful load lifted was noted as 1RM. Participants were given
162 three minutes rest between lifts.

163 *SJ and CMJ*

164 After a 10-min warm-up on a cycle ergometer, SJ and CMJ jumps were performed on a force
165 plate (SG-9, Advanced Mechanical Technologies, Newton, MA, USA, sampling frequency of
166 1kHz). After 3-5 submaximal warm up jumps, the participants performed three SJ and three
167 CMJ with 2-3 min rest between each jump. The mean of the two highest SJ and CMJ were
168 chosen for statistical analyses. During all jumps the participants were instructed to keep their
169 hands placed on their hips and aim for maximal jumping height. The SJ was performed from
170 approximately 90 degrees knee angle. In this position, they paused for 3 s before the jump
171 was performed. No downward movement was allowed prior to the jump and the force curves
172 were inspected to verify this. During the eccentric phase of the CMJ the runners were
173 instructed to turn at a knee angle they felt was optimal for achieving maximal jump height.

174 *Maximal isometric torque and isokinetic torque at an angular velocity of $240^{\circ}\cdot\text{sec}^{-1}$*

175 After the jump tests MVC and peak torque during a knee extension at $240^{\circ}\cdot\text{sec}^{-1}$ were tested
176 in a dynamometer (Cybex 6000, Cybex International, Medway, USA). During these test the
177 participants were seated with a 90° hip angle and were stabilized in this position using chest,
178 hip and thigh straps. The input axis of the dynamometer was aligned with the participants'
179 knee joint and the ankle was strapped to a lever arm. The participants held their arms in front

180 of their chest during all tests. First, the participants performed three maximal knee extensions
181 against the lever arm with a 90° knee angle. The contractions lasted for 5 s and 1 min rest was
182 given between each attempt. The participants were instructed to perform the muscle action as
183 forcefully and quickly as possible. The attempt with the highest maximal torque was chosen
184 for statistical analyses. Two min following the last MVC three maximal isotonic knee
185 extensions from 90° knee angle to full extension were performed against the lever arm at an
186 angular velocity of 240 °·sec⁻¹. One min rest was given between each attempt and the attempt
187 with the highest torque recorded during the contraction was chosen for statistical analyses.
188 Strong verbal encouragement were given to the participants at all attempts.

189 **Dietary intake**

190 In the 6th training week, the participants recorded their daily dietary intake for four days using
191 the weighed food intake method. These four days included three weekdays (not Fridays) and
192 either Saturday or Sunday. The participants weighed all their consumed food using digital
193 food weights (Wilfa KW-4, Wilfa AS, Hagan, Norway) and kept food record journals. All
194 participants were given detailed written and verbal guidelines about how to carry out this
195 method. Dietary assessment data were analyzed using a nutrient analysis software
196 (Kostholdsplanleggeren 2014, Norwegian Food Safety Authority & The Norwegian
197 Directorate of Health, Oslo, Norway)

198 **Statistics**

199 All data in the text, figures and tables are presented as mean ± standard deviation, unless
200 otherwise stated.

201 To test for differences between groups at pre, at post and differences in changes from pre to
202 post, unpaired Students t-test were used. Within-group analyses were performed using paired
203 t-tests. Effect sizes (ES) between groups were calculated for percent changes for key

204 performance and physiological adaptations to elucidate on the practical significance of
205 strength training. ES were calculated as Cohen's d and the criteria to interpret the magnitude
206 were the following: 0-0.2 = trivial, 0.2-0.6 = small, 0.6-1.2 = moderate, 1.2-2.0 = large and >
207 2 = very large (Hopkins et al. 2009).

208 **Results**

209 **Lean mass in the legs, training load and maximal strength**

210 Body mass remained unchanged in *E+S* (from 62.4 ± 5.2 kg to 63.1 ± 5.6 kg) and in *S* (from
211 67.8 ± 13.5 kg to 68.0 ± 12.3 kg). Leg_{LM} increased in *E+S* with $3.1 \pm 4.0\%$ ($p < 0.05$) and in *S*
212 with $3.3 \pm 3.3\%$ ($p < 0.05$, fig. 1). There were no difference in these changes between the
213 groups. The percent increase in the 6RM load from week 2 to week 11 (fig. 2) was similar
214 between the groups (*E+S*: $39 \pm 11\%$, $p < 0.01$, *S*: $40 \pm 11\%$, $p < 0.01$).

215 Both *E+S* and *S* increased 1RM in one-legged leg press (*E+S*: $39 \pm 19\%$, $p < 0.05$, *S*: $42 \pm$
216 17% , $p < 0.05$) and maximal isometric torque (*E+S*: $12 \pm 11\%$, $p < 0.05$, *S*: $8 \pm 10\%$, $p <$
217 0.05) to the same degree (fig. 1).

218 *Insert fig. 1 around here*

219 *Insert fig. 2 around here*

220 **SJ and CMJ**

221 Before the intervention period *E+S* performed better than *S* ($p < 0.05$, fig. 3) in both SJ (*E+S*
222 24.3 ± 6.0 cm, *S*: 18.9 ± 3.2 cm) and CMJ (*E+S* 25.6 ± 4.2 cm, *S*: 21.0 ± 3.6 cm).

223 *E+S* increased SJ and CMJ height by $8 \pm 6\%$ and $6 \pm 6\%$ respectively ($p < 0.05$) while the
224 corresponding numbers in *S* were $14 \pm 7\%$ and $11 \pm 8\%$ respectively ($p < 0.05$, fig. 3). The
225 increase in SJ was larger in *S* than in *E+S* ($p < 0.05$) while there were no statistically
226 difference in the increase in CMJ ($p = 0.11$). The effect size analyses revealed a moderate

227 practical effect in favor the *S* group in both SJ (ES = 0.95) and CMJ (ES = 0.73). *E+S*
228 increased peak torque at $240^{\circ}\cdot\text{sec}^{-1}$ with $8 \pm 5\%$ ($p < 0.01$) and *S* increased with $15 \pm 7\%$ ($p <$
229 0.01 , fig. 3). The change in *S* was significantly larger than in *E+S* ($p < 0.05$) and the practical
230 effect of the percent change of *S* compared to *E+S* was moderate (ES = 1.11).

231 *Insert fig. 3 around here*

232 **Dietary intake**

233 There were no differences between *E+S* and *S* in total energy, carbohydrate or fat intake
234 either in absolute values or normalized to body mass (table 2). *E+S* had, however, higher
235 protein intake than *S* both in absolute values ($p < 0.05$, table 2) and normalized to body mass
236 ($p < 0.05$, table 2).

237 *Insert table 2 around here*

238 **Discussion**

239 The main findings of the current study were that there were no differences in changes in the
240 ability to develop force at low contraction velocities or in muscle hypertrophy after a strength
241 training program in endurance athletes maintaining their normal endurance training compared
242 to previously untrained participants. However, the ability to develop muscular power
243 increased more in the previously untrained participants as shown by the greater increase in
244 maximal isokinetic torque at high contraction velocities and jumping performance.

