

Ritva Sofia Taipale

Acute Neuromuscular, Cardiorespiratory and  
Endocrine Responses and Chronic Adaptations  
to Combined Strength and Endurance Training in  
Recreationally Endurance Trained Men and Women



STUDIES IN SPORT, PHYSICAL EDUCATION AND HEALTH 196

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kysy aamulta aika  
mistään neuvoa et  
saa parempaa*

## ABSTRACT

Taipale, Ritva Sofia

Acute neuromuscular, cardiorespiratory and endocrine responses and chronic adaptations to combined strength and endurance training in recreationally endurance trained men and women

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Finnish summary

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The aim of the present series of studies was to examine 1) chronic neuromuscular, cardiorespiratory and hormonal adaptations to prolonged periodized combined strength and endurance training using a longitudinal study design; and 2) acute neuromuscular, cardiorespiratory and hormonal responses to combined sessions using a cross-sectional study design. In the longitudinal study, following a preparatory period of strength training, recreationally endurance trained men and women were divided into specific strength training groups. Men: maximal (M), explosive (E), Men and women: mixed maximal and explosive (MM, MW) and circuit training control (CM, CW). Subjects completed an 8-week strength training intervention after which M, E and CM completed 14 weeks of marathon training. Periodized maximal, explosive, and mixed maximal and explosive strength training were more effective than circuit training in improving maximal and explosive strength as well as maximal muscle activation of the lower extremities despite a progressively increasing running volume. Overall endurance performance improved (peak running speed,  $S_{\text{peak}}$ ) in all groups. After the 14-week marathon period, only M made further gains in  $S_{\text{peak}}$  and running economy (RE). The cross-sectional study showed that when mixed strength and endurance were combined into a single session, the order of loadings, (endurance followed by strength (ES) or strength followed by endurance (SE)), led to different acute responses and recovery patterns. At recovery of 24 and 48 h, suppressed testosterone concentrations were still observed in SE men. A delayed decrease in explosive strength was observed in ES women at recovery of 24 and 48 h. Thus, the order of exercise appeared to influence the time course of neuromuscular and hormonal responses and recovery. The present results suggest that periodized maximal, explosive, and mixed strength training combined with progressive endurance training lead to improvements in neuromuscular running characteristics while the order of exercises should be considered for optimization of training.

**Keywords:** combined training, endurance, interference effect, order effect, strength

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Jyväskylä, July 2013  
Ritva Sofia Taipale

## LIST OF ORIGINAL PUBLICATIONS

This thesis is based on the following original articles, which are referred to in the text by Roman numerals:

- I. Taipale, R.S., Mikkola, J., Nummela, A., Vesterinen, V., Capostagno, B., Walker, S., Gitonga, D., Kraemer, W.J. & Häkkinen, K. (2010) Strength Training in Endurance Runners. *International Journal of Sports Medicine*. 31(7):468-76.
- II. Taipale, R.S., Mikkola, J., Vesterinen, V., Nummela, A. & Häkkinen, K. (2013) Neuromuscular adaptations of strength vs. power training or a combination of both during combined strength and endurance training in endurance runners. *European Journal of Applied Physiology*. 113(2): 325-335.
- III. Taipale, R.S., Mikkola, J., Salo, T., Hokka, L., Vesterinen, V., Kraemer, W.J., Nummela, A. & Häkkinen, K. (2013) Mixed maximal and explosive strength training added to an endurance training regimen. *Journal of Strength and Conditioning Research*. Epub ahead of print.
- IV. Taipale, R.S. & Häkkinen, K. (2013) Acute hormonal and force responses to combined strength and endurance loadings in men and women: the "order effect". *PloS one* 8.2 (2013): e55051.
- V. Taipale, R.S., Schumann, M., Mikkola, J., Nyman, K., Kyröläinen, H., Nummela, A. & Häkkinen, K. Acute neuromuscular and metabolic responses to combined strength and endurance loadings: examination of the "order effect" in recreationally endurance trained runners. Submitted for publication.

## ABBREVIATIONS

|                        |  |
|------------------------|--|
| 1RM                    | 1 repetition maximum                                   |
| $\mu$ IU               | micro international units                              |
| ACTH                   | adrenocorticotrophic hormone                           |
| ANOVA                  | analysis of variance                                   |
| AnT                    | anaerobic threshold                                    |
| AnT <sub>speed</sub>   | anaerobic threshold speed                              |
| ATP                    | adenosine triphosphate                                 |
| a-vO <sub>2</sub> diff | arterial venous oxygen difference                      |
| Ca <sup>2+</sup>       | calcium ions   |
| CO <sub>2</sub>        | carbon dioxide   |
| DHEAS                  | dehydroepiandrosterone sulfate                         |
| EMG                    | electromyography                                       |
| ES                     | endurance training session + strength training session |
| FSH                    | follicle stimulating hormone                           |
| GH                     | growth hormone   |
| HR                     | heart rate   |
| HR <sub>max</sub>      | maximum heart rate                                     |
| IGF-1                  | insulin like growth factor 1                           |
| IGFBP3                 | insulin like growth factor binding protein 3           |
| LH                     | luteinizing hormone                                    |
| mmol                   | millimole  |
| mV                     | millivolt  |
| MVC                    | maximal voluntary contraction                          |
| N                      | newton   |
| nmol                   | nanomole   |
| O <sub>2</sub>         | oxygen   |
| pH                     | measure of acidity                                     |
| Q                      | cardiac output   |
| RE                     | running economy  |
| RER                    | respiratory exchange ratio                             |
| RFD                    | rate of force development                              |
| RM                     | repetition maximum                                     |
| SD                     | standard deviation                                     |
| SE                     | strength training session + endurance training session |
| SHBG                   | sex hormone binding globulin                           |
| SV                     | stroke volume  |
| T/C                    | testosterone to cortisol ratio                         |
| TSH                    | thyroid stimulating hormone                            |
| VL                     | vastus lateralis                                       |
| VM                     | vastus medialis  |
| VO <sub>2</sub> max    | maximal oxygen uptake                                  |

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ABSTRACT

ACKNOWLEDGEMENTS

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# 1 INTRODUCTION

Comparing values of maximal oxygen uptake ( $VO_{2max}$ ) is common among endurance athletes and coaches. In more recent years, however, other components of endurance performance have been recognized. Maximal oxygen uptake and other cardiorespiratory characteristics, while essential elements of endurance and anaerobic parameters, in addition to running economy, for evaluating and predicting overall performance capacity in distance runners (Paavolainen et al. 1999b; Bassett & Howley 2000; Nummela et al. 2006). In addition, both strength and endurance based sport disciplines have recognized the contribution of concurrently developing strength, power, and endurance characteristics for optimal performance.

It has been documented that strength training can positively influence endurance performance, although strength training itself does not appear to have a significant impact on endurance capacity in terms of  $VO_{2max}$  (Katch et al. 1985). Similarly, endurance training typically does not induce significant gains in muscle strength and power even using maximal efforts such as uphill running (Sloniger et al. 1997). In fact, the responses and adaptations to strength and endurance training are documented as being, to some degree, divergent (e.g. Hickson 1980; Hawley 2009; Nader 2006; Putman et al. 2004). When a high volume and prolonged period of strength and endurance training are combined, a phenomenon commonly referred to as the “interference effect” in which strength and/or endurance development is negatively affected, may be observed (Hickson 1980; Wilson et al. 2012).

Some of the contributing factors to the interference effect are thought to be the hormonal responses that initiate various physiological cascades linked to e.g. muscle hypertrophy (Kraemer & Ratamess 2005) capillarization (Andersen & Henriksson 1977) and mitochondrial biogenesis (Goffart & Wiesner 2003). These responses are somewhat different and even divergent between strength and endurance training (Hawley 2009; Nader 2006; Putman et al. 2004). The fundamental force production requirements and movement patterns associated with strength and endurance training also vary, suggesting that neuromuscular ad-

aptations to combined strength and endurance training may be affected (Gergley 2009). If the end goals of training for strength or endurance are markedly different, how can they be combined to elicit the best possible results? There remains conflict about the extent to which strength and endurance training may inhibit the other's development (Nelson et al. 1990), although a number of studies have shown the benefits of combined training utilizing different training modes. In recent years, strength training for endurance disciplines and general health/wellbeing has been examined with primarily favorable results. Strength training added to endurance training has been shown to improve endurance performance in runners (Paavolainen et al. 1999c; Støren et al. 2008), Nordic skiers (Hoff et al. 2002; Mikkola et al. 2007; Østerås et al. 2002), triathletes (Millett et al. 2002) and recreational athletes (Häkkinen et al. 2003; Mikkola et al. 2011), while untrained individuals (Sillanpää et al. 2009) have also benefited from combining strength and endurance training.

Although benefits have been observed from combining training over relatively short and progressive training periods, somewhat less is known about the prolonged effects of combined strength and endurance training over multiple unique training periods. Still less is known about the acute responses to combined strength and endurance training and how the order of intra-session exercise might influence responses. Neuromuscular fatigue may be expected following both individual strength and endurance training sessions, but what might be expected from endocrine responses is less clear as response to a second exercise session may be blunted (Cadore et al. 2012) or magnified (Ronsen et al. 2001). The "order effect", or influence of the sequence of exercises in a single session, is a relatively new phenomenon that is currently being examined within combined strength and endurance training.

Many sports require a combination of strength and endurance for optimal performance, while current exercise recommendations (Kraemer et al. 2002; Garber et al. 2011) call for at least a moderate intensity of both strength and endurance training to be completed on a weekly basis for basic fitness and health. The examination of combined strength and endurance training is complex due to the numerous combinations of exercise modes, and intensities as well as training durations. Furthermore, information regarding the acute responses to combined training sessions has received relatively little attention. Each study examining exercise and training combinations helps to improve understanding of the mechanisms behind the acute responses and prolonged training adaptations which, in turn, may provide further insight into practical methods for optimizing training programs and performance in both recreational and elite populations while also helping to improve training methods in clinical populations. This thesis examined combined strength and endurance training from both prolonged and acute perspectives with an emphasis on neuromuscular, cardiorespiratory and endocrine variables in subjects with a recreational background in endurance running.



## 2 REVIEW OF THE LITERATURE

Endurance and strength training offer unique and shared contributions to distance running performance (Figure 1). Neuromuscular, cardiorespiratory and endocrine responses and adaptations to these training modes are, however, even more complex when training methods are mixed. Thus, the responses and adaptations to strength and endurance training will briefly be examined separately before a brief discussion of responses and adaptations to combined strength and endurance training.

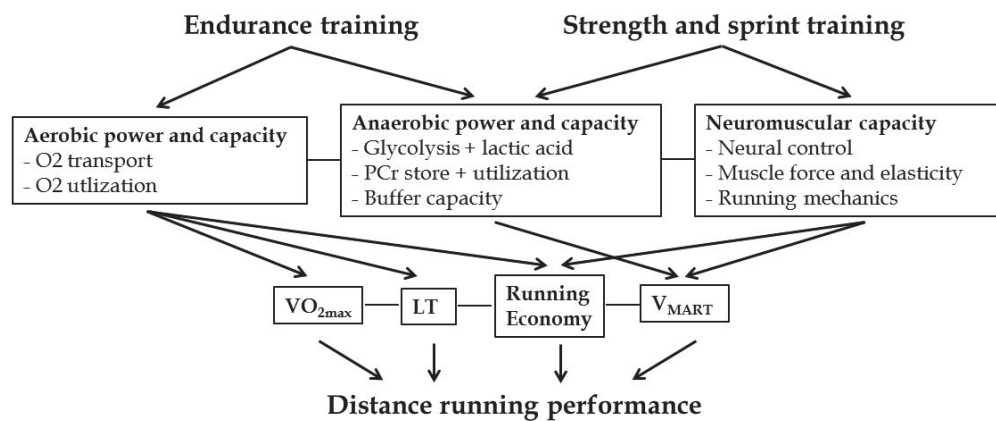


FIGURE 1 Factors influencing distance running (endurance) performance (from Paavolainen et al. 1999c).

## **2.1 Neuromuscular responses and adaptations to strength and endurance training**

The neuromuscular system is essential for human locomotion and is made up of central and peripheral mechanisms including the brain and nerves (central) and muscles (peripheral). Muscles are made up of multiple fibers enclosed in fascicles. Muscle fibers have been classified into different types and subtypes using methods based on e.g. myosin heavy chain content. The primary fiber types that have been identified include: type I, type IIa, type IIx and type IIb, while hybrid types have also been identified (Pette & Staron 2000). More simply, type II fibers and type II subtypes are often referred to as “fast twitch” fibers that primarily use anaerobic metabolism to produce energy while generating rapid contractions. Type I fibers, in contrast, are referred to as “slow twitch” fibers that use aerobic metabolism to produce energy and produce slower contractions. Type II fibers are capable of generating more force per cross-sectional area than type I fibers, whereas type I fibers generate less force, but are less fatigable (Kraemer & Häkkinen 2002). Thus, strength and power require type II fibers, whereas endurance relies on capacity of type I fibers.

Men are typically stronger than women in terms of absolute strength thus, it is important to remember gender differences when evaluating the literature. Furthermore, as daily physical activity and training background may have an influence on fiber types and their distribution (Tesch & Karlsson 1985) it may be of importance to consider the influence of daily physical activity and the difference between typical daily tasks of men and women. A high correlation between muscle cross-sectional area and strength has been observed suggesting that the typically greater muscle cross-sectional area of men is one of the contributing factors to their superior strength (Miller et al. 1993). In contrast, it appears that women are less fatigable than men and seem to recover more quickly (Linnamo et al. 1998; Bosco et al. 2000; Häkkinen & Pakarinen 1995). This greater endurance in women may be credited to the larger proportion of type I muscle fibers typically observed, which may, in turn, influence fatigue mechanisms (Martin & Rattey 2007; Hicks et al. 2001). As men and women are innately different in terms of strength, muscle mass and particularly from an endocrinological perspective, it is logical to acknowledge that both responses and adaptations to training may differ. Nevertheless, young men and women seem to have a similar capacity for making strength gains via training (Lemmer et al. 2000).

### **2.1.1 Neuromuscular responses to a strength training session**

Neuromuscular fatigue is a common consequence of strength and endurance exercise sessions (loadings). Understanding the type of fatigue, its duration and the time-course of recovery are necessary to aid in planning effective training. The neuromuscular response to a single bout of strength training generally

show evidence of fatigue in terms of large decreases in force production, but the mechanisms behind this fatigue may differ (Linnamo et al. 1998; McCaulley et al. 2009). Fatigue can result from central factors such as decreases in recruitment of new motor units and reduced firing frequency of the currently active units (Gandevia 2001), while peripheral factors like lactic acid accumulation inside the muscle, decreased pH, and decreased  $\text{Ca}^{2+}$  transport may also contribute to the inhibition of muscle contractile characteristics (Sahlin 1992). Both types of fatigue may be present after any given loading, although central drive can compensate for some peripheral fatigue in terms of maintaining force production via recruitment of higher threshold motor units (Henneman et al. 1965) or an increased in motor unit discharge rate (De Ruiter et al. 2005).

Within strength training, there is a “specificity effect” of the training mode and its responses and adaptations within the neuromuscular system (Kraemer & Häkkinen 2002). The magnitude and nature of neuromuscular responses and adaptations are determined by the type of strength training utilized including loads, volume, and duration of rest between sets. Loadings that require great neuromuscular activation will induce fatigue more quickly than less strenuous loadings (Häkkinen et al. 1988). Maximal strength training with high loads (such as 70–90 % 1RM) and low repetitions per set generally result in substantial acute fatigue evidenced by decreased muscle activation and increased blood lactate accumulation (Linnamo et al. 1998; Häkkinen & Komi 1983; Ahtiainen et al. 2003). In contrast, explosive resistance training with low to medium loads (such as 30–60 % 1RM) and high action velocity results in acute fatigue evidenced by decreased muscle activation accompanied by a less pronounced increase in blood lactate accumulation (Linnamo et al. 1998; Mikkola et al. 2007). Hypertrophic strength training, while not generally utilized by endurance athletes, is performed using relatively high loads (such as 70–80%) and a moderate number of repetitions (e.g. 8–12) with short periods of rest (e.g. 1–2 minutes) (Kraemer & Häkkinen 2002). This type of training induces acute neuromuscular fatigue as well as a pronounced increase in blood lactate accumulation (McCaulley et al. 2009).

Circuit type strength training, or muscle endurance training, consists of a series of repetitive exercises using only body weight as a load. For example: squats, lunges, step-ups, push-ups, sit-ups, calf-raises, back-extensions, and planks. This mode of training requires only low force production and generally causes less fatigue than maximal or explosive type resistance training protocols, although heart rate response to circuit training indicates that this type of training can be quite strenuous (Gettman & Pollock 1978) and may even be performed as high intensity endurance training (Klika & Jordan 2013).

The time-course of recovery from an acute strength training session is important to know for optimizing training and reducing the chances for injury or overtraining. The time needed for recovery is generally dependent on the intensity and duration of the training session. Recovery of force production capabilities appears to be near complete within approximately 48 h following high in-

tensity strength training in strength trained and untrained individuals (Ahtiainen et al. 2004; Häkkinen & Pakarinen 1993), while the delayed onset of muscle soreness caused by muscle damage typically peaks 24-48 h post exercise session (Byrne et al 2004). In more extreme cases in which eccentric loadings are used, recovery of force production capabilities can take more than 10 days (Howell et al. 1993).

### **2.1.2 Neuromuscular adaptations to strength training**

When strength training sessions are performed repeatedly over a prolonged period of time, adaptations in neuromuscular function are observed if the training stimulus is adequate. Training status has a significant impact on trainability. Typically, untrained individuals are able to make more substantial gains in strength within a relatively short period of time than trained individuals (Häkkinen 1985). In untrained individuals early-phase adaptations may be observed in as little as 4-12 weeks of regular training (Häkkinen et al. 1985a; Häkkinen et al. 1985b). These early-phase adaptations to strength training in untrained individuals may be unspecific to training mode (Cormie et al. 2010b) because a "base" of maximal strength and power appears to be necessary in order to develop more specific neuromuscular characteristics (Newton & Kraemer 1994). Interestingly, there seems to only be a trend towards stronger individuals improving e.g. jump performance more than weaker individuals who complete the same training regimen over a 10 week period (Cormie et al. 2010a). From a practical perspective, and for optimizing training, an adequate level of maximal strength may be of great importance when trying to develop explosive strength. It appears that over a more prolonged period of time the type of strength training performed may have a more marked and specific influence on neuromuscular adaptations than short-term training.

Maximal strength training typically results in early-phase neural adaptations that may be accompanied by muscle hypertrophy after prolonged training (Häkkinen 1994, Moritani & deVries 1979). Similarly, explosive strength training results in specific neural adaptations that are evidenced by increases in muscle activation and especially rapid activation of the muscles due to increased motor unit recruitment. Explosive strength training is focused on improving fast force production/explosive power (Häkkinen et al. 1985b; Häkkinen & Komi 1985; Kyröläinen et al. 2005) but may also induce modest gains in maximal strength due to increases in motor unit firing frequency and increased motor unit recruitment (Van Cutsem et al. 1998), while muscle hypertrophy is minimal (Sale 1988). Mixing maximal and explosive strength training strategies, or high velocity actions with heavy loads may be a more potent stimulus for producing explosive strength than maximal or explosive strength training alone (Newton & Kraemer 1994; Kyröläinen et al. 2005; Heggelund et al. 2013; Newton et al. 2002). Although endurance runners do not typically use hypertrophic strength training, it is a common form of strength training that deserved a brief mention. As the name suggests, hypertrophic strength training causes signifi-

cant increases in muscle cross-sectional area as well as some neural adaptations (e.g. Ahtiainen et al. 2003). Significant hypertrophy is not desired by endurance athletes, because extra mass in endurance sport is often considered non-functional load, however, it is interesting to note that there is ample evidence to suggest that endurance training, and particularly long distance running, blunts the hypertrophic response by way of decreased serum testosterone concentrations that may be accompanied by increased concentrations of serum cortisol (Hackney et al. 2003).

Circuit training with body weight as a load (muscle endurance) may cause increased strength in untrained individuals although changes in muscle activation and muscle mass are not expected to be quite as large in magnitude as those produced by maximal or explosive training. These more modest gains are due, in part, to a smaller degree of muscle recruitment related to the smaller loads moved, i.e. the classic study by Henneman and colleagues (1965) regarding the importance of muscle cell size more commonly known as the “size principle” (Henneman et al. 1965). Nevertheless, in untrained individuals, circuit training with body weight as a load can cause some gains in strength and power, whereas in more well-trained individuals, this type of training can be supplementary and important in, for example, injury prevention. It is worth noting that circuit training, although partially considered to be a form of strength training, may have greater cardiorespiratory benefits than actual strength benefits (see section 2.2.4).

Endurance athletes may use a variety of different strength training methods, however, some endurance athletes avoid strength training due to the commonly held belief that strength training will significantly increase muscle mass, which is not desirable for competitors in most endurance events. Only a few studies (e.g. Rønnestad et al. 2010; Losnegard et al. 2011) have shown a significant increase in the muscle mass of endurance athletes due to their use of heavy resistance training programs. In fact, as previously mentioned, intensive endurance training has been shown to blunt muscle hypertrophy (Hackney et al. 2003), and to cause some degree of muscle atrophy (Kraemer et al. 1995). Interestingly, the majority of studies regarding combined strength and endurance training in which significant muscle hypertrophy has been observed, have used an endurance training mode other than running like cycling. In untrained individuals, the combination of strength training and cycling appears to induce significant muscle hypertrophy (Häkkinen et al. 2003) similar to the previously mentioned studies (Rønnestad et al. 2010; Losnegard et al. 2011). More often than not, however, strength training in endurance athletes/runners has been shown to improve the neuromuscular characteristics of muscles including improved strength and muscle activation, as well as increased muscle stiffness and stability (e.g. Paavolainen et al. 1999c; Mikkola et al. 2007; Spurrs et al. 2003) with minimal or no hypertrophy. Often these improvements have transferred into improved sport-specific neuromuscular performance such as running economy (RE), which will be discussed later.

Regardless of strength training type, in order to continue making strength gains, training programs are typically customized to an individual's specific needs, planned to progress in intensity, and are periodized in order to avoid over- or undertraining (Kraemer & Häkkinen 2002). In order to maintain or continue strength and power gains, an adequate volume, frequency and intensity of training needs to be performed. If the training stimulus is not adequate, detraining may result (Kraemer et al. 2002). Detraining is characterized by decreases in strength and muscle activation (e.g. Häkkinen & Komi 1983; Häkkinen et al. 1985a), in addition to possible muscle atrophy (e.g. Häkkinen et al. 1985a). The time-course of detraining seems to differ depending on training status (Coyle et al. 1984); however, typically neural adaptations to strength training decrease before muscle atrophy occurs. The time course of the loss of adaptations tends to mirror the time course of training adaptations. Strength gains may be maintained using a small but adequate volume and intensity of strength training despite an increase in concurrently performed endurance training (Bell et al. 1993).

### **2.1.3 Neuromuscular responses to endurance exercise**

There are a number of modes of exercise used for endurance training including e.g. running, walking, biking, Nordic skiing, rowing, and Nordic-walking. Even body-weight "circuit training" is considered to be (muscle) endurance training. These modes of exercise are able to stress the cardiorespiratory/cardiovascular system but are fundamentally different in terms of movement patterns. Thus, neuromuscular responses to different endurance exercise modes may differ. For the purpose of brevity, this thesis will focus on running, biking and body-weight "circuit training" in the following section.

Neuromuscular responses to endurance exercise are typically somewhat smaller in magnitude than those observed following strength exercise, although it is important to remember that the magnitude of fatigue is dependent on the intensity and / or duration of exercise. While large decreases in strength and neural activation are almost always observed following strength exercise, only small-scale decreases in strength and neural activation are typically observed following endurance exercise sessions like running (Paavolainen et al. 1999a). Nevertheless, running can induce a significant amount of fatigue such as marathon running (e.g. Nicol et al. 1991). Endurance running typically requires a smaller amount of force production than strength exercise; and even maximal uphill running cannot induce maximal muscle activation (Sloniger et al. 1997). Running, however, does demand repetitive force production using the stretch-shortening cycle (Komi 2000), which can be quite strenuous. The previously mentioned central (Gandevia 2001) and peripheral factors (Sahlin 1992) of fatigue may also play a role in different ratios in response to endurance exercise.

Following a 5 km time-trial, the maximal voluntary contraction (MVC) of the lower extremities has been shown to decrease by 15% (Nummela et al. 2008), after 30 km of high intensity trail running, the decrease in MVC was 24% (Millet

et al. 2003), and a 22% decrease in MVC and 16% decrease in drop jump were observed after simulated marathon racing (Nicol et al. 1991). These decreases in isometric and dynamic performance are partly the result of changes in muscle stiffness (Nicol et al. 1991; Avela & Komi 1998), reduced economy/efficiency and a reduced ability to use elastic energy. This information suggests that the repetitive stretch-shortening of muscles during running actions may cause significant fatigue (Avela & Komi 1998). Although it appears that strength loss is highly correlated with decreases in voluntary muscle activation (Millet et al. 2003), the average decreases in MVC are not necessarily related to decreases in running velocity during a running trial (Paavolainen et al. 1999a; Nummela et al. 2008). In addition, it has been shown that the activity of the leg extensor muscles, particularly during the braking phase, exceeds that of which is recorded during MVC (Kyröläinen et al. 2005). As such, the traditionally used isometric tests may not be precise enough to detect specific muscle-contraction related fatigue in runners, although they clearly show decreases in force-production capabilities following endurance running. While decreases in force-production and muscle activation are often observed after endurance running, trained middle and long distance runners who completed intensive running for 20-40 minutes actually increased jump and half-squat performance, although muscle activation was observed to decrease (Vuorimaa et al. 2006). The differences observed in these results may be related to differences in coordination strategies between agonist and synergist muscle activities (Nicol et al. 1991; Vuorimaa et al. 2006) or perhaps even slight differences in laboratory testing procedures. Nevertheless, an endurance training session may, in some cases, cause potentiation suggesting that fatigue after running might be more peripheral than central in nature (Mettler & Griffin 2012; Škof & Strojnik 2005). More commonly, it appears that at least 48 h are needed after e.g. a 10 km running at race pace in trained runners in order for neuromuscular characteristics to recover fully (Gomez et al. 2002). A greater amount of time may be necessary to recover from longer distances or intensities of running due to, for example, the possibility of accumulating a greater amount of muscle damage from repetitive stretch-shortening cycle actions (Horita et al. 1999).

As previously mentioned, running consists of rapid and repetitive eccentric-concentric muscle actions (stretch-shortening cycle actions), while cycling consists of primarily concentric muscle actions (Bijker et al. 2002). In endurance running, isometric force production capabilities appear to decrease in a non-linear relationship with exercise duration, while the same does not appear to be true of cycling. The muscle contraction strategy of the endurance exercise, however, may not be responsible for this difference; rather the type of fatigue appears to be responsible (Millet & Lepers 2004). Unfortunately, it is difficult to conclusively separate central and peripheral fatigue, as the mechanisms of both central and peripheral fatigue seem to be affected by different kinds of physical activity. For example, decreased force production after 30 min of moderate in-

tensity cycling at different cadences appears to be the result of both central and peripheral mechanisms (Lepers et al. 2001).

#### **2.1.4 Neuromuscular adaptations to endurance training**

As the neuromuscular responses to an endurance exercise session are somewhat smaller than those typically observed following strength exercise, it is reasonable to expect that neuromuscular adaptations to endurance training will also be somewhat smaller in magnitude. Again, endurance training mode has a significant influence on various physiological adaptations to endurance training including neuromuscular adaptations that are specific to movement patterns. Muscle morphological changes may also be different between modes of training due to the “task dependency” related to muscle contractions and resulting in fatigue as well as differences in training modes affecting muscle damage in different ways (Millet & Lepers 2004; Millet et al. 2009).

In recreational (novice) runners, it appears that 16 weeks of moderate marathon training induces specific muscle adaptations that would support endurance performance including the size of the muscle, contractile properties and fiber type distribution, that depend on the muscle examined (Luden et al. 2012). Based on studies comparing e.g. sprinters to distance runners, we can deduce that distance running either induces or favors lean muscles with shorter fascicle lengths and smaller pennation angles than what might be observed in sprinters (Abe et al. 2000). Meanwhile, trained strength athletes and sprinters are able to produce a greater absolute maximal force, as well as a higher maximal force per unit of cross-sectional area than endurance athletes (Häkkinen & Keskinen 1989). This supports the idea that training mode favors specific muscle characteristics or that these characteristics are the result of prolonged specific training. As muscular adaptations appear to be specific to the force and velocity of movements rather than actual movement patterns or exercises used, we can infer that athletes involved in different types of training (e.g. running vs. biking or strength training) will have different muscular adaptations (Blazevich et al. 2003). Muscles appear to be highly plastic; however, there are not many studies that specifically examine the neuromuscular adaptations to distance running, possibly because controlling for running technique (foot strike patterns), footwear, and daily physical activity is quite difficult. Fortunately, due to the stretch-shortening nature of running mechanics, studies involving repetitive jumping may provide some insight into the neuromuscular adaptations to running. For example, drop-jump training for 4 weeks induced training adaptations specific to stretch-shortening type exercises (Schmidtbleicher et al. 1988). Movements similar in terms of force and velocity, like running, may produce similar specific stretch-shortening adaptations.

While the endurance training mode of running is the focus of the present thesis, cycling is a popular and practical endurance training mode used in studies of combined strength and endurance training. Studies utilizing cycling seem to show greater increases in both dynamic and isometric strength of the lower



extremities than studies examining strength combined with running (e.g. Häkkinen et al. 2003; Karavirta et al. 2009). Even 10 weeks of cycling induces increases in strength of the lower extremities in untrained males (Farup et al. 2012). The greater increases typically observed in dynamic and isometric strength in cycling are likely due to the previously mentioned differences in force production and muscle actions required between the two training modes of running and cycling (Bijker et al. 2002). In addition, in cycling the quadriceps femoris and gluteus muscles play a more significant role than in running where the biceps femoris is doing more significant work (Kyröläinen et al. 2001).

## 2.2 Cardiorespiratory and metabolic responses and adaptations to strength and endurance training

Maximal oxygen consumption is the traditional measure for evaluating endurance performance capacity. Values are typically reported in absolute terms in  $L \cdot \text{min}^{-1}$  or relative to body weight in  $\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$  or  $\text{ml} \cdot \text{kg}^{-0.75} \cdot \text{min}^{-1}$ . The highest  $\text{VO}_{2\text{max}}$  values have been reported in elite male Nordic skiers, cyclists and runners as high as  $90 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ , while values reported for the general population range from 52 to  $55 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$  for the 90<sup>th</sup> percentile in men 20 - 39 years of age and 46 to  $49 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$  for the 90<sup>th</sup> percentile in women 20 - 39 years of age (Armstrong 2006, 79). Aerobic capacity is known to decline with age and disuse (detraining) due to sedentary behavior. As a result of the specific nature of training adaptations, endurance exercise is required to develop and maintain aerobic capacity. Clinically,  $\text{VO}_{2\text{max}}$  is defined as 1) a plateau in oxygen consumption during the final minutes of a graded exercise test, 2) when the ratio of  $\text{CO}_2$  produced to  $\text{O}_2$  consumed (respiratory exchange ratio or RER) increases to 1.15 or higher, 3) when heart rate reaches maximum or within 10 beats per minute of the age predicted maximum, and 4) when blood lactate exceeds  $8 \text{ mmol} \cdot \text{L}^{-1}$  (Armstrong 2006, 123; Nieman 2003, 80) regardless of endurance exercise mode.

While  $\text{VO}_{2\text{max}}$  often dominates discussions on endurance performance, another important component of endurance performance of particular interest in terms of coaching is the anaerobic threshold. Anaerobic threshold is the intensity at which an individual can e.g. run, at which oxygen is the primarily contributor to energy production and the rate of lactic acid production and clearance is equal (Svedahl & MacIntosh 2003). As previously stated, lactic acid accumulation decreases the pH of the muscle and reduces  $\text{Ca}^{2+}$  transport to inhibit muscle contractile characteristics, which causes peripheral fatigue (Sahlin 1992). Thus, running at or just below the anaerobic threshold in race situations is an effective strategy for distance running events.

### 2.2.1 Cardiorespiratory responses to endurance exercise

Heart rate is the most common and easily accessible measure of training intensity as it rises linearly with an increase in training intensity/workload (Nieman 2003, 202). Heart rate monitors are a regular tool in both training and research for monitoring training intensity and recovery because heart rate is not a subjective measure and it has the ability to give more feedback than e.g. speed or time. In addition, heart rate is easier to measure and less invasive than e.g. blood lactate, serum hormone concentrations or inflammatory response. During exercise, failure of the heart rate to increase with increasing workload or a rapid increase in heart rate may indicate significant health problems (Armstrong 2006, 123). In contrast, the lower heart rates observed in athletes are generally the result of increased stroke volume meaning that each beat of the heart is able to pump more blood (Nieman 2003, 210-211). It should be noted, however, that an abnormally low heart rate can also indicate various cardiac pathologies.

Although heart rate increases in proportion to workload, the relationship between heart rate and running speed is not constant (Lambert et al. 1998). Over the duration of an exercise session, heart rate may be increased due to cardiac drift resulting from changes in hydration status (Lambert et al. 1998). In addition, sleep, stress, diet, caffeine consumption, physical activity (or lack thereof), prolonged training and aging can influence the heart rate response to a training session.

### 2.2.2 Cardiorespiratory adaptations to endurance training

One of the fundamental aspects of endurance training is related to the Fick Principle, which is explained using the equation:  $VO_2 = Q \cdot a-vO_{2diff}$  (where  $Q = HR \cdot SV$ ). This principle suggests that in order to improve oxygen consumption ( $VO_2$ ) one needs to improve cardiac output ( $Q$ ) by increasing either heart rate ( $HR$ ) or stroke volume ( $SV$ ) or that one needs to improve arteriovenous oxygen difference ( $a-vO_{2diff}$ ) by increasing oxygen delivery and/or improving oxygen utilization in the working muscles. Using this principle, training for endurance capacity generally means training in order to increase cardiac output and to increase capillarization and mitochondrial density. While resting heart rate is typically lower in trained individuals, stroke volume is consistently higher in trained athletes than their sedentary counterparts and appears to increase systematically until  $VO_{2max}$  (Wang et al. 2012) suggesting that this cardiorespiratory characteristic develops with endurance training (Weiner & Baggish 2012).

Maximal aerobic capacity has traditionally been used as the predictor of endurance performance, but along with the neuromuscular factors previously discussed, anaerobic capacity has also been shown to be associated with endurance performance (Nummela et al. 2007; Rusko et al. 1993; Houmard et al. 1991). Anaerobic running tests have, in fact, been found to be more reliable predictors of endurance performance than  $VO_{2max}$  tests due to their more multifaceted na-

ture. Interestingly, lactate threshold / anaerobic threshold has been reported to be one of the best predictors for endurance performance (Yoshida et al. 1987).

In addition to anaerobic performance, running economy is helpful for evaluating endurance performance capacity and for comparing athletes to each other. Running economy is described as the energy needed to run at a given sub-maximal velocity (Bassett & Howley 2000; Saunders et al. 2004) and is typically measured by sub-maximal steady-state running on a motorized treadmill in a controlled lab environment (Saunders et al. 2004). Running economy is expressed in  $\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$  or in  $\text{ml} \cdot \text{kg}^{-0.75} \cdot \text{min}^{-1}$ , a value that corrects for body mass, making it possible to compare RE values between athletes (Svedenhag 1995). Running economy takes into consideration neuromuscular factors and has been shown to be a better predictor of endurance performance than  $\text{VO}_{2\text{max}}$  alone (Paavolainen et al. 1999b; Saunders et al. 2004). In addition to running economy, the velocity at  $\text{VO}_{2\text{max}}$ , which combines running economy and  $\text{VO}_{2\text{max}}$  into the same term, is useful in comparing performance differences between athletes (Billat & Koralsztein 1996).

### 2.2.3 Metabolic responses and adaptations to endurance training

Repeated endurance sessions lead to increased capillary density (Andersen & Henriksson 1977) and the initiation of mitochondrial biogenesis (Goffart & Wiesner 2003) as well as improvements in stroke volume/cardiac output (Weiner & Baggish 2012) due to the physiological cascades initiated by endurance exercise. The increased capillary density improves the distribution of energy sources, while simultaneously enhancing the clearance of  $\text{H}^+$  and  $\text{K}^+$  ions as well as lactate while helping to maintain muscle contractile properties. Similarly, a greater concentration of mitochondria increases the potential for the production of adenosine triphosphate (ATP) (Ingjer 1979). Long slow aerobic endurance training is performed at a low intensity over long distances/a long duration of time with sessions lasting for multiple hours. While distance training is considered the “foundation” for endurance training, to our knowledge there are no long-term studies examining the specific responses and adaptations to long slow endurance training versus e.g. high intensity training. A well-controlled 8-week study comparing endurance training methods was, however, carried out by Helgerud and colleagues (2007) and will be discussed shortly. Aerobic endurance training is performed below 60%  $\text{VO}_{2\text{max}}$  and uses primarily muscle fat stores as fuel. At the higher end of aerobic training, both fat and carbohydrates are used almost equally for ATP production. Anaerobic training is typically performed at  $\geq 80\%$  of  $\text{VO}_{2\text{max}}$  and tends to use carbohydrates as the primary fuel source (McCaulley et al. 2009). From a muscle perspective, slow-twitch muscle fibers (type I) and fat are primarily being used while training at or below anaerobic threshold while anaerobic training (above anaerobic threshold) utilizes more fast-twitch muscle fibers (type II and type IIx) and a greater proportion of carbohydrates for fuel.

High intensity endurance training is the “maximal training” of endurance training. It is used for developing speed and improving maximal oxygen uptake including the ability to transfer oxygen to the working muscles via an increase in cardiac output (e.g. Daussin et al. 2008). This training is typically executed using shorter intervals that also make use of fast-twitch muscle fibers (type II and type IIx). Currently, there is some debate regarding whether continuous low intensity training or interval training is more effective in improving endurance performance. A study comparing continuous training with interval training over an 8-week period found that both types of training increased  $VO_{2max}$ , but that the increases were explained by different mechanisms. While continuous aerobic training appeared to improve arteriovenous oxygen difference ( $a-vO_{2diff}$ ), interval training appeared to improve cardiac output (Q) as well as  $a-vO_{2diff}$  (Daussin et al. 2008). Time to exhaustion improved more in the interval training group along with mitochondrial function, however, the magnitude of capillarization was higher in the group performing continuous training. Another study by Helgerud et al. (2007) examined the differences between four different training protocols matched for energy consumption. The training protocols included: long slow aerobic distance running at 70% of  $HR_{max}$  for 40 min, lactate (anaerobic) threshold running at 85% of  $HR_{max}$  for 24.25 min, 47 repetitions of 15 s intervals at 90-95% of  $HR_{max}$  alternated with 15 s of active rest at 70% of  $HR_{max}$  and 4 repetitions of 4 min intervals at 90-95%  $HR_{max}$  alternated with 3 min of active rest at 70% of  $HR_{max}$ . The latter two high intensity training modalities were more effective in improving  $VO_{2max}$  and stroke volume than the lower intensity training modalities suggesting that intensity of training is more important than actual training duration (Helgerud et al. 2007). High intensity training is reported to improve both aerobic and anaerobic capacity better than even moderate intensity training (Tabata et al. 1996). Still other studies suggest that similar metabolic adaptations occur following low intensity training and high intensity but lower volume “sprint” interval training over a period of 6 weeks (Burgomaster et al. 2008). Again, no long term studies have examined the differences between aerobic training and high-intensity training adaptations, although it may be suggested that a properly periodized and balanced mixture of training appears to be the most practical.

Decreases in aerobic capacity may result after a reduction or cessation in training. Aerobic capacity can begin to decline in as little as 14 days in trained endurance athletes that stop training (Mujika & Padilla 2001). For example, Mujika and Padilla (2001) reported that the decline in  $VO_{2max}$  of highly trained endurance athletes is proportional to the initial  $VO_{2max}$  with a decline in aerobic capacity typically observed between 4 and 20%. Further changes including increased working heart rate, reduced stroke volume, cardiac output, ventilatory function and aerobic performance influence this decline in aerobic capacity and may occur already within less than two weeks after training cessation (Coyle et al. 1984; Mujika & Padilla 2001). Changes in metabolic responses to endurance

training have also been observed following detraining and appear to be chronic within a relatively short period of time (Petibois & Deleris 2003).

#### **2.2.4 Cardiorespiratory responses and adaptations to strength training**

Although the primary focus of strength training is to improve force production characteristics of the muscles, strength training also has an effect on cardiorespiratory measures. For example, maximal type strength training causes acute increases in heart rate and oxygen consumption that may be high enough, though short in duration, to induce modest gains in cardiorespiratory fitness if performed over a prolonged period of time (Katch et al. 1985). Additional changes in stroke volume/cardiac output may also be observed (Weiner & Baggish 2012) with strength training. As with endurance training, current training status may influence individual cardiorespiratory responses and adaptations to strength training.

Circuit type strength training has been reported to cause modest gains in aerobic capacity because of the heart rate response that can reach approximately 80% of maximum heart rate (Gettman & Pollock 1978). A recent article suggested that using only body weight (muscle endurance) circuit training for 7 minutes at a high intensity will yield similar or even better results than even traditional combinations of strength and endurance training methods, although a training study comparing this method of training to traditional training methods has not been published (Klika & Jordan 2013).

### **2.3 Endocrine responses and adaptations to strength and endurance training**

The endocrine system has an integral role in daily physiological function of humans and plays a substantial role in regulating training adaptations. At rest, the release of hormones is regulated by circadian rhythms/diurnal variations that are influenced by age, sex and climate/environment. Physical stressors like strength and/or endurance training stimulate unique responses from the endocrine system. More specifically, exercise mode, intensity (Tremblay et al. 2004; Linnamo et al. 2005) and duration (Tremblay et al. 2005; Viru et al. 2001) influence hormonal concentrations. When serum hormone concentrations increase, the probability for interaction between hormones and receptors also increases (Smilios et al. 2003). These responses initiate several physiological cascades that may, over time, contribute to training adaptations such as an increase in protein synthesis and muscle hypertrophy (Kraemer & Ratamess 2005), increased capillary density (Andersen & Henriksson 1977), and increased mitochondrial density (Goffart & Wiesner 2003). The subsequent rest/recovery after exercise (Kraemer & Ratamess 2005) plays a considerable role in helping to adapt to acute training-related responses and to maintain physiological homeostasis. In addi-

tion, training status may augment or attenuate specific acute responses (Tremblay et al. 2004) as well as the chronic adaptations to training. In well-trained endurance athletes, for example, suppressed basal testosterone levels in combination with elevated cortisol levels are a typical observation (Hackney et al. 2003), while strength athletes may have higher levels of basal testosterone (Häkkinen et al. 1988b). These resting levels of hormones like testosterone are associated with changes in strength and muscle mass (e.g. Häkkinen et al. 1988b).

Hormonal responses in women are typically more subtle than in men (Häkkinen & Pakarinen 1995; Linnamo et al. 2005; Häkkinen & Pakarinen 1993; Kraemer et al. 1998) and unlike men, the menstrual cycle may have an effect on hormonal responses in women. It appears, however, that e.g. pituitary-adrenal responses to short-term, moderate intensity exercise are not significantly influenced by the menstrual cycle (Galliven et al. 1997), although strict control and observation of the menstrual cycle is still recommended when studying hormonal responses and adaptations to exercise/training in women. Estrogen, while not examined in the current thesis, is worth a brief mention as it may have an effect on muscle contractile properties and has been reported to positively influence muscle repair processes including attenuation of muscle damage and inflammation (Enns & Tiidus 2010), which may warrant further research.

The hypothalamic-pituitary-adrenal (HPA) axis, growth hormone-IGF-1 (GH-IGF-1) axis, and reproductive axis together are affected by physical and psychological stress. The anterior pituitary is responsible for producing follicle stimulating hormone (FSH), luteinizing hormone (LH), adrenocorticotrophic hormone (ACTH), thyroid stimulating hormone (TSH) and growth hormone (GH) (Kraemer & Rogol 2008). Follicle stimulating hormone (FSH) affects growth and development as well as the reproductive processes in the body while luteinizing hormone (LH) plays a role in secretion of sex hormones, like testosterone, produced by the testes and ovaries (Kraemer & Rogol 2008).

Testosterone is an anabolic sex steroid produced primarily by the testes in men (Kraemer & Rogol 2005) and the ovaries in women (Consitt et al. 2002) by conversion of testosterone precursors, androstenedione and dehydroepiandrosterone (DHEA). Testosterone has been linked to the trainability of neuromuscular characteristics of strength and muscle mass (Cumming et al. 1986; Kraemer et al. 1990). This anabolic hormone stimulates growth and protein synthesis in both men and women, while promoting the development of male sex characteristics during puberty (Kraemer & Rogol 2008). Testosterone may increase remarkably as an acute response to both heavy resistance exercise (Kraemer & Ratamess 2005) and endurance exercise (Tremblay et al. 2005), however, endurance exercise (Vuorimaa et al. 1999) and submaximal or explosive type resistance exercise do not always induce as great testosterone response as heavy resistance exercise (Linnamo et al. 2005).

Growth hormone is considered to be an anabolic “family of related polypeptides” and has many variants, the most prominent of which is the 22kDa

version (Nindl 2007). Growth hormone is secreted by the anterior pituitary gland in a pulsatile manner that is influenced by a number of external stimuli including exercise and psychological stress (Hartman et al. 2008) to stimulate protein synthesis, cell reproduction and renewal. Growth hormone and insulin like growth factor-1 (IGF-1) work synergistically and independently to stimulate many metabolic functions (Mauras & Haymond 2005; Kraemer et al. 1995). Insulin like growth factor-1 is produced primarily by the liver and is positively associated with parameters of good health including good cardiovascular fitness and body composition (Nindl et al. 2011). Another important factor in cell growth is TSH, which stimulates thyroid hormone production and secretion, and is regulated by thyrotropin-releasing hormone, which is produced by the hypothalamus. Thyroid hormones stimulate cell growth and are a factor in mitochondrial oxidative metabolism (Kraemer & Rogol 2008). Thyroid stimulating hormone is linked to both physical (Pakarinen et al. 1991) and psychological (Nadolnik 2011) stress. Thus, a temporary state of hypothyroidism may be observed following prolonged endurance exercise (like marathons) (Moore et al. 2005) or other kinds of strenuous exercise like interval training sessions (Hackney et al. 2012) or running at anaerobic threshold until exhaustion (Hackney & Dobridge 2009).

Cortisol release is stimulated by ACTH, and is the primary glucocorticoid in humans. Cortisol is produced by the adrenal cortex, is a catabolic steroid that promotes protein breakdown and glucose production while stimulating fat metabolism (Kraemer & Rogol 2008). High concentrations of cortisol relative to testosterone have been reported to interfere with the anabolic processes that promote muscle hypertrophy, which may in turn negatively affect strength development (Daly et al. 2005; Izquierdo et al. 2004; Häkkinen et al. 1985). Cortisol has a negative effect on the primary immune response (Gleeson 2007) that may affect immunity and may be a maker of overtraining (Häkkinen et al. 1985c, Stone et al. 1991, Adlercreutz et al. 1986). In adequate concentrations; however, cortisol has also been shown to aid in recovery from training (Nindl et al. 2001). Interestingly, a relationship between increased cortisol and decreased thyroid hormone concentrations also appears to exist (Hackney et al. 2012) illustrating how interwoven and complex the various hormones and their responses to exercise or stress are.

Serum hormone concentrations are influenced by the availability of hormone transporters and receptors. Sex hormone binding globulin (SHBG) has a specific "high-affinity" binding site for transporting sex hormones of testosterone and estradiol while IGF-1 binding protein 3 (IGFBP3) transports and helps to control the distribution of the majority of IGF-1 in circulation (Clemmons 1997). Binding of hormones to proteins/globulins allows for an extended period of transport throughout the body, consequently increasing the potential for action.

Hormonal analysis can be affected by a number of possibly confounding factors. The diurnal variations / pulsatile secretion of some hormones have led

researchers to believe that physical testing should always be performed at the same time of day (Sedliak et al. 2007b). Previous studies have shown a substantial decrease in morning testosterone concentrations between 04:00 and 08:00 and a more gradual decrease from 08:00 until 22:00 (Kraemer et al. 2001) while cortisol also appears to be affected by diurnal variations in both young men and women (Häkkinen & Pakarinen 1995; Kanaley et al. 2001), and GH responses are not (Kanaley et al. 2001). Diurnal variations, including nocturnal responses, may obscure some physiological responses (Häkkinen & Pakarinen 1995), however, this is not necessarily true of all hormones. The magnitude of GH response to exercise, for example, appears to be independent of time of day (Kanaley et al. 2001). Finally, nutrition, overtraining/excessive fatigue and stress should also be considered because of their ability to alter normal hormonal responses (Kraemer & Ratamess 2005; Kanaley et al. 1997). These factors that may influence hormones should be taken into consideration in study designs in order to preserve the legitimacy of the hormonal data (Hackney & Viru 2008).

### **2.3.1 Endocrine responses to endurance exercise**

Describing endocrine responses to endurance exercise is challenging due to the many ways of training for endurance. For example, both long slow aerobic distance type workouts and interval training sessions are considered endurance training. Nevertheless, endurance exercise typically induces an acute increase in testosterone and cortisol but after the initial increase of testosterone, a decrease is typically observed (Tremblay et al. 2005). A higher intensity of endurance exercise at or above anaerobic threshold leads to greater stress and, therefore, greater responses than aerobic exercise despite the typically shorter duration (Kindermann et al. 1982). This finding suggests that there may be a specific intensity threshold, possibly related to anaerobic threshold, which is required for hormone release (Weltman et al. 1997). In trained male runners, however, intermittent running at a velocity associated with  $VO_{2max}$  did not change serum concentrations of testosterone, luteinizing hormone, follicle stimulating hormone, or cortisol during 3 days of follow-up (Vuorimaa et al. 1999). In contrast, longer duration running at a submaximal effort appears to result in a greater increase in the catabolic state in trained male runners. When the duration of exercise is long (e.g. 2 h) testosterone levels are typically observed to decrease, while cortisol levels have a tendency to increase (Tremblay et al. 2005). The response of cortisol may parallel those observed in GH concentrations (Viru et al. 2001). Growth hormone concentrations have been observed to increase at the start of aerobic exercise with peak concentrations being identified at or close to the end of aerobic exercise (e.g. Weltman et al. 1992). Growth hormone release magnitude and patterns are similar in men and women during 30 min of aerobic exercise above the anaerobic threshold despite differences in basal serum GH concentrations (Wideman et al. 1999).



### 2.3.2 Endocrine adaptations to endurance training

After a prolonged period of endurance training, a decrease, or no change, in basal concentrations of hormones has been observed in women (Consitt et al. 2002), while decreased testosterone and increased cortisol has been observed in endurance trained men (Hackney et al. 2003). The suppressed testosterone production is suggested to be an endocrinal adaptation to endurance training (Hackney et al. 2003), however, it is not clear whether or not this adaptation is permanent following training cessation. Whether or not suppressed testosterone is a positive or negative adaptation is also debatable. It is possible that suppressed testosterone is actually a positive adaptation for endurance runners due to the influence of a less anabolic environment on anthropometrical adaptations including a decreased tendency towards developing muscle mass (Hackney et al. 2003). Suppressed testosterone and/or increased cortisol have not, however, been observed in all studies on endurance trained athletes. Trained swimmers, for example, had no significant changes in basal levels of cortisol during the training year (Häkkinen et al. 1989). Drastic changes in basal hormonal concentrations are not expected in trained athletes except, perhaps, in the case of overreaching/ overtraining (Stone et al. 1991).

While testosterone and cortisol are the most commonly discussed hormones in training studies, other hormones may also be affected by physical training and/or related stress. Growth hormone response to endurance training appears to be blunted within 3 weeks of starting moderate intensity endurance training on a bicycle. This may be the result of a decreased secretion or enhanced clearance of GH (Weltman et al. 1997). It appears, however, that 1 year of endurance training at an intensity above anaerobic threshold augments the pulsatile release of GH (Weltman et al. 1992). Interestingly, basal levels of TSH are reportedly unaffected by prolonged strength training (Pakarinen et al. 1988) but intensive short-term (1 - week) strength training decreased levels of TSH (Pakarinen et al. 1991), suggesting that lowered TSH is linked to stress (i.e. Nadolnik 2011). Additionally, dehydroepiandrosterone sulfate (DHEAS) has been shown to decrease with exercise related stress and aging, possibly as a result of increased oxidative stress (Aldred et al. 2009). In psychological studies, decreased DHEAS concentrations are also associated with greater stress while higher levels are suggested to have protective qualities against the stress hormone cortisol (Bonne et al. 2004).

### 2.3.3 Endocrine responses to strength exercise

Hormonal responses to strength exercise are typically greater in magnitude than hormonal responses to endurance exercise (Tremblay et al. 2004). In particular, the acute response of anabolic hormones has been of interest because of their role in increasing protein synthesis in the muscles. Anabolic hormones like testosterone, GH, IGF-1 and cortisol are typically observed to increase following strength exercise (Häkkinen & Pakarinen 1995), while testosterone

levels tend to stay elevated following heavy strength exercise (Kraemer & Ratamess 2005; Häkkinen et al. 1998). Studies have indicated that the magnitude of GH response is greater in strength training sessions that cause significant fatigue (Häkkinen & Pakarinen 1993; Kraemer et al. 1990) and amplified with repeated bouts of strength training (Kanaley et al. 1997). Growth hormone concentrations generally increase in response to strength exercise thereby stimulating IGF-1 production, which occurs through both autocrine and paracrine actions.

The acute response of serum hormones to strength training is unique based on the type of loading (McCaulley et al. 2009; Smilios et al. 2003; Häkkinen & Pakarinen 1993). Lower volume strength and power loadings appear to induce a smaller hormone response than high volume loadings with short periods of rest (Kraemer et al. 2010). Similarly, submaximal or explosive type resistance exercises do not always induce as great an anabolic response as more intense strength exercise (Linnamo et al. 2005). Depending on the type of strength training protocol used, an increase in the number of sets may increase hormonal responses, although it appears that there is a limit to the magnitude of increase in hormonal responses that can be achieved (Smilios et al. 2003). In addition to different types of strength loadings, muscle actions appear to influence hormonal responses. For example, larger acute increases in GH have been observed in groups performing strength training that utilized both concentric and eccentric motions, than groups using only concentric movements (Kraemer et al. 2001). Clearly, strength training session intensity and rest periods and different muscle actions stimulate different hormonal responses to strength loadings (McCaulley et al. 2009; Kraemer et al. 1990) (Figure 2), which may be important to consider when prescribing training regimens to different kinds of athletes.

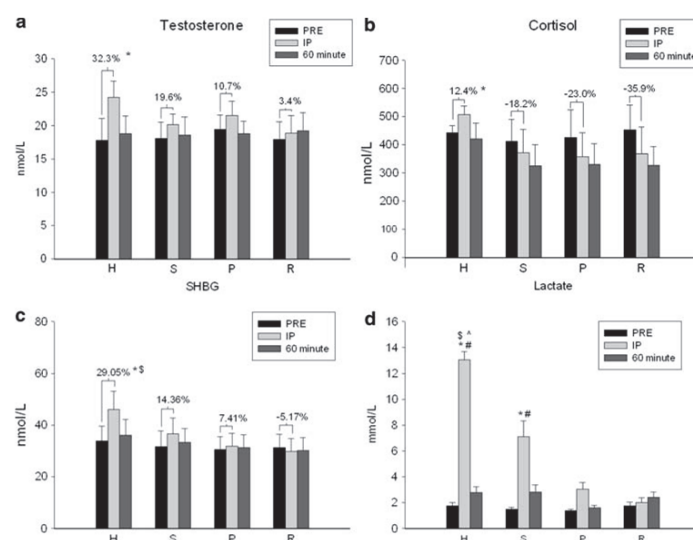


FIGURE 2 Acute response of testosterone, cortisol, sex hormone binding globulin and lactate to hypertrophic (H), strength (S), and power (P) protocols with time of day rest (R) as control. # = significant ( $p < 0.001$ ) difference from pre value, \* = significant ( $p < 0.05$ ) difference from R, \$ = significant ( $p < 0.05$ ) difference from P, ^ = significant ( $p < 0.05$ ) difference from S (from McCauley et al. 2009, reproduced with permission from Springer.)

As previously mentioned, hormonal responses observed in women are typically more subtle than those observed in men. Serum testosterone concentrations in women, for example, might not increase significantly even after heavy resistance training (Häkkinen & Pakarinen 1995; Häkkinen et al. 2000). In women, cortisol has even been observed to decrease in response to strength and endurance exercise (Copeland et al. 2002), although brief increases (Kraemer et al. 1993) and no change (Häkkinen & Pakarinen 1995) have also been observed. It should be noted that the differences in hormones and hormonal responses between men and women are not limited to sex-hormones. For a thorough review of hormone responses to strength and endurance exercise, see Consitt et al. (2002) or Kraemer & Rogol (2005).

### 2.3.4 Endocrine adaptations to strength training

Acute stress may cause hormones to fluctuate, but because the body is under relatively strict homeostatic control, drastic fluctuations in basal levels are not always expected. Basal serum levels of hormones generally change only after a prolonged period of training (Häkkinen 1985). The literature does not give a definitive description of the time-course of prolonged adaptations of the endocrine system to strength training. Due to the complex nature of the endocrine system, it is also difficult to determine if adaptations observed occur in synthesis and storage of hormones or if the mechanisms of transport and clearance have been altered, among other possibilities (Kraemer & Mazetti 2003). Adap-

tations in basal levels of testosterone, however, appear to be dependent, in part, on training status and training period (Häkkinen et al. 1987), whereas basal levels of testosterone have been observed to increase and remain constant following prolonged training (see Kraemer & Ratamess 2005 for a review).

Basal serum concentrations of GH do not appear to be affected by prolonged training (Kraemer et al. 1998), while cortisol appears to be the most dynamic and sensitive of hormones. Cortisol has been reported to increase (Häkkinen & Pakarinen 1991), decrease (Häkkinen et al. 1985c) and remain constant (Häkkinen et al. 1988b) over various training periods. Decreases in TSH may also be linked to both physiological and psychological stress, although prolonged strength training does not appear to significantly alter TSH levels (Pakarinen et al. 1988).

When prolonged training elicits significant stress, hormones may be able to assist in determining the degree of stress and may also be used in monitoring subsequent recovery. For example, the ratio of testosterone to cortisol (a decrease in testosterone and increase in cortisol) may be used as an indicator of overreaching/ overtraining (Stone et al. 1991) and is an indicator of a protein catabolic state (Adlercreutz et al. 1986), while also showing a relationship between strength and hormonal balance over prolonged training (Häkkinen et al. 1985c).

## **2.4 Combined strength and endurance training**

Strength and endurance are needed for daily physical activities as well as a number of different sport disciplines. Current exercise recommendations even advise the healthy adults to perform strength and endurance exercises on a weekly basis for healthy living (Kraemer et al. 2002; Garber et al. 2011). Although endurance training is the primary form of training for an endurance athlete, both strength and endurance training provide unique and shared contributions to distance running performance. Responses and adaptations to strength and endurance, as has already been established, are unique. Thus, we can expect that performing strength and endurance training concurrently should also produce unique responses and adaptations.

### **2.4.1 Neuromuscular responses to a combined training session**

Neuromuscular responses to a single session of combined strength and endurance training may be expected to elicit decreases in force production capabilities and muscle activation similar to a single session of strength or endurance training performed separately. Fatigue at the end of a combined training session may even be hypothesized to be the sum of two individual training sessions (strength + endurance). In healthy recreationally active men, no difference was observed in maximal voluntary force production of the knee extensors when

endurance exercise preceded strength exercise versus endurance exercise alone (Zory et al. 2010). The intensity and volume of endurance and strength exercise employed in this study was relatively low with a mean heart rate of 63% of maximum for endurance exercise for 30 min versus 15 min of aerobic exercise followed immediately by 6 x 10 maximal knee extensions (with 3 min of rest between sets). Aerobic cycling exercise for 45 min at 75% of maximal heart rate appears to negatively affect subsequent back squat performance, but not bench press performance suggesting that potential “interference” between training modes is localized to the concurrently loaded muscles (Schilling et al. 2013).

#### **2.4.2 Cardiorespiratory responses to a combined training session**

Similar to neuromuscular responses, cardiorespiratory responses to a single session of combined strength and endurance training, particularly sessions of low volume and intensity may be expected to produce similar responses. Depending on exercise intensity and duration, substrate utilization expressed in terms of the respiratory exchange ratio might not be expected to change significantly, although there is some evidence that metabolism may be altered, at least immediately post training session, depending on the order of combined exercises performed (Di Blasio et al. 2012). Heart rate response, on the other hand, would likely only be affected by cardiac drift due to changes in hydration status (Lambert et al. 1998). It is worth mentioning that stress, in terms of heart rate variability may also be affected, but discussion of this parameter goes beyond the scope of this thesis.

#### **2.4.3 Endocrine responses to a combined training session**

The theory of training specificity suggests that strength and endurance exercise should elicit specific neuromuscular, cardiorespiratory and endocrine responses. Thus, combining strength and endurance training is complex because of the plethora of training combinations possible. As both strength and endurance are needed for daily living, as well as a number of different sport disciplines, further research regarding combined strength and endurance training is warranted. The primary goal of endurance training is to increase endurance and speed by developing oxidative energy metabolism through increased capillary and mitochondrial density in addition to increased enzyme activity (Bassett & Howley 2000). In contrast, the goal of strength training is to produce considerable gains in strength of the trained muscle groups (Kraemer et al. 2002) by inducing functional and structural adaptations including improved muscle activation and increased protein synthesis via augmented hormonal responses that may result in muscle hypertrophy.

Training specificity is observable in endocrine responses and adaptations to strength and endurance training. These unique responses influence physiological cascades and appear to be at least partially responsible for the interference effect. While these physiological cascades and molecular responses to

training go beyond the scope of this thesis, it is important to note the downstream responses/adaptations to training that are clearly illustrated in a review by Hawley (Hawley 2009), supported by Nader (Nader 2006) (Figure 3) and demonstrated by Putnam (Putman et al. 2004). These molecular responses, as previously mentioned, lead to e.g. muscle hypertrophy (Kraemer & Ratamess 2005) capillarization (Andersen & Henriksson 1977) and mitochondrial biogenesis (Goffart & Wiesner 2003).

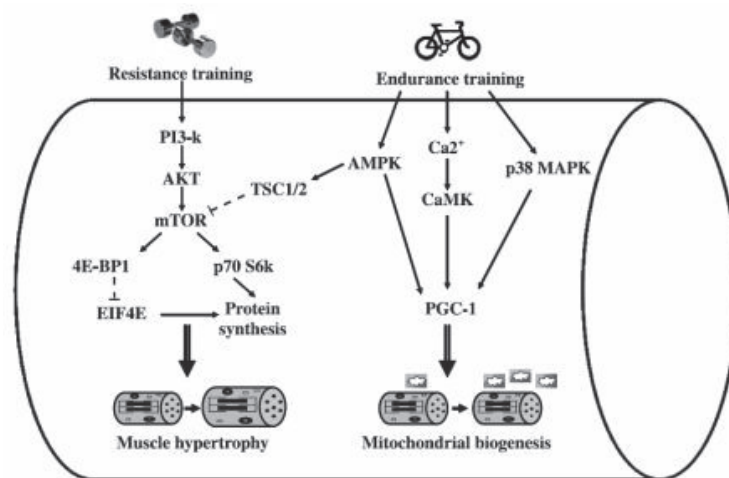


FIGURE 3 The intracellular signaling networks involved in responses to strength and endurance training (from Hawley 2009, reproduced with permission from NRC Research Press).

Clearly, the responses to training are unique with different end results, so what happens when both stimuli are introduced in a single session? In healthy males, performing 60 min of cycling endurance exercise at 50%  $VO_{2max}$  prior to 4 x 10RM of bench press and bilateral leg press, versus resistance training only, no differences were observed in serum testosterone or cortisol responses, but serum GH response was attenuated in the combined group (Goto et al. 2005). In another study, endurance exercise followed by strength exercise has been shown to negatively influence the magnitude of the testosterone, but not the cortisol response in strength trained men (Cadore et al. 2012). When performing two endurance training sessions in a single day (morning and afternoon), the hormone response to the second bout of exercise was greater in terms of ACTH, cortisol and GH, while IGF-1, SHBG, LH, FSH, TSH and FT4 responses were similar to a single bout of exercise (Ronsen et al. 2001). These observations suggest that the physiological milieu of the body can be affected enough by one training session that the following training session will be affected in terms of hormonal response.

#### 2.4.4 Neuromuscular adaptations to combined training

Combined strength and endurance training appears to elicit different adaptations than strength or endurance training alone. Different study designs including unique combinations of strength and endurance training in diverse study populations have presented a variety of results. In untrained individuals, combined strength and endurance training improved both neuromuscular and cardiorespiratory fitness with no indication of interference (Sillanpää et al. 2009). Improvements in force production capabilities in this study by Sillanpää and colleagues (2009) were attributed primarily to enhanced neural activation. In healthy men, over a 21-week training period, combined strength and endurance training caused significant increases in maximal strength and muscle activation, but fast force production appeared to be inhibited by the low volume and intensity endurance training performed on the bicycle (Häkkinen et al. 2003). Bell et al. (2000) examined combined strength and endurance training with strength and endurance only groups as controls. The study showed that strength and hypertrophy were smaller in the combined training groups and suggested that these characteristics were blunted by endurance training (Bell et al. 2000). These findings are in agreement with research by Hunter et al. (1987) and Kraemer et al. (1995). In contrast, McCarthy et al. (2002), using similar groups, found that strength, muscle activation, and hypertrophy were unaffected over a short period of combined training.

Both maximal and explosive strength training combined with endurance training have been shown to be more effective in improving strength, power and muscular activation in recreational endurance runners than combined circuit and endurance training (Mikkola et al. 2011), while an 8-week study of recreational runners performing either combined training or endurance training only found that there were no benefits in running economy despite improvements in strength of the lower extremities (Ferrauti et al. 2010). In this study, strength training for the lower extremities was performed  $1 \cdot \text{week}^{-1}$  with 4 sets of 3-5 RM completed explosively, while a second training session was performed for the trunk only. Endurance exercise was relatively low in volume with an average of  $4 \text{ h} \cdot \text{week}^{-1}$ . The strength training volume and intensity in this study, however, was suggestive of a strength maintenance program. Nevertheless, improved neuromuscular function via strength training may be beneficial over a prolonged period of time. A review of the effects of strength training on endurance capacity in top-level endurance athletes suggests that enhanced endurance performance is the result of improvements in maximal and explosive strength characteristics that are generally accompanied by gains in muscle activation (Aagaard & Andersen 2010). Strength training added to endurance training has been shown to improve endurance performance in runners (Paavolainen et al. 1999c; Støren et al. 2008), Nordic skiers (Hoff et al. 2002, Mikkola et al. 2007; Østerås et al. 2002), and triathletes (Millet et al. 2002) without consistent increases in maximal oxygen uptake. This observation supports the contention that strength training is beneficial for improving performance in endur-

ance athletes. While the vast majority of studies have focused on the lower extremities, the benefits of maximal strength training on endurance performance of the upper body have also been observed in Nordic skiers (Hoff et al. 2002).

#### 2.4.5 Cardiorespiratory adaptations to combined training

Studies examining combined strength and endurance training appear to primarily target neuromuscular capabilities and performance, particularly when subjects are endurance athletes. These neuromuscular capabilities may be independent of, for example,  $VO_{2max}$ . The importance of  $VO_{2max}$  is well established, however, combined strength and endurance training does not appear to induce large increases in  $VO_{2max}$ , when the subjects are already physically active or elite (e.g. Hoff et al. 2002; Millet et al. 2002; Mikkola et al. 2007; Losnegard et al. 2011). This lack of improvement may be related to the relatively low volume and intensity of the endurance training involved compared to the normal training of the subjects, or may be a result of combined strength and endurance training study designs often “replacing” some percentage of endurance training with strength training.