245 **The ability to develop force at low contraction velocities and leg_{LM}**

246 The finding of similar increases in 1RM and MVC in *E+S* and *S* indicate that the concurrent
247 endurance training performed by *E+S* had no negative effect on the improvement in the
248 ability to develop force at low contraction velocities. This is not in accordance to many
249 studies reporting that concurrent training interferes with increases in maximal strength after a

250 strength training period (Bell et al. 2000; Cadore et al. 2010; Gergley 2009; Hickson 1980;
251 Hunter et al. 1987; Izquierdo et al. 2005; Jones et al. 2013; Kraemer et al. 1995). Hickson
252 (1980) first highlighted this phenomenon in a study where a concurrent training group
253 combined five strength training sessions per week with six endurance training sessions per
254 week for 10 weeks. The concurrent training group had smaller increases in 1RM squat during
255 the last 3 weeks of the intervention period compared to a group performing only strength
256 training. However, there are also many studies reporting no negative effect of concurrent
257 endurance training on changes in maximal strength at low contraction velocities after a
258 strength training intervention (Abernethy and Quigley 1993; Cantrell et al. 2014; Holviala et
259 al. 2012; McCarthy et al. 1995; McCarthy et al. 2002; Nelson et al. 1990; Shaw et al. 2009).
260 The reason for these conflicting results in the literature is unclear, but probably relates to
261 numerous methodical differences between these studies.

262 The typical classic concurrent training study includes a strength-training group, an endurance
263 training group and a concurrent training group. The concurrent training group typically
264 performs the same amount of endurance training as the endurance training group and the same
265 amount of strength training as the strength training group. Therefore, the total training volume
266 will be considerable larger in the concurrent groups, and for untrained individuals this is an
267 unusual and quite large increase in total training volume. Among these concurrent training
268 studies it is difficult to find a common feature regarding the studies that reports attenuated
269 adaptations in strength development after concurrent training. However, although exceptions
270 exists (de Souza et al. 2007; Gergley 2009; Izquierdo et al. 2005), there is a trend that the
271 studies reporting impaired increases in maximal strength include more total training sessions
272 and endurance training sessions per week (Bell et al. 2000; Hickson 1980; Hunter et al. 1987;
273 Jones et al. 2013; Kraemer et al. 1995) than the studies that do not report any attenuated
274 adaptations (Holviala et al. 2012; McCarthy et al. 1995; McCarthy et al. 2002; Shaw et al.

275 2009). Therefore, the total amount of endurance training performed in the concurrent groups
276 is probably an important factor deciding if impairments in strength training adaptations
277 occurs. In fact, in a recent study it was reported that combining strength training with one
278 endurance training session per week had no negative effects on strength gains in
279 recreationally strength-trained men. However, increasing the numbers of endurance training
280 session to three negatively affected the strength gains (Jones et al. 2013).

281 The design of the current study is different from most previous concurrent training studies in
282 some important aspects. In this study, we investigated whether well-trained endurance athletes
283 who maintained a steady level of endurance training would have different adaptations to a
284 strength training intervention than previously untrained individuals. Consequently, the actual
285 increase in training volume was not different between the groups by introducing strength
286 training. In a previous study with similar design (Rønnestad et al. 2012), well-trained male
287 cyclists had reduced increases in 1RM after 12 weeks of heavy strength training compared to
288 a group of recreational active individuals only performing strength training. The reasons for
289 the different findings probably relates to the amount of endurance training performed. In the
290 current study, the athletes in *E+S* performed about 5 h of endurance training per week,
291 whereas in the study by Rønnestad et al. (2012) the cyclists performed endurance training for
292 about 10 h per week. In recreationally active runners, 1-3 h of endurance training did not lead
293 to any impaired strength training adaptations compared to a group only performing strength
294 training (Hunter et al. 1987).

295 The lack of attenuated gains in maximal strength after concurrent training in the current study
296 is probably explained by the lack of attenuated muscle hypertrophy. Many studies report that
297 endurance athletes who add heavy strength training to their normal training do not increase
298 body mass and argues that this indicates lack of muscle hypertrophy (Hoff et al. 2002; Storen
299 et al. 2008; Sunde et al. 2010). Furthermore, no changes in muscle fiber area have been

300 reported in endurance athletes after periods of strength training (Aagaard et al. 2011; Bishop
301 et al. 1999; Hickson et al. 1988). However, recent studies including hypertrophy measures at
302 the whole muscle levels report increased muscle CSA in the triceps brachii muscle in cross
303 country skiers (Losnegard et al. 2011) or increased lean mass in cyclists (Aagaard et al. 2011;
304 Ronnestad et al. 2015) after strength training. However, the reported muscle CSA increase of
305 about 5% (Losnegard et al. 2011) is quite low compared to what is expected in normal active
306 individuals after periods of heavy strength training (e.g. Wernbom et al. 2007), indicating that
307 the endurance training may have affected the hypertrophic response. Unfortunately, these
308 studies did not include a control group performing the same strength training intervention as
309 the endurance athletes, and it is difficult to assess the exact impact of the endurance training.
310 No impairment of muscle hypertrophy in *E+S* is in contrast to impaired hypertrophy reported
311 after strength training in the cyclists in the study by Ronnestad et al. (2012). Again, the
312 difference between these results are probably explained by the different amount of endurance
313 training performed. The “classic” concurrent training studies also yield conflicting results
314 regarding the impact on muscle hypertrophy. Some report attenuated hypertrophy after
315 concurrent training (Bell et al. 2000; Karavirta et al. 2011; Kraemer et al. 1995; Putman et al.
316 2004) whereas others do not (McCarthy et al. 1995; McCarthy et al. 2002). The reason behind
317 these conflicting results are probable the same as discussed above regarding the different
318 findings on maximal strength.

319 The reason for no attenuated effect of the strength training intervention on muscle
320 hypertrophy in the current study could be related to the nutrition of the participants. Even
321 though both groups had protein intakes that were within ACSM’s recommendations for
322 endurance and strength trained athletes (Rodriguez et al. 2009), the daily protein intake was
323 significantly higher in *E+S*. On the other hand, since the total energy intake was similar
324 between the groups despite *E+S* performing 5 h of endurance training per week the energy

325 balance was probably more positive in *S*. Furthermore, the carbohydrate intake in *E+S* was
326 lower than the ACSM's recommendations for endurance athletes. A positive energy balance is
327 important for optimal hypertrophy (Garthe et al. 2013; Rodriguez et al. 2009). However, some
328 underestimation of energy intake might occur, and the unchanged body mass in both groups
329 suggests that both groups were in positive energy balance. Overall, the nutrition of the
330 participants in the current study probably does not explain why no interference effect
331 occurred.

332 **The ability to develop forces at high contraction velocities and jumping ability**

333 The finding of attenuated increase in peak torque at high contraction velocities and jumping
334 performance after concurrent training is in accordance with previous "classical" concurrent
335 training studies (Dudley and Djamil 1985; Glowacki et al. 2004; Hunter et al. 1987). Dudley
336 and Djamil (1985) reported attenuated gains in maximal knee extension torque at angular
337 velocities between 140 and 240 °·s⁻¹ in untrained individuals performing concurrent training
338 compared to individuals performing strength training only. Also in accordance with our
339 results, the changes in peak torque were similar between the groups at lower angular
340 velocities.