Strength training has been reported to have influence on anaerobic performance characteristics (Mikkola et al. 2007; Tanaka & Swensen 1998), while a number of studies have shown that maximal and/or explosive strength training have a positive influence on sport-specific economy related to improvements in neural characteristics (Nummela et al. 2006; Paavolainen et al. 1999c; Støren et al. 2008; Hoff et al. 2002; Østerås et al. 2002; Millet et al. 2002; Heggelund et al. 2013; Saunders et al. 2004; Ferrauti et al. 2010; Hoff et al. 1999; Sunde et al. 2010). Few studies have reported no changes in economy (e.g. Losnegard et al. 2011). It has been suggested that strength training programs that are successful in increasing maximal strength and fast force production are most effective in improving work economy (Heggelund et al. 2013). It is unclear, however, if improvements in anaerobic characteristics are only a result of neuromuscular adaptations or if other muscular mechanisms behind these changes may also be present. A well-designed study by Barrett-O’Keefe and colleagues (2012) investigated mechanisms behind improved economy by measuring both pulmonary  $VO_2$  and  $VO_2$  of the lower extremities. It was concluded that improved economy was solely the result of decreased use of  $O_2$  in the muscles investigated and not related to actual changes in cardiorespiratory measures (Barrett-O’Keefe et al. 2012) suggesting that the changes in economy are primarily muscular or neuromuscular in nature.

Although there is evidence suggesting that combined strength and endurance training actually helps to improve parameters of endurance performance, improvements in aerobic capacity may actually be blunted by strength training (Glowacki et al. 2004). This finding, however, is not consistent as a greater number of studies have reported that endurance capacity or its development are not interfered with by strength training (e.g. Wilson et al. 2012; Paavolainen et al. 1999c; Mikkola et al. 2007; Millet et al. 2002; Kraemer et al.



1995; Spurrs et al. 2003; McCarthy et al. 1995). Nevertheless, the prospect that resistance training can actually decrease capillary and mitochondrial volume density (MacDougall et al. 1979) or increase muscle mass (e.g. Häkkinen 1994) may deter endurance runners from incorporating strength training into their training program.

#### **2.4.6 Endocrine adaptations to combined training**

As the acute hormonal responses to exercise and combinations of exercises are unique, one might hypothesize that the perturbations in hormonal balance might have an effect on subsequent adaptations. Whether or not these adaptations are observable in basal hormonal levels or in training responses remains somewhat unclear. Basal levels of hormones like testosterone, human GH and SHBG have been shown to remain statistically unaltered while urinary cortisol has increased following combined training (Bell et al. 2000). Changes in the muscle such as an increase in type IIa fibers (Kraemer et al. 1995) and increased capillarization (Bell et al. 2000) suggest that while basal levels of hormones may not be disturbed, the acute responses to training are of great importance. It is clear that the downstream responses/adaptations to strength and endurance training performed separately are different (Hawley 2009; Nader 2006; Putman et al. 2004), and the adaptations to combined strength and endurance training versus strength or endurance training alone are unique. Even though molecular responses to strength training and combined training have been shown to be different, muscle strength and hypertrophy were similar in a study by de Souza et al. (2013) in which a low intensity and low volume of combined training was examined in rats.

#### **2.4.7 Significance of training mode**

As previously described, training mode may have a significant influence on training adaptations. The same is true when combining exercise/training modes. There is some evidence that different combinations of strength and endurance training elicit different responses and adaptations, which can be observed when comparing strength gains across the literature. It appears, however, that when endurance training and strength training modes “match” or are biomechanically similar in terms of movement patterns, interference may be reduced (Gergley 2009). Dissimilar movement patterns, on the other hand, may contribute to interference (Bell et al. 2000). When low volume combined training (2 sessions  $\cdot$  week<sup>-1</sup>) is performed, strength gains may not be affected regardless of endurance training mode and intensity (Silva et al. 2012). Interestingly there is ample evidence to suggest that running will have a greater effect on blunting lower body strength, power and muscle hypertrophy than cycling or strength training alone as evidenced by a recent meta-analysis (Figure 4) (Wilson et al. 2012). A high volume of endurance training in well-trained cyclists, however, also appears to impair adaptations to maximal strength training,

particularly fast force production over 12 weeks of training (Rønnestad et al. 2012).

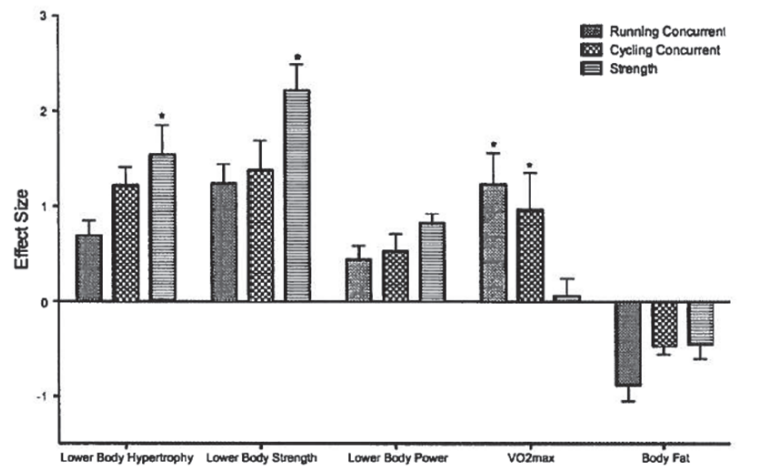


FIGURE 4 Overall effect sizes for running c cycling concurrent (combined), and strength only training: mean  $\pm$  standard error for lower-body strength, lower-body hypertrophy, power,  $VO_{2max}$ , and body fat. \* = Significant  $p < 0.05$  difference from the running concurrent group (from Wilson et al. 2012 reproduced with permission from Wolters Kluwer Health).

Endurance training mode and movement patterns clearly have an influence on strength development. Thus, when combining strength and endurance training, periodization may be a key factor in optimizing training (Armstrong 2006). Biomechanically “matching” of specific strength training exercises with endurance training modes may also contribute to more positive training outcomes (Gergley 2009). In addition, modifying strength programs by not training until failure may help to optimize gains in performance (Izquierdo et al. 2010) by avoiding muscle damage and excessive training fatigue/ stress that could cause interference in training adaptations as well as increasing the risk for overreaching / overtraining.

#### 2.4.8 The “interference effect”

In 1980, Hickson described the “interference effect” suggesting that when a high volume and prolonged period of strength and endurance training are combined, the endurance exercise may cause the typical chronic adaptations to strength training to be blunted (Hickson 1980). In Hickson’s 1980 study, strength development plateaued following 6-8 weeks of training in the combined strength and endurance training groups, while strength development continued after 10 weeks of training in the strength training only group (Figure 5). Hickson (1980) has typically been credited with being the first to document this “interference” phenomenon but subsequent studies (e.g. Hunter et al. 1987)

have demonstrated that combined training does indeed blunt adaptations that are typically observed following single-mode training in terms of hormonal and muscle fiber/ intramuscular adaptations. This observation has also been made by (e.g. Hawley 2009; Nelson et al. 1990; Häkkinen et al. 2003; Kraemer et al. 1995; Chilibeck et al. 2001; Leveritt et al. 1999). While a number of individual studies have demonstrated that interference exists, a recent meta-analysis has confirmed that interference is indeed a function of training mode, frequency and duration of the endurance training mode utilized (Wilson et al. 2012).

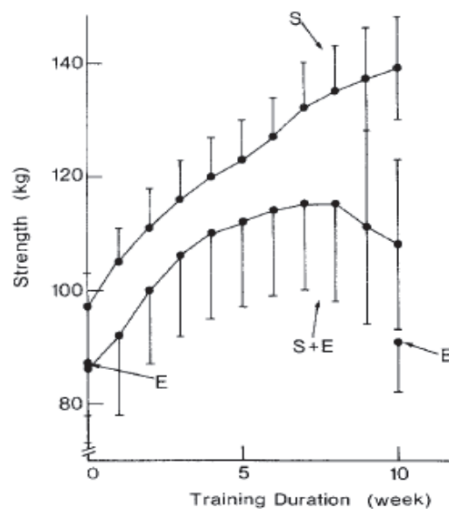


FIGURE 5 The classic interference effect figure illustrating changes in strength over 10 weeks of training in a strength training group (S), endurance training group (E), and a combined strength and endurance training group (S+E) (from Hickson 1980 reproduced with permission from Springer).

Though interference has been observed, research has shown that many combinations of strength and endurance training can actually increase characteristics of strength and endurance including that of Hickson et al. (1988). It appears that when a lower volume and intensity of strength training is combined with a lower volume and intensity of endurance training interference does not occur (e.g. McCarthy et al. 2002; de Souza et al. 2013). In fact, strength training added to endurance training has been shown to improve endurance performance in cross-country runners (orienteerers) (Paavolainen et al. 1999c), Nordic skiers (Hoff et al. 2002; Mikkola et al. 2007; Østerås et al. 2002), triathletes (Millet et al. 2002) and recreational athletes (Häkkinen et al. 2003) while untrained individuals (Sillanpää et al. 2009) have also benefited from combining strength and endurance training. When strength training is added to low intensity endurance training, endurance capacity in terms of  $VO_{2max}$  is not expected to change (unless a decrease in body mass is observed). Instead, augmented neuromuscular

performance including strength, power and muscle activation appears to be responsible for improvements in speed and increased time to exhaustion.

The interference effect has often been studied in terms of adaptations to training, but interference may actually be happening already as an acute response to combined training (Leveritt & Abernethy 1999), meaning that training for both strength and endurance in a single session may attenuate specific neuromuscular, cardiorespiratory and hormonal responses leading to blunted adaptations. Between the accumulation of central or peripheral fatigue possibly affecting the second training session and possible influence of changes in hormonal responses, it is logical to suspect that training responses and adaptations might be affected from neuromuscular and hormonal responses down to the actions of satellite cells (Babcock et al. 2012). On the other hand, it is possible that some combinations of training, over a short period of time, actually enhance responses and/or adaptations (Lundberg et al. 2013). For example, when resistance training with 6 sets of leg press using loads of 70-80% 1RM follows endurance cycling at  $\sim 65\%$   $VO_{2max}$  it appears that the processes that promote muscle oxidative capacity and proteins synthesis are enhanced when compared to endurance cycling alone (Wang et al. 2011).

#### **2.4.9 The “order effect”**

A slightly more recent phenomenon of interest regarding combined strength and endurance training is the “order effect”. How might the order of combined strength and endurance training sessions affect or influence the responses and subsequent adaptations to training? Sale et al. (1990) have speculated that when strength and endurance are combined, fatigue may affect the quality of the subsequent training session. Does performing a training session blunt or intensify responses to a second training session? It may be hypothesized that the order of combined exercise will induce fatigue that is specific and may differ in magnitude. This fatigue may subsequently affect the next training session (Schilling et al. 2013). Running economy, for example, appears to be impaired following a bout of strength training exercise, which may be related to neuromuscular fatigue resulting from a temporary decrease in contractile properties of the muscles (Palmer & Sleivert 2001). How much decreased running economy might affect the actual endurance training session is unclear, but one might hypothesize that if the magnitude of fatigue is significant, the training session may be compromised and the likelihood of e.g. injury or even unintentional overreaching may be increased.

While running economy appears to be impaired, a study investigating the cardiorespiratory and metabolic effects of the intra-session order of exercise found that energy expenditure,  $VO_2$ , and ventilation were similar following loadings in which moderate intensity strength (using 55% 1RM) and endurance (30 min at 60% HR reserve) were combined. Only rating of perceived exertion was reported to be higher following the loadings when strength training preceded endurance training and this finding was paralleled by significantly high-

er fat oxidation (Di Blasio et al. 2012). Another study combining short intervals during 20 minutes of cycling with muscle endurance strength training found no difference between any of the measured cardiorespiratory parameters (Vilacxa Alves et al. 2012). Unfortunately, these studies did not examine any neuromuscular variables or parameters of recovery.

Decreased force production was reported to be similar following a combined endurance and strength session as a single endurance session when the endurance training mode was cycling (Zory et al. 2010). Similarly, no difference was observed in maximal isometric force production, rapid force production, rate of force development or power when a combined power, maximal strength, and hypertrophic strength training session of 30 min in duration and 30 min of moderate intensity (65% of each subject's individual maximal watts) cycling on an ergometer were performed in a single session using two different orders (Schumann et al. 2013). Interestingly, however, serum testosterone concentrations were observed to be decreased relative to baseline during 2 days of recovery following the loading in which the strength training session preceded the endurance training session (Schumann et al. 2013). These studies clearly suggest that the combination, mode and intensity of training may influence acute responses to a training session.

The "order effect" is observed both on an acute level, as well as after training. Bell et al. (1988) examined the "order effect" over a short period of training by examining high-velocity resistance training in combination with endurance training in oarsmen. Half the group completed endurance training followed by strength training (ES) within a single training session while the other half completed strength training followed by endurance training (SE). Following five weeks of this combined training, Bell and colleagues (1988) concluded that the sequence of training can indeed influence the physiological responses and adaptations to training. The study demonstrated that the improvements in peak torque for knee extension and flexion following resistance training were larger in the SE group than those in ES.

A pair of studies examining intra-session order of exercise over 12 weeks of training in young males showed that when endurance was performed prior to strength in a single session, improvements in endurance performance and aerobic capacity were superior to single session strength performed prior to endurance (Chtara et al. 2005). In contrast, it was observed that the order of exercise did not influence maximal and explosive strength development (Chtara et al. 2008). The strength training utilized in these studies consisted of strength endurance and explosive type exercises. In contrast, in elderly males, performing strength prior to endurance appears to be more beneficial for strength development when progressing from 20RM to 6RM over 12 weeks (Cadore et al. 2011).

While there is some evidence that the order of exercises may influence training adaptations, the acute responses to combined strength and endurance exercise performed in a single training session and the effects that the order of

training might have on these acute responses have not yet been fully elucidated. From a scientific perspective and for practical purposes, the influence of the order of exercises may be of importance in order to optimize training.

### **3 PURPOSE OF THE STUDY**

The purpose of this study was to examine the chronic neuromuscular, cardiorespiratory and hormonal adaptations to combined strength and endurance training and to evaluate the acute neuromuscular, cardiorespiratory, and hormonal responses to combined strength and endurance training sessions using two different intra-session loading orders.

#### **Papers I, II, III**

The first three papers examined the long-term adaptations to periodized combined strength and endurance training in recreational endurance trained men and women with specific reference to the effects of strength training induced neuromuscular adaptations on parameters of endurance performance and hormonal adaptations.

The main objectives were to:

- 1) Examine the effects of periodized maximal, explosive, and mixed maximal and explosive strength training combined with endurance training in male recreational endurance runners. This included examination of the effects of prolonged reduced strength training volume, accompanied by increased endurance training volume, on strength maintenance and endurance performance.
- 2) Evaluate the differences in adaptations between men and women to mixed maximal and explosive strength training combined with endurance training.
- 3) Assess possible changes in serum basal hormone concentrations over the prolonged combined strength and endurance training periods in both men and women.

**Papers IV and V**

The purpose of the final two papers was to examine the acute neuromuscular, cardiorespiratory and hormonal responses to strength (S) and endurance (E) loadings performed in a single combined training session.

The main objectives were to:

- 1) Examine the phenomenon known as the “order effect” including the possible differences in acute neuromuscular, cardiorespiratory and hormonal responses to a training session when the order of loadings in a single session are reversed (ES vs. SE). This investigation also took into consideration the time-course of fatigue and recovery (24 and 48 h post-loading) resulting from ES and SE.
- 2) Examine the possible differences in responses between male and female subjects with a recreational background in endurance training.



## **4 RESEARCH METHODS**

### **4.1 Subjects**

Healthy men and women, 20-45 years of age with a recreational background in endurance training, were recruited to participate in these studies comprising papers I-V. Exclusion criteria included BMI > 28 kg/m<sup>2</sup> (to exclude pronounced overweight), illness, disease, injury, and use of any medications that might contraindicate strenuous exercise. A physician screened each subjects' health, health history and electrocardiogram (ECG) prior to giving clearance to participate in the study.

### **4.2 Experimental design**

#### **4.2.1 Papers I, II and III**

A longitudinal study design was used to examine the chronic adaptations to combined strength and endurance training over a prolonged period of time in both men and women (Figure 6). The first 6-8 weeks of the study were used for familiarizing subjects with strength training. The purpose of this preparatory strength training period was for subjects to learn proper lifting technique prior to the intervention. As subjects were previously untrained in terms of strength, it was important to include this period in order to help subjects avoid injury while reducing the effect of learning on strength gains that may have otherwise occurred during the intervention period.

The 8-week strength training intervention was the primary focus of the research and was used because 8-weeks of strength training after a 6-8 week preparatory period of strength training should be long enough to show differences between training modes (e.g. Häkkinen & Komi 1981). The 14-week "marathon

training” and reduced strength training follow-up period (for maximal and explosive experimental groups as well as the control group) was used to observe the possible lasting effects of strength training for endurance runners while they trained by increasing endurance training volume and intensity and reducing strength training volume in preparation for a marathon or half marathon. During the 8-week intervention, male and female subjects were divided into groups (Table 1).

TABLE 1 Training groups for papers I-III.

| Men                              | Women                            |
|----------------------------------|----------------------------------|
| Maximal (M)                      | Mixed maximal and explosive (MW) |
| Explosive (E)                    | Circuit training control (CW)    |
| Mixed maximal and explosive (MM) |                                  |
| Circuit training control (CM)    |                                  |

Testing at pre, weeks 0, 4, 8 and post included blood samples for serum hormones, body composition measurements including body fat %, body weight and muscle thickness, maximal bilateral isometric force (MVC) and rate of force development (RFD), maximal bilateral dynamic strength (1RM), countermovement jump (CMJ) and muscle activation (EMG). A test for maximal oxygen uptake (including aerobic and anaerobic thresholds and running economy) was measured at pre, weeks 0 and 8, as well as post. Only M, E and CM completed the 14-week “marathon training” period.

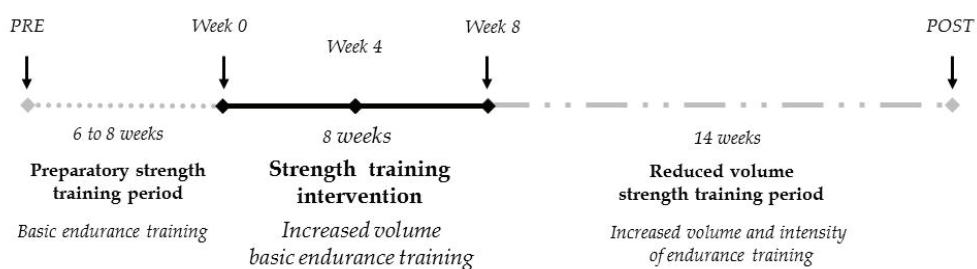


FIGURE 6 The effects of periodized combined strength and endurance training on neuromuscular, cardiorespiratory and hormonal variables were examined in recreationally trained endurance subjects.

### 4.2.2 Papers IV and V

A cross-sectional study design was used to examine the acute responses to combined endurance and strength exercise when performed concurrently in a single training session by recreational endurance runners (Figure 7). Male and female subjects performed two combined sessions in random order and separated by at least one week. One combined session started with an endurance loading, which was immediately followed by a strength loading (ES), while the other combined session started with a strength loading, which was immediately followed by an endurance loading (SE). The combined sessions for papers IV and V were performed in random order, thus we do not expect that there is any kind of training adaptation or “repeated bout effect” that is influencing the results (Proske & Allen 2005).

Testing at pre, mid, post, 24 and 48 h included MVC, RFD, CMJ, and blood samples for blood lactate and serum hormones. Electromyography was measured at pre, mid, and post, while running economy and respiratory exchange ratio (using a portable gas analyzer) as well as heart rate were measured during the endurance loading only.

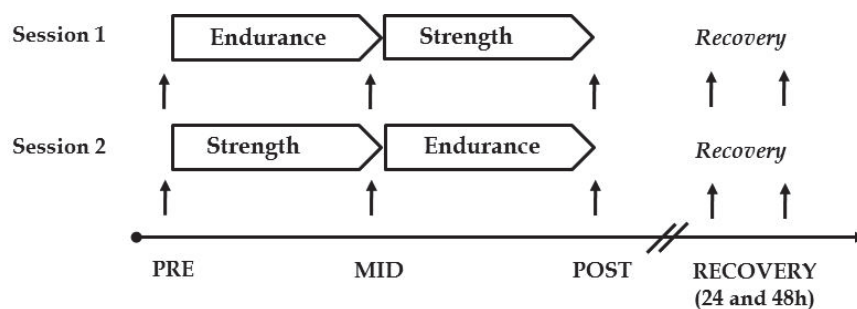


FIGURE 7 Cross-sectional study of combined strength and endurance training sessions including endurance + strength (ES) and strength + endurance (SE).

## 4.3 Data collection

### 4.3.1 Cardiorespiratory performance

Maximal aerobic capacity was measured by maximal oxygen uptake ( $VO_{2max}$ ) using a treadmill running protocol (Mikkola et al. 2007). Running velocity began at  $7 \text{ km} \cdot \text{h}^{-1}$  for women and  $8 \text{ km} \cdot \text{h}^{-1}$  for men. Running velocity was increased by  $1 \text{ km} \cdot \text{h}^{-1}$  every third minute until volitional exhaustion. Treadmill incline remained a constant 0.5 degrees. Heart rate was recorded continuously using a heart rate monitor (Suunto t6, Vantaa, Finland). Mean heart rate values

from the last minute of each stage were used for analysis. Oxygen consumption was measured breath-by-breath throughout the test using a portable gas analyzer (Oxycon Mobile®, Jaeger, Hoechberg, Germany) and  $\dot{V}O_{2\max}$  was accepted as the highest average 60 s  $\dot{V}O_2$  value. For blood lactate sampling, fingertip samples were taken every 3 min while the treadmill was stopped for approximately 15-20 s (see 4.3.5 for details on blood lactate analysis). Running economy (RE) and the velocity of running at  $\dot{V}O_{2\max}$  ( $S_{\text{peak}}$ ) were calculated:  $S_{\text{peak}} = \text{speed of the last whole completed stage (km} \cdot \text{h}^{-1}) + (\text{running time (s) of the speed at exhaustion} - 30 \text{ seconds}) / (180 - 30 \text{ seconds}) * 1 \text{ km} \cdot \text{h}^{-1}$ . Aerobic (AerT) and anaerobic (AnT) thresholds were determined using blood lactate, ventilation,  $\dot{V}O_2$  and  $\dot{V}O_2$  according to Aunola and Rusko (1986).

### 4.3.2 Neuromuscular performance

#### Concentric one repetition maximum

Concentric one repetition maximum (maximal dynamic bilateral horizontal leg press in a seated position) was measured using a David 210 dynamometer (David Sports Ltd., Helsinki, Finland) (Häkkinen et al. 1998). Prior to attempting 1RM, subjects completed a warm-up, after which no more than 5 attempts to reach 1RM were made. The knee angle at the beginning of the leg extension was approximately 65 degrees. Subjects were instructed to grasp handles located by the seat of the dynamometer and to keep constant contact with the seat and backrest during leg extension to a full 180 degrees. The greatest weight that the subject could successfully lift (knees fully extended) was accepted as 1RM.

#### Isometric strength

An electromechanical isometric leg extension device (Department of Biology of Physical Activity, University of Jyväskylä, Finland) was used in all studies to measure maximal strength and rate of force development. The subjects' knee angle was 107° measured using the greater trochanter, lateral tibiofemoral joint space and lateral malleolus as reference points. The hip angle was 110°. In pre-testing, subjects performed at least three maximal voluntary contractions (see Häkkinen et al. 1998 for details). Subjects were instructed to push "as fast and as hard as possible" for approximately 3 s. If the maximum force during trials was greater than 5% compared to the previous trial, an additional trial was performed until a plateau was reached.

#### Countermovement jump

A force platform (Department of Biology of Physical Activity, Jyväskylä, Finland) was used to measure maximal dynamic explosive force by countermovement jump height (Komi & Bosco 1978). Subjects were instructed to stand with their feet approximately hip-width apart with their hands on their hips. The countermovement jump action was then demonstrated and explained to be quick and explosive so that knee angle for the jump was no less than 90°. Force data was collected and analyzed by computer software (Signal 2.14, CED, Cam-

bridge, UK), which used the equation  $h = I^2 \cdot 2gm^{-2}$  to calculate jump height from impulse ( $I$  = impulse,  $g$  = gravity and  $m$  = mass of subject).

### **Electromyographic activity**

Electromyographic activity (EMG) was recorded from the vastus lateralis (VL), vastus medialis (VM) of the right leg during 1RM, isometric MVC and CMJ at a sampling frequency of 2000 Hz, and then filtered (20 Hz low pass filter for force and 20–350 Hz band pass filter for EMG signals following amplification at gain of 500 and sampling bandwidth of 10–500 Hz). Electrode positions were marked with small ink tattoos (Häkkinen & Komi 1983) on the skin during the first testing session to ensure that electrode placement over the entire experimental period would be consistent. The guidelines published by SENIAM (Hermens et al. 1999) were followed for skin preparation, electrode placement and orientation. Inter-electrode distance was 20 mm (input impedance < 10 k $\Omega$ , common mode rejection ratio 80 dB, 1000 gain). Raw signals were passed from a transmitter, positioned around the subjects' waist, to a receiver (Telemetry 2400R, Noraxon, Scottsdale, AZ, USA) from which the signal was relayed to a computer via an AD converter (Micro1401, CED, Cambridge, UK). EMG was recorded and subsequently analyzed by computer software (Signal 2.14, CED, UK). During 1RM, whole range EMG was recorded from the starting knee angle approximately 65 degrees to full leg extension of 180 degrees. In CMJ, EMG was examined during the concentric phase of the movement as determined by the force signal (knee angle of approximately 90 degrees to full leg extension of 180 degrees). Maximal root mean square EMG from the concentric phase of each movement was analyzed by a customized script (Signal 4.04, CED, UK) and was used in further analysis.

### **4.3.3 Anthropometrics**

#### **Body composition**

Standing height was measured using standard laboratory techniques. Body mass and body composition were measured using a bioimpedance device (In-Body720 body composition analyzer, Biospace Co. Ltd, Seoul, South Korea). Measurements were taken in conjunction with blood tests between 07.30 - 08.00 am. Subjects always arrived for testing in a fasted state, thus keeping the possible confounding variables of diet and hydration status to a minimum.

#### **Muscle thickness**

Muscle thickness of vastus lateralis (VL) and vastus intermedius (VI) were measured using a compound ultrasound scanner (Aloka SSD-2000, Aloka Co., Tokyo, Japan) (Häkkinen et al. 2006) (Studies IV and V). The subject's legs were secured with a belt at the ankles and the knees were supported to avoid movement. Thickness was measured at the anterior surface of the leg at 50% length of the femur measured from the lateral aspect of the distal diaphysis to the greater trochanter (Seynnes et al. 2007), which was marked with an ink tattoo. Water-

soluble transmission gel was used to avoid unnecessary tissue compression by the probe and the probe was adjusted manually until a clear image was achieved. Muscle thickness was calculated from the average of three consecutive muscle thickness measurements (VL + VI).

#### 4.3.4 Blood samples

##### Basic blood count

Venous blood samples (10 ml) were drawn from the antecubital vein using sterile needles into serum tubes (Venosafe, Terumo Medical Co., Leuven, Belgium) by a qualified lab technician for examination of basic blood count (KN-21N Sysmex, Japan). Subjects were tested after 12 h of fasting between 07.30 - 08.00 am (papers I-IV).

##### Blood lactate

Blood lactates were analyzed using a Biosen S\_line Lab+ lactate analyzer (EKF Diagnostic, Magdeburg, Germany).

##### Serum hormones

Venous blood samples (10 ml) were drawn from the antecubital vein using sterile needles into serum tubes (Venosafe, Terumo Medical Co., Leuven, Belgium) by a qualified lab technician for examination of serum hormones. Subjects were tested after 12 h of fasting between 07:30-08:00 am (papers I-IV) and also the same time of day as their respective loadings and follow-up measurements in order to take into consideration normal daily variation in hormone concentrations (paper IV). Samples were taken from a seated position with the arm extended. Whole blood was centrifuged at 2500 g (Megafuge 1.0R, Heraeus, Germany) for 10 min after which serum was removed and stored at -80°C until analysis. Samples were used for determination of serum hormone concentrations. Analyses were performed using chemical luminescence techniques (Immunlite 1000, DCP Diagnostics Corporation, Los Angeles, California, USA) and hormone specific immunoassay kits (Siemens, New York, NY, USA). For papers I-III blood samples were used for the determination of testosterone, cortisol and thyroid stimulating hormone. The sensitivity of testosterone, cortisol and TSH assays were  $0.5 \text{ nmol} \cdot \text{l}^{-1}$ ,  $5.5 \text{ nmol} \cdot \text{l}^{-1}$  and  $0.004 \text{ mIU} \cdot \text{l}^{-1}$ , respectively. The intra-assay coefficients of variation for testosterone, cortisol, and TSH were: 5.7 %, 4.6 % and 3.9 %, respectively. For paper IV, blood samples were used for determination of serum testosterone, cortisol, GH, IGF-1 and SHBG. The sensitivity of testosterone, cortisol, GH, IGF-1, SHBG and IGF binding protein 3 assays were:  $0.5 \text{ nmol} \cdot \text{l}^{-1}$ ,  $5.5 \text{ nmol} \cdot \text{l}^{-1}$ ,  $0.026 \text{ mIU} \cdot \text{l}^{-1}$ ,  $2.6 \text{ nmol} \cdot \text{l}^{-1}$ ,  $5.5 \text{ nmol} \cdot \text{l}^{-1}$ , and  $0.1 \mu\text{g} \cdot \text{ml}^{-1}$ , respectively. The intra-assay coefficients of variation for testosterone, cortisol, GH, IGF-1, SHBG, and IGF-1 BP-3 were: 5.7, 4.6, 4.2, 3.1, 2.4, and 4.4 % respectively.

#### **4.3.5 Ethical considerations**

Ethical approval for methodology and procedures was granted by the Ethical Committee of the University of Jyväskylä. All studies were conducted according to the provisions of the most recent Declaration of Helsinki. Subjects received written and oral information about the study design and measurement procedures including information on the possible risks and benefits of participation. Subjects then signed two copies of an informed consent document (for the subjects' records and research records). A resting electrocardiogram and health history questionnaires filled out by each subject were reviewed and approved by a physician prior to participation in the study.

### **4.4 Training programs (Papers I, II and III)**

#### **4.4.1 Strength training**

Throughout the study, strength training was focused on the lower extremities, because of the major role in running as well as daily locomotion of humans. During the 6 to 8-week preparatory period, all subjects trained using exercises similar to those used by the experimental groups during the strength training intervention. Loads during the preparatory strength training period progressed from 50 to 70 % 1RM. The planned strength training frequency for the preparatory period was 3 sessions  $\cdot$  2 weeks<sup>-1</sup> (1.5 sessions  $\cdot$  week<sup>-1</sup>).

Following the preparatory period, the experimental groups began their specified maximal, explosive or mixed maximal and explosive strength training and the control groups began circuit training for a period of 8 weeks (training programs in Table 2). Strength training for all participants was planned as 2 sessions  $\cdot$  week<sup>-1</sup> (training adherence is reported in the results section).

Maximal, explosive and circuit training control groups of men continued using the same strength exercises with reduced training frequency and volume during the 14-week "marathon training" period.

TABLE 2 Strength training programs.

| <b>STRENGTH TRAINING INTERVENTION PROGRAMS</b>   |             |                    |  |                       |
|--|-------------|--------------------|--|-----------------------|
|  | <b>Sets</b> | <b>Repetitions</b> | <b>Load</b>                                    | <b>Recovery (min)</b> |
| <b>MAXIMAL STRENGTH</b>  |             |                    |  |                       |
| <b>Squat:</b>  | 3           | 4-6                | 80 - 85% 1RM                                   | 2                     |
| <b>Leg Press:</b>  | 3           | 4-6                | 80 - 85% 1RM                                   | 2                     |
| <b>Calf Exercise</b>   | 2           | 12-15              | 50 - 60% 1RM                                   | 2                     |
| <b>Sit-ups, back-extension</b>   | 3           | 20-30              | Body weight                                    | 2                     |
| <b>EXPLOSIVE POWER</b>   |             |                    |  |                       |
| <b>Squat:</b>  | 3           | 6                  | 30 - 40% 1RM                                   | 2                     |
| <b>Leg Press:</b>  | 3           | 6                  | 30 - 40% 1RM                                   | 2                     |
| <b>Scissor jump:</b>   | 2-3         | 10s                | 20 kg  | 2                     |
| <b>Squat jump:</b>   | 2-3         | 5 single           | Body weight                                    | 2                     |
| <b>Squat jump:</b>   | 2-3         | 5 in series        | *Body weight                                   | 2                     |
| <b>Sit-ups, back-extension</b>   | 3           | 20-30              | Body weight<br><i>*(20 kg between 4 and 8)</i> | 2                     |
| <b>MIXED MAXIMAL STRENGTH + EXPLOSIVE POWER</b>  |             |                    |  |                       |
| <b>Squat</b> (week 0 – week 4):  | 2           | 6                  | 80 - 85% 1RM                                   | 3                     |
| <b>Leg press</b> (week 0 – week 4):  | 2           | 6                  | 80 - 85% 1RM                                   | 3                     |
| <b>Squat/Leg Press</b> (week 4- week 8):   | 3           | 4                  | 4RM  | 3                     |
| <b>Box jumps</b>   | 2-3         | 8-10               | Body weight                                    | 2-3                   |
| <b>Squat jumps</b>   | 2-3         | 8-10               | Body weight                                    | 2-3                   |
| <b>Sit-ups, back-extension</b>   | 3           | 20-30              | Body weight                                    | 2                     |
| <b>CIRCUIT TRAINING</b>  |             |                    |  |                       |
| <b>Squats, lunges, step-ups, push-ups, sit-ups, calf-raises, back-extensions, planks</b> | 3           | 45-50s             | Body weight                                    | 15-10s                |

#### 4.4.2 Endurance training

All subjects performed endurance training throughout the study. Endurance training was typically performed on non-strength training days with a training intensity below the aerobic threshold throughout the study. On strength training days, a low intensity warm-up of 20-30 min was typically performed involving running or biking. Training intensities, based on aerobic and anaerobic thresholds (Aunola & Rusko 1986), were individually determined for each subject each time they were tested for maximal oxygen uptake. Subjects were informed of their individual heart rate ranges between which to train, and used a heart rate monitor (Suunto t6, Vantaa, Finland) to monitor and record each training session. Endurance training was individualized for subjects based on their training background and current fitness level. The relative volume and



intensity of training, however, were similar between subjects. Running volume increased progressively for all subjects over the entire study starting with an average of  $20 \pm 13 \text{ km} \cdot \text{week}^{-1}$  of endurance running during the 6 to 8-week preparatory period, increasing to  $24 \pm 14 \text{ km} \cdot \text{week}^{-1}$  of running during the strength training intervention and  $38 \pm 5 \text{ km} \cdot \text{week}^{-1}$  of running during the marathon training period (14 weeks, for group details, see results). Subjects kept personal training diaries throughout the study recording strength training sessions, kilometers and duration of running and “other” endurance activity including cycling, cross-country skiing and Nordic walking.

## 4.5 Combined session protocols (Papers IV and V)

Subjects performed two combined strength and endurance sessions of equal volume, duration, and intensity. The order of the combined sessions was random with each subject performing one session in which the strength loading was immediately followed by the endurance loading (SE), and a second session in which the endurance loading was immediately followed by the strength loading (ES).

### 4.5.1 Strength loading

The strength loading focused primarily on the leg extensors and included both maximal and explosive exercises (Table 3). Strength exercises were performed in a circuit such that leg press exercises were performed at the beginning, middle and end of both combined sessions. The total duration of the strength loading was approximately 45 min. Sets, repetitions, loads and rest were recorded from a familiarization session and used to calculate workload (sets x repetitions x load) in order to match the strength loadings between combined ES and SE sessions.

TABLE 3 Strength loading for combined sessions (papers IV-V).

| <b>STRENGTH LOADING</b>   |             |                    |              |                       |
|---------------------------|-------------|--------------------|--------------|-----------------------|
|                           | <b>Sets</b> | <b>Repetitions</b> | <b>Load</b>  | <b>Recovery (min)</b> |
| <b>MAXIMAL STRENGTH</b>   |             |                    |              |                       |
| <b>Leg Press:</b>         | 3           | 5-8                | 70 - 85% 1RM | 2                     |
| <b>Squat:</b>             | 3           | 5-8                | 70 - 85% 1RM | 2                     |
| <b>Calf Exercise:</b>     | 2           | 5-8                | 70 - 85% 1RM | 2                     |
| <b>EXPLOSIVE STRENGTH</b> |             |                    |              |                       |
| <b>Leg Press:</b>         | 3           | 8-10               | 30 - 40% 1RM | 2                     |
| <b>Loaded Squat Jump:</b> | 3           | 8-10               | 30 - 40% 1RM | 2                     |

#### **4.5.2 Endurance loading**

The endurance loading was performed by steady-state running on a 200 m indoor track. The intensity of running was between each subjects' previously determined individual aerobic and anaerobic thresholds for a duration of 60 min.

### **4.6 Statistical analyses**

Standard statistical methods were used for calculation of means and standard deviations (SD) in all studies. Group differences were analyzed using a one-way analysis of variance (One-way ANOVA) and within group differences (group-by-training interaction) was analyzed using repeated measures factorial ANOVA (time  $\times$  sex  $\times$  group) in papers I, II and III. In studies IV and V, group differences and group-by-loading interaction were analyzed by a repeated analysis using mixed models and an unstructured covariance matrix. Groups were compared with least significant difference post hoc analysis in a mixed models analysis when appropriate. In the presence of a significant F-value, post-hoc comparison of means was provided by the appropriate test. The criterion for significance was  $p \leq 0.05$ . Statistical analyses were completed with SPSSWIN 14.0 - 18.0 (SPSS Inc., Chicago, IL, USA).

## 5 RESULTS

### 5.1 Training (I, II, III)

After baseline testing, subjects were divided into groups (Table 4) in which they performed the same type of combined strength and endurance training for the 6 to 8-week preparatory period followed by specified strength training during the actual 8-week strength training intervention. The maximal (M), explosive (E) and control group of men (CM) continued combined strength and endurance training for an additional 14 weeks, while the mixed maximal and explosive strength training men (MM) and women (MW) and the women's control group (CW) did not. Two subjects were discarded from analysis due to low training compliance.

TABLE 4 Baseline data for subjects in papers I, II and III.

|                       | AGE        | HEIGHT (cm) | WEIGHT (kg) | % FAT      |
|-----------------------|------------|-------------|-------------|------------|
| Maximal (n = 11)      | 35.5 ± 6.1 | 178.5 ± 4.7 | 77.2 ± 5.5  | 17.1 ± 4.4 |
| Explosive (n = 10)    | 36.5 ± 6.5 | 180.5 ± 6.4 | 78.4 ± 6.3  | 17.2 ± 5.5 |
| Mixed Men (n = 9)     | 31.3 ± 8.9 | 178.1 ± 5.3 | 79.5 ± 6.5  | 18.2 ± 4.2 |
| Control Men (n = 7)   | 33.7 ± 8.8 | 180.0 ± 4.8 | 83.8 ± 10.5 | 21.1 ± 5.9 |
| Mixed Women (n = 9)   | 29.1 ± 7.0 | 168.1 ± 4.4 | 62.9 ± 4.3  | 24.8 ± 4.1 |
| Control Women (n = 9) | 35.2 ± 6.1 | 164.4 ± 6.7 | 59.9 ± 7.4  | 24.3 ± 6.3 |

During the preparatory strength training period, strength training in all groups was performed an average of  $1.0 \pm 0.6$  sessions  $\cdot$  week<sup>-1</sup>, while during the strength training intervention, strength training was performed an average of  $1.5 \pm 0.5$  sessions  $\cdot$  week<sup>-1</sup> (Table 5). Endurance running volume (average kilometers  $\cdot$  week<sup>-1</sup>) over the preparatory period and strength training intervention increased progressively in all groups (Table 5).

The 14-week period of reduced volume strength training and increased volume and intensity of endurance training included strength training  $0.6 \pm 0.1$ ,  $0.5 \pm 0.1$  and  $0.5 \pm 0.1$  times  $\cdot$  week<sup>-1</sup> for M, E and CM groups, respectively. The average running distances were  $38.1 \pm 3.4$ ,  $40.8 \pm 5.6$  and  $36.4 \pm 5.3$  kilometers  $\cdot$  week<sup>-1</sup> for M, E and CW groups, respectively. Running volume per week increased significantly in all groups over the preparatory period, strength training intervention and 14-week "marathon training period" ( $p < 0.01$ ). "Other" endurance activity including cycling, cross-country skiing and Nordic walking remained a small portion of training in each group over the 3 training periods.

TABLE 5 Strength and endurance training volume and frequency over the preparatory strength training period (6-8 weeks), 8-week strength training intervention and 14-week marathon training period. From preparatory \*\* =  $p < 0.01$ , \*\*\* =  $p < 0.001$ , from intervention ++ =  $p < 0.001$ , +++ =  $p < 0.001$ . Group differences a-a, b-b =  $p < 0.05$ .

|  | PREPARATORY<br>(6-8weeks) | INTERVENTION<br>(8 weeks) | MARATHON PERIOD<br>(14 weeks) |
|--|---------------------------|---------------------------|-------------------------------|
| <b>Strength training frequency (sessions <math>\cdot</math> week<sup>-1</sup>)</b>               |                           |                           |                               |
| Maximal  | $1.2 \pm 0.7$             | $1.6 \pm 0.4^{***}$       | $0.6 \pm 0.1$                 |
| Explosive  | $1.2 \pm 0.7$             | $1.6 \pm 0.4^{***}$       | $0.5 \pm 0.1$                 |
| Mixed Men  | $0.7 \pm 0.5$             | $1.4 \pm 0.5^{***}$       | ---                           |
| Control Men  | $0.8 \pm 0.6$             | $1.3 \pm 0.7^{***}$       | $0.5 \pm 0.1$                 |
| Mixed Women  | $1.0 \pm 0.6$             | $1.5 \pm 0.4^{***}$       | ---                           |
| Control Women  | $0.9 \pm 0.6$             | $1.4 \pm 0.6^{***}$       | ---                           |
| <b>Running volume (average kilometers <math>\cdot</math> week<sup>-1</sup>)</b>                  |                           |                           |                               |
| Maximal  | $19.7 \pm 13.8$           | $27.0 \pm 15.5^{**}$      | $38.1 \pm 3.4^{***,+++}$      |
| Explosive  | $23.5 \pm 17.3$           | $32.4 \pm 19.1^{***,b}$   | $40.8 \pm 5.6^{***,++}$       |
| Mixed Men  | $19.0 \pm 12.1$           | $17.6 \pm 10.7^b$         | ---                           |
| Control Men  | $19.9 \pm 9.1$            | $24.9 \pm 14.6$           | $36.4 \pm 5.3^{***,++}$       |
| Mixed Women  | $22.3 \pm 14.0$           | $22.6 \pm 13.1$           | ---                           |
| Control Women  | $18.0 \pm 10.6$           | $19.0 \pm 12.9$           | ---                           |
| <b>Other endurance training volume (average kilometers <math>\cdot</math> week<sup>-1</sup>)</b> |                           |                           |                               |
| Maximal  | $12.9 \pm 15.8$           | $15.6 \pm 27.7$           | $11.9 \pm 12.1^a$             |
| Explosive  | $13.0 \pm 20.4$           | $8.2 \pm 14.4$            | $6.7 \pm 7.0$                 |
| Mixed Men  | $9.1 \pm 21.0$            | $8.2 \pm 19.4$            | ---                           |
| Control Men  | $6.1 \pm 13.8$            | $6.4 \pm 13.1$            | $7.8 \pm 17.8^a$              |
| Mixed Women  | $2.9 \pm 6.3$             | $7.9 \pm 12.3$            | ---                           |
| Control Women  | $10.7 \pm 18.0$           | $16.0 \pm 26.9$           | ---                           |

## 5.2 Neuromuscular measures (I, II, III)

### 5.2.1 Maximal force and power (dynamic and isometric)

Concentric 1 repetition maximum as measured by maximal bilateral dynamic leg press increased in all training groups over the preparatory strength training period and strength training intervention (Figure 8). In men, significant gains in 1RM were observed after the preparatory period in M, MM and CM ( $3.5 \pm 3.5\%$ ,  $p < 0.05$ ;  $3.0 \pm 3.6\%$ ,  $p < 0.05$  and  $1.8 \pm 5.8\%$ ,  $p < 0.05$ , respectively), but not in E ( $-0.5 \pm 6.1\%$ ,  $p > 0.05$ ). During the strength training intervention between weeks 0 and 4, significant gains in 1RM were observed in M, E, MM and CM ( $4.3 \pm 4.5\%$ ,  $p < 0.001$ ;  $3.7 \pm 4.7\%$ ,  $p < 0.01$ ;  $2.5 \pm 2.7\%$ ,  $p < 0.05$  and  $3.7 \pm 3.7\%$ ,  $p < 0.05$ , respectively). In women, significant gains in 1RM were observed after the preparatory period in MW ( $6.9 \pm 6.8\%$ ,  $p < 0.001$ ), but not in CW ( $4.4 \pm 5.4$ ,  $p > 0.05$ ). Further gains in 1RM were observed in MW in the first 4 weeks of the strength training intervention ( $7.2 \pm 6.2\%$ ,  $p < 0.001$ ,) while strength gains in CW between weeks 0 and 4 ( $2.7 \pm 2.9\%$ ) made the total strength gains from pre significant ( $p < 0.05$ ).

A plateau in strength development occurred in all groups between weeks 4 and 8. Differences in absolute maximal strength were not observed between groups at pre, however, a significant difference between the E and MM groups was observed at weeks 0, 4 and 8 ( $p < 0.05$ ).

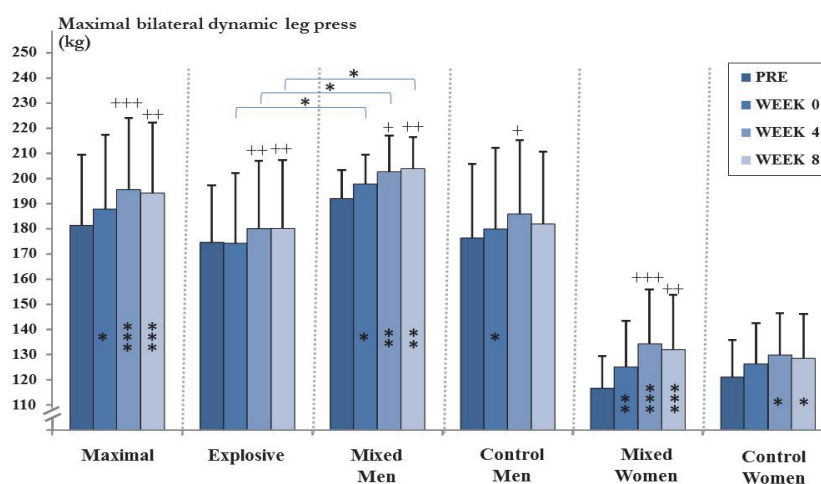


FIGURE 8 Maximal strength as maximal bilateral dynamic leg press 1RM load (kg, mean  $\pm$  SD) during the 6-8 week preparatory period (pre) and 8-week strength training intervention (week 0, week 4 and week 8). Group differences marked above columns \* =  $p < 0.05$ . From pre within group differences inside columns \* =  $p < 0.05$ , \*\* =  $p < 0.01$  and \*\*\* =  $p < 0.001$ , from week 0 within group differences above columns + =  $p < 0.05$ , ++ =  $p < 0.01$  and +++ =  $p < 0.001$ .

Changes in maximal bilateral isometric force and rate of force development were less systematic than those observed in maximal dynamic force (Table 6).

TABLE 6 Maximal bilateral isometric force and rate of force development during the 6 to 8-week preparatory strength training period and 8-week strength training intervention week 0, week 4, week 8). From pre within group differences \* =  $p < 0.05$  and \*\* =  $p < 0.01$ , from week 0 + =  $p < 0.05$  and ++ =  $p < 0.01$ , and from week 4 ¥¥ =  $p < 0.01$ . Difference between groups a-a, b-b, c-c, d-d =  $p < 0.05$ .

|   | PRE                     | WEEK 0                       | WEEK 4                       | WEEK 8                      |
|---|-------------------------|------------------------------|------------------------------|-----------------------------|
| <b>Maximal bilateral isometric force (N)</b>          |                         |                              |                              |                             |
| Maximal   | 3587 ± 817              | 3511 ± 652 <sup>a</sup>      | 3434 ± 640 <sup>a</sup>      | 3666 ± 718 <sup>a,¥¥</sup>  |
| Explosive   | 3867 ± 819              | 3760 ± 757 <sup>b,c</sup>    | 3854 ± 682                   | 3786 ± 719 <sup>b</sup>     |
| Mixed Men   | 4058 ± 875 <sup>a</sup> | 4340 ± 921 <sup>a,d*</sup>   | 4303 ± 794 <sup>b</sup>      | 4371 ± 728 <sup>a,c,*</sup> |
| Control Men   | 3240 ± 828 <sup>a</sup> | 2893 ± 329 <sup>b,c,d*</sup> | 3195 ± 622 <sup>b,+</sup>    | 3081 ± 724 <sup>b,c</sup>   |
| Mixed Women   | 2337 ± 397              | 2432 ± 410                   | 2694 ± 624 <sup>*,+</sup>    | 2752 ± 599 <sup>**,++</sup> |
| Control Women   | 2417 ± 545              | 2509 ± 532                   | 2669 ± 640                   | 2613 ± 558                  |
| <b>Rate of force development (N · s<sup>-1</sup>)</b> |                         |                              |                              |                             |
| Maximal   | 27065 ± 8284            | 25064 ± 8742                 | 21876 ± 7858 <sup>**</sup>   | 24477 ± 14021               |
| Explosive   | 24395 ± 6193            | 23361 ± 9166                 | 23220 ± 10808                | 26774 ± 10342               |
| Mixed Men   | 27966 ± 8803            | 30545 ± 16195                | 26698 ± 11092 <sup>a</sup>   | 26893 ± 5985 <sup>a</sup>   |
| Control Men   | 23657 ± 9718            | 16154 ± 4444 <sup>*</sup>    | 15619 ± 4410 <sup>a,**</sup> | 19360 ± 9586 <sup>a</sup>   |
| Mixed Women   | 12238 ± 4811            | 12275 ± 3447                 | 13014 ± 4210                 | 16196 ± 4194                |
| Control Women   | 14124 ± 4968            | 15178 ± 3870                 | 17277 ± 5348                 | 15959 ± 3761                |

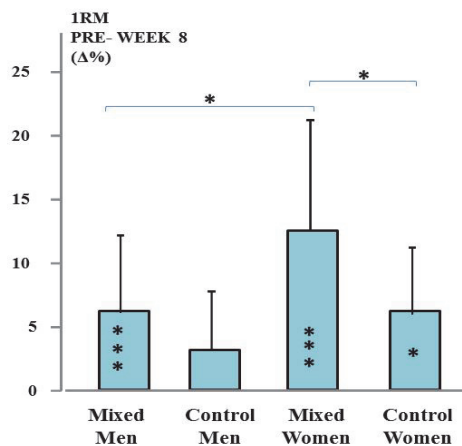


FIGURE 9 Relative change in maximal bilateral dynamic strength. Increase in 1RM from pre to week 8 (the entire 6-8 week preparatory period and 8-week strength training intervention) inside columns \* =  $p < 0.05$  and \*\*\* =  $p < 0.001$ . Differences between groups above columns \* =  $p < 0.05$ .

A significant difference in relative strength development over the entire experimental period (pre to week 8) was observed between MM and MW ( $p < 0.05$ ) as well as between MW and CW ( $p < 0.05$ , Figure 9).

Jump height improved significantly over the preparatory period in M, E, MM, and CM ( $7.1 \pm 10.1\%$ ,  $p < 0.01$ ;  $6.9 \pm 5.1\%$ ,  $p < 0.01$ ;  $5.2 \pm 4.4\%$ ,  $p < 0.01$ ;  $9.3 \pm 8.5\%$ ,  $p < 0.01$ , respectively), but not in MW or CW ( $4.1 \pm 5.1\%$  and  $0.5 \pm 6.1\%$ , Figure 10). During the first 4 weeks of the strength training intervention, significant gains in jump height were observed in M and E ( $3.3 \pm 4.3\%$ ,  $p < 0.05$  and  $2.5 \pm 5.0\%$ ,  $p < 0.05$ ), as well as in MW and CW ( $7.1 \pm 7.4\%$   $p < 0.01$  and  $7.1 \pm 8.0\%$ ,  $p < 0.05$ ).

A plateau in jump height was observed in CM after the preparatory period and first 4 weeks of the strength training intervention in M, E, MW and CW. Only the MM group made significant gains in jump height in the final 4 weeks of the strength training intervention ( $4.7 \pm 6.6\%$ ,  $p < 0.01$ ).

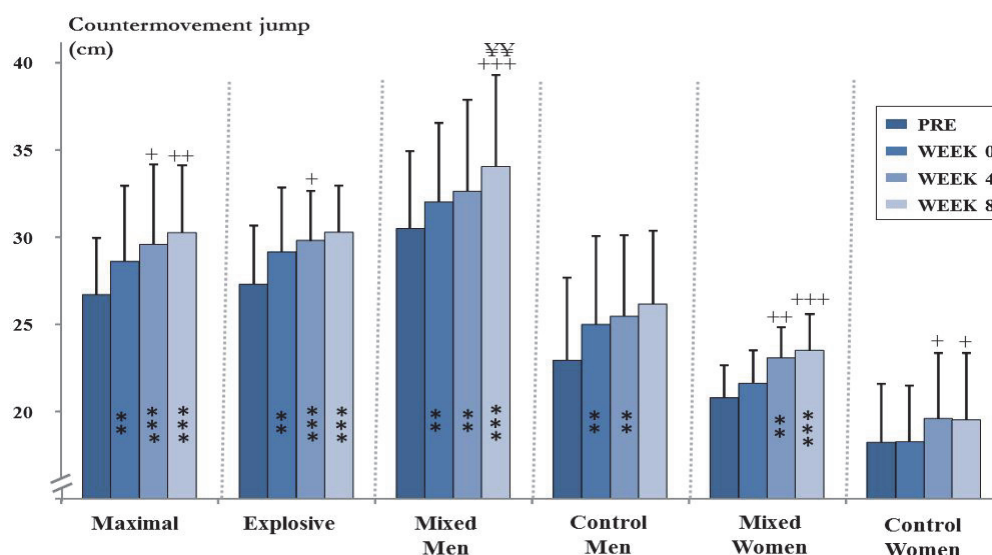


FIGURE 10 Countermovement jump height (cm, mean  $\pm$  SD) during the 6-8 week preparatory period (pre) and 8-week strength training intervention (week 0, week 4 and week 8). From pre within group differences inside columns \*\* =  $p < 0.01$  and \*\*\* =  $p < 0.001$ , from week 0 within group differences above columns + =  $p < 0.05$ , ++ =  $p < 0.01$  and +++ =  $p < 0.001$ , from week 4 within group differences above columns ¥¥¥ =  $p < 0.01$ .

## 5.2.2 Muscle activation during dynamic and isometric actions

Muscle activation of VL+VM during the concentric phase of maximal bilateral dynamic leg press increased from pre to week 8 in M ( $p < 0.05$ ) and E ( $p < 0.05$ ,

Figure 11). A statistical trend was observed from pre to week 4 in MM ( $p = 0.056$ ). No changes in muscle activation were observed in CM, MW or CW.

Muscle activation of VL+VM did not change significantly in any of the groups during maximal bilateral isometric leg press.

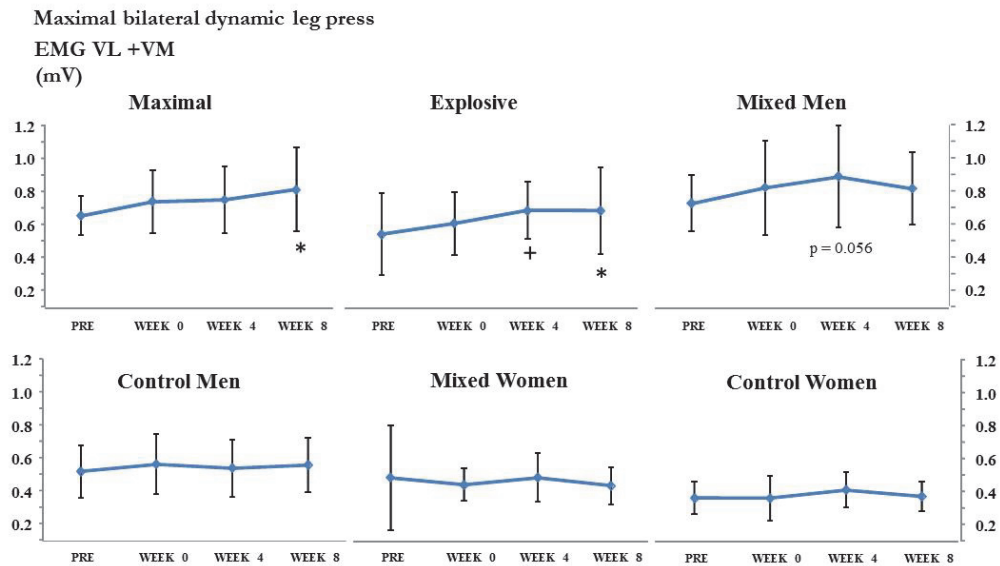


FIGURE 11 Muscle activation as the sum of vastus lateralis (VL) and vastus medialis (VM) during the concentric phase of maximal bilateral dynamic leg press over the 6 to 8-week preparatory strength training period and 8-week strength training intervention. Within group differences from pre \* =  $p < 0.05$ , and from week 0 +=  $p < 0.05$ .

In men, muscle activation of VL+VM during the concentric phase of a counter-movement jump increased significantly (Figure 12) in M, E and MM over the preparatory period and strength training intervention ( $p < 0.05$ ,  $p < 0.01$  and  $p < 0.01$ , respectively). In MW, a significant increase in VL+VM muscle activation during counter-movement jump ( $p < 0.05$ ) was also observed. No change in muscle activation was observed in either CM or CW.



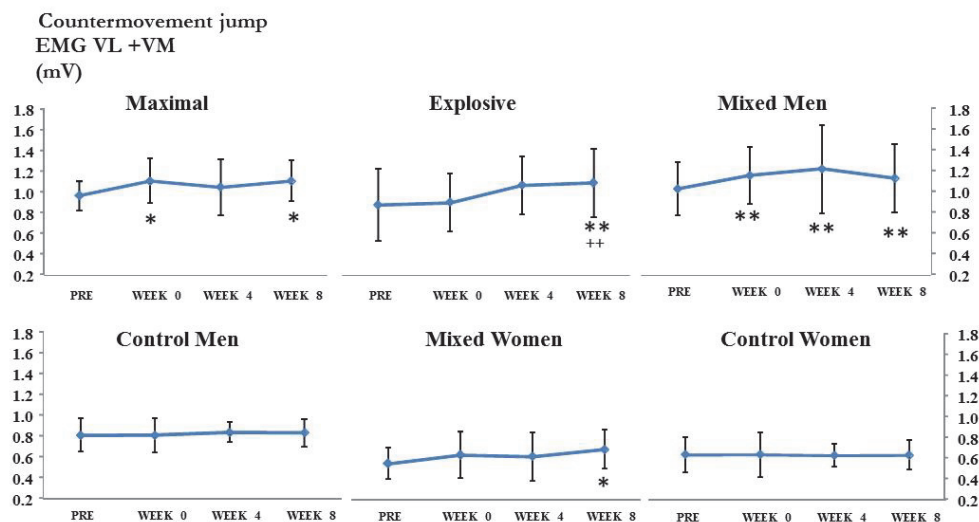


FIGURE 12 Muscle activation as the sum of vastus lateralis (VL) and vastus medialis (VM) during the concentric phase of countermovement jump over the 6 to 8-week preparatory strength training period and 8-week strength training intervention. Within group differences from pre \* =  $p < 0.05$ , \*\* =  $p < 0.01$ , and from week 0 +=  $p < 0.01$  from week 0.

### 5.3 Cardiorespiratory measures (I, II, III)

During the preparatory period (6 to 8 weeks) and strength training intervention (8 weeks), submaximal and maximal running characteristics showed improvement in most groups (Tables 7 and 8).  $VO_{2max}$  in terms of  $ml \cdot kg \cdot min^{-1}$  improved in M, CM and CW ( $4.3 \pm 6.0\%$ ,  $p < 0.05$ ;  $8.7 \pm 9.8\%$ ,  $p < 0.01$  and  $4.4 \pm 3.4\%$ ,  $p < 0.05$ , respectively). In MW, the significant improvement was observed after the strength training intervention at week 8 ( $4.1 \pm 8.5\%$   $p < 0.05$ ).  $VO_{2max}$  in terms of  $L \cdot min^{-1}$  improved in only M, CM and CW. Peak running speed at  $VO_{2max}$  improved significantly in all training groups over both training periods ( $4.3 \pm 3.0\%$ ,  $p < 0.001$ ;  $3.3 \pm 2.9\%$ ,  $p < 0.001$ ;  $4.7 \pm 3.5\%$ ,  $p < 0.001$ ;  $7.8 \pm 3.3\%$ ,  $p < 0.001$ ;  $5.1 \pm 2.8$ ,  $p < 0.001$  and  $3.6 \pm 2.7\%$ ,  $p < 0.05$ , for M, E, MM, CM, MW and CW, respectively). Running speed at anaerobic threshold increased in all groups but CW, while running economy at  $8 km \cdot h^{-1}$ ,  $10 km \cdot h^{-1}$  and  $12 km \cdot h^{-1}$  improved primarily only in CM.

TABLE 7 Maximal oxygen uptake and running economy at submaximal running speeds of 8, 10 and 12 km · h<sup>-1</sup> during the 6 to 8-week preparatory strength training period and 8-week strength training intervention. Differences within groups from pre \* = p < 0.05, \*\* = p < 0.01 and \*\*\* = p < 0.001, and from week 0 + = p < 0.05, ++ = p < 0.01 and +++ = p < 0.001. Difference between groups a-a, b-b = p < 0.05.

|  | Maximal                 | Explosive               | Mixed Men                | Control Men                  | Mixed Women             | Control Women |
|--|-------------------------|-------------------------|--------------------------|------------------------------|-------------------------|---------------|
| <b>VO<sub>2max</sub> (ml · kg<sup>-1</sup> · min<sup>-1</sup>)</b>                         |                         |                         |                          |                              |                         |               |
| Pre  | 49.9 ± 2.8              | 50.6 ± 4.5 <sup>a</sup> | 50.9 ± 5.4 <sup>b</sup>  | 45.7 ± 3.0 <sup>a,b</sup>    | 43.7 ± 2.4              | 42.8 ± 5.9    |
| Week 0   | 51.4 ± 3.8*             | 50.6 ± 5.2              | 51.3 ± 5.2               | 47.0 ± 6.2                   | 43.6 ± 1.8              | 44.2 ± 6.2    |
| Week 8   | 52.1 ± 4.9*             | 51.7 ± 4.0              | 51.7 ± 5.4               | 49.8 ± 7.0 <sup>***,++</sup> | 45.4 ± 2.7 <sup>+</sup> | 44.7 ± 6.4*   |
| <b>VO<sub>2max</sub> (L · min<sup>-1</sup>)</b>  |                         |                         |                          |                              |                         |               |
| Pre  | 3.9 ± 0.4               | 3.9 ± 0.2               | 3.9 ± 0.5                | 3.9 ± 0.5                    | 2.7 ± 0.3               | 2.6 ± 0.4     |
| Week 0   | 4.0 ± 0.4*              | 3.8 ± 0.3               | 3.9 ± 0.5                | 3.9 ± 0.4                    | 2.8 ± 0.3               | 2.7 ± 0.5*    |
| Week 8   | 4.0 ± 0.4 <sup>**</sup> | 4.0 ± 0.4               | 4.0 ± 0.5                | 4.1 ± 0.5 <sup>***,++</sup>  | 2.8 ± 0.3               | 2.7 ± 0.5*    |
| <b>Running Economy at 8 km · h<sup>-1</sup> (ml · kg<sup>-1</sup> · min<sup>-1</sup>)</b>  |                         |                         |                          |                              |                         |               |
| Pre  | 30.2 ± 1.2              | 31.1 ± 2.2              | 32.0 ± 2.8               | 32.8 ± 3.8                   | 32.0 ± 2.3              | 30.9 ± 2.9    |
| Week 0   | 30.9 ± 1.9              | 30.3 ± 2.2              | 30.6 ± 1.9               | 29.3 ± 2.7 <sup>**</sup>     | 31.3 ± 2.4              | 29.4 ± 2.4    |
| Week 8   | 30.9 ± 1.9              | 30.5 ± 2.5              | 30.9 ± 2.9               | 30.0 ± 3.3*                  | 31.0 ± 1.7              | 29.3 ± 2.8    |
| <b>Running Economy at 10 km · h<sup>-1</sup> (ml · kg<sup>-1</sup> · min<sup>-1</sup>)</b> |                         |                         |                          |                              |                         |               |
| Pre  | 37.0 ± 1.9              | 37.0 ± 2.8              | 38.9 ± 3.7               | 36.6 ± 1.9                   | 38.4 ± 2.5              | 36.3 ± 3.7    |
| Week 0   | 37.4 ± 1.7              | 36.1 ± 2.9              | 36.4 ± 2.2 <sup>**</sup> | 35.8 ± 2.2 <sup>**</sup>     | 37.4 ± 2.1              | 35.5 ± 4.1    |
| Week 8   | 37.6 ± 1.8              | 36.1 ± 3.3              | 36.9 ± 3.0               | 35.9 ± 3.3*                  | 37.9 ± 2.6              | 35.5 ± 3.4    |
| <b>Running Economy at 12 km · h<sup>-1</sup> (ml · kg<sup>-1</sup> · min<sup>-1</sup>)</b> |                         |                         |                          |                              |                         |               |
| Pre  | 43.1 ± 2.0              | 42.7 ± 2.9              | 44.1 ± 4.1               | 42.2 ± 1.7                   | 42.7 ± 1.5              | 42.0 ± 1.9    |
| Week 0   | 43.5 ± 1.9              | 42.4 ± 3.3              | 42.6 ± 2.7               | 41.3 ± 2.8 <sup>**</sup>     | 42.4 ± 1.3              | 42.0 ± 2.0    |
| Week 8   | 43.8 ± 2.3 <sup>a</sup> | 42.0 ± 3.3              | 42.9 ± 3.3               | 41.0 ± 3.4 <sup>*,a</sup>    | 43.1 ± 2.0              | 42.4 ± 2.4    |

TABLE 8 Peak running speed and anaerobic threshold ( $\text{km} \cdot \text{h}^{-1}$ ) during the 6 to 8-week preparatory strength training period and 8-week strength training intervention. Differences within groups from pre \* =  $p < 0.05$ , \*\* =  $p < 0.01$  and \*\*\* =  $p < 0.001$  and from week 0 + =  $p < 0.05$ , ++ =  $p < 0.01$  and +++ =  $p < 0.001$ . Difference between groups a-a, b-b =  $p < 0.05$ .

|   | Maximal          | Explosive        | Mixed Men        | Control Men       | Mixed Women     | Control Women |
|---|------------------|------------------|------------------|-------------------|-----------------|---------------|
| <b>Peak Running Speed at <math>\text{VO}_{2\text{peak}}</math> (<math>\text{km} \cdot \text{h}^{-1}</math>)</b> |                  |                  |                  |                   |                 |               |
| <b>Pre</b>  | 14.7 ± 1.0       | 15.1 ± 1.0       | 14.9 ± 1.2       | 14.0 ± 0.8        | 12.9 ± 0.9      | 13.0 ± 1.6    |
| <b>Week 0</b>   | 15.1 ± 1.2**     | 15.3 ± 1.0       | 15.2 ± 1.2**     | 14.4 ± 0.9**      | 13.3 ± 0.9**    | 13.4 ± 1.5**  |
| <b>Week 8</b>   | 15.4 ± 1.1***,+  | 15.6 ± 1.0***,++ | 15.6 ± 1.2***,++ | 15.1 ± 1.1***,+++ | 13.5 ± 0.8***,+ | 13.4 ± 1.4**  |
| <b>Anaerobic Threshold Running Speed (<math>\text{km} \cdot \text{h}^{-1}</math>)</b>                           |                  |                  |                  |                   |                 |               |
| <b>Pre</b>  | 12.0 ± 1.2       | 12.3 ± 1.2       | 11.8 ± 1.5       | 11.1 ± 0.7        | 10.7 ± 1.0      | 10.7 ± 1.3    |
| <b>Week 0</b>   | 12.7 ± 1.2***    | 12.7 ± 0.9*      | 12.1 ± 1.6       | 11.5 ± 0.7        | 11.1 ± 0.9*     | 11.4 ± 0.8*   |
| <b>Week 8</b>   | 13.2 ± 1.2***,++ | 13.2 ± 1.0**,+   | 12.4 ± 1.7**     | 12.1 ± 0.9**,+    | 11.1 ± 1.2**    | 11.0 ± 1.3    |

## 5.4 Serum hormone concentrations (I, II, III)

Basal serum concentrations of testosterone increased significantly in MM over the preparatory strength training period and first 4 weeks of the strength training intervention ( $p < 0.05$ , Table 9). At week 8, concentrations of testosterone had decreased back to baseline. In all other groups, basal serum concentrations of testosterone remained statistically unaltered during the preparatory strength training period and strength training intervention. Serum cortisol concentrations remained statistically unaltered in all groups. Significant differences in basal testosterone levels were observed between the men's groups.