341 Increase in jumping performance is a common finding after a period of heavy strength
342 training in untrained individuals (e.g. Tricoli et al. 2005; Wilson et al. 1993). However, in
343 endurance athletes many studies report no improvements in jumping performance after
344 addition of strength training (Francesca et al. 2012; Guglielmo et al. 2009; Millet et al. 2002),
345 supporting our finding of a smaller improvements of jumping height in *E+S* compared to *S*.
346 However, these studies in endurance athletes did not include a control group performing the
347 same strength training program without concurrent endurance training. Attenuated increase in
348 jumping performance have also been reported in well-trained male cyclists compared to

349 individuals only performing strength training after addition of strength training (Rønnestad et
350 al. 2012). However, one study in previously endurance trained athletes that maintained
351 endurance training and added strength training did not report any impaired increases in
352 jumping ability (Hunter et al. 1987). The most likely reason for this finding was the low
353 volume (1-3 h per week) of endurance training performed.

354 Because the *E+S* group jumped higher than *S* before the intervention period, it may be argued
355 that the superior gain in SJ in *S* was because they were at a lower performance level at
356 baseline. However, none of the participants in neither group had performed any jump training
357 or explosive training during the last year leading up to the study. In addition, all other strength
358 measurements were similar between the groups at baseline. The superior jumping
359 performance in *E+S* before the intervention was therefore probably because of better motor
360 skills and non-significant lower body mass. Therefore, the larger improvement in jumping
361 ability in *S* is most likely not because of their lower jumping performance before the start of
362 the intervention.

363 Taken together, the findings from the current study support the notion that concurrent training
364 impairs power related adaptations more than the ability to produce high forces at low muscle
365 contraction velocities. The reason for this is unclear, and the measurements and design of the
366 current study is not suited to answer this question. However, some speculations are possible.
367 The ability to produce high power output and force rapidly is, in addition to maximal
368 muscular strength, dependent on percent of type II muscle fibers (Fitts and Widrick 1996),
369 rapid neural activation of the muscles (Folland and Williams 2007; Rhea et al. 2008) and
370 muscle fascicle length (Blazevich 2006). Hakkinen et al. (2003) reported attenuated
371 adaptations in RFD during isometric knee extension in untrained men after 21 weeks of
372 concurrent training compared to participants performing strength training only. The strength
373 training only group also increased iEMG in *m. vastus lateralis* muscle during the first 500 ms

374 of the isometric contraction with no changes in the concurrent group. The authors therefore
375 suggested that the lack of improved RFD after concurrent training was due to lack of
376 improved rapid voluntary neural activation.

377 There appear to be only small to none differences in the adaptations in fiber type composition
378 after strength training only compared to concurrent training (Kraemer et al. 1995; Putman et
379 al. 2004). In addition, the concurrent studies reporting impaired hypertrophy in muscle fibers
380 indicate that this happens predominately in the type I fibers (Bell et al. 2000; Kraemer et al.
381 1995). Therefore, concurrent training does not seem to induce adaptations in muscle fiber type
382 composition compared to strength training only that should have a larger negative impact on
383 maximal power and rapid force production compared to force productions at low contraction
384 velocities.

385 Long muscle fascicles with a large numbers of serially arranged sarcomeres is advantageous
386 for developing forces at high contraction velocities compared to shorter muscle fascicles
387 (Blazevich 2006). Strength training usually leads to increased anatomical muscle CSA and
388 increased pennation angels (e.g. Aagaard et al. 2001; Blazevich et al. 2007; Franchi et al.
389 2014). These adaptations will have opposite effects on fascicle length. Therefore, fascicle
390 length often remains unchanged (Kawakami et al. 1995) or increases slightly (Blazevich et al.
391 2007; Franchi et al. 2014) after strength training. Research on the effects of endurance
392 training on fascicle length is sparse (Murach et al. 2015). However, long-distance runners
393 have been reported to have shorter fascicles than sprinters and untrained individuals (Abe et
394 al. 2000), and a recent study indicates that endurance running leads to increased pennation
395 angle and shorter fascicles in *m. gastrocnemius* (Murach et al. 2015). On the other hand,
396 running training did not affect fascicle length in *m. vastus lateralis* (Murach et al. 2015), and
397 10 weeks of cycle training did not affect pennation angel in *m. vastus lateralis* or muscle
398 CSA, indicating no changes in fascicle length (Farup et al. 2012). Therefore it is unknown

399 whether different adaptations in muscle architecture after concurrent training compared to
400 strength training only can explain why concurrent training seems to impair adaptations in the
401 ability to develop power. To our best knowledge, no concurrent training study have
402 investigated this.

403 A limitation in the current study is that there may be differences in genotype and/or phenotype
404 between endurance athletes and untrained individuals that might affect strength training
405 adaptations. For example, the fiber type distribution between the groups might have been
406 different. Endurance athletes have been reported to have larger proportions of type I fibers
407 than untrained (Tesch and Karlsson 1985), and endurance training is reported to lead to fiber
408 type shift from type IIX to type IIA (Baumann et al. 1987; Howald et al. 1985). Since the
409 hypertrophic response to strength training may differ between fiber types (Folland and
410 Williams 2007) this could affect strength training adaptations. Another potential limitation of
411 the current study is the fact that *E+S* performed their strength tests 20 min after a VO_{2max} test
412 while *S* performed the strength test in a non-fatigued state. Fatigue after the VO_{2max} test might
413 have affected performance in the strength test. However, this was similar at both pre and post
414 and should therefore not affect the relative changes in strength from pre to post in *E+S*
415 compared to *S*. These limitations need to be kept in mind when the results are interpreted.

416 **Conclusion**

417 The results from the present study showed that well-trained female endurance athletes who
418 maintained a steady state endurance training consisting of about 5 h per week had similar
419 increases in the ability to develop force at low muscular contractions velocities, and muscle
420 hypertrophy after 11 weeks of heavy strength training as previously untrained individuals.

421 However, performing concurrent endurance training led to smaller increases in jump
422 performance and the ability to develop torque at high contraction velocities. Therefore, this

423 study further support the notion that concurrent training can interfere with the power related
424 adaptations to a strength-training program compared to performing strength training only.

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431 **Conflict of interest**

432 None declared.

433

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598

599 **Figure legends**

600 **Fig. 1** Individual values (dotted lines) and mean values (solid lines) before (Pre) and after
601 (Post) the intervention period for athletes adding strength training to their normal endurance
602 training (*E+S*), and previously untrained individuals performing strength training only (*S*). A:
603 Lean mass in the legs. B: 1 repetition maximum (1RM) in one-legged legpress. C: Maximal
604 isometric torque in knee extension (MVC). * Larger than pre ($p < 0.05$).

605

606 **Fig. 2** Percent change in 6 repetition maximum (6RM) load from training week 2 to training
607 week 11 during the intervention period for athletes adding strength training to their normal
608 endurance training (*E+S*), and previously untrained participants performing strength training
609 only (*S*). * Different from pre ($p < 0.01$).

610

611 **Fig. 3** Individual values (dotted lines) and mean values (solid lines) before (Pre) and after
612 (Post) the intervention period for athletes adding strength training to their normal endurance
613 training (*E+S*), and previously untrained individuals performing strength training only (*S*). A:
614 Counter movement jump (CMJ). B: Squat jump (SJ). C: Peak torque in isokinetic knee
615 extension at an angular velocity of $240^\circ \cdot s^{-1}$. * Larger than pre ($p < 0.05$), # the percent
616 change from pre is different between *E+S* and *E* ($p < 0.05$).