In women, TSH decreased significantly in CW between pre and week 0 ( $p < 0.01$ ) while in MW, a decrease in TSH was observed at week 4 ( $p < 0.01$  from pre). Both groups of women had decreased TSH at the end of the experimental period.

TABLE 9 Basal serum hormone concentrations over the preparatory strength training period (6 to 8 weeks) and 8-week strength training intervention. Differences within groups from pre \* =  $p < 0.05$ , \*\* =  $p < 0.01$ , from week 0 + =  $p < 0.05$ , and from week 4  $\forall$  =  $p < 0.05$ . Differences between groups a-a, b-b, c-c =  $p < 0.05-0.001$ .

|  | PRE                         | WEEK 0                  | WEEK 4                    | WEEK 8                          |
|--|-----------------------------|-------------------------|---------------------------|---------------------------------|
| <b>Testosterone (nmol · L<sup>-1</sup>)</b>                |                             |                         |                           |                                 |
| Maximal  | 17.0 ± 4.3 <sup>a</sup>     | 18.7 ± 5.7              | 17.4 ± 5.4                | 17.7 ± 3.3                      |
| Explosive  | 15.7 ± 2.0 <sup>b</sup>     | 15.9 ± 3.7              | 17.1 ± 3.2                | 16.8 ± 3.0                      |
| Mixed Men  | 21.9 ± 5.0 <sup>a,b,c</sup> | 23.2 ± 4.2              | 23.8 ± 5.0*               | 20.1 ± 5.7 <sup>+,\forall</sup> |
| Control Men  | 14.6 ± 4.7 <sup>c</sup>     | 16.5 ± 4.8              | 16.0 ± 2.6                | 14.5 ± 3.8                      |
| Mixed Women  | 1.8 ± 0.6                   | 1.7 ± 0.6               | 1.7 ± 0.6                 | 1.6 ± 0.6                       |
| Control Women  | 1.6 ± 0.7                   | 1.7 ± 0.7               | 1.8 ± 0.9                 | 1.8 ± 0.7                       |
| <b>Cortisol (nmol · L<sup>-1</sup>)</b>                    |                             |                         |                           |                                 |
| Maximal  | 451.1 ± 65.0                | 440.4 ± 98.1            | 412.8 ± 79.9              | 401.9 ± 94.9                    |
| Explosive  | 414.8 ± 93.5                | 414.6 ± 78.9            | 452.5 ± 134.5             | 437.6 ± 120.2                   |
| Mixed Men  | 496.0 ± 158.8               | 496.6 ± 148.8           | 542.9 ± 103.2             | 482.0 ± 119.2                   |
| Control Men  | 462.2 ± 77.7                | 468.3 ± 118.5           | 497.3 ± 108.4             | 456.7 ± 124.7                   |
| Mixed Women  | 659.3 ± 202.1               | 620.9 ± 127.3           | 650.1 ± 174.7             | 630.3 ± 170.8                   |
| Control Women  | 615.3 ± 153.5               | 600.0 ± 158.4           | 603.4 ± 95.3              | 578.3 ± 139.3                   |
| <b>Thyroid Stimulating Hormone (μIU · ml<sup>-1</sup>)</b> |                             |                         |                           |                                 |
| Maximal  | 2.6 ± 1.2                   | 2.1 ± 0.8               | 2.2 ± 0.8                 | 2.0 ± 0.8                       |
| Explosive  | 2.3 ± 1.1                   | 1.8 ± 0.4               | 2.0 ± 0.8                 | 2.1 ± 1.0                       |
| Mixed Men  | 2.6 ± 1.0                   | 2.3 ± 1.1               | 2.5 ± 1.1                 | 2.5 ± 1.1                       |
| Control Men  | 2.8 ± 0.9                   | 2.7 ± 1.0               | 2.0 ± 0.8                 | 1.9 ± 1.0                       |
| Mixed Women  | 4.2 ± 2.6                   | 3.8 ± 2.0               | 3.2 ± 2.3 <sup>**,+</sup> | 3.2 ± 1.3*                      |
| Control Women  | 3.8 ± 1.9                   | 2.8 ± 1.1 <sup>**</sup> | 3.3 ± 1.3                 | 2.8 ± 1.0*                      |

## 5.5 Overall strength and endurance development

During the preparatory period and strength training intervention, no significant correlations were found between strength development (1RM, MVC and CMJ) and running characteristics/parameters ( $VO_{2max}$ ,  $S_{peak}$  and  $AnT_{speed}$ ). Combined strength and endurance over the preparatory period and strength training intervention, however, caused overall improvements in averaged strength and running parameters in all groups (Figure 13), while mixed maximal and explosive strength training improved strength parameters significantly more than endurance running characteristics/ parameters.

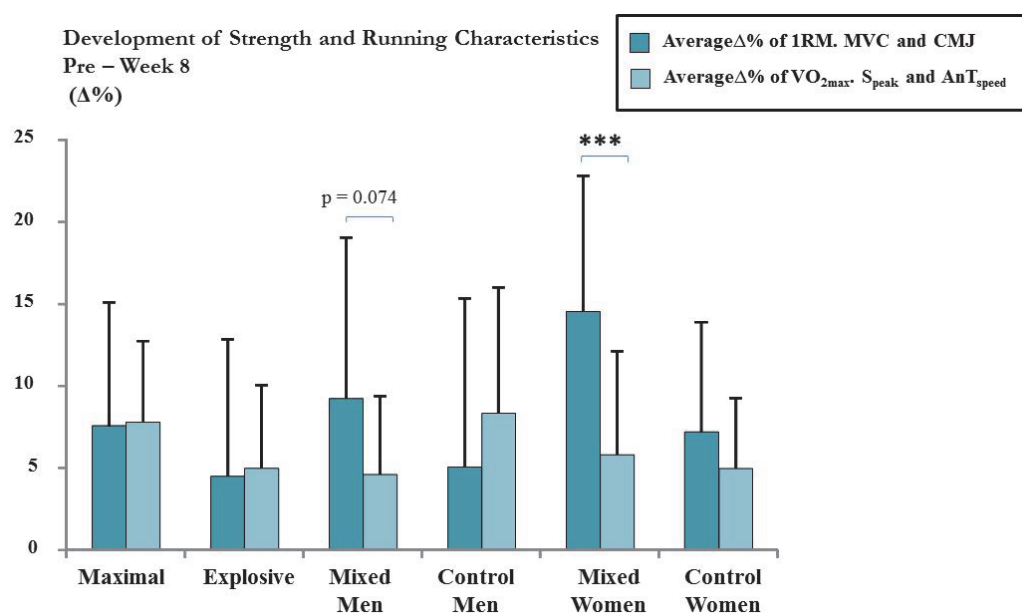


FIGURE 13 Development of strength and running characteristics over the combined preparatory strength training period and strength training intervention (Pre-Week 8). Strength characteristics are an average of  $\Delta\%$  maximal bilateral dynamic leg press (1RM),  $\Delta\%$  maximal bilateral isometric leg press (MVC) and  $\Delta\%$  countermovement jump height (CMJ). Running characteristics are an average of  $\Delta\%$  maximal oxygen uptake ( $VO_{2peak}$ ),  $\Delta\%$  maximal running speed at  $VO_{2peak}$  ( $S_{peak}$ ) and  $\Delta\%$  running speed at anaerobic threshold ( $AnT_{speed}$ ). \*\*\* =  $p < 0.001$  difference between average  $\Delta\%$  of 1RM, MVC and CMJ and  $\Delta\%$  of  $VO_{2max}$ ,  $S_{peak}$  and  $AnT_{speed}$ .

## 5.6 Body composition

Body mass fluctuated significantly over the preparatory period and strength training intervention in M, E, MM, and CW, but only the group of men performing explosive strength training was significantly lighter at the end of the two training periods than at baseline. Body mass of CM and MW did not change significantly during the two training periods. Body fat % changed significantly in E, MM, and MW over the two training periods and was significantly lower than baseline in E and MM following the two training periods, but body fat % did not change significantly in M, CM, and CW (Table 10).

TABLE 10 Body mass and body fat % during the preparatory strength training period (6 to 8 weeks) and 8-week strength training intervention (week 0, week 4, week 8). Differences within groups from pre \* =  $p < 0.05$ , \*\* =  $p < 0.01$ , \*\*\* =  $p < 0.001$ , from week 0 + =  $p < 0.05$ , ++ =  $p < 0.01$ , and from week 4 ¥ =  $p < 0.05$ , ¥¥¥ =  $p < 0.001$ .

|                       | PRE         | WEEK 0                    | WEEK 4                    | WEEK 8                          |
|-----------------------|-------------|---------------------------|---------------------------|---------------------------------|
| <b>Body Mass (kg)</b> |             |                           |                           |                                 |
| Maximal               | 77.2 ± 5.5  | 78.8 ± 5.9**              | 78.3 ± 6.0*               | 77.4 ± 5.9 <sup>¥</sup>         |
| Explosive             | 78.4 ± 6.3  | 78.6 ± 6.3                | 78.4 ± 5.7                | 76.9 ± 5.4 <sup>** ++ ¥¥¥</sup> |
| Mixed Men             | 79.5 ± 6.5  | 78.4 ± 6.8*               | 78.6 ± 7.1                | 78.6 ± 7.0                      |
| Control Men           | 83.8 ± 10.5 | 84.3 ± 9.5                | 84.0 ± 9.9                | 82.9 ± 9.6                      |
| Mixed Women           | 62.9 ± 4.3  | 63.1 ± 4.8                | 63.4 ± 4.1                | 62.7 ± 4.1                      |
| Control Women         | 59.9 ± 7.4  | 60.7 ± 7.9                | 61.4 ± 7.2 <sup>**</sup>  | 60.6 ± 7.2 <sup>¥</sup>         |
| <b>Body Fat (%)</b>   |             |                           |                           |                                 |
| Maximal               | 17.1 ± 4.4  | 17.2 ± 4.0                | 17.1 ± 4.2                | 16.7 ± 4.3                      |
| Explosive             | 17.2 ± 5.5  | 16.0 ± 6.1 <sup>**</sup>  | 16.0 ± 5.9 <sup>*</sup>   | 15.4 ± 5.7 <sup>***</sup>       |
| Mixed Men             | 18.2 ± 4.2  | 16.2 ± 5.4 <sup>***</sup> | 15.9 ± 5.5 <sup>***</sup> | 15.8 ± 4.0 <sup>***</sup>       |
| Control Men           | 21.1 ± 5.9  | 20.8 ± 5.4                | 20.5 ± 5.7                | 19.8 ± 5.6                      |
| Mixed Women           | 24.8 ± 4.1  | 23.9 ± 4.6                | 23.5 ± 4.6 <sup>*</sup>   | 23.5 ± 4.4                      |
| Control Women         | 24.3 ± 6.3  | 24.0 ± 5.9                | 23.6 ± 5.7                | 23.5 ± 5.7                      |

Muscle thickness increased significantly from baseline between pre and week 4 in M, after which a plateau was observed. Following the 14-week marathon training period in which the strength training frequency was reduced, muscle thickness decreased in all groups, but statistical significance was achieved only in the explosive strength training group (Figure 14).

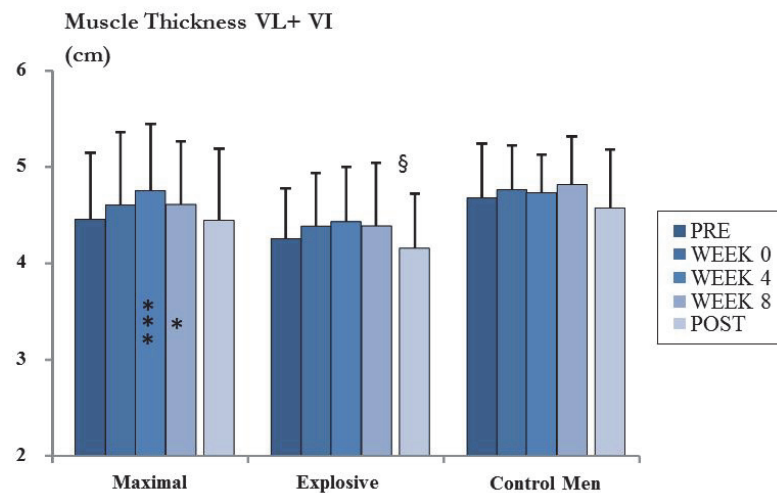


FIGURE 14 Muscle thickness as the sum of vastus lateralis and vastus intermedius during the preparatory strength training period (6 to 8 weeks) and 8-week strength training intervention (week 0, week 4, week 8) as well as the 14-week marathon training period with reduced strength training (post). Differences within groups from pre \* =  $p < 0.05$ , \*\* =  $p < 0.001$ , and from week 8 § =  $p < 0.05$ .

## 5.7 Marathon training period - reduced strength training

The M, E and CM groups performed similar endurance training for ~14 weeks following the preparatory period and strength training intervention. During this period, maximal strength decreased significantly in M ( $-4.5 \pm 3.4\%$ ,  $p < 0.01$ ), but not in E or CM ( $-3.1 \pm 5.9\%$  and  $0.4 \pm 5.4\%$ , Figure 15), while parallel changes were observed in muscle activation (Figure 14). Decreases in countermovement jump height were also observed, but these decreases were statistically insignificant (Figure 16).

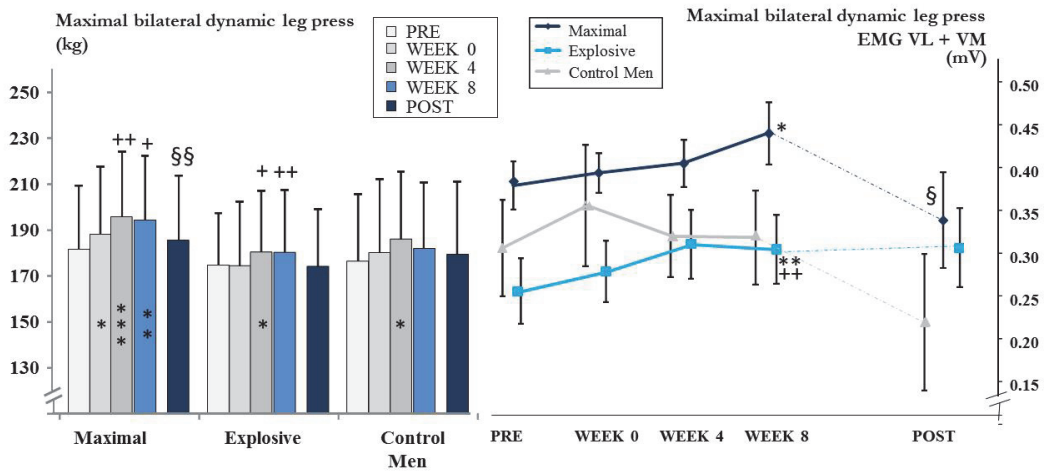


FIGURE 15 Maximal bilateral dynamic leg press and muscle activation of vastus lateralis during the preparatory strength training period (pre = 6 to 8 weeks), the 8-week strength training intervention (week 0, week 4, week 8) and the 14-week marathon training period with reduced strength training (post). Differences within groups from pre \* =  $p < 0.05$ , \*\* =  $p < 0.01$  and \*\*\* =  $p < 0.001$ , from week 0 + =  $p < 0.05$ , ++ =  $p < 0.01$  and +++ =  $p < 0.001$ , and from week 8 § =  $p < 0.05$ , §§ =  $p < 0.01$ .

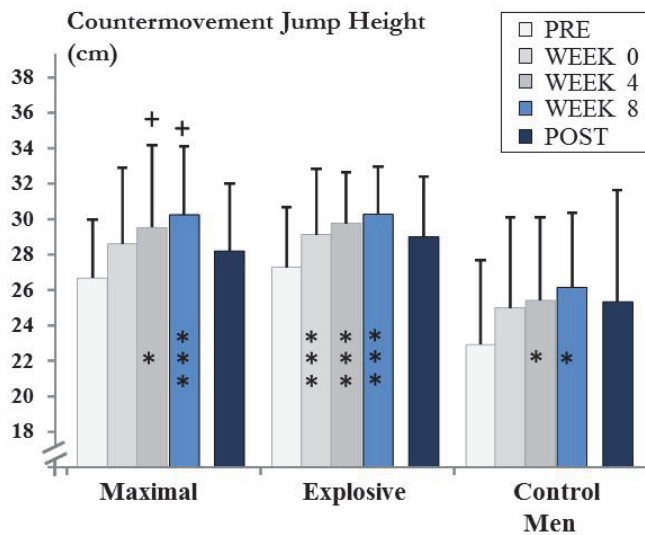


FIGURE 16 Countermovement jump height during the preparatory strength training period (pre = 6 to 8 weeks), the 8-week strength training intervention (week 0, week 4, week 8) and the 14-week marathon training period with reduced strength training (post). Differences within groups from pre \* =  $p < 0.05$ , \*\* =  $p < 0.01$  and \*\*\* =  $p < 0.001$ , and from week 0 + =  $p < 0.05$ .



During the marathon period  $VO_{2max}$  in  $ml \cdot kg \cdot min^{-1}$  or  $L \cdot min^{-1}$  did not increase significantly in any of the groups. Peak running speed, however, increased by  $4.7 \pm 2.6\%$  ( $p < 0.001$ ) in M,  $2.4 \pm 2.7\%$  ( $p < 0.05$ ) in E and decreased in CM by  $0.7 \pm 4.6\%$  (n.s., Figure 17). Anaerobic threshold running speed and running economy at  $8 km \cdot h^{-1}$ ,  $10 km \cdot h^{-1}$  and  $12 km \cdot h^{-1}$ , improved significantly in only M ( $p < 0.001$  at  $10 km \cdot h^{-1}$ , Figure 18), while  $AnT_{speed}$  and running economy at  $8 km \cdot h^{-1}$  improved significantly in E, and only  $AnT_{speed}$  improved significantly in CM.

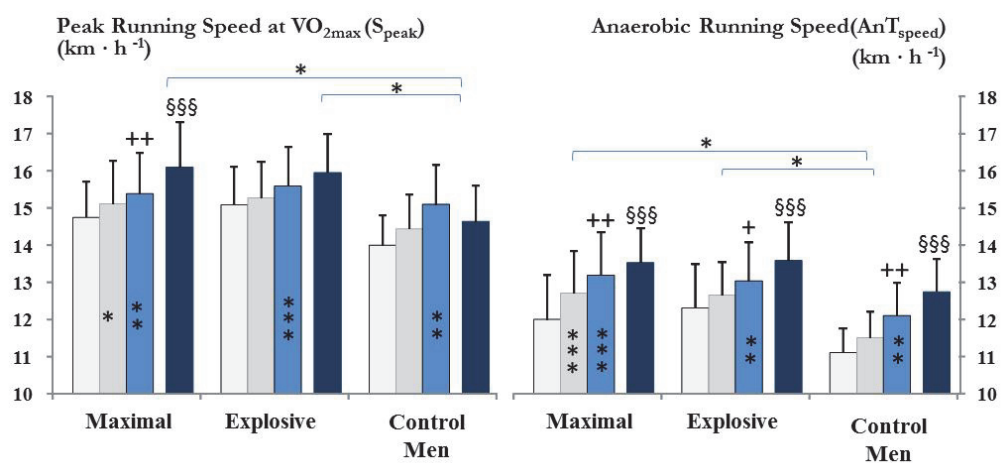


FIGURE 17 Peak running speed at  $VO_{2max}$  ( $S_{peak}$ ) and anaerobic running speed ( $AnT_{speed}$ ) during the preparatory strength training period (pre = 6 to 8 weeks), the 8-week strength training intervention (week 0, week 8) and the 14-week marathon training period with reduced strength training (post). Differences between groups marked above columns \* =  $p < 0.05$ . Differences within groups from pre \* =  $p < 0.05$ , \*\* =  $p < 0.01$  and \*\*\* =  $p < 0.001$ , and from week 8 \$\$\$ =  $p < 0.001$ .

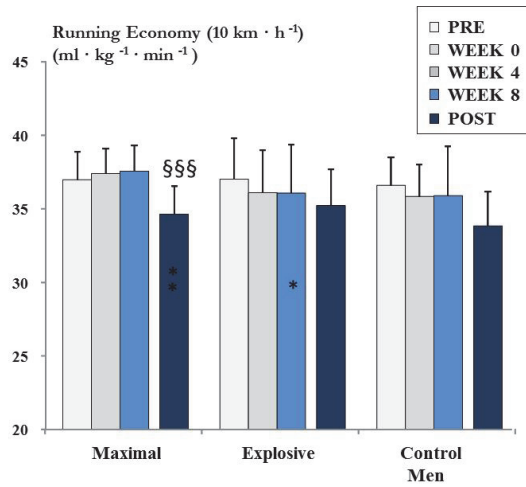


FIGURE 18 Running economy in  $\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$  at  $10 \text{ km} \cdot \text{h}^{-1}$  during the preparatory strength training period (pre = 6 to 8 weeks), the 8-week strength training intervention (week 0, week 8) and the 14-week marathon training period with reduced strength training (post). Differences within groups from pre \* =  $p < 0.05$ , and from week 8 §§§ =  $p < 0.001$ .

Basal levels of testosterone and cortisol did not change significantly in either maximal, explosive or circuit training groups over the entire study including the preparatory period (6 weeks), 8-week strength training intervention and marathon training period with reduced strength training (14 weeks). The ratio of testosterone to cortisol decreased significantly during the marathon training period with reduced strength training (14 weeks) in the maximal strength training group ( $p < 0.05$ , Figure 19).

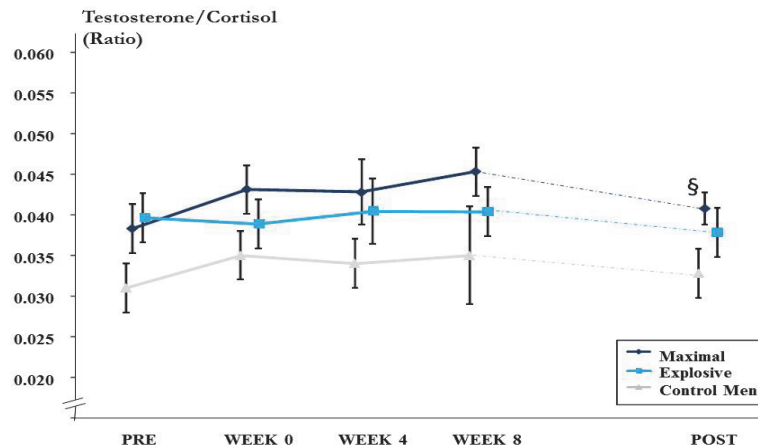


FIGURE 19 Testosterone cortisol ratio during the preparatory strength training period (pre = 6 to 8 weeks), the 8-week strength training intervention (week 0, week 8) and the 14-week marathon training period with reduced strength training (post). Differences within groups from week 8 § =  $p < 0.05$ .

## 5.8 Combined sessions (IV, V)

Men and women with a recreational background in endurance training participated in the study (Table 11). Naturally, physical characteristics between men and women were different. Only subjects that had successfully completed both combined ES and SE sessions were included in the analysis and men and women are only compared statistically in terms of relative change.

TABLE 11 Subject characteristics for studies (IV and V)

|  | Men (n = 12) | Women (n = 10) |
|--|--------------|----------------|
| <b>Age</b>                                       | 38.8 ± 7.1   | 33.5 ± 8.3     |
| <b>Height (cm)</b>                               | 177.4 ± 6.4  | 165.9 ± 7.6    |
| <b>Weight (kg)</b>                               | 75.7 ± 3.6   | 59.8 ± 5.1     |
| <b>% Fat</b>                                     | 12.9 ± 3.6   | 22.0 ± 3.8     |
| <b>VO<sub>2max</sub> (L · min<sup>-1</sup>)</b>  | 4.1 ± 0.3    | 2.9 ± 0.4      |
| <b>(ml · kg<sup>-1</sup> · min<sup>-1</sup>)</b> | 54.5 ± 4.0   | 48.5 ± 4.6     |

## 5.9 Acute neuromuscular responses (IV, V)

Following the endurance loading, absolute maximal bilateral isometric (MVC) strength in the ES men decreased significantly ( $-8.1 \pm 7.3\%$ ,  $p < 0.01$  at mid) but not in women (Figure 20). After completion of both E and S, however, absolute strength decreased significantly in both men and women ( $-20.7 \pm 6.1\%$ ,  $p < 0.001$  and  $-12.4 \pm 9.3\%$ ,  $p < 0.01$ , respectively). In SE, absolute strength decreased significantly following S in both men and women ( $-18.9 \pm 9.2\%$ ,  $p < 0.001$  and  $-14.4 \pm 7.9\%$ ,  $p < 0.05$ ).

After both S and E had been completed, absolute strength remained significantly reduced in men, and only a trend was observed in women ( $-19.3 \pm 9.4\%$ ,  $p < 0.001$  and  $-11.6 \pm 12.0\%$ ,  $p = 0.074$ , respectively). The relative loading-induced decreases in strength between ES and SE were significantly different at mid in men ( $p < 0.001$ ). The relative decreases in strength in men and women were similar at mid but at post, ES men had significantly greater reductions in strength than ES women ( $p < 0.05$ ). MVC remained significantly decreased in SE and ES men at 24 h ( $-11.8 \pm 8.2\%$ ,  $p < 0.01$  and  $-10.2 \pm 7.8\%$ ,  $p < 0.001$ , respectively) and in ES men at 48 h ( $-6.7 \pm 5.6\%$ ,  $p < 0.01$ ). No differences between combined sessions were observed at 24 h and 48 h of recovery.

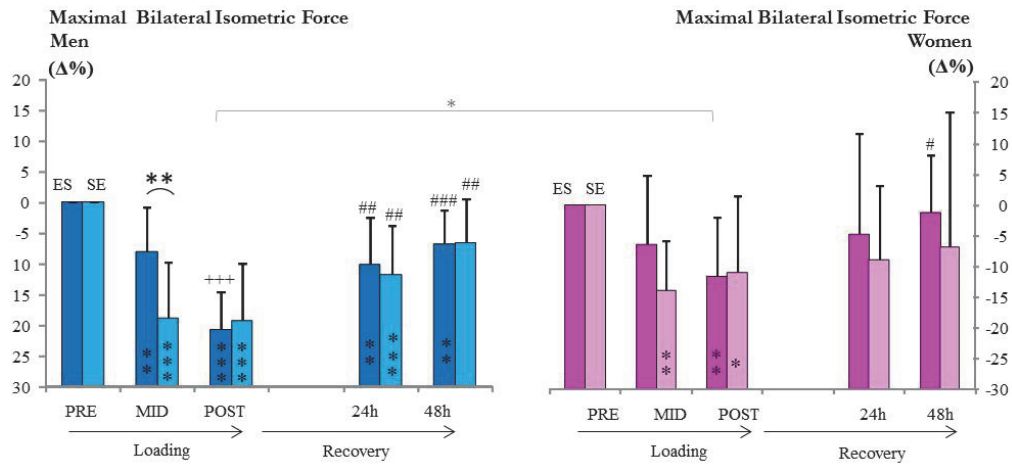


FIGURE 20 Maximal isometric force. Differences within groups in absolute maximal isometric force from pre inside columns \*\* =  $p < 0.01$ , \*\*\* =  $p < 0.001$ , and from mid above columns +++ =  $p < 0.001$ . Differences between relative changes from pre in ES and SE sessions \*\* above columns =  $p < 0.01$ . Difference between men and women \* above graph =  $p < 0.05$ .

The rate of force development (RFD) during bilateral isometric leg press decreased during both combined sessions in men following E and S at mid ( $-19.0 \pm 15.6\%$ ,  $p < 0.001$  and  $-18.8 \pm 21.2\%$ ,  $p < 0.05$  respectively) and from pre-post in ES and SE ( $-18.9 \pm 18.5\%$ ,  $p < 0.01$  and  $-26.4 \pm 20.3\%$ ,  $p < 0.01$ , respectively). Relative changes in RFD were not significantly different between combined sessions in men. In women, RFD decreased significantly only after E (at mid  $-12.4 \pm 21.6\%$ ,  $p < 0.05$ ) and differences in relative RFD response to ES and SE was observed at post ( $p < 0.05$ ). Relative RFD was decreased significantly more in men than women at post in ES ( $p < 0.05$ ). RFD returned to baseline in both men and women following ES and SE during recovery at 24 h and 48 h.

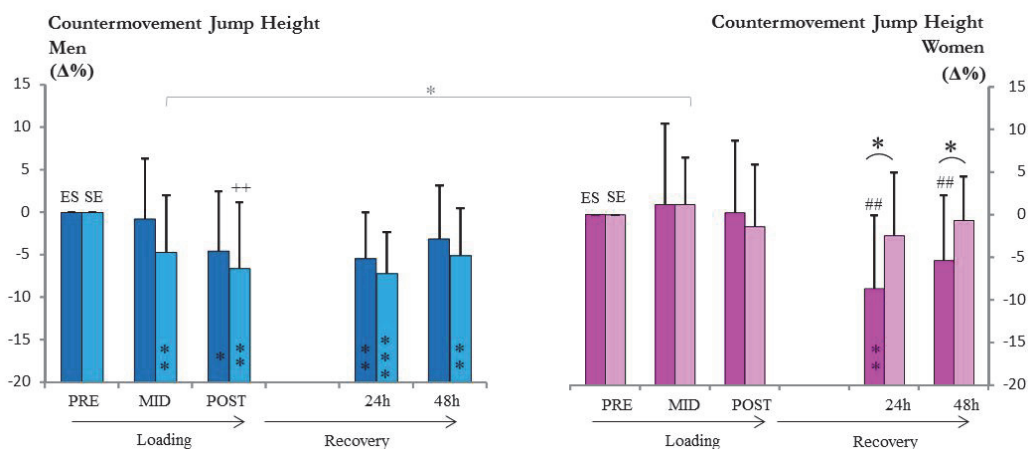


FIGURE 21 Counterovement jump height. Differences within groups from pre inside columns \* =  $p < 0.05$ , \*\* =  $p < 0.01$ , \*\*\* =  $p < 0.001$ , from mid above columns ++ =  $p < 0.01$ , and from post ## =  $p < 0.01$ . Above columns \* =  $p < 0.05$  between changes in CMJ height from pre in men and women. Above graph \* =  $p < 0.05$  between relative changes from pre in ES and SE sessions.

In men, a significant decrease in counterovement jump height was observed in SE following the S loading ( $-4.7 \pm 6.7\%$ ,  $p < 0.01$ , Figure 21), while no significant decrease was observed following the E loading of SE. Jump height was significantly decreased at post of ES and SE ( $-4.5 \pm 7.0\%$ ,  $p < 0.05$ ;  $-6.6 \pm 7.7\%$ ,  $p < 0.01$ , respectively), but no significant differences between combined sessions were observed. At 24 h of recovery, jump height remained reduced in both ES and SE ( $-5.4 \pm 5.3\%$ ,  $p < 0.01$ ;  $-7.2 \pm 4.9\%$ ,  $p < 0.001$ , respectively). No changes in jumping height were observed in either group of women during the combined sessions, however, at 24 h of recovery, jumping height in ES was significantly reduced ( $-8.7 \pm 8.6\%$ ,  $p < 0.01$ ) and at both 24 and 48 h of recovery, a significant difference in relative response was observed between ES and SE ( $p < 0.05$ ).