617

618

619

620 **Table 1.** Characteristics of the athletes adding strength training to their normal endurance training (*E+S*) and the
 621 untrained individuals performing strength training only (*S*).

Group	N	Age (years)	Height (m)	Body mass (kg)	BMI (kg/m ²)
<i>E+S</i>	11	31.5 ± 8.0	1.69 ± 0.05	62.2 ± 5.2	21.7 ± 1.3
<i>S</i>	10	31.0 ± 9.9	1.72 ± 0.04	67.8 ± 13.5	22.8 ± 3.9

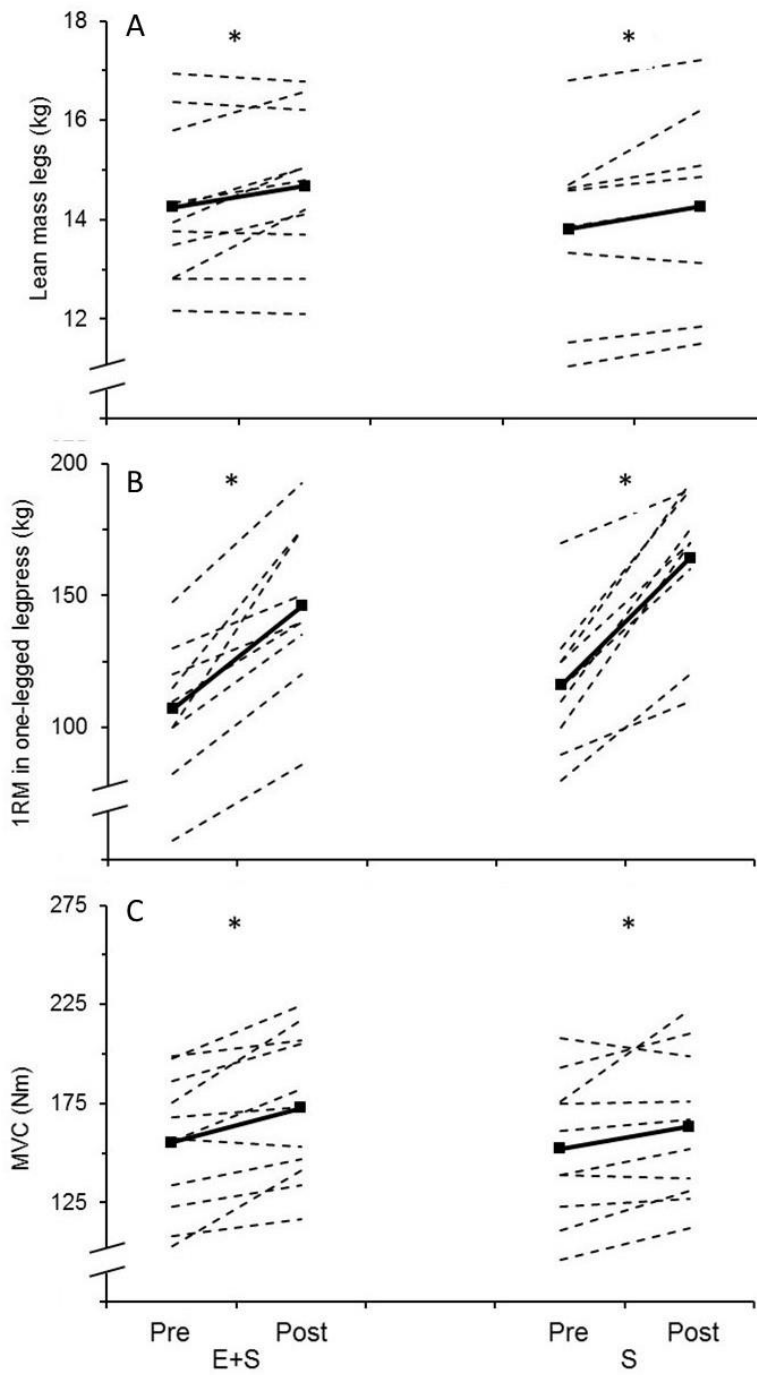
622 Values are mean ± SD

623 **Table 2.** Energy and macro-nutrient intake for athletes adding strength training to their normal endurance
 624 training (*E+S*) and previously untrained individuals performing strength training only (*S*).

Nutrient	<i>E+S</i>	<i>S</i>
Energy intake (kJ·day ⁻¹)	8901 ± 2119	7752 ± 811
Energy intake (kJ·kg ⁻¹ ·day ⁻¹)	141 ± 24	123 ± 32
Carbohydrate (g·day ⁻¹)	218 ± 93	232 ± 35
Carbohydrate (g·kg ⁻¹ ·day ⁻¹)	3.5 ± 1.5	3.6 ± 0.9
Protein (g·day ⁻¹)	104 ± 26*	67 ± 26
Protein (g·kg ⁻¹ ·day ⁻¹)	1.7 ± 0.4*	1.2 ± 0.3
Fat (g·day ⁻¹)	80 ± 26	75 ± 11
Fat (g·kg ⁻¹ ·day ⁻¹)	1.3 ± 0.3	1.2 ± 0.3

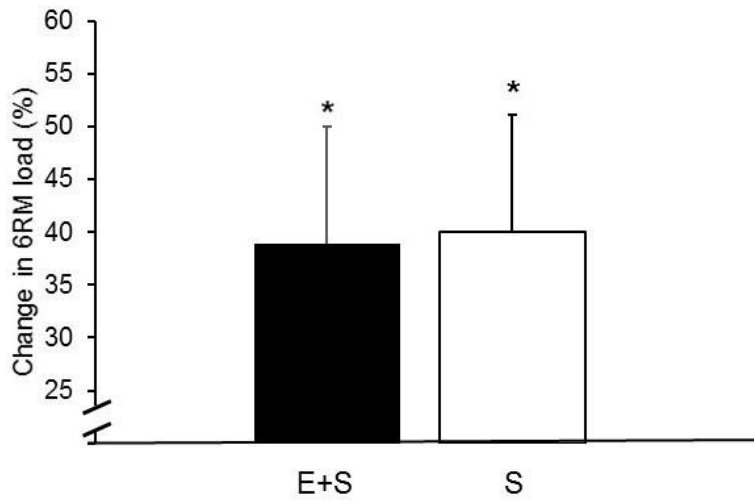
625 Values are mean ± SD. *Larger than *S* (p < 0.05).