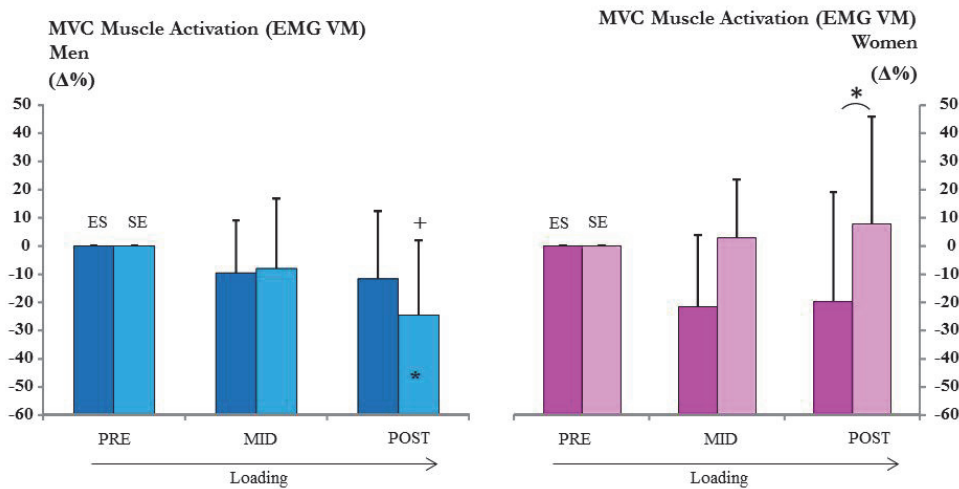


FIGURE 22 Muscle activation of vastus medialis during MVC. Differences within groups from pre inside columns \* =  $p < 0.05$ , from mid above columns + =  $p < 0.05$ . Above columns \* =  $p < 0.05$  difference between groups.

In men, muscle activation of vastus medialis during maximal bilateral isometric leg press decreased significantly in SE, but not ES ( $p < 0.05$ , Figure 22). In women, no significant changes were observed in muscle activation, but a significant difference between ES and SE was observed at post ( $p < 0.05$ ).

### 5.10 Acute cardiorespiratory responses (IV, V)

Running economy ( $\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ ) at the beginning of the endurance loading was reduced in both men and women when strength was performed before the endurance loading ( $p < 0.01$ , Table 12). Running economy in SE women was significantly decreased in comparison to ES women at the end of the E loading ( $p < 0.05$ ). Respiratory exchange ratio decreased significantly ( $p < 0.01$ ) during the E loading, but no differences between women were observed. Heart rate increased significantly over the endurance loading for all groups of men and women ( $p < 0.01$ ); however, no differences were observed between combined sessions.

TABLE 12 Running economy, respiratory exchange ratio and heart rate during the endurance loading (mean  $\pm$  SD). Differences within groups from pre to post and mid to post \* =  $p < 0.05$ , \*\* =  $p < 0.01$ , \*\*\* =  $p < 0.001$ . Difference between loadings a-a, b-b =  $p < 0.01$  and c-c =  $p < 0.05$ .

|  | PRE -E                      | POST - E                       |
|--|-----------------------------|--------------------------------|
| <b>Running economy (ml <math>\cdot</math> kg<sup>-1</sup> <math>\cdot</math> min<sup>-1</sup>)</b> |                             |                                |
| Men ES   | 43.3 $\pm$ 3.7 <sup>a</sup> | 44.7 $\pm$ 3.7 <sup>*</sup>    |
| Men SE   | 45.0 $\pm$ 4.0 <sup>a</sup> | 45.3 $\pm$ 4.4                 |
| Women ES   | 37.9 $\pm$ 3.3 <sup>b</sup> | 38.5 $\pm$ 3.8 <sup>c</sup>    |
| Women SE   | 39.1 $\pm$ 3.4 <sup>b</sup> | 39.3 $\pm$ 3.5 <sup>c</sup>    |
| <b>Respiratory exchange ratio</b>  |                             |                                |
| Men ES   | 0.93 $\pm$ 0.03             | 0.89 $\pm$ 0.03 <sup>***</sup> |
| Men SE   | 0.93 $\pm$ 0.03             | 0.87 $\pm$ 0.04 <sup>**</sup>  |
| Women ES   | 0.90 $\pm$ 0.03             | 0.85 $\pm$ 0.03 <sup>***</sup> |
| Women SE   | 0.89 $\pm$ 0.04             | 0.84 $\pm$ 0.04 <sup>***</sup> |
| <b>Heart rate (bpm)</b>  |                             |                                |
| Men ES   | 151 $\pm$ 8                 | 160 $\pm$ 10 <sup>***</sup>    |
| Men SE   | 154 $\pm$ 11                | 162 $\pm$ 10 <sup>**</sup>     |
| Women ES   | 159 $\pm$ 13                | 166 $\pm$ 12 <sup>***</sup>    |
| Women SE   | 160 $\pm$ 17                | 167 $\pm$ 17 <sup>**</sup>     |

## 5.11 Serum hormone responses (IV, V)

### 5.11.1 Time of day responses

In men, serum testosterone levels fluctuated during both combined sessions (Figure 23). A significant decrease in serum testosterone was observed at mid in SE ( $p < 0.01$ ); while a significant decrease in serum testosterone was observed at post in ES ( $p < 0.01$ ). No significant differences between groups were observed during the combined sessions. During recovery at 24 and 48 h, serum testosterone was decreased relative to pre in SE ( $p < 0.01$  and  $p < 0.05$ , respectively). At both 24 and 48 h, a significant difference between ES and SE groups of men was observed ( $p < 0.05$ ). Serum testosterone levels did not change significantly in women during the combined sessions or recovery, but a significant difference between ES and SE was observed at post ( $p < 0.05$ ).

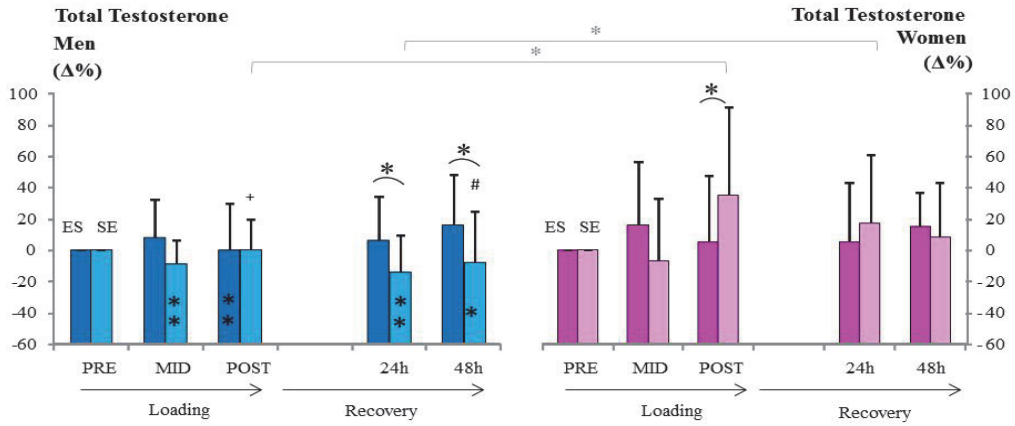


FIGURE 23 Serum total testosterone. Differences within groups from pre inside columns \*\* =  $p < 0.01$ , and from mid above columns + =  $p < 0.05$ . Above graph \* =  $p < 0.05$  between relative changes in serum testosterone concentrations from pre in men and women. Above columns \* =  $p < 0.05$  between relative changes from pre in ES and SE sessions.

The relative cortisol response in ES and SE was significantly different in men at post ( $p < 0.05$ ), but did not differ at mid (Figure 24). In women, no significant differences in differences in serum cortisol response were observed between combined sessions. A significant difference in the magnitude of cortisol response between men and women was observed at both mid and post in both ES and SE ( $p < 0.05$ ). Absolute serum cortisol concentrations in men were significantly different in ES and SE at post ( $p < 0.05$ ).

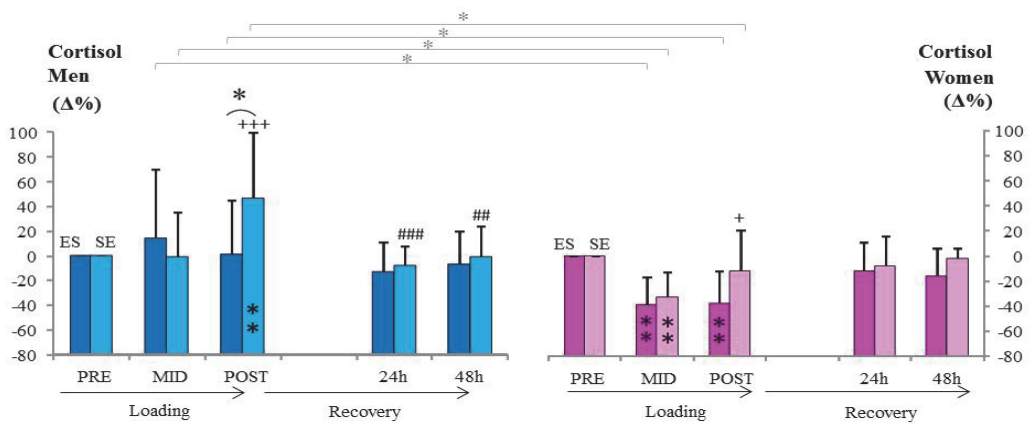


FIGURE 24 Serum cortisol. Differences within groups from pre inside columns \*\* =  $p < 0.01$ , and from mid +++ =  $p < 0.001$ . Above graph \* =  $p < 0.05$  between relative changes in serum cortisol concentrations from pre in men and women. Above columns \* =  $p < 0.05$  between relative changes from pre in ES and SE sessions.



Serum GH concentrations in men were significantly different between ES and SE ( $p < 0.001$ , Figure 25) following a similar increase in GH concentrations following the onset of exercise. At 24 and 48 h, GH concentrations had returned to baseline. In women, GH concentrations were statistically unaltered during both loadings and recovery. Differences were observed in the magnitude of GH responses between men and women at mid and post in both ES and SE ( $p < 0.05$ ), while the difference between both ES and SE responses in men was greater ( $p < 0.01$ ) than in women. Absolute GH concentrations in men were significantly different between ES and SE at post ( $p < 0.01$ ).

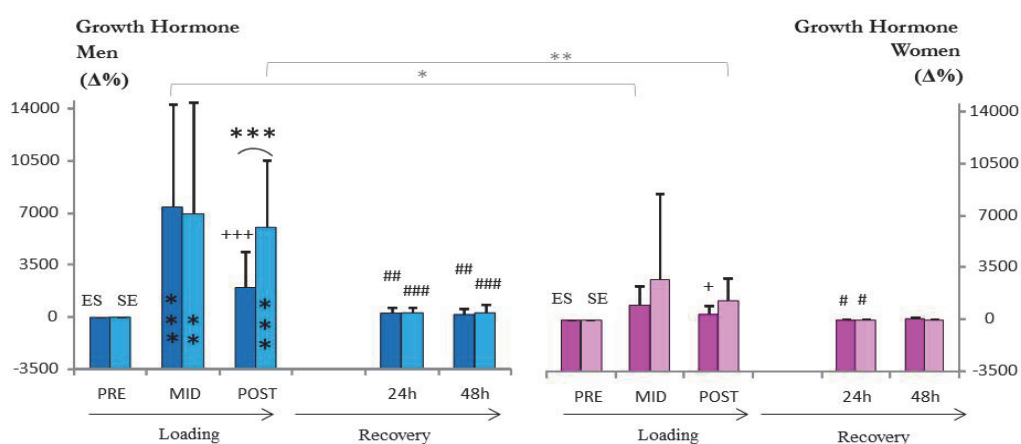


FIGURE 25 Growth hormone. Differences within groups from pre inside columns \*\* =  $p < 0.01$ , \*\*\* =  $p < 0.001$ , from mid above columns +++ =  $p < 0.001$ , and from post ## =  $p < 0.01$ , ### =  $p < 0.001$ . Above graph \* =  $p < 0.05$  and above graph \*\* =  $p < 0.01$  between changes in GH concentrations from pre in men and women. Above columns \*\*\* =  $p < 0.001$  between relative changes from pre in ES and SE sessions.

### 5.11.2 Morning serum hormone concentrations

Morning serum testosterone concentrations were significantly reduced in the SE group of men at 24 h-AM ( $p < 0.01$ ) resulting in a significant difference between morning testosterone concentrations between combined sessions ( $p < 0.05$ , Table 13). No significant changes in serum morning testosterone concentrations were observed in either group of women.

TABLE 13 Absolute serum hormone values from morning measurements.  $\square = p < 0.05$  between combined sessions. Differences within groups from Pre-AM measurements  $* = p < 0.05$ ,  $** = p < 0.001$ , and from 24hAM measurements  $+ = p < 0.05$ . Significant difference between men and women in T at all time-points, in GH at pre in SE  $p < 0.05$ , and in SHBG at all time-points.

|  | PRE-AM       | 24H-AM                      | 48H-AM                           |
|--|--------------|-----------------------------|----------------------------------|
| <b>Testosterone (nmol · l<sup>-1</sup>)</b>  |              |                             |                                  |
| Men ES                                       | 16.0 ± 4.2   | 15.5 ± 3.4 $\square$        | 15.7 ± 4.1                       |
| Men SE                                       | 15.8 ± 2.9   | 13.8 ± 2.4 $**$ , $\square$ | 14.5 ± 5.1                       |
| Women ES                                     | 1.2 ± 0.5    | 1.2 ± 0.5                   | 1.2 ± 0.5                        |
| Women SE                                     | 1.1 ± 0.4    | 1.0 ± 0.2                   | 1.1 ± 0.3                        |
| <b>Cortisol (nmol · l<sup>-1</sup>)</b>      |              |                             |                                  |
| Men ES                                       | 480 ± 99     | 502 ± 113                   | 485 ± 109                        |
| Men SE                                       | 505 ± 121    | 479 ± 83                    | 489 ± 90                         |
| Women ES                                     | 478 ± 70     | 453 ± 89                    | 386 ± 128 $**$ , $+$ , $\square$ |
| Women SE                                     | 449 ± 62     | 429 ± 61                    | 477 ± 64 $+$ , $\square$         |
| <b>Growth Hormone (mIU · l<sup>-1</sup>)</b> |              |                             |                                  |
| Men ES                                       | 0.41 ± 0.33  | 0.48 ± 0.40                 | 0.92 ± 1.45                      |
| Men SE                                       | 0.72 ± 1.05  | 1.00 ± 1.38                 | 1.15 ± 1.29 $+$                  |
| Women ES                                     | 8.84 ± 13.21 | 3.04 ± 6.55 $**$            | 1.80 ± 1.49                      |
| Women SE                                     | 4.68 ± 5.19  | 2.27 ± 2.96 $*$             | 2.68 ± 3.16                      |

No significant changes in morning serum cortisol were observed in men. A significant decrease in serum morning cortisol was observed in the ES group of women between 24 h-AM and 48 h-AM ( $p < 0.05$ ), while a significant increase in serum morning cortisol was observed in the SE group of women ( $p < 0.05$ ). These divergent responses resulted in a significant difference between serum morning cortisol concentrations between the ES and SE groups of women at 48 h-AM ( $p < 0.05$ ).

Serum morning GH concentrations increased significantly in the SE group of men between 24 h-AM and 48 h-AM ( $p < 0.05$ ). In women, serum morning GH decreased significantly in both ES and SE groups at 24 h-AM ( $p < 0.01$  and  $p < 0.05$ , respectively).

No significant correlations between the relative changes in serum hormonal concentrations and relative changes in force variables over the combined sessions and recovery period were observed.

## 6 DISCUSSION

### 6.1 Prolonged adaptations to combined strength and endurance training

In the present study, a preparatory strength training period of 6-8 weeks in which strength training was performed  $\sim 1 \cdot \text{week}^{-1}$  was completed in order for all of the subjects to become accustomed to strength training with weights. Following the preparatory period, subjects were divided into experimental training groups matched for physical characteristics and training background. During the 8-week strength training intervention, strength training was performed  $\sim 1.5 \cdot \text{week}^{-1}$ . After the strength training intervention, M, E and C continued strength training  $0.5 \cdot \text{week}^{-1}$  during a 14-week marathon training period. The preparatory strength training period, experimental strength training intervention and marathon period were combined with a progressively increasing volume of endurance training.

The present results demonstrated that strength training is effective in improving neuromuscular characteristics of recreational endurance runners despite a progressively increasing volume of endurance running, when combined strength and endurance training is periodized. Initial significant gains in maximal and explosive strength were observed in almost all groups following the preparatory period. The strength training intervention then demonstrated that maximal, explosive, and mixed maximal and explosive strength training are more effective in improving neuromuscular characteristics including maximal and explosive strength, than circuit training with only body weight as a load as carried out by our control groups. The circuit training groups maintained the initial gains in strength caused by strength training during the preparatory period, but further improvements in neuromuscular performance were minimal and statistically insignificant during the actual strength training intervention. Only small and insignificant improvements in  $\text{VO}_{2\text{max}}$  were observed in the experimental strength training groups of the present study, but overall endurance performance improved via peak running speed at maximal oxygen uptake

( $S_{\text{peak}}$  at  $\text{VO}_{2\text{max}}$ ) in all groups. The groups performing circuit training with body weight improved their  $\text{VO}_{2\text{max}}$  and peak running speed at  $\text{VO}_{2\text{max}}$  ( $S_{\text{peak}}$ ) over the combined 6-8 week preparatory period and the actual 8-week strength training intervention. After a 14-week marathon period only the maximal strength training group was able to make further gains in  $S_{\text{peak}}$  as well as running economy without further gains in  $\text{VO}_{2\text{max}}$ .

### *Neuromuscular adaptations*

Maximal strength, as measured by dynamic bilateral leg press, increased significantly following the preparatory strength training period (pre to week 0) in M  $4 \pm 4\%$ , MM  $3 \pm 4\%$ , CM  $2 \pm 6\%$  and MW  $7 \pm 7\%$ . The strength training intervention (week 0 to week 8) caused significant gains only in M  $4 \pm 5\%$ , E  $4 \pm 3\%$ , MM  $3 \pm 4\%$ , CM  $2 \pm 4\%$  and CW  $5 \pm 3\%$ . The overall increase of 13% and 7% in 1RM were the greatest in MW and M, respectively. Initial gains in strength during a prolonged training period are typically attributed to improvements in muscle activation (Häkkinen 1994; Moritani & deVries 1979). In the present study, significant increases in muscle activation of vastus lateralis and vastus medialis were detected only in maximal and explosive strength training groups from the beginning of the study to the end of the strength training intervention (pre to week 8), while a trend was observed in the mixed maximal and explosive strength training group. The gains in maximal strength accompanied by increased muscle activation of the trained muscles indicate training-induced changes in firing-frequency and increased recruitment of motor units (Van Cutsem et al. 1998).

The current gains in strength and muscle activation were observed despite the progressively increasing endurance training volume of approximately 17% in all groups between the preparatory period (pre to week 8) and the strength training intervention (weeks 0 to 8). After the initial strength gains, a plateau in maximal strength development was observed by 10-12 weeks of training, or halfway through the strength training intervention starting at week 4, in all training groups. This finding is similar to the observations in the classic study by Hickson (1980) in which an interference effect was reported. Maximal strength has been observed to fluctuate daily up to 5% (Sedliak et al. 2007a) suggesting that strength gains in our control group of men and explosive strength training group may not be fully attributed to training, as the average magnitude of change in these groups was less than 5%. The observed plateau in maximal strength gains at week 4 suggests that the strength training stimulus was not sufficient enough in volume, frequency, or intensity to produce further gains (Kraemer & Ratamess 2004) or could indicate that the present endurance training stimulus interfered with strength development (Hunter et al. 1987; Hickson et al. 1988). Interference, however, cannot conclusively be responsible for the plateau in strength gains in the present study because we do not have any groups from the same population performing strength training only. Nevertheless, gains in maximal dynamic strength observed in the present subject

population were noticeably smaller than the 10-20% improvements typically observed in untrained individuals performing only strength training (Ahtiainen et al. 2003; Häkkinen 1994). The present strength gains were also smaller than those observed when strength training has been combined with cycling (Häkkinen et al. 2003) which, in untrained populations, appears to yield greater increases in both dynamic and isometric maximal strength than those observed in the present study (e.g. Häkkinen et al. 2003; Karavirta et al. 2009). The greater increases observed in dynamic and isometric strength resulting from strength training combined with cycling may be the result of the differences in force production between the two training modes of running and cycling (Bijker et al. 2002). While the leg extensors produce a large amount of force during the concentric phase in cycling, running utilizes rapid and repetitive stretch-shortening cycle actions, which does not fully activate the leg muscles (Sloniger et al. 1997; Blazeovich et al. 2003). This difference in force production in the endurance training modes may affect training adaptations and may also be influenced by the specific body positions used for measurements (Blazeovich 2012) that may not match the body positions used in training.

Like maximal strength, a significant increase in explosive strength measured by jump height was observed following the preparatory strength training period (pre to week 0) in M  $7 \pm 10\%$ , E  $7 \pm 5\%$ , MM  $5 \pm 4\%$  and CM  $9 \pm 9\%$ , but no increase in jump height were observed in women. The strength training intervention (week 0 to week 8) caused significant gains only in M  $5 \pm 5\%$ , E  $5 \pm 8\%$ , MM  $6 \pm 7\%$ , MW  $9 \pm 7\%$  and CW  $7 \pm 7\%$ . Again, a plateau in explosive strength was observed at approximately 10-12 weeks of combined training (at week 4) in all groups except for the group of mixed men that made further significant gains in jumping height in the final 4 weeks of the strength training intervention ( $5 \pm 7\%$ ) between week 4 and week 8). The plateau in explosive strength may be attributable to an insufficient training stimulus (Kraemer & Ratamess 2004) or interference (Hunter et al. 1987; Hickson et al. 1988) as discussed above in terms of maximal strength. Concomitant with the increase in jump height, muscle activation of vastus lateralis and vastus medialis was observed to increase significantly in all strength training groups. While specific adaptations to explosive resistance training may be hindered by endurance training (Hunter et al. 1987; Häkkinen et al. 2006), similar improvements in explosive performance and muscle activation following combined explosive strength training and endurance running have previously been observed (e.g. Paavolainen et al. 1999c; Mikkola et al. 2007). These improvements, like the improvements in maximal strength, may be credited to a higher firing-frequency and increased recruitment of motor units (Van Cutsem et al. 1998).

Improvements in neuromuscular performance were highly individual as is suggested by the standard deviations observed in the present study. Thus, in the current population of recreational endurance runners, the more multifaceted nature of mixed maximal and explosive strength training combined with endurance running may have provided a slight but statistically insignificant advantage over maximal and explosive strength training at least in the explo-

sive strength of men. Previously mixed methods of training have been shown to be effective in improving strength and power (Newton & Kraemer 1994; Kyröläinen et al. 2005; Heggelund et al. 2013; Newton et al. 2002). The experimental strength training groups (M, E, MM, MW) improved in both maximal and explosive strength; however, significant differences between the magnitudes of improvements were not observed. The lack of significant differences between the training groups of men in terms of strength development is, however, not entirely surprising. The current population of recreational endurance runners, being untrained in terms of strength, may not have had an adequate level of strength and power that is required for specific adaptations to explosive strength training to occur (Newton & Kraemer 1994) as there seems to be a tendency towards greater adaptations in explosive strength when a “base” of maximal strength has already been established (Cormie et al. 2010a). In addition, the movement patterns of exercises used in the present training programs may not have been different enough to show specificity of training (Blazevich et al. 2003). Furthermore, it is possible that a longer duration such as 8-12 weeks of training is needed prior to observing differences in adaptations to different strength training modalities (Häkkinen 1994). Both maximal and explosive strength training target fast-twitch muscles, so there may also be some crossover in adaptations to these strength training methods. The significant difference in magnitude of increase in maximal strength in women showed that mixed maximal and explosive strength training was more effective in women in developing strength than circuit training with only body weight. This also shows that untrained women have a high capacity for strength improvements. Unfortunately, the study did not include maximal and explosive strength training groups for women, so it is unclear as to whether or not another strength training stimulus combined with endurance training would have been more effective for recreationally endurance trained women.

Over the current prolonged study, improvements in strength and power capabilities were accompanied by improvements in neural activation. The present study, however, used only surface electromyography to measure changes in muscle activation of vastus lateralis and vastus medialis. While this method is both reliable and valid (Hermens et al. 1999; Merletti et al. 2001), it gives somewhat limited information on the mechanisms behind the present neuromuscular adaptations i.e. surface electromyography is only able to measure activity at the level of the muscle (Millet et al. 2012). Muscle stimulation using the superimposed twitch technique during maximum isometric contraction, could have provided further information regarding the mechanisms behind increased forced production. The technique superimposes an electrical “twitch” over the maximal isometric contraction, and by comparing the resulting signal to a resting signal; one is able to determine voluntary activation as a percentage of maximum activation (Millet et al. 2012; Harridge et al. 1999). Even using the superimposed twitch technique, the source of changes in muscle activation during a maximal voluntary isometric contraction may not be fully elucidated (Harridge et al. 1999).

In addition to changes in muscle/neural activation, we might expect to observe changes in fiber type composition following a prolonged period of combined training including e.g. an increase in type IIa fibers as was observed by Kraemer et al. (Kraemer et al. 1995) or even augmented capillarization as observed by Bell et al. 2000 (Bell et al. 2000). Unfortunately, the only reliable method for determining fiber type transformations is via the invasive muscle biopsy technique (e.g. Blomstrand & Ekblom 1982) that we did not make use of in the present study. Although we did not measure muscle hypertrophy using muscle biopsies, further muscular changes resulting from the present combined strength and endurance training were observed via ultrasound measurements of the quadriceps including vastus lateralis and vastus intermedius. The ultrasound measurements detected significant hypertrophy in the maximal strength training group between pre and week 4 (~7% at total of 10 weeks of combined training). Although magnetic resonance imaging is the gold standard for measuring changes in muscle hypertrophy, use of ultrasound is a valid and easily accessible technique for a study of this nature (Reeves et al. 2004). We can speculate that these significant changes in muscle mass might also reflect changes in muscle fibers as suggested above, as well as changes in muscle morphology. Possible changes in muscle morphology include increased pennation angle and lengthening of fascicle length with hypertrophy (Abe et al. 2000).

Following the 14-week marathon training period in men, a significant decrease in maximal strength ( $-5 \pm 3\%$ ) and muscle activation of vastus lateralis and vastus medialis was observed in the maximal strength training group while insignificant decreases were observed the explosive ( $-3 \pm 6\%$ ) and control groups ( $-3 \pm 5\%$ ). Explosive strength (jump height) was also observed to decrease; however, the decreases were insignificant. These decreases in strength and power were likely influenced by the progressive increase in endurance running volume (running kilometers  $\cdot$  week<sup>-1</sup>) and intensity (training above aerobic and anaerobic thresholds) (Hickson et al. 1988) that occurred over the 14-week marathon training period. This significant increase in running kilometers coincided with the plateaus and subsequent decreases in maximal strength, explosive strength and muscle activation observed throughout the study. It is possible that the increased running volume resulted in mechanical stress that interfered with muscle strength (Bell et al. 2000). As the muscles of the legs are not fully activated even during high-intensity running (on horizontal or up-hill), there is a limitation in the number of muscle fibers that can be recruited in order to increase force production capabilities via running (Sloniger et al. 1997; Blazeovich et al. 2003). With only a low frequency and low volume strength training stimulus during the 14-week marathon training period, it is possible that a detraining of the previously strength trained muscle fibers occurred (Kraemer & Ratamess 2005; Lemmer et al. 2000; Häkkinen & Komi 1983; Häkkinen et al. 1985a; Häkkinen 1994; Kraemer et al. 2001). The ultrasound measurements detected significant atrophy that occurred over the 14-week marathon training period in the explosive strength training group ( $-5\%$ ), which may also indicate

changes in muscle morphological characteristics including a decrease in pennation angle and shortening of fascicle length (Abe et al. 2000).

Body composition as a whole was observed to change significantly primarily over the 6-8 week preparatory period in E and MM including a significant decrease in overall body fat percentage. In MW, a significant decrease in body fat was observed slightly later, at week 4. Actual total body weight decreased only in the explosive strength training group with respect to baseline. Body weight and % body fat were measured using bioimpedance. Measurements were always taken in conjunction with fasting blood samples to limit the possible confounding factors related to e.g. hydration status. Body composition, of course, may have a marked influence on athletic performance. Endurance athletes typically aim to be lean because excessive muscle mass or body fat is considered to be non-functional load and may be detrimental to performance (Eisenmann & Malina 2000) as greater body mass requires more oxygen for the same endurance performance.

### *Cardiorespiratory adaptations*

Our subject population was representative of recreational endurance athletes, which is evidenced by aerobic fitness values that are slightly higher than population norms (Armstrong 2006), but lower than what would typically be observed of elite endurance athletes. Based on the study design, only minor changes in  $VO_{2max}$  were expected because the training intensity for the majority of the duration of the study was low (below the aerobic threshold). Endurance performance capacity has traditionally been determined by measuring maximal oxygen uptake ( $VO_{2max}$ ), the fractional utilization of  $VO_{2max}$ , and economy, (Bassett & Howley 2000), however, maximal anaerobic capacity and neuromuscular characteristics have become additional predictors of endurance performance. While an increase in  $VO_{2max}$  is certainly positive and desired, it is important to recall that improvements in running performance are not always attributed to increases in  $VO_{2max}$  alone (Daniels et al. 1978). Increases in endurance performance may occur without increases in  $VO_{2max}$  by way of improvements in sport-specific economy and neuromuscular function (Paavolainen, 1999b; Nummela et al. 2006; Paavolainen et al. 2000) including maximal strength, power, and muscular activation as well as  $S_{peak}$ .

In the present study, significant increases in  $VO_{2max}$  ( $ml \cdot kg^{-1} \cdot min^{-1}$  and  $L \cdot min^{-1}$ ) were observed only in the maximal strength training group of men and both control groups over the preparatory period and strength training intervention. The goal of lower intensity training is not to increase  $VO_{2max}$  as much as it is to improve substrate utilization (Horowitz & Klein 2000), whereas intermittent high-intensity training (interval training) is more effective than moderate intensity or low intensity training in improving  $VO_{2max}$  over a shorter duration of time (Helgerud et al. 2007; Tabata et al. 1996). Changes or improvements in other endurance characteristics such as  $S_{peak}$  and RE may, nevertheless, improve via lower intensity training (Bassett & Howley 2000). The in-



crease in  $VO_{2max}$  of the control group is likely due to the endurance nature of their strength training mode. Circuit training with body weight is “endurance training” and can increase HR in a similar way as intervals. This training, combined with actual endurance running, resulted in the circuit training group performing a greater volume and intensity of overall endurance type training than that performed in the experimental group, which may explain the increase in  $VO_{2max}$  of the control groups.

In two runners with the same or similar  $VO_{2max}$ , the runner with better economy will generally have an advantage, while running economy can also explain some of the sex-difference in performance between men and women with similar  $VO_{2max}$  (Helgerud et al. 1990). In performance-matched male and female marathoners, better running economy, training, and ability to perform at a higher percentage of  $VO_{2max}$  explained why the performance level in men was the same in women despite a greater  $VO_{2max}$  (Helgerud 1994). In the present study, running economy was shown to improve significantly primarily in the control group of men following the preparatory strength training period. Interestingly, over a more prolonged period of time, the group performing maximal strength training during the strength training intervention had the greatest improvement in running economy during the 14-week marathon training period. Previously, improved economy has been associated with improved 1RM and fast-force production, while the velocity of movements (intentional velocity) has been identified as a possible training program variable responsible for improvements in economy between different strength training modes (Heggelund et al. 2013). It is possible that the neuromuscular improvements observed in the maximal strength training group, such as improved 1RM and CMJ, prepared subjects for increased endurance running intensity and volume, which was similar between groups at the end of the marathon training period. Based on these and previous observations, we might speculate that mixed maximal and explosive strength training would have had a similar effect. Maximal strength training has previously been linked to improvements in work economy, although this does not always seem to be the case (e.g. Losnegard et al. 2011). It has been suggested that strength training programs that are successful in increasing maximal strength and fast force production are most effective in improving work economy (Heggelund et al. 2013). In the present study, we might expect that the endurance training intensity and volume may have aided in improving endurance performance parameters, but strength training appears to have had a marked influence on running performance as well because the marathon running period itself did not induce further significant gains in  $VO_{2max}$ . In addition, running speed at anaerobic threshold was shown to improve significantly in all training groups while  $S_{peak}$  and running economy at  $10 \text{ km} \cdot \text{h}^{-1}$  improved significantly over the marathon training period in the maximal strength training group.  $S_{peak}$ , which combines  $VO_{2max}$  and RE as a single value (Billat & Koralsztejn 1996) increased in all groups, which suggests an increase in endurance performance capabilities, as greater values have been shown to predict performance (Noakes et al. 1990).

Fast force production has been found to be an important characteristic for peak running speed as well as running economy (Nummela et al. 2007), while jump ability is significantly correlated with middle and long distance running performance (Hudgins et al. 2012). Improvements in neuromuscular characteristics via strength training should transfer to the neuromuscular characteristics of endurance performance like  $S_{\text{peak}}$  and running economy. These improvements appear to be relatively similar between maximal and explosive strength training modes, although adaptations to each strength training mode showed some degree of specificity of training. This observation is in line with Cormie et al. (2010b) who observed that in untrained individuals, the mechanisms that contribute to improved athletic performance may be different, although the improvement in the outcome measures are similar.

#### *Serum hormone concentrations*

Concentrations of serum hormones have been used to monitor training stress, while also providing other information about training status or giving clues about possible pathological conditions that may be affecting training responses and adaptations. Increased cortisol, for example, may suggest overreaching, overtraining, or significant psychological stress. In the present study, serum testosterone concentrations increased significantly between pre and week 4 in the mixed group of men, which was followed by a significant decrease back to baseline. No significant changes in serum testosterone concentrations were observed in the other groups. The increase in serum testosterone may be indicative of an increase in the anabolic milieu of the body related to the increase in training intensity/volume (e.g. Ahtiainen et al. 2003; Häkkinen et al. 1988b; Häkkinen et al. 1987). Serum cortisol remained statistically unaltered over the combined training periods as they have in other studies (Bell et al. 2000) suggesting that the combined training loads were well tolerated by our subjects. In women, TSH decreased following “the onset of strength training” in both groups of women indicating possible feedback related to the added stress of strength training (Pakarinen et al. 1991). Subsequently, the return to baseline showed evidence of strict homeostatic control via the thyroid gland (Kraemer et al. 1995). During the marathon training period, testosterone to cortisol ratio decreased significantly in the maximal strength training group but was not significantly lower than at baseline during the preparatory period and strength training intervention. This indicates that some stress may have contributed to strength and power decreases (Häkkinen et al. 1985c), however, as the ratio stayed above baseline, we can assume that overtraining had not occurred.