626 **Fig 1**



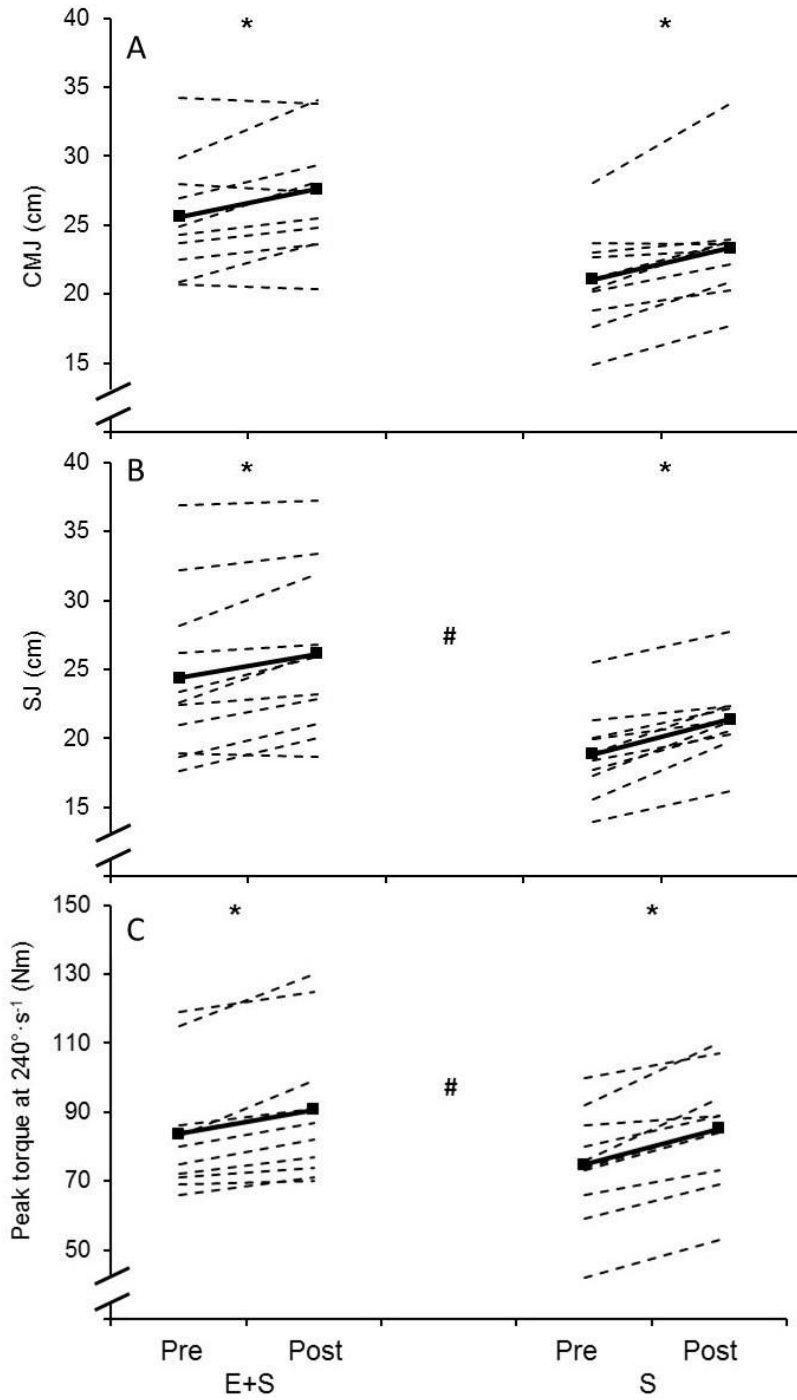
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628 **Fig 2**



629

630 Fig 3



631

Appendix I-V

Appendix I

Muscle biopsy sampling, handling and analyses

Appendix I

Muscle biopsy sampling and handling

Muscle biopsies were sampled from *m. vastus lateralis* using the Bergström procedure [1]. The participants were instructed to refrain from physical activity during the last 24 h before biopsy sampling. During each biopsy sampling-event, two separate muscle biopsies were retrieved and pooled in a petri dish filled with sterile physiological saline. An appropriately sized muscle sample was selected for quantitative real-time PCR (qRT-PCR) analyses (average wet weight \pm SD: 38 ± 7 mg), and a sample of about 50 mg (average wet weight \pm SD: 51 ± 5 mg) was selected for homogenization/fractionation and later Western Blotting. Biopsies for qRT-PCR analyses were immediately immersed in RNAlater® (Ambion) and were treated according to manufacturer's protocol before storage at -80° C until RNA extraction. Biopsies for Western Blotting analyses were snap-frozen in isopentane, cooled with dry ice, before storage at -80° C until extraction. Two samples was selected for immunohistochemical analyses. One was formalin fixed (average wet weight \pm SD: 34 ± 13 mg), and was immediately immersed in 10% buffered formaldehyde solution (Chemi-teknik AS), wherein they were left to fixate for 3-4 days before further preparation. One was mounted in Tissue-Tek (Sakura Finetek USA, Inc., Torrance, CA, USA) and quickly frozen in isopentane cooled on liquid nitrogen (average wet weight \pm SD: 29 ± 8 mg). Pre- and post-biopsies were sampled at the same time of day for each particular athlete.

Muscle biopsy analyses

Fiber type composition by immunohistochemistry

Formalin-fixed muscle biopsies were processed using an Shandon Excelsior ES (Thermo Scientific, Hanover Park, IL USA), before it was paraffin-embedded and sectioned, whereupon transverse, serial sections were immuno-labelled for myosin heavy chain I (MyHC1) (*A4.840*), MyHCIIA (*EPR5280*) and MyHCIIIX (*6HI*), as previously described [2]. The detections system used for determination of muscle fiber types was EnVision™ Flex+ (Dako, Glostrup, Denmark) using the immunostainer, Autostainer Link 48 (Dako, Glostrup, Denmark). Determination of muscle fiber composition was based on analysis of a minimum of 200 fibers, performed using Photoshop CS6 Extended (Adobe, San Jose, CA, USA). The investigator performing image analyses were blinded for which group the participants

belonged. Because of technical problems with some analyses the number of participants in the fiber type composition data is 8 in *E+S* and 8 in *E*.

For fibers that labeled for the IIX antibody, a particular issue became evident during analyses. All these fibers were found to co-label for the IIA-antibody, but not vice versa. This means one of two things: i) either all IIX-positive fibers are IIX hybrids or ii) the IIA-antibody recognizes the IIX antigen in addition to recognizing the IIA antigen. In this study we have chosen to refer to all these fibers type as IIX-IIX fibers.

Western blotting analyses

For Western Blotting analyses, ~50 mg of muscle tissue was homogenised and fractionated into cytosol, membrane, nuclear and cytoskeletal fractions using ProteoExtract Subcellular Proteo Extraction Kit (Cat. no. 539790, Calbiochem; EMD Biosciences GmbH, Schwalbach, Germany), performed according to the manufacturer's protocol. Protein concentrations were assessed with a commercial kit (BioRad DC protein micro plate assay, nos 0113, 0114, 0115; Bio-Rad Laboratories, Inc., Hercules, CA, USA), a filter photometer (Expert 96; ASYS Hitech Cambridge, UK), and the provided software (Kim Version 5.45.0.1; Daniel Kittrich). Membrane fractions (including the mitochondrial components) were analyzed by the Western Blotting technique. Equal amounts of protein from pre and post biopsies (7–12 μg) were loaded into wells and separated on 4–12% SDS-PAGE gels for 35–45 min at 200 volts in cold MES running buffer (NuPAGE MES SDS running buffer; Invitrogen, Inc., Carlsbad, CA, USA) under denaturing conditions. Thereafter, proteins were blotted onto a PDVF-membrane (Immuno-blot, cat. no. 162-0177; Bio-Rad Laboratories, Inc.), at 30 volts for 90 min in cold transfer buffer (NuPAGE transfer buffer, cat. no. NP0006-1; Life Technologies, Inc.). Membranes were blocked at room temperature for 2 h in a 5% fat-free skimmed milk and 0.05% TBS-T solution [TBS, cat. no. 170–6435 (Bio-Rad Laboratories, Inc.); Tween 20, cat. no. 437082Q (VWR International, Radnor, PA, USA); skimmed milk, cat. no. 1.15363 (Merck KGaA, Darmstadt, Germany)]. Blocked membranes were incubated with antibodies against Citrate Synthase (CS, rabbit anti-Citrate Synthase, cat. no. Ab96699, diluted 1:1000; Abcam Plc, Cambridge, UK), Hydroxyacyl-CoA dehydrogenase (HADH, rabbit anti-HADH cat. no. Ab15088, diluted 1:8000 Abcam Plc, Cambridge, UK) and Cytochrome c oxidase subunit IV (COX 4, mouse anti-COX4, cat. no. Ab14744, diluted 1:1000; Abcam Plc, Cambridge, UK) overnight at 4°C, followed by incubation with secondary antibody (COX4: goat anti-mouse, cat. no. 31430, diluted 1:30000; Thermo Fisher Scientific, Inc., Hanover

Park, IL, USA, CS and HADH: Anti rabbit cat. No. 70745 diluted 1:3000 Cell signaling Technology, Inc Danvers, MA. USA) at room temperature for 1 h. All antibodies were diluted in a 1% fat-free skimmed milk and 0.05% TBS-T solution. Between stages, membranes were washed in 0.05% TBS-T solution. Bands were visualised using an HRP-detection system (Super SignalWest Dura Extended Duration Substrate, cat. no. 34076; Thermo Fisher Scientific, Inc., Waltham, MA, USA). Chemiluminescence was measured using a CCD image sensor (Image Station 2000R or Image Station 4000R; Eastman Kodak, Inc., Rochester, NY, USA), and band intensities were calculated with Carestream molecular imaging software (Carestream Health, Inc., Rochester, NY, USA). All samples were run as duplicates and mean values were used for statistical analyses

Capillarization and muscle fiber CSA measured by immunohistochemistry

Cross-sections 8 μm thick were cut using a microtome at -20°C (CM3050; Leica Microsystems GmbH, Wetzlar, Germany) and mounted on microscope slides (Superfrost Plus; Thermo Fisher Scientific, Inc., Waltham, MA, USA). The sections were then air-dried and stored at -80°C . The muscle sections were blocked for 30 min with 1% BSA (cat. no. A4503; Sigma-Aldrich Corp., St Louis, MO, USA) and 0.05% PBS-T solution (cat. no. 524650; Calbiochem, EMD Biosciences, Inc., San Diego, CA, USA).

They were then incubated with antibodies against CD31 (capillaries; 1 : 200; clone JC70A, M0823; Dako A/S, Glostrup, Denmark) overnight at 4°C followed by incubation with appropriate secondary antibodies (Alexa Fluor, cat. no. A11005).

Muscle sections were visualized and pictures taken using a high-resolution camera (DP72; Olympus Corp., Tokyo, Japan) mounted on a microscope (BX61; Olympus Corp.) with a fluorescence light source (X-Cite 120PCQ; EXFO Photonic Solutions Inc., Mississauga, Ontario, Canada).

The muscle sections were then incubated with antibodies against MyHCII (1 : 1000; SC71; gift from Professor S. Schiaffino) and dystrophin (1 : 1000; cat. no. ab15277; Abcam Plc) for 1 h in room temperature followed by incubation with appropriate secondary antibodies (Alexa Fluor, cat. no. A11005 or A11001; Invitrogen, Inc.). Between stages, sections were washed for 3×5 min in 0.05% PBS-T solution.

Muscle sections were then covered with a coverslip and glued with ProLong Gold Antifade Reagent with DAPI (cat.no. P36935; Invitrogen Molecular Probes, Eugene, OR, USA) and left

to dry overnight at room temperature. Muscle sections were again visualized and new pictures was taken at the exactly same location in the section as for the CD31 picture.

Fiber type distribution, fiber cross-sectional area and capillaries were identified using TEMA software (CheckVision, Hadsund, Denmark). Capillarization was expressed as capillaries around each fiber (CAF) and capillaries related to fiber area (CAFA), for type I and type II (IIA and IIX) fibres. Because of technical problems with some analyses, the number of athletes in the immunohistochemistry data is 8 in *E+S* and 5 in *E*.

Gene expression

Gene-specific primers for reference genes and target genes were designed as previously described [1, 2], using Primer3 Plus [253]. For each gene three to five primer pairs were designed. To avoid genomic contamination from affecting gene expression analyses, every primer pair was either located to span an exon-exon boundary containing a genomic intron >~1000 nucleotides *or* to include at least one primer positioned directly across an exon-exon boundary, where possible. Primers were ordered from Thermo Scientific (Waltham, MA, USA) (HPLC-purified; in our hands this level of purification provides primers with much more consistent performance than desalted primers). All primer pairs were tested using the below described qRT-PCR protocol, employing a primer concentration of 100 nM and an annealing temperature of 60° C. The primer pair showing the lowest Ct value and at the same time showing distinct melting curves were chosen.

Total RNA was extracted from muscle biopsies using TRIzol reagent (Invitrogen, Life technologies, Carlsbad, CA, USA), as previously described [2]. Care was taken to remove all remnants of *RNAlater* from biopsies. RNA quantities were obtained using Nanodrop (Thermo Scientific, Waltham, MA, USA), whereupon reverse transcription was performed on 500 µg total RNA using Superscript III Reverse Transcriptase (Invitrogen, Life technologies, Carlsbad, CA, USA), primed with both random hexamers and oligo d(T) (Ambion, Life technologies, Carlsbad, CA, USA), according to manufacturer's protocol. For each sample, duplicate cDNA syntheses were performed.

qRT-PCR was performed on 1/20 dilutions of cDNA using SYBR® Select Master Mix (Invitrogen, Life technologies, Carlsbad, CA, USA) and the 7500 Fast Real-Time PCR System (Applied Biosystems, Life technologies, Carlsbad, CA, USA). Cycling consisted of initial UDG-activation at 50°C for 2 min, followed by denaturation at 95°C for 2 min and 39

repeats of 94°C for 3 s and 60°C for 30 s. For each cDNA synthesis one qRT-PCR reaction was performed for each gene, meaning that two qRT-PCR reactions were performed *per gene per* muscle biopsy. For each qRT-PCR reaction, the cycle threshold (Ct) was calculated using the 7500 Fast Real-Time PCR System software in an automated manner and the priming efficiency was calculated using the LinRegPCR software [3, 4]. For final calculations of target gene expression average priming efficiencies were utilized, calculated separately for each primer pair. Calculations of target gene expression was performed as described by Vandensompele et al. [5].

In order to identify the most suitable internal reference gene, and hence to achieve accurate normalization of target gene expression, GeNorm [5] was utilized to evaluate the geometric expression stability of six frequently utilized reference genes: β_2 -microglobulin (β_2m), peptidylprolyl isomerase A (PPIA, cyclophilin A), TATA box binding protein (TBP), glyceraldehyde-3-phosphate dehydrogenase (GAPDH), β -actin (β -a) and ribosomal protein L32 (RPL32). β_2m and RPL32 was evaluated to be the two most stable reference genes with an M-value well below the limit set by Vandensompele et al. [5]. β_2m and RPL32 were therefore included in all qRT-PCR runs, and were then utilized for calculation of normalisation factors using GeNorm, which were subsequently utilized for calculation of target gene expression.

References

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Appendix II

Approval letter from the Local Ethics Committee at Section for Sport Sciences at
Lillehammer University College.