A number of limitations exist when monitoring hormones including diurnal variation, pulsatile secretion and nocturnal response of specific hormones that may affect measurements. Nutrition, overtraining/excessive fatigue and stress may also influence measurements. Possible confounding factors should have been taken into consideration in the study designs when possible in order to preserve the legitimacy of the hormonal data (Hackney & Viru 2008). The

present hormonal data is specific to the present combined strength and endurance training and the current population of recreational endurance runners as changing any of the present training variables could influence different responses.

### *Interference effect?*

Although the present study cannot prove the existence of interference because the study design does not include groups that performed only strength training, based on previous studies using maximal or explosive strength training programs, we might expect a greater magnitude of development in neuromuscular characteristics (Hickson 1980; Wilson et al. 2012; Häkkinen et al. 2003; Leveritt et al. 1999). In addition, in untrained subjects, we would expect the plateau in strength and power development to occur somewhat later considering that the strength training programs were progressive in nature. The present strength training frequency of 1.5 sessions · week<sup>-1</sup> may have also influenced the relatively small magnitude of changes as it, combined even with a relatively low volume and intensity of endurance training, may not have been adequate enough to produce greater changes. Furthermore, running as an endurance training mode has been shown to be the most detrimental to strength development because of e.g. muscle damage in a recent meta-analysis of combined strength and endurance training (Wilson et al. 2012). This suggests that at least a small degree of interference would exist in the present study, although the overall outcome for the recreational endurance runners was positive.

It may be suggested that strength training interfered with endurance capacity in terms of  $VO_{2max}$ , as there were no significant increases in  $VO_{2max}$  of the groups of men performing explosive and mixed maximal and explosive strength training as has sometimes been observed (Glowacki et al. 2004). Lower intensity endurance training as used in the present study, however, is not expected to induce significant gains in  $VO_{2max}$  (Helgerud et al. 2007; Tabata et al. 1996) and strength training is not often associated with blunted endurance performance/capacity over a short period of time.

## **6.2 Acute responses to combined sessions of strength and endurance**

The examination of combined strength and endurance in a single combined session using a cross-sectional study design showed that the order of exercises in a single-session, either endurance followed by strength (ES) or strength followed by endurance (SE), may lead to differing acute responses and recovery even when combined sessions are of the same relative duration and intensity. The present results suggest that neuromuscular and hormonal responses follow a different time course of recovery. In addition, the results demonstrate that the

intra-session order of exercises affects some of these responses. In men, suppressed serum testosterone concentrations were observed following SE during the recovery period at 24 and 48 h post combined session; while in women, a delay in neuromuscular fatigue was observed in explosive strength measured by countermovement jump height following ES during the recovery period at 24 and 48 h post combined session. Running economy was impaired in both men and women when the strength loading was performed before the endurance loading, however, no other evidence of an order effect or influence of intra-session order of exercises was observed in the present cardiorespiratory measures.

### *Neuromuscular responses*

In the present study, both ES and SE sessions led to significant decreases in maximal and explosive strength. The cumulative neuromuscular fatigue, as measured by maximal bilateral isometric force and countermovement jump height, was the same post combined session in ES and SE. A greater magnitude of fatigue was observed in men than in women in both variables. In both men and women, it is clear that the strength loading led to greater acute decreases in maximal strength than the endurance loading, although the difference was only significant in men. Interestingly, countermovement jump height decreased only in men and not women during the combined sessions. Again, the decrease in jump height was greater following the strength loading than the endurance loading, but the difference was not significant.

In men, maximal force was significantly reduced during the recovery period at 24 and 48 h after the combined sessions, but maximal force returned to baseline only in SE by 48 h. In women, maximal force returned to baseline already 24 h after the combined sessions. Countermovement jump was significantly reduced in men at 24 and 48 h post combined sessions except for ES which had returned to baseline at 48 h. Interestingly, countermovement jump height of women was reduced 24 h after the combined sessions and jump height ES was significantly lower than in SE at both 24 and 48 h. In part, we can only speculate about the reason for the delayed fatigue response, however, as both combined strength and endurance training sessions contain eccentric muscle actions, we can infer that some degree of muscle damage may have occurred (Proske & Allen 2005). Currently unpublished data does suggest that muscle damage plays a role in the delayed fatigue response observed in ES women because the absolute values for creatine kinase activity, an indirect marker for muscle damage, are well above resting norms during the recovery period at 24 and 48 h. This creatine kinase response appears to be somewhat individual, but as it may also explain the continued decrease in maximal and explosive strength also observed in men, it appears to be of importance as it is an indicator of muscle damage that may cause impairments in muscle contractile function (Byrne et al. 2004).

In addition to maximal bilateral isometric force, rate of force development and countermovement jump, the present study used surface electromyography to evaluate changes in muscle activation during the loading to detect fatigue. Muscle activation of vastus medialis was shown to decrease in men following the combined loading, but significance was achieved only in SE. In women, muscle activation did not change significantly. The reason for maintained muscle activation in women and ES men may be related to recruitment of higher threshold motor units or an increase in motor unit discharge rate (De Ruiter et al. 2005), however, this does not explain the decrease in force production that occurred concomitantly in 1RM in ES men and ES and SE women. Muscle activation was not examined at 24 and 48 h post loading, but it is possible that activation was impaired as the decrease in maximal muscle activation often parallels the decrease in maximal strength (Häkkinen & Komi 1986). The greater decreases in maximal and explosive strength, as well as muscle activation observed in men demonstrated that men have the ability to fatigue themselves more than women, a common finding (Bosco et al. 2000; Häkkinen & Pakarinen 1995; Linnamo et al. 2005). This ability is likely due, in part, to fiber type distribution and size between men and women. Men generally have a greater cross-sectional area of muscle fibers with type IIa fibers being the largest, while women generally have smaller cross-sectional area of muscle fibers with the largest fibers being type I fibers (Staron et al. 2000). Type II fibers and type II subtypes generate rapid contractions while type I fibers produce slower contractions. Type II fibers are capable of generating more force per cross-sectional area than type I fibers, but they are more fatigable, whereas type I fibers generate less force, but are less fatigable (Kraemer & Häkkinen 2002). This greater endurance and presence of less fatigue during the combined sessions in women may be credited to a larger proportion of type I muscle fibers, which may act as a “protective” mechanism against fatigue (Martin & Rattey 2007; Hicks et al. 2001).

Significant decreases in strength and power are a clear indication of fatigue, however, we are unable to determine with certainty whether the fatigue is central or peripheral in nature, or if both central and peripheral mechanisms were contributing to overall fatigue. While surface electromyography measures muscle activation, using the superimposed twitch technique mentioned earlier, we could have determined whether or not the decrease in muscle activation was centrally mediated or due to a reduction in contractile properties on a peripheral level (Millet et al. 2012; Taylor & Gandevia 2008). From the data collected we can suggest that both central factors including decreased recruitment of motor units and reduced firing frequency of active motor units (Gandevia 2001) and peripheral factors such as possible lactic acid accumulation inside the muscle, decreased pH, and decreased  $\text{Ca}^{2+}$  transport may have contributed to inhibited muscle contractile characteristics (Sahlin 1992). Both types of fatigue may have been present, although central drive could have compensated for some peripheral fatigue by maintaining force production via recruitment of higher threshold motor units (Henneman et al. 1965) or an increased in motor unit discharge rate (De Ruiter et al. 2005).

### *Cardiorespiratory responses*

Running economy in men was negatively affected when the strength loading preceded the endurance loading, although there was no significant difference in running economy at the end of the endurance loading. In women, running economy was compromised for the entire endurance loading when the strength loading was performed first. Increased consumption of, or need for, oxygen is an indicator of fatigue or decreased running economy; however, it is unclear as to whether or not the source of this fatigue was central or peripheral in nature. An increase in blood lactate induced by the present mixed maximal and explosive strength training may be hypothesized to decrease running economy while neuromuscular fatigue may also be a contributing factor (Saunders et al. 2004) as discussed above.

Respiratory exchange ratio behaved similarly regardless of the intra-session order of loadings used in the combined sessions. This indicates that performing the current type of strength loading prior to endurance running does not significantly affect substrate utilization. Respiratory exchange ratio may not be sensitive enough to be used in this context to demonstrate fatigue. Similarly, heart rate during the endurance loading increased steadily throughout the loading and appeared to be unaffected by the intra-session order of exercises. This increase in heart rate during the loading demonstrated what may be normal "cardiac drift" that is associated, in part, with hydration status (Lambert et al. 1998).

### *Hormone responses*

The serum testosterone response in men was similar from pre to post in both ES and SE sessions with no differences observed between combined sessions. In women, a difference in the magnitude of serum testosterone response was observed at post ES and SE with a greater increase observed following SE. Typically, an acute increase in serum testosterone concentrations associated with strength development and muscle growth after a period of training (Kraemer & Ratamess 2005; Ahtiainen et al. 2003) is observed immediately after a single strength training session. While maximal force production capabilities returned to baseline, it appears that serum testosterone concentrations continued to respond to the exercise stimulus. In men, the serum testosterone responses in ES and SE were observed to differ significantly during recovery at 24 and 48 h post combined session with both absolute and relative serum testosterone concentrations being lower in SE than ES on both days. In contrast, serum concentrations of testosterone were similar at 24 and 48 h of recovery in women. The recovery of serum testosterone concentrations has been observed after 1-2 days of rest in some studies (McCaulley et al. 2009; Häkkinen et al. 1988a) but very stressful resistance loading conditions may suppress serum testosterone levels for up to 2 days despite rest (Häkkinen & Pakarinen 1993). This finding suggests that serum testosterone concentrations may not return to baseline values following

strenuous training in men (Häkkinen & Pakarinen 1993), however, the mechanism causing the suppressed testosterone is unknown, although an increase in catabolic state may stimulate recovery and gluconeogenesis (Nindl et al. 2001). In addition, we might speculate that the hypothalamic-pituitary axis is partially responsible due to attenuation in luteinizing hormone secretion as was observed by Nindl and colleagues (2001) following a heavy strength loading (Nindl et al. 2001). Suppressed serum testosterone concentrations suggest that the hormone is being metabolized for physiological processes (like promoting muscle growth) (Kraemer & Rogol 2008) but may also suggest the presence of a protein catabolic state (Adlercreutz et al. 1986), which may be harmful over a prolonged period. It is important to note, however, that during recovery, the decreased serum testosterone observed in the present study was not accompanied by significantly elevated cortisol.

Serum cortisol concentrations in men were similar at mid ES and SE sessions, but significantly different at post, with a greater concentration of serum cortisol observed in SE than ES. The greater increase in cortisol observed in SE suggests that the combined session may have been more demanding than the combined ES session (Izquierdo et al. 2009). The changes in serum cortisol concentrations following the acute combined sessions paralleled changes observed in serum growth hormone (GH) concentrations similar to Viru et al. (2001). In women, no differences were observed between ES and SE sessions in terms of serum cortisol response, however, cortisol decreased during the combined session similarly to Copeland et al. (2002) who examined separate strength and endurance loadings in women. Recovery of serum cortisol at 24 and 48 h was complete in both loadings in men and women. As cortisol is known to be an important factor in the activation of cascades leading to mitochondrial biogenesis in skeletal muscle (Goffart & Wiesner 2003), the greater magnitude of increase in serum cortisol stimulated by the SE loading in men and the similar increase observed in women may be physiologically important for endurance development should it occur after multiple training bouts.

The initial serum GH response in the present study following E and S loadings at mid in men was the same, whereas serum GH concentrations decreased significantly when E was followed by S (ES) while remaining elevated when S was followed by E (SE) at post. Similar GH responses were observed by Goto et al. (2005) when 60 min of low intensity cycling was performed prior to a strength loading. In women, serum GH responses were minor, while recovery was complete in both men and women by 24 h. The sustained elevation of serum GH in men during the SE session is difficult to explain, but may be linked to the previous observation that the start of aerobic exercise initiates an increase in serum GH concentrations that generally peaks at or close to the end of aerobic exercise (Weltman et al. 1992). Following fatiguing heavy-resistance protocols, however, GH concentrations have been observed to remain elevated for 1-2 h (Häkkinen & Pakarinen 1993). The decrease in serum GH concentrations observed after mid in ES may partially be explained by the same phenomena because aerobic exercise had stopped and the present strength loading used

relatively short contraction times and 2 min of recovery between sets. It is important to recall that the loading for strength was made up of both maximal and explosive exercises, and included several periods of rest between each set of exercises. Serum GH response magnitude is generally larger in “more fatiguing” strength loadings (Kraemer et al. 1990) and is amplified with repeated loadings of strength (Kanaley et al. 1997). As the GH variant examined in this study was the most abundant 22kDa variant of the many GH variants, it is important to acknowledge that other variants of GH might respond differently. As such, it is important to exercise some caution when interpreting these results. In light of the present observation, however, it seems reasonable to suggest that the extended period of GH elevation induced by the SE session could have some physiological significance if it is repeated during subsequent SE sessions.

As expected, the hormonal responses in women were somewhat smaller in magnitude than those observed in men (Häkkinen & Pakarinen 1995; Linnamo et al. 2005; Kraemer et al. 1998; Häkkinen 1993). The primary reason for the difference in responses is likely due to the innate differences between men and women, although the present combined sessions may also be responsible. Lower volume strength and power loadings induce smaller hormone responses than high volume loadings with shorter periods of rest (Kraemer et al. 2010). For the subjects that are slightly better trained, the intensity of the present combined sessions may not have been adequate to induce a great response.

In addition to measuring hormones at the same time of day as the combined sessions at 24 and 48 h, hormones were measured in the morning 24 and 48 h after the combined sessions. Interestingly, parallel changes in the morning and time of day concentrations of testosterone were observed suggesting that the SE session produced a larger stress response than the ES session. This finding also suggests that serum testosterone concentrations may not return to baseline values following training that induces physiological stress (Häkkinen & Pakarinen 1993). The mechanism behind the suppressed testosterone, in this case, is unknown, but an increase in catabolic state may stimulate recovery and gluconeogenesis (Nindl et al. 2001).

Serum cortisol concentrations did not change significantly in men during recovery whereas in women, serum cortisol was observed to decrease significantly in ES and increase significantly in SE. Interestingly, time of day hormone response and neuromuscular responses did not reflect any additional stress or fatigue suggesting that the observed elevated cortisol in men might be related to typical diurnal variation. Cortisol is controlled by diurnal variations in both young men and women (Häkkinen & Pakarinen 1995; Kanaley et al. 2001; Hackney & Viru 1999), however, because measurements were consistently taken at the same time of day, the changes observed in serum concentrations are expected to be true representations of the physiological responses, especially since cortisol appears to be stable during morning measurements despite training (Hackney & Viru 1999).

The concentration of serum GH was significantly elevated in the morning in men 48 h after the SE session, while in women GH was significantly lower



than baseline during recovery at 24 h post session in ES and SE. In previous studies, serum GH concentrations have stabilized already 1-2 h after more fatiguing loadings (Häkkinen & Pakarinen 1993). Serum GH is also secreted in a pulsatile manner throughout the day influenced by numerous external stimuli (Hartman et al. 2008), meaning that the full response of the hormone may not have been observed. Further responses may also have occurred immediately after the combined training sessions (e.g. (Kraemer et al. 1990)). Fluctuation in hormones may be a function of the previously discussed diurnal variations or response to psychological stress, as physiological functions appear to follow daily rhythms while trying to maintain homeostasis. It is well known that diurnal variations and nocturnal responses may conceal some physiological responses to exercise (Häkkinen & Pakarinen 1995), however, there is evidence that the magnitude of e.g. GH response to exercise is independent of time of day (Kanaley et al. 2001).

The blood samples for this investigation were only taken at specific times during and post combined sessions, which limits, to some extent, our ability to identify the precise overall responses of hormones. At the same time, taking both morning and time of day measurements allows for better monitoring of hormone responses (Sedliak et al. 2007b). The typical diurnal variation of e.g. serum testosterone concentrations include an increase during sleep (overnight) and a significant decrease in testosterone concentrations is typically observed in the morning between 04:00 and 08:00 with more gradual decreases observed from 08:00 until 22:00 (Kraemer et al. 2001). Our hormone measurements were taken between 07:30 and 08:00 when only minor changes in serum testosterone concentrations are expected, thus the observed decrease in morning testosterone concentrations indicate that the regulatory feedback loop of pituitary-testicular action was actually inhibited. It is also possible that the metabolic clearance was somehow accelerated or that hemodilution occurred.

### **6.3 Methodological strengths and limitations**

The present longitudinal study (papers I-III) examined 3 unique training periods of combined strength and endurance training. The 6-8 week preparatory strength training period was used for learning and becoming accustomed to training and equipment limiting the learning effect on strength gains during the subsequent 8-week strength training intervention. In addition, a 14-week marathon training period in which strength training frequency was reduced to examine the long-term training adaptations and influence on performance. The present cross-sectional study (papers IV-V) was also unique in that it examined the responses to different intra-session orders of combined strength and endurance training sessions in both men and women. Both studies used multiple neuromuscular, cardiorespiratory and hormonal variables to examine the mechanisms behind responses and adaptations. Both studies were well controlled and closely supervised and contribute additional information to the current scien-

tific body of knowledge. Subjects were always given thorough instructions about training and testing procedures and were asked not to undertake any other exercise or weight-loss programs over the duration of the study in order to minimize the effect of other external stimuli.

In the present studies, treadmill testing was used rather than running on the ground although actual training and loadings were carried out by running outside. While treadmill running is the most accessible and controllable method of testing running parameters in the lab, it is also known that there are some differences between the kinematics of running on the ground versus on a treadmill (Nigg et al. 1995, Wank et al. 2007). Nevertheless, our subjects were accustomed to running on the treadmill as well as the ground, so we are confident that the present results are reliable. In papers I-III, performance in terms of an actual marathon or other time-trial event following the combined training was not utilized. While measures of  $VO_{2max}$ ,  $S_{peak}$ , running economy and determination of aerobic and anaerobic thresholds are strong indicators of changes in endurance performance, when examining the effects of strength training on endurance performance in individuals training for a marathon, using a long distance race or time trial event may be warranted for even more practical results. On the other hand, laboratory testing is more strictly controlled for air temperature and humidity and is not affected by external meteorological phenomena, so laboratory testing is highly justified.

Nutrition is extremely important in terms of both body composition and acute hormonal responses. Nutrition was not strictly controlled in our studies; however, the previously described instructions to adhere to a normal diet were followed. Thus, there is no reason to believe that nutrition was a confounding factor in the present studies. In addition, the present subject population was relatively homogenous and the typical Finnish diet is quite normal, thus drastic changes in basic nutrition were not expected. As with diet, controlling all daily physical activity and "outside stress" over a prolonged study is almost impossible while it is very difficult even in short term or acute studies. Nevertheless, as subjects were not tested while sick and laboratory and weight room staff always communicated with subjects, there is no reason to believe that outside stressors significantly affected the present results.

Diurnal variations and time of day of testing were considered when examining possible hormonal changes in the present study. In addition, nutrition, overtraining, fatigue and stress (Kraemer & Ratamess 2005) as well as training status (Kraemer et al. 1999) were taken into consideration. Subjects were given specific instructions on how to prepare for each set of measurements with the goal of minimizing the possible confounding factors. With these procedures, the observed changes and differences in serum concentrations of hormones, although sometimes relatively small in magnitude, are believed to be accurate and may have a dynamic role in subsequent physiological processes and training adaptations. In women it is important to note that the study of serum hormones is made more difficult due to the different phases of the menstrual cycle possibly influencing hormonal responses. It appears, however, that pituitary-adrenal

responses to short-term, moderate intensity exercise are not significantly influenced by the menstrual cycle (Galliven et al. 1997) suggesting that menstrual cycle may not affect an acute loading such as the ones presented in papers IV and V.

## 7 PRIMARY FINDINGS AND CONCLUSIONS

### *Papers I, II and III: Prolonged combined strength and endurance training*

Recreational endurance runners were recruited to perform periodized combined strength and endurance training over a longitudinal study. A preparatory strength training period of 6-8 weeks, an 8-week strength training intervention, and a 14-week marathon period (including strength maintenance) were performed concurrently with a progressively increasing volume of endurance training. Periodized combined strength and endurance training appears to improve maximal and explosive strength of recreational endurance runners with concomitant improvements in parameters of endurance performance associated with neuromuscular performance.

- 1) Periodized maximal, explosive, and mixed maximal and explosive strength training combined with endurance training in male recreational endurance runners is effective in improving maximal and explosive strength, muscle activation and characteristics of running that are linked to neuromuscular performance such as peak running speed at  $VO_{2max}$  ( $S_{peak}$ ) and running economy.
- 2) Reduced strength training volume, accompanied by increased endurance training volume caused decreases in maximal and explosive strength, however,  $S_{peak}$  and running economy continued to improve in maximal and explosive strength training groups due to improved maximal and explosive strength as well as muscle activation.
- 3) The improvements in neuromuscular characteristics associated with strength training appeared to benefit our endurance runners by preparing them for a higher volume and intensity of endurance training as demonstrated over the 14-week “marathon training” period.

- 4) Mixed maximal and explosive strength training combined with endurance training is more effective in men and women in increasing maximal and explosive strength as well as muscle activation than circuit training with body weight as a load combined with endurance training. Periodized strength training (maximal and explosive) is beneficial for recreational endurance runners in improving overall performance capacity, perhaps because of its more multifaceted nature and ability to target multiple neuromuscular characteristics including both strength and power.
- 5) Serum basal hormone concentrations over the prolonged combined strength and endurance training periods in both men and women remained relatively stable. Significant fluctuations were observed in serum concentrations of testosterone of the mixed maximal and explosive strength training group of men and in serum concentrations of thyroid stimulating hormone in women. These fluctuations are indicative of possible training stress, but as they had stabilized at the end of the combined preparatory strength training period and strength training intervention, it appears that the endocrine system had adapted to the new training stimulus.
- 6) A significant decrease in testosterone to cortisol ratio was observed in the maximal strength training group during the marathon training period, but as this ratio remained above baseline, it appears that the decrease coincided with increased volume and intensity of training and does not indicate overreaching or overtraining.

#### *Papers IV and V: Acute combined strength and endurance training sessions*

Strength and endurance loadings were combined into a single session. Using a cross-sectional study design, the possible differences in acute responses and recovery 24 and 48 h after the combined session were examined. Combined sessions included an endurance loading followed by a strength loading (ES) or a strength loading followed by an endurance loading (SE). Order of intra-session loadings appears to influence acute neuromuscular and hormonal responses and subsequent short-term recovery. These findings may be important in order to optimize training sessions in prolonged training programs.

- 1) Different responses to ES and SE sessions were observed between male and female recreational endurance athletes. As expected, men demonstrated a greater degree of fatigue in terms of both maximal and explosive strength than women. A delayed fatigue response in explosive strength was observed in the ES group of women, while both maximal and explosive strength were still decreased during recovery in men 24 and 48 h after the ES and SE sessions.

- 2) Acute hormonal responses were greater in men than in women. Interestingly, the SE session caused a greater increase in serum cortisol concentrations in men at post, which was followed by significantly suppressed levels of testosterone at 24 and 48 h of recovery (both time of day and morning measurements) possibly due to suppressed function of the hypothalamic-pituitary axis.
- 3) Running economy is significantly reduced when strength is performed before endurance in both men and women, although respiratory exchange ratio and heart rate are unaffected. Reduced running economy may be linked to neuromuscular fatigue as strength and power characteristics were also reduced.

## YHTEENVETO (FINNISH SUMMARY)

### **Hermolihasjärjestelmän, sydän- ja verenkiertoelimistön ja hormonaaliset vasteet ja adaptaatiot yhdistetyssä voima- ja kestävyysharjoittelussa kestävyysharjoittelutaustaa omaavilla miehillä ja naisilla**

Hapenottokyky on yksi keskeisimmistä tekijöistä kestävyysuorituskyvyssä mutta myös hermolihasarjestelmän voimantuotolla on havaittu olevan merkittävä vaikutus mm. kestävyysuorituksen taloudellisuuteen. Näin ollen voima- ja kestävyysharjoituksia yhdistetään usein sekä kuntoilijoiden että huippu-urheilijoiden harjoittelussa. Nykyisten kansainvälisten ja kotimaisten liikuntasuosittelusten täyttäminen edellyttää sekä voiman että kestävyiden viikoittaista harjoittelua. Kestävyysharjoittelulla kehitetään hengitys- ja verenkiertoelimistön kuntoa. Voimaharjoittelulla kehitetään hermolihasarjestelmän ominaisuuksia kuten voimaa ja nopeutta parantuneen lihasaktiivisuuden ja lihaskasvun ansioista. Sekä kestävyys- että voimaharjoittelun on havaittu edistävän koko kehon suorituskykyä ja terveyttä. Sopivalla yhdistetyn harjoittelun määrällä on havaittu, että voima- ja kestävyysharjoittelun yhdistäminen esim. kestävyysjuoksijoilla voi tuottaa kehitystä sekä voima- että kestävyysominaisuuksissa.

Voima- ja kestävyysharjoittelun aiheuttamat erilaiset vasteet ja adaptaatiot saattavat kuitenkin häiritä toistensa kehittymistä. Eri harjoittelumuodot (voima ja kestävyys) voivat aiheuttaa toisensa kumoavia harjoitteluvasteita ja/tai adaptaatioita varsinkin silloin, kun harjoittelun määrä on suuri tai intensiteetti korkea. Nämä vastakkaiset voima- ja kestävyysharjoittelun vasteet herättävät kysymyksen siitä, kumpi harjoittelumuoto kannattaa tehdä ensimmäisenä yhdistetyssä harjoittelusessiossa. Onko harjoituksen järjestyksellä (voimaharjoitus, jonka jälkeen kestävyysharjoitus (VK) tai kestävyysharjoitus, jonka jälkeen voimaharjoitus (KV)) vaikutusta akuutteihin vasteisiin? Voiko toinen harjoittelumuodoista olla tehokkaampi kuin toinen?

Tämän väitöskirjan tarkoituksena oli tutkia voima- ja kestävyysharjoittelun yhdistämisen vaikutuksia 20-45-vuotiaiden terveiden miesten ja naisten hermolihasarjestelmän, sydän- ja verenkiertoelimistön sekä hormonaalisiin vasteisiin ja adaptaatioihin. Tutkittavat harrastivat kestävyysjuoksua säännöllisesti ennen tutkimusta, mutta eivät olleet aiemmin osallistuneet säännölliseen voimaharjoitteluun.

Ensimmäisessä vaiheessa tutkimuksen tarkoituksena oli selvittää, miten pitkäkestoinen jaksotettu yhdistetty voima- ja kestävyysharjoittelu vaikuttaa hermolihasarjestelmän, sydän- ja verenkiertoelimistön ja hormonaalisiin adaptaatioihin sekä miehillä että naisilla. Alkumittausten jälkeen tutkittavat satunnaisesti neljään harjoitteluryhmään. Miesten harjoitteluryhmät olivat: maksimaalinen voima (n = 11), räjähtävä voima (n = 10) ja yhdistetty maksimaalinen ja räjähtävä voima (n = 9). Naisten harjoitteluryhmät olivat: yhdistetty maksimaalinen ja räjähtävä voima (n = 9). Lisäksi oli kaksi kontrolliryhmää (lihaskestävyysharjoittelua kuntopiiriperiaatteella): miehet (n = 7) ja naiset (n = 9). Yhdistetty voima- ja kestävyysharjoittelu toteutettiin kolmessa jaksossa. Ensimmäisessä jaksossa harjoitettiin voimaa ja kestävyysuorituskykyä.

mäisessä vaiheessa kaikki tutkittavat toteuttivat voimaharjoittelua ohjatusti 1.5 kertaa viikossa 6-8 viikon ajan. Tämän harjoittelujakson tavoitteena oli voimaharjoitteiden suoritustekniikan oppiminen, harjoiteltavien lihasten totuttelu voimaharjoitteluun, ja voimatasojen kehittäminen. Tämän vaiheen jälkeen seurasi toinen 8 viikon pituinen voimaharjoittelujakso, jonka aikana tutkittavat harjoittelivat voimaa ohjatusti omissa ryhmissään kaksi kertaa viikossa. Miesten maksimi- ja räjähtävävoima -ryhmät sekä kontrolliryhmä toteuttivat varsinaisen voimaharjoittelujakson jälkeen vielä 14 viikon jakson, jolloin kestävyysjuoksun määrä nousi merkitsevästi ja voimaharjoittelun määrä pieneni (kerran viikossa). Voimaharjoitteet keskittyivät koko tutkimuksen ajan alaraajoihin. Tutkimuksen aikana kaikki koehenkilöt harjoittelivat kestävyyttä juosten 3-5 kertaa viikossa. Kestävyysharjoittelun määrä ja intensiteetti kasvoivat nousujohteisesti tutkimuksen aikana. Suurin osa kestävyysharjoittelusta toteutettiin intensiteetillä, joka oli aerobisen kynnyksen alapuolella.

Alaraajojen voimatasoja seurattiin maksimaalisella dynaamisella ojentajalihas- lihasten horisontaalisella jalkaprässillä. Lisäksi räjähtävän voiman kehitystä seurattiin kevennyshypyn avulla. Maksimaalista isometristä voimaa ja voimantuottonopeutta mitattiin jalkadynamometrillä ja lihasaktiivisuutta EMG:llä. Kestävyysuorituskyvyn muutoksia tutkittiin juoksumatolla suoralla maksimaalisella hapenottokykytestillä, jonka aikana mitattiin veren laktaattipitoisuudet. Juoksun taloudellisuutta kuvaavat muuttujat ja kynnykset määritettiin testien jälkeen. Kehon koostumuksen muutoksia mitattiin bioimpedanssilla ja lihasten poikkipinta-alan muutoksia tutkittiin ultraäänimittauksella. Veren seerumista määritettiin testosteroni-, kortisoli- ja kilpirauhashormonipitoisuudet koko tutkimusjakson aikana.

Tutkimuksen toisessa vaiheessa tarkoituksena oli selvittää, miten yhdistetyn voima- ja kestävyysharjoittelusession järjestys vaikuttaa hermolihasjärjestelmän, sydän- ja verenkiertoelimistön sekä hormonaalisiin vasteisiin ja palautumiseen. Alkumittausten jälkeen koehenkilöt suorittivat satunnaisesti kaksi eri kuormitusta. Kuormituksissa tehtiin joko voimaharjoitus ennen kestävyysharjoitusta (VK) tai kestävyysharjoitus ennen voimaharjoitusta (KV). Maksimimaalisen isometrisen voiman ja voimantuottonopeuden, kevennyshypyn, lihasaktivaation, juoksun taloudellisuuden, hengitysosamäärän ja sykkeen muutosta sekä testosteroni-, kortisoli- ja kasvuhormonipitoisuuksien muutoksia seurattiin yhdistetyn kuormituksen aikana. Lisäksi palautumista seurattiin 24 ja 48 tuntia kuormituksen jälkeen.

Ensimmäisen tutkimuksen päätulokset osoittivat, että nousujohteisella, jaksotetulla ja ohjatusti toteutetulla yhdistetyllä voima- ja kestävyysharjoittelulla saatiin hyötyä kestävyysjuoksua harrastaville. Maksimaalinen, räjähtävä ja yhdistetty maksimi- ja räjähtävä voimaharjoittelu olivat lihaskestävyysharjoittelua tehokkaampia hermolihasjärjestelmän ominaisuuksien kehittämisen kannalta. Kestävyysominaisuuksia kuten taloudellisuutta ja nopeutta voitiin kehittää myös ilman hapenottokyvyn kehittymistä hermolihasjärjestelmän adaptaatioiden kautta. Voimaominaisuudet kehittyivät 6-8 viikon valmistavalla voimaharjoittelulla ja 8 viikon varsinaisella voimaharjoittelulla, vaikka juoksumäärää



nostettiin nousujohteisesti. 14 viikon maratonharjoittelun aikana, kun juoksumäärää lisättiin ja voimaharjoittelun määrä oli pienempi, voimatasot ja lihasaktiivisuus heikkenivät. Sen sijaan juoksun taloudellisuus ja nopeus paranivat eniten niissä ryhmissä, missä toteutettiin maksimaalista tai räjähtävää voimaharjoittelua.

Vaikka tässä tutkimuksessa käytetyllä yhdistetyllä voima- ja kestävyysharjoittelulla saatiin aikaan merkitsevää voimantuotto-ominaisuuksien kehittymistä tutkittavilla koehenkilöllä, niin maksimivoimatasojen kehitys oli vähäisempi kuin mitä yleensä havaitaan silloin, kun voimaharjoittelu toteutetaan ilman samanaikaista kestävyysharjoittelua. Yhdistetyllä maksimaalisella ja räjähtävällä voimaharjoittelulla saavutetaan mahdollisesti vielä suurempi etu kestävyysjuoksijoilla kuin pelkästään maksimaalisella tai räjähtävällä voimaharjoittelulla, sillä se kehittää voimaominaisuuksia monipuolisemmalla tavalla. Seerumin hormonipitoisuudet pysyivät suhteellisen tasaisena koko tutkimuksen aikana. Testosteronitasot miehillä ja kilpirauhashormonitasot naisilla kohoivat merkitsevästi harjoittelujakson aikana. Näiden hormonipitoisuuksien palautuminen ennalleen ennen 8 viikon voimaharjoittelujakson päättymistä viittaa siihen, että keho oli sopeutunut uuteen voimaharjoitteluvasteeseen. Testosteroni-kortisolisuhte laski merkitsevästi maksimivoimaryhmässä 14 viikon maratonharjoittelujakson aikana. Tämä johtuu luultavasti juoksumäärän nousujohteisesta kasvusta jakson aikana, mikä on mahdollisesti aiheuttanut hetkellistä ylikuormitusta. Muutos ei ole kuitenkaan ollut niin suuri, että ylirasitusta voitaisiin olettaa ilmenneen.