Lokal etisk komite
Seksjon for idrettsvitenskap
PB 952
2604 Lillehammer

Lillehammer 29. mars 2012

Vedrørende prosjekt «Effekten av styrketrening på prestasjonen i løp og sykling og effekten av samtidig utholdenhetstrening på endringer i styrkeparametere og muskelmasse ved styrketrening»

Prosjektleder:
Stian Ellefsen
Førsteamanuensis
Høgskolen i Lillehammer

Olav Vikmoen
PhD-student
Høgskolen i Lillehammer

Det vises til innsende søknad om godkjenning av doktorgradsprosjektet til stipendiat Olav Vikmoen.

Til søknaden ble vedlagt prosjektplan og informasjonsskriv til deltagere

Lokal etisk komite har behandlet søknaden og tilrår at prosjektet gjennomføres i henhold til prosjektets plan og intensjoner.

På vegne av lokal etisk komite



Marit Roland Udnæs
Seksjonsleder, høgskolelektor
Seksjon for Idrettsvitenskap

marit.roland.udnes@hil.no
Telefon: 61 28 81 74

Appendix III

Letter from the Regional Committee for Medical and Health Research Ethics stating that the research project falls outside the “Act on medical and health research” and that the project therefore could be performed without their consent.

Region: REK sør-øst	Saksbehandler: Katrine Ore	Telefon: 22845517	Vår dato: 31.01.2012	Vår referanse: 2011/2630/REK sør-øst A
			Deres dato: 13.12.2011	Deres referanse:

Vår referanse må oppgis ved alle henvendelser

Stian Ellefsen
Gudbrandsdalsvegen 350

2011/2630 A Effekt av styrketrening på prestasjonen i sykling og løping

Vi viser til søknad om forhåndsgodkjenning av ovennevnte forskningsprosjekt. Søknaden ble behandlet av Regional komité for medisinsk og helsefaglig forskningsetikk i møtet 12.01.2012.

Forskningsansvarlig: Høgskolen i Lillehammer ved øverste ledelse
Prosjektleder: Stian Ellefsen

Prosjektomtale (revidert av REK):

Prosjektets hovedmål er å bedre forstå hvordan styrketrening som et supplement til utholdenhetstrening kan påvirke prestasjonen i utholdenhetssidretter. I tillegg vil prosjektet undersøke nærmere hvordan utholdenhetstrening samtidig med styrketrening påvirker styrkeendring, muskelvekst og molekylære aspekter av muskelcellefunksjon. Det skal rekrutteres 45 friske ikke-styrketrente kvinner som aktivt har drevet med både sykling og løp de siste 12 måneder. De skal rekrutteres fra studenter på høgskolen i Lillehammer, Norges Idrettshøyskole, og i løp- og sykkelmiljøet og fordeles på 3 grupper, en som trener styrke og sykkeløp, en som bare trener sykkeløp og en som bare trener styrke.

Forskningsetisk vurdering

Komiteen har tolket prosjektet som treningsintervensjon, som ikke vil gi ny kunnskap om medisin og helse. Det er komiteens mening at prosjektet er utenfor helseforskningslovens mandat.

Det gis derfor ikke tillatelse til opprette forskningsbiobank etter bestemmelsene i helseforskningsloven.

Vedtak

Etter søknaden fremstår prosjektet å være forskning på treningsmetoder med friske forskningsdeltakere, som ikke vil gi ny kunnskap om helse og sykdom. Det faller derfor utenfor helseforskningslovens virkeområde, jf. § 2, og kan gjennomføres uten godkjenning av REK.

Komiteens vedtak kan påklages til Den nasjonale forskningsetiske komité for medisin og helsefag, jfr. helseforskningsloven § 10, 3 ledd og forvaltningsloven § 28. En eventuell klage sendes til REK Sørøst A.

Klagefristen er tre uker fra mottak av dette brevet, jfr. forvaltningsloven § 29.

Vi gjør oppmerksom på at den forskningsansvarlige institusjon er ansvarlig for at personopplysningene behandles forsvarlig og lovlig i henhold til personopplysningsloven og personopplysningsforskriftens bestemmelser, og må derfor vurdere om prosjektet må forelegges personvernombud eller Datatilsynet.

Komiteens vedtak kan påklages til Den nasjonale forskningsetiske komité for medisin og helsefag, jfr.

Besøksadresse:
Gullhaug torg 4 A,
Nydalens, 0484 Oslo

Telefon: 22845511
E-post:
post@helseforskning.etikkom.no

Web:
<http://helseforskning.etikkom.no/>

All post og e-post som inngår i saksbehandlingen, bes adressert til REK sør-øst og ikke til enkelte personer

Kindly address all mail and e-mails to the Regional Ethics Committee, REK sør-øst, not to individual staff

helseforskningsloven § 10, 3 ledd og forvaltningsloven § 28. En eventuell klage sendes til REK sør-øst. Klagefristen er tre uker fra mottak av dette brevet, jfr. forvaltningsloven § 29.

Vi ber om at alle henvendelser sendes inn via vår saksportal: <http://helseforskning.etikkom.no> eller på e-post til: post@helseforskning.etikkom.no.

Vennligst oppgi vårt referansenummer i korrespondansen.

Med vennlig hilsen,

Gunnar Nicolaysen (sign.)
Professor dr med
Komitéleder

Katrine Ore
Rådgiver

Kopi til: kari.kjenndalen@hil.no; lise.sofie.woie@nih.no

Appendix IV

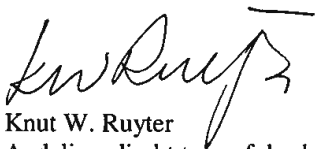
Letter from the Regional Committee for Medical and Health Research Ethics confirming that muscle biopsies and analyses could be performed in the project.

Stian Ellefsen
Høgskolen i Lillehammer

25/4-2012

**VEDRØRENDE OPPBEVARING AV BIOLOGISK MATERIAL I PROSJEKTET
"EFFEKT AV STYRKETRENING PÅ FYSISK PRESTASJONSEVNE OG
MUSKELMASSE HOS KVINNER"**

I møte mellom representanter fra REK sør-øst, Norges idrettshøgskole og Høgskolen i Lillehammer mandag 6/2-2012 ble det avklart at prosjektet "*Effekt av styrketrening på fysisk prestasjonsevne og muskelmasse hos kvinner*" faller utenfor Helseforskningsloven. Det vil derfor, *per nå*, ikke være mulig å opprette biobank i prosjektet. Representantene ble allikevel enige om at muskelbiopsidelen kan gjennomføres under forutsetning av at de aktuelle analysene gjennomføres innen rimelig tid av prosjektavslutning (siste biopsitaking) og at materialet destrueres i etterkant.



Knut W. Ruyter
Avdelingsdirektør, prof.dr.philos
REK sør-øst
Sign.

Appendix V

Approval letter from the Norwegian Social Science Data Services

Norsk samfunnsvitenskapelig datatjeneste AS

NORWEGIAN SOCIAL SCIENCE DATA SERVICES



Harald Hårfagres gate 29
N-5007 Bergen
Norway
Tel: +47-55 58 21 17
Fax: +47-55 58 96 50
nsd@nsd.uib.no
www.nsd.uib.no
Org.nr. 985 321 884

Stian Ellefsen
Avdeling for samfunnsvitenskap
Høgskolen i Lillehammer
Postboks 952
2604 LILLEHAMMER

Vår dato: 13.10.2011

Vår ref: 28070 / 3 / SSA

Deres dato:

Deres ref:

TILRÅDING AV BEHANDLING AV PERSONOPPLYSNINGER

Vi viser til melding om behandling av personopplysninger, mottatt 18.09.2011. Meldingen gjelder prosjektet:

28070	<i>Effekt av styrketrening på fysiske prestasjonsevne og muskelmasse hos kvinner</i>
Behandlingsansvarlig	<i>Høgskolen i Lillehammer, ved institusjonens øverste leder</i>
Daglig ansvarlig	<i>Stian Ellefsen</i>
Student	<i>Olav Vikmoen</i>

Personvernombudet har vurdert prosjektet, og finner at behandlingen av personopplysninger vil være regulert av § 7-27 i personopplysningsforskriften. Personvernombudet tilrår at prosjektet gjennomføres.

Personvernombudets tilråding forutsetter at prosjektet gjennomføres i tråd med opplysningene gitt i meldeskjemaet, korrespondanse med ombudet, eventuelle kommentarer samt personopplysningsloven/-helseregisterloven med forskrifter. Behandlingen av personopplysninger kan settes i gang.

Det gjøres oppmerksom på at det skal gis ny melding dersom behandlingen endres i forhold til de opplysninger som ligger til grunn for personvernombudets vurdering. Endringsmeldinger gis via et eget skjema, http://www.nsd.uib.no/personvern/forsk_stud/skjema.html. Det skal også gis melding etter tre år dersom prosjektet fortsatt pågår. Meldinger skal skje skriftlig til ombudet.

Personvernombudet har lagt ut opplysninger om prosjektet i en offentlig database, <http://www.nsd.uib.no/personvern/prosjektoversikt.jsp>.

Personvernombudet vil ved prosjektets avslutning, 31.12.2020, rette en henvendelse angående status for behandlingen av personopplysninger.

Vennlig hilsen


Atle Alvheim


Sondre S. Arnesen

Kontaktperson: Sondre S. Arnesen tlf: 55 58 25 83

Vedlegg: Prosjektvurdering

Kopi: Olav Vikmoen, Stavsvegen 17, 2635 TRETEN

Avdelingskontorer / District Offices:

OSLO: NSD, Universitetet i Oslo, Postboks 1055 Blindern, 0316 Oslo. Tel: +47-22 85 52 11. nsd@uio.no

TRONDHEIM: NSD, Norges teknisk-naturvitenskapelige universitet, 7491 Trondheim. Tel: +47-73 59 19 07. kyrre.svarva@svt.ntnu.no

TROMSØ: NSD, HSL, Universitetet i Tromsø, 9037 Tromsø. Tel: +47-77 64 43 36. martin-arne.andersen@uit.no

Personvernombudet for forskning



Prosjektvurdering - Kommentar

Prosjektnr: 28070

Utvalget består av 30 kvinner i alderen 18-40 år. Formålet med prosjektet er å studere muskulære tilpasninger til 12 ukers styrketrening hos kvinner. Data samles inn ved hjelp av laboratorietester av fysisk prestasjonsevne, biobank-materiale og ved logging av kosthold.

Førstegangskontakt opprettes av prosjektleder og forsker ved utsending av e-post til studenter og opphenging av plakater ved høgskolen og studentboliger. Interessenter tar selv kontakt med forsker for å være med i studiet.

Personvernombudet finner at behandlingen av personopplysninger i prosjektet kan hjemles i personopplysningsloven §§ 8 første alternativ og 9 a) (samtykke). Det vil i prosjektet bli registrert sensitive personopplysninger om helseforhold, jf. personopplysningsloven § 2 nr. 8 c).

Ombudet forutsetter at det innhentes godkjenning fra den regionale komiteen for medisinsk og helsefaglig forskningsetikk (REK) før opprettelsen av en forskningsbiobank finner sted jf. helseforskningsloven § 25. Personvernombudet ber om at vedtaket fra REK ettersendes.

Personvernombudet finner informasjonsskrivet tilfredsstillende.

Tilgang til datamaterialet vil også gis til førsteamanuensis Bent Rønnestad, Høgskolen i Lillehammer.

Prosjektet skal avsluttes 31.12.2020 og innsamlede opplysninger skal da anonymiseres og slettes. Anonymisering innebærer at direkte personidentifiserende opplysninger som navn/koblingsnøkkel slettes, og at indirekte personidentifiserende opplysninger (sammenstilling av bakgrunnsopplysninger som f.eks. yrke, alder, kjønn) fjernes eller endres.

Appendix VI

Confirmation on “Notice of change” letter from the Norwegian Social Science Data Services

Norsk samfunnsvitenskapelig datatjeneste AS

NORWEGIAN SOCIAL SCIENCE DATA SERVICES



Harald Hårfagres gate 29
N-5007 Bergen
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Tel: +47-55 58 21 17
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www.nsd.uib.no
Org.nr. 985 321 884

Stian Ellefsen
Avdeling for samfunnsvitenskap
Høgskolen i Lillehammer
Postboks 952
2604 LILLEHAMMER

Vår dato: 13.05.2015

Vår ref: 28070/5/AH/LR

Deres dato:

Deres ref:

BEKREFTELSE PÅ ENDRINGSMELDING

Vi viser til endringsmelding mottatt 21.02.2012 for prosjektet:

28070 *Effekt av styrketrening på fysisk prestasjonsevne og muskelmasse hos kvinner*

Personvernombudet for forskning beklager at vi ikke har svart på endringsmeldingen tidligere. Dette skyldes en rutinesvikt som vi nå retter opp.

Av endringsmeldingen mottatt i 2012 går det fram at prosjektet i tillegg til å inkludere friske utrente kvinner, også skulle inkludere friske trente kvinner. Vi mottok oppdatert samtykkeskriv sammen med endringsmeldingen og vi har ingen merknader til denne utvidelsen av prosjektet.

Av samme endringsmelding går det fram at det opprinnelig var planlagt å opprette en forskningsbiobank i prosjektet for oppbevaring av vevs- og blodprøver. Siden prosjektet er vurdert å falle utenfor Helseforskningsloven, har ikke Regional etisk komite kunnet hjemle denne delen av prosjektet. Personvernombudet for forskning forstår det imidlertid slik at vevs- og blodprøvene kun trenger å oppbevares en kort periode for analyse (2-5 mnd). Biologisk materiale som oppbevares i så korte perioder trenger ikke godkjenning, jf. Helseforskningsloven § 4, pkt. c. Det forutsettes da at biologisk materiale destrueres fortløpende innenfor nevnte tidsramme. Dette var også slik vi forstår konklusjonen i møte mellom REK-sør-øst, Norges idrettshøgskole og Høgskolen i Lillehammer, jf. møtereferat fra 25.04.2012. Slik vi forstår det kan altså det biologiske materialet benyttes i prosjektet innenfor en slik kort tidsramme.

Vi håper dette er oppklarende. Ta gjerne kontakt dersom noe er uklart.

Vennlig hilsen

Vigdis Namtvedt Kvalheim

Åsne Halskau

Kopi: Olav Vikmoen, Stavsvegen 17, 2635 TRETEN