Tutkimuksen toisessa osassa todettiin, että yhdistetyn voima- ja kestävyysharjoituksen järjestys vaikutti akuutteihin vasteisiin. Lisäksi akuutit vasteet olivat miesten ja naisten välillä erilaisia. Molemmat yhdistetyt VK ja KV harjoitukset aiheuttivat kuormituksen aikana yhteensä saman verran sekä maksimivoiman että räjähtävän voiman akuuttia heikkenemistä miehillä. Naisilla maksimivoiman heikkeneminen oli pienempää kuin miehillä ja räjähtävässä voimassa naisilla ei havaittu muutosta lainkaan. Palautumisen aikana 24 ja 48 tuntia kuormitusten jälkeen miesten maksimi- ja räjähtävävoima oli edelleen lähtötasoa heikompi sekä VK:ssä että KV:ssä. Naisilla maksimivoima oli palautunut 24 ja 48 tuntia kuormitusten jälkeen, kun taas räjähtävä voima oli tilastollisesti merkitsevästi heikompi naisten KV ryhmässä. Räjähtävän voimantuoton lasku, mikä ilmeni naisilla vasta viiveellä palautumisen aikana, saattaa johtua joko viivästyneestä hermolihasjärjestelmän väsymyksestä ja/tai jopa lihasvaurioista. Lihasvaurion ilmenemistä tukee se, että seerumin kreatiini-aktiivisuus oli samanaikaisesti lähtötasoa korkeammalla tasolla ja viitearvojen yläpuolella.

Yhdistetyssä voima- ja kestävyysharjoituksessa akuutit hormonivasteet olivat suurempia miehillä kuin naisilla. Miesten VK -ryhmässä havaittiin tilastollisesti merkitsevä nousu kortisolipitoisuuksissa yhdistetyn kuormituksen jälkeen. Tämä oli tilastollisesti suurempi kuin KV -ryhmässä. Palautumisen aikana (24 ja 48 tuntia) havaittiin alentunut seerumin testosteronipitoisuus miesten VK -ryhmässä. Miehillä seerumi kortisolipitoisuuden nousu ja testosteronin lasku saattavat merkitä kovempaa stressiä VK -harjoituksessa kuin KV- harjoituksessa.

tuksessa. Naisilla hormonivasteet olivat pienempiä. Naisilla ryhmien välinen ero havaittiin ainoastaan KV ja VK -harjoituksen päätyttyä testosteronitasoissa mutta testosteronipitoisuuksien muutokset eivät olleet tilastollisesti merkitseviä. Muuten naisten hormonivasteet olivat kuormituksesta riippumatta samanlaisia ja hormonitasot palautuivat 48 tuntia kuormitusten jälkeen lähtötasoille.

Juoksun taloudellisuus kärsii merkittävästi, kun voimaharjoitus edeltää kestävyysharjoittelua sekä naisilla että miehillä, vaikka hengitysosamäärä ja syke pysyvät tilastollisesti samana. Tutkimustulosten perusteella kestävyysharjoitus ennen voimaharjoitusta voi olla kestävyysjuoksijan kehityksen kannalta hyödyllisempää. Mikäli voimaharjoitusta toteutetaan ennen kestävyysharjoitusta, miesten tulee kiinnittää erityisesti huomiota riittävään palautumiseen. Naisilla puolestaan KV järjestys saattaa aiheuttaa viivästynyttä räjähtävän voimantuoton heikkenemistä yhdistetyn harjoituksen jälkeen.

Molemmista tutkimuksista saatuja tuloksia voidaan hyödyntää optimoimassa harjoitteluohjelmien suunnittelua kestävyysjuoksua harrastavilla ihmisillä. Maksimimaalinen, räjähtävä tai yhdistetty maksimi ja räjähtävä voimaharjoittelu parantavat hermolihasjärjestelmän toimintaa, mikä heijastuu kestävyysominaisuuksiin kuten juoksun taloudellisuuteen ja nopeuden kehittymiseen. Yhdistetyn voima- ja kestävyysharjoituksen järjestyksellä saattaa olla merkitystä palautumiseen. Miehillä kestävyysharjoitus ennen voimaharjoittelua ja naisilla vastakkainen järjestys voi olla palautumisen kannalta optimaalinen. Tutkimustulokset osoittavat sitä, että kestävyyttä harjoittelevan juoksijan kannattaa harjoitella joko maksimaalista, räjähtävää tai yhdistettyä maksimi- ja räjähtävä voimaa, kun he valmistautuvat juoksumäärän lisäämiseen. Tämän lisäksi yhdistetyn voima- ja kestävyysharjoittelun järjestystä kannattaa miettiä mm. kulloisenkin harjoitusjakson ja harjoittelun tavoitteen kannalta.

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## ORIGINAL PAPERS

### I

#### STRENGTH TRAINING IN ENDURANCE RUNNERS

by

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## **Strength Training in Endurance Runners**

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**ABSTRACT**

This study examined effects of periodized maximal versus explosive strength training and reduced strength training, combined with endurance training, on neuromuscular and endurance performance in recreational endurance runners. Subjects first completed 6 weeks of preparatory strength training. Then, groups of maximal strength (MAX, n=11), explosive strength (EXP, n=10) and circuit training (C, n=7) completed an 8-week strength training intervention, followed by 14 weeks of reduced strength training. Maximal strength (1RM) and muscle activation (EMG) of leg extensors, countermovement jump (CMJ), maximal oxygen uptake ( $VO_{2MAX}$ ), velocity at  $VO_{2MAX}$  ( $vVO_{2MAX}$ ) running economy (RE) and basal serum hormones were measured. 1RM and CMJ improved ( $p<0.05$ ) in all groups accompanied by increased EMG in MAX and EXP ( $p<0.05$ ) during strength training. Minor changes occurred in  $VO_{2MAX}$ , but  $vVO_{2MAX}$  improved in all groups ( $p<0.05$ ) and RE in EXP ( $p<0.05$ ). During reduced strength training 1RM and EMG decreased in MAX ( $p<0.05$ ) while  $vVO_{2MAX}$  in MAX and EXP ( $p<0.05$ ) and RE in MAX ( $p<0.01$ ) improved. Serum testosterone and cortisol concentrations remained unaltered. Maximal or explosive strength training performed concurrently with endurance training was more effective in improving strength and neuromuscular performance and in enhancing  $vVO_{2MAX}$  and RE in recreational endurance runners than concurrent circuit and endurance training.

**Key words:** concurrent training, neuromuscular performance, endurance performance, running economy, strength



## INTRODUCTION

Strength and endurance training are commonly known to produce divergent adaptations. The primary adaptation to endurance training is improved oxygen transportation and utilization by way of increased capillary and mitochondrial density, as well as increased enzyme activity, which improves oxidative energy metabolism [3,12]. The primary adaptation to strength training with high loads includes increases in maximal strength, resulting from improvements in voluntary neuromuscular activation, which is usually followed by muscle hypertrophy during prolonged training periods [17]. Unlike strength training, endurance running requires only repetitive low force production, and even intensive up-hill running does not induce maximal activation of the leg muscles [37]. Nevertheless, even in endurance sports, strength training for increases in strength and power has been reported to be beneficial in leading to increases in rapid force production e.g. contributing to increases in running speed. Thus, recreational and elite endurance athletes may perform both types of training concurrently to optimize endurance performance.

Many different models exist for periodization of resistance training, particularly when it is performed concurrently with endurance exercise. Endurance runners often include either very little or no resistance training in their exercise programs, especially when increases in running volume occur, which makes the periodization of concurrent strength and endurance training in endurance runners very different from that of strength and power athletes. Periodized training typically calls for periods of higher intensity/volume of training alternated with periods of lower intensity/volume of training. The periods of lower intensity/volume of training may not provide an adequate training stimulus for development, or even maintenance of strength, a phenomenon referred to as “detraining”. Detraining from strength training is further characterized by decreases in strength, muscle mass, and muscle activation [18] that mirrors the time-course of the preceding training adaptations [30].

It has been suggested that endurance training may interfere with strength development when strength and endurance training are performed concurrently [4,13,15,30]. When present, this interference effect is predominantly attributed to a high volume of training, high intensity training or prolonged training duration [4,13,21], and may also be related to hormonal adaptations, mechanical stress, and muscle damage [4,23]. It might be hypothesized that changes in concentrations of the hormones of testosterone and cortisol also play a role in this interference effect. Strength exercise typically stimulates an acute increase in testosterone [24] associated with anabolic processes in the body such as muscle growth. In contrast, a chronic increase in circulating basal levels of cortisol, indicating an increase in catabolic activity, has been reported with prolonged endurance training [40].

Several studies have shown increases in endurance performance resulting from the addition of various types of strength training to endurance training regimens in endurance runners (orienteers) [33] cross-country skiers [14,26], triathletes [28] and previously untrained men [15,21]. These studies have examined maximal or explosive strength training combined with endurance training, but no studies have been conducted to compare these two different strength training modes when they are combined with endurance training. Improvements in endurance performance in the previously mentioned studies have been attributed to enhanced neuromuscular activation and improved sport-specific economy rather than increases in maximal oxygen uptake ( $VO_{2max}$ ). Moreover, individual performance differences may be further explained by running speed at  $VO_{2max}$  ( $vVO_{2max}$ ), which is often used in the analysis of distance running performance, and for monitoring training [5].

The primary purpose of the present study was to examine the effects of periodized maximal versus explosive strength training, combined with endurance training, on neuromuscular adaptations and changes in endurance performance in male recreational endurance runners. In addition, this study

examined the effect of reduced strength training volume, accompanied by increased endurance training volume, on strength maintenance and endurance performance.

## **METHODS**

### **Subjects**

A total of twenty-eight male recreational endurance runners (age 21-45 years) were recruited from the Jyväskylä, Finland region as part of a marathon training school, and completed the study. Subjects were fully informed about the study design, including information on the possible risks and benefits of participation, prior to signing an informed consent document. Ethical approval was granted by the University of Jyväskylä Ethical Committee, and the study was conducted according to the most recent Declaration of Helsinki. Most of the subjects had previously completed either a running marathon or half-marathon. Subjects were divided into three groups matched for age, anthropometrics, training experience, strength and  $\text{VO}_{2\text{max}}$  following baseline testing at -6 weeks. Groups included a maximal strength training group (MAX,  $n = 11$ , age:  $35.5 \pm 5.8$  years, height:  $178.6 \pm 4.6$  cm (mean  $\pm$  SD)), an explosive strength training group (EXP,  $n = 10$ ,  $36.4 \pm 6.1$  years,  $180.5 \pm 6.1$  cm) and a circuit training group that acted as a control and used only their own body weight as a load following the preparatory period (C,  $n = 7$ ,  $33.7 \pm 8.2$  years,  $180.0 \pm 4.5$  cm). Subjects were not using medications nor did they have any injuries that would affect physical performance. Following the period of reduced strength training and increased endurance training, 2 subjects from the circuit training group were unable to complete all testing due to minor injuries; thus, statistics following this period are calculated separately.

### **Study design and training**

Strength training throughout the 28-week study was focused on the leg extensors, a major muscle groups at work in human locomotion and in running, and was preceded by 20-30 minutes of low-intensity endurance exercise (below aerobic threshold) [2]. The preparatory strength training period

consisted of approximately 9 training sessions completed over 6 weeks in which all subjects performed strength training exercises using loads that progressed from 50 to 70% 1RM, and that were similar to those used in the training intervention (Table 1). The preparatory period was followed by an 8-week strength training intervention in which groups began their specified maximal, explosive or circuit training programs, and in which strength training was to be completed approximately twice per week (Table 2). No statistically significant differences in training frequency were observed between training groups. In MAX and EXP, two to three minutes of rest separated exercise sets throughout the study. C completed exercises in series including 10 - 15 seconds of rest in between each exercise.

Throughout the study, subjects concurrently performed endurance training, typically on non-strength training days. During the preparatory period and strength training intervention, endurance training in all groups was primarily performed below the aerobic threshold, which was individually determined for each subject each time they were tested for maximal oxygen uptake. Endurance training volume during the preparatory period was at its lowest in the study in terms of both running kilometers (km) (Table 3) and endurance training time (average hours:min  $\pm$  SD, 3:02  $\pm$  0:51, 3:45  $\pm$  2:19, 2:34  $\pm$  0:46 for MAX, EXP and C, respectively). Training volume, in terms of time, increased ( $p < 0.01$  in MAX,  $p = 0.056$  (n.s) in EXP and  $p = 0.128$  (n.s.) in C) from the preparatory period into the actual 8-week strength training intervention (up to 4:49  $\pm$  1:27, 4:43  $\pm$  1:57, 4:03  $\pm$  2:01 hours:min). In terms of running km, training volume increased from the beginning of the study to the end of the strength training intervention in all groups, although significantly only in MAX and EXP (Table 3). There were no group differences in training volume (time or km) in these two periods. Endurance training consisted primarily of running, but also occasionally included typical outdoor activities in Finland such as cross-country skiing, cycling and Nordic walking. This “other training” ranged an average of 6 to 15 km/week throughout the entire experimental period.

\*FIGURE 1 and TABLE 1 ABOUT HERE\*

Following the main training periods, including the preparatory training period and strength training intervention, a 14-week reduced strength and increased endurance training period was completed as part of marathon preparation. Subjects completed strength training  $\leq 1$  time per week for 14 weeks (Table 2) for strength maintenance purposes. At this time, running volume in km increased significantly in all groups (Table 3). Endurance training volume in time during this period was 5:20  $\pm$  1:36, 4:52  $\pm$  1:41, 4:50  $\pm$  1:29 (hours:min) in MAX, EXP and C, respectively. From the beginning of the study to the end of the study, this increase in running volume was significant ( $p < 0.05$ ) in all three training groups. No statistical differences in training volume either in terms of km or time of endurance training between groups were observed during this 14-week period. Intensity of endurance training was at its highest during this period including training sessions that were performed above aerobic or anaerobic thresholds. In addition, longer lower intensity training sessions were included as part of marathon preparation. Subjects kept a training diary throughout the study recording strength training sessions, weekly kilometers of running and “other” endurance activity (cycling, cross-country skiing and Nordic walking). Training plans were personalized based on training ability and background and were furthermore adjusted after each aerobic testing session (-6 weeks, weeks 0 and 8). Measurements took place prior to the preparatory period (-6 weeks), before, during and after the strength training intervention (weeks 0, 4 and 8) and after the reduced strength training period (+14 weeks).

\*TABLES 2, 3 ABOUT HERE\*

## **MEASUREMENTS**

### **Body composition**

In addition to standing height, body mass and body composition were measured using bioimpedance (InBody720 body composition analyzer, Biospace Co. Ltd, Seoul, South Korea). Measurements were always taken in conjunction with blood tests between 07.30-08.00. Thus, subjects always arrived for testing in a fasted state helping to keep the possible confounding variables of diet and hydration status to a minimum. Subjects were instructed to remove excess clothing, watches, jewelry, shoes and socks prior to the measurement.

### **Muscle thickness**

Muscle thickness of vastus lateralis (VL) and vastus intermedius (VI) were measured using a compound ultrasound scanner (Aloka SSD-2000, Aloka Co., Tokyo, Japan) [16]. The subject's legs were secured with a belt at the ankles and the knees were supported with a foam pad to avoid movement during the measurements. Thickness was measured at a point marked with an ink tattoo (similar to EMG placement) that was placed on the anterior surface of the leg at 50% length of the femur measured from the lateral aspect of the distal diaphysis to the greater trochanter [38]. All measurements were performed by the same individual. Water soluble transmission gel was used to avoid unnecessary tissue compression by the probe, and the probe was adjusted manually until a clear image was achieved. Muscle thickness was calculated from the average of three consecutive muscle thickness measurements (VL + VI).

## **PERFORMANCE MEASURES**

### **Aerobic capacity:**

Endurance capacity was measured by maximal oxygen uptake ( $VO_{2max}$ ) were determined using a treadmill running protocol [27]. The running velocity began at  $8 \text{ km}\cdot\text{h}^{-1}$  and was increased by  $1 \text{ km}\cdot\text{h}^{-1}$  every third minute until volitional exhaustion. Treadmill incline remained a constant 0.5

degrees throughout the test. Heart rate was recorded continuously using a heart rate monitor (Suunto t6, Vantaa, Finland). Mean heart rate values from the last minute of each stage were used for analysis. Oxygen consumption was measured breath-by-breath throughout the test using a portable gas analyzer (Oxycon Mobile®, Jaeger, Hoechberg, Germany) and  $\dot{V}O_{2max}$  was accepted as the highest average 60 s  $\dot{V}O_2$  value. Fingertip blood samples were taken every 3rd minute to measure blood lactate concentrations. For blood sampling, the treadmill was stopped for approximately 15-20 seconds. Blood lactates were analyzed using a Biosen S\_line Lab+ lactate analyzer (EKF Diagnostic, Magdeburg, Germany). Running economy (RE) was evaluated by examining  $\dot{V}O_2$  at 10 and 12  $\text{km}\cdot\text{h}^{-1}$ , speeds comparable to the marathon running speeds of our subjects. The velocity of running at  $\dot{V}O_{2max}$  ( $v\dot{V}O_{2max}$ ) was calculated as follows  $v\dot{V}O_{2max} = \text{speed of the last whole completed stage (km}\cdot\text{h}^{-1}) + (\text{running time (s) of the speed at exhaustion} - 30 \text{ seconds}) / (180 - 30 \text{ seconds}) * 1 \text{ km}\cdot\text{h}^{-1}$ . Aerobic (AerT) and anaerobic (AnT) thresholds were determined using blood lactate, ventilation,  $\dot{V}O_2$  and  $\dot{V}CO_2$  (production of carbon dioxide) according to Aunola and Rusko (1986) [2].

#### **Strength and power measurements:**

##### **One repetition maximum**

One repetition maximum (maximal dynamic bilateral horizontal leg press in a seated position) was measured using a David 210 dynamometer (David Sports Ltd., Helsinki, Finland) [20]. Prior to attempting 1RM, subjects completed a warm-up consisting of 5 x 70% 1RM, 1 x 80-85% 1RM and 1 x 90-95% of estimated 1RM, with one minute of rest between sets. Following this warm-up, no more than 5 attempts to reach 1RM were made. Leg extension action started from a knee angle of  $65.4 \pm 1.5$  degrees. Subjects were instructed to grasp handles located by the seat of the dynamometer and to keep constant contact with the seat and backrest during leg extension to a full extension of 180 degrees. Verbal encouragement was given to promote maximal effort. The greatest weight that the subject could successfully lift (knees fully extended) to the accuracy of 2.5 kilograms was accepted as 1RM.

### **Countermovement jump**

A force platform (Department of Biology of Physical Activity, Jyväskylä, Finland) was used to measure maximal dynamic explosive force by countermovement jump height [7]. Subjects were instructed to stand with their feet approximately hip-width apart with their hands on their hips. Subjects were then instructed to perform a quick and explosive countermovement jump on verbal command so that knee angle for the jump was no less than 90 degrees. Force data was collected and analyzed by computer software (Signal 2.14, CED, Cambridge, UK), which used the equation  $h = I^2 / 2gm^2$  to calculate jump height from impulse ( $I$  = impulse,  $g$  = gravity and  $m$  = mass of subject).

### **Electromyographic activity**

Electromyographic activity (EMG) was recorded from the vastus lateralis (VL) and vastus medialis (VM) of the right leg during 1RM. Electrode positions were marked with small ink tattoos [18] on the skin during the first testing session to ensure that electrode placement over the entire experimental period would be consistent. The guidelines published by SENIAM [36] were followed for skin preparation, electrode placement and orientation. Inter-electrode distance was 20 mm (input impedance < 10 k $\Omega$ , common mode rejection ratio 80 dB, 1000 gain). Raw signals were passed from a transmitter, positioned around the subjects' waist, to a receiver (Telemetry 2400R, Noraxon, Scottsdale, AZ, USA) from which the signal was relayed to the computer via an AD converter (Micro1401, CED, Cambridge, UK). Whole range EMG was recorded from the starting knee angle between  $65.4 \pm 1.5$  degrees to full leg extension of 180 degrees, and subsequently analyzed by computer software (Signal 2.14, CED, UK).

### **Serum hormones**

Venous blood samples (10 ml) were collected using sterile needles into serum tubes (Venosafe, Terumo Medical Co., Leuven, Belgium) by a qualified lab technician. Subjects were tested after 12



hours of fasting between 07.30-08.00. Whole blood was centrifuged at 3500 rpm (Megafuge 1.0R, Heraeus, Germany) for 10 min after which serum was removed and stored at -80°C until analysis. Samples were used for determination of serum testosterone and serum cortisol. The ratio of testosterone and cortisol (testosterone/cortisol) was also calculated. Analyses were performed using chemical luminescence techniques (Immunlite 1000) and hormone specific immunoassay kits (Siemens, New York, NY, USA). The sensitivity of testosterone and cortisol assays were 0.05 nmol·l<sup>-1</sup> and 5.5 nmol·l<sup>-1</sup> respectively. The intra-assay coefficients of variation for testosterone and cortisol were 3.9% and 4.6%, respectively. The inter-assay coefficients for testosterone and cortisol were 2.2% and 7.6%, respectively.

#### **Statistical methods**

Standard statistical methods were used for calculation of means, standard deviation (SD) and standard error (SE). Group differences were analyzed using a one-way analysis of variance (One-way ANOVA) and within group differences (group-by-training interaction) were analyzed using repeated measures ANOVA. Significance was set at \* =  $p \leq 0.05$ , \*\* =  $p < 0.01$  and \*\*\* =  $p < 0.001$ . Statistical analysis was completed with SPSSWIN 15.0 (SPSS Inc., Chicago, IL, USA).

#### **RESULTS**

Significant gains in 1RM were observed after the preparatory period in MAX ( $p < 0.05$ ), but not in EXP or C (Fig. 2). During the strength training intervention between weeks 0 and 4, significant gains were observed in MAX and EXP ( $p < 0.01$  and  $p < 0.05$ , respectively) after which strength gains plateaued. Following reduced strength training, progressive decreases in strength were observed, but this decrease was significant only in MAX ( $p < 0.01$ ).

\*FIGURE 2 ABOUT HERE\*

Increases in muscle activation of VL were significant from weeks -6 to 8 in MAX and EXP ( $p < 0.05$  and  $p < 0.01$ , respectively) (Fig. 3). Activation of VM also increased significantly over the preparatory period and the strength training intervention in MAX and in EXP (results not shown,  $p < 0.05$  and  $p < 0.01$ , respectively). A significant decrease in muscle activation of VL was observed in MAX after the period of reduced strength training and increased endurance training volume ( $p < 0.05$ ) (Fig. 3). No significant changes in muscle activation were observed in either VL or VM in C.

\*FIGURE 3 ABOUT HERE\*

Jump height improved significantly over the preparatory period and the strength training intervention in MAX, EXP and C ( $p < 0.001$ ,  $p < 0.001$  and  $p < 0.05$ , respectively) (Fig. 4). During the strength training intervention; however, significant gains were only observed in MAX ( $p < 0.05$ ). A plateau in jump height was observed in MAX and C after week 4 and in EXP after week 0.

\*FIGURE 4 HERE\*

Body mass at the beginning of the experimental period was  $77.2 \pm 5.5$  kg in MAX,  $78.4 \pm 6.3$  kg in EXP, and  $83.8 \pm 10.5$  kg in C. A small but significant increase in body mass was observed in MAX (1.4%,  $p < 0.01$ ) after 10 weeks of training (at week 4) which was accompanied by a significant increase in muscle thickness of VL + VI (6.7%,  $p < 0.001$ ). Body mass then decreased significantly during the final 14 weeks of training (-1.6%,  $p < 0.05$ ) though no significant changes in body fat % or muscle thickness were observed. In EXP, body mass decreased significantly over both strength training periods (-2.0%,  $p < 0.05$ ) which was accompanied by a significant decrease in body fat % that occurred during the preparatory period (-7.0%,  $p < 0.01$ ). Following reduced strength training, only a significant decrease in muscle thickness was observed (-5.3%,  $p < 0.05$ ). Body mass and

muscle thickness of C remained statistically unaltered throughout the study while % body fat decreased significantly over both strength training periods (-6.5%,  $p < 0.05$ ).

Maximal oxygen uptake ( $\text{VO}_{2\text{max}}$ ) improved progressively in all groups; however, significant improvement was observed only between weeks -6 and 8 in MAX (Fig. 5).

\*FIGURE 5 HERE\*

Maximal running speed at exhaustion ( $v\text{VO}_{2\text{max}}$ ) improved progressively throughout the study from week -6 to the end of the strength training intervention (week 8) in MAX, EXP and C ( $p < 0.01$ ,  $p < 0.001$  and  $p < 0.05$ , respectively) (Fig. 6). Significant increases were also observed after reduced strength training in MAX,  $p < 0.001$  and in EXP and  $p < 0.05$ , whereas in C,  $v\text{VO}_{2\text{max}}$  was statistically unaltered.

\*FIGURE 6 ABOUT HERE\*

Significant improvements in running economy (RE) occurred at  $10 \text{ km}\cdot\text{h}^{-1}$  over the entire study from week -6 to +14 ( $p < 0.01$ ) in MAX while in EXP, these improvements occurred only from week -6 to 8 ( $p < 0.05$ ) (Fig. 7). Significant improvements in RE also occurred at  $12 \text{ km}\cdot\text{h}^{-1}$  in MAX over the entire study from week -6 to +14 ( $p < 0.01$ ), but not in EXP (results not shown). No significant improvements in RE were observed in C at either speed.

\*FIGURE 7 ABOUT HERE\*

Basal levels of testosterone and cortisol (Table 4) did not change throughout the study. The ratio of testosterone/cortisol decreased significantly during reduced strength training in MAX ( $p < 0.05$ ) (Fig. 8).

\*TABLE 4 ABOUT HERE\*

\*FIGURE 8 ABOUT HERE\*

## DISCUSSION

The primary findings of the present study demonstrated that maximal (MAX) and explosive (EXP) strength training groups improved strength, power, and maximal muscle activation systematically during both the preparatory period and the strength training intervention. Concomitantly, running velocity at  $VO_{2max}$ , ( $vVO_{2max}$ ) and running economy (RE) systematically and significantly improved in both MAX and EXP with only minor changes in  $VO_{2max}$ . The neuromuscular and strength changes associated with improvements in  $vVO_{2max}$  and RE seemed to be more important in augmenting endurance performance than increases in  $VO_{2max}$ . The circuit training control group (C) made smaller, but statistically significant improvements in strength and power over the preparatory strength training period and the strength training intervention. The changes in  $VO_{2max}$  and RE in C were not significant; however, the change in  $vVO_{2max}$  was significant. Despite the apparent detraining of strength that occurred with reduced strength training in MAX and EXP, the overall gains from strength training remained somewhat above pre-training values over the entire experimental period. This period, which also included an increase in endurance training volume and intensity, was associated with further improvements in  $vVO_{2max}$  and RE.

The total improvement in 1RM was 8% in MAX over both strength training periods and 3% in EXP, but the difference between the groups was not significant. The increases in 1RM during the strength training intervention alone (week 0 to 8) were similar in both groups (~4%) despite different strength training programs. A difference in the magnitude of improvement over these combined training periods was expected considering the progressively higher loads that MAX used for training (80-85% 1RM) versus the lighter loads (30-40% 1RM) used by EXP during the

intervention; however, movement patterns may not have differed enough to distinctly show specificity of training [6]. The increases in strength that occurred in MAX and EXP, though significant, were not as great in magnitude as in studies examining only strength training in previously untrained individuals [1,17]. The smaller magnitude of increase observed in our study may be related to differences in exercise protocols in addition to concurrently performed endurance training. The plateau in strength gains that occurred in all groups following 10 weeks of training suggests that the strength training stimulus was either not adequate enough to induce additional changes, or it may indicate some interference of endurance training on strength development, an observation in line with e.g. Hickson (1980) and Hunter et al. (1987). The significant 6% increase in 1RM load that C experienced following 10 weeks of training indicates that even low load/intensity strength training (using only body weight for 4 of those weeks) was sufficient to stimulate maximal strength improvements in individuals who have not previously used any type of resistance training.

Significant increases in muscle activation of VL and VM (an average of 18% and 34% in VL and VM, respectively) accompanied the improvements in strength over the entire strength training period in both MAX and EXP. Although caution should be exercised with regards to the present ultrasound method for measurement of muscle mass, our results indicated that in MAX, strength development may have also been influenced by a significant increase observed in muscle thickness of VL and VI. More drastic increases in muscle mass were not expected because training was not designed to be hypertrophic, and subjects were participating concurrently in endurance exercise.

Significant increases in CMJ jump height were observed in both strength training groups; however, changes in jumping height indicated that MAX made somewhat greater (n.s.) improvements (13%) in explosive strength over 10 weeks of strength training than EXP (11%). Although increases in jump height seem to be more systematic in MAX than in EXP, the overall similarity in the increases in jumping height are attributed, in part, to 6 weeks of common training. Furthermore, this overall

similarity indicates that movement patterns in the subsequent maximal and explosive strength training intervention programs may not have differed enough [6], or that the nature of CMJ testing might not show specificity of training. It has been suggested that specific adaptations to explosive strength training may not occur unless a subject already has an “adequate” level of strength and power [31]. As a result, in recreationally trained endurance runners with no strength training background, maximal strength training may have had more of an effect on CMJ jump height than more specific explosive strength training. Subjects were also concurrently participating in endurance training activities, which have been reported to hinder specific adaptations to explosive resistance training [15,21].

Following the period of reduced strength training volume and increased endurance training volume (and intensity), significant decreases were observed in maximal strength and muscle activation of the trained muscle groups (quadriceps femoris, VL) in MAX while maximal strength in EXP and C remained statistically unaltered. Decreases in maximal strength, muscle activation [18], and CMJ jump height are typically associated with reduced strength training volume [19]. Nevertheless, it is possible that these decreases were influenced by the progressive increase in endurance running volume (running kilometers per week) and intensity (training above aerobic and anaerobic thresholds) [13] (Table 3) from the beginning of the study to the end of the study all three groups. This significant increase in running kilometers coincided with plateaus/decreases in maximal strength, explosive strength and muscle activation. The increased endurance training (especially running) may have also resulted in greater mechanical stress that has previously been reported to interfere with muscle strength [4]. This finding is in agreement with e.g. Hickson (1980) and Hunter et al. (1987) who stated that early strength development may be hindered if aerobic training volume is high. Endurance running involves repeated low force production and impact loading which provides a different type of stimulus than that of strength training. Furthermore, leg muscles are not fully activated even during high-intensity running (on horizontal and up-hill), which indicates that

there is a limit to how many muscle fibers can be recruited to increase force production capabilities by running [6]. Thus, the 14-week period of reduced strength training and increased volume (and intensity) of endurance training appeared not to have provided a sufficient strength training stimulus to maintain increases in muscle activation and strength made during the preparatory period and strength training intervention.

The present study showed minimal increases in maximal oxygen uptake in all three training groups with a significant increase occurring only in MAX, and only over the preparatory period and strength training intervention. However, significant increases in  $vVO_{2max}$  were continuously systematic over the preparatory period and strength training intervention in both MAX and EXP. In C, the increase was significant only between the beginning of the preparatory period and the end of the strength training intervention. Interestingly, the increase in  $vVO_{2max}$  continued only in MAX and EXP over the period of reduced volume strength training and increased volume and intensity of endurance training. In addition, improvements in running economy (RE) were observed over the preparatory training period and the strength training intervention in EXP (at  $10 \text{ km}\cdot\text{h}^{-1}$ ) and in MAX over the entire 28 weeks of training (at  $10 \text{ km}\cdot\text{h}^{-1}$  and  $12 \text{ km}\cdot\text{h}^{-1}$ ). Since increases observed in  $VO_{2max}$  were minimal, improvements that occurred in strength, power, and muscular activation likely contributed to improved  $vVO_{2max}$  and RE, and prepared subjects for increased endurance training volume. These observations are consistent with previous research by Daniels et al. [9] who observed that improvements in running performance are not necessarily related to increases in  $VO_{2max}$  alone. Furthermore, research by Paavolainen et al. [34,35] and Nummela et al. [32] attributes improved endurance performance to sport-specific economy and improved neuromuscular function. While endurance performance has typically been determined by measurement of maximal oxygen uptake ( $VO_{2max}$ ), fractional utilization of  $VO_{2max}$  and sport-specific economy [3], these more recent studies have suggested that maximal anaerobic capacity and neuromuscular characteristics are additional predictors of endurance performance.

It should be noted that although the values of aerobic fitness measured at the beginning of this study were representative of recreational endurance athletes (non-elite), only minor changes in  $\text{VO}_{2\text{max}}$  were expected since the training intensity for much of the study was relatively low (below the aerobic threshold). Intermittent high-intensity training, such as interval training, is reported to be more effective than moderate intensity training in improving  $\text{VO}_{2\text{max}}$  [39]. On the other hand, changes in other endurance parameters such as  $\dot{v}\text{VO}_2$  and RE may still be positively influenced by low intensity training [3]. Improvements in body composition, such as a decrease in mass or decreased %fat, such as those observed in all training groups in the present study, may also positively influence endurance performance [10].

Serum concentrations of testosterone and cortisol remained statistically unaltered over the entire 28-week study in all three groups indicating maintained homeostatic control. Over 10 weeks of strength training; however, the testosterone/cortisol ratio tended to increase concomitantly with concurrent strength and endurance training in MAX. Yet, following the reduced strength training and increased endurance training period, the serum ratio of testosterone/cortisol decreased significantly 10% in MAX, which indicates an increase in catabolic activity. Though significant, the decrease in serum testosterone/cortisol ratio was not below baseline; but it may have still contributed to strength and power decreases [22]. Suppressed resting concentrations of testosterone and elevated levels of cortisol have been reported to occur in male endurance athletes [8,11]. Mode (strength versus endurance), intensity [40] and duration of training [40,41] influence these hormonal concentrations.

In conclusion, the findings of this study show that both maximal and explosive strength training performed concurrently with endurance training are more effective in improving strength, power and muscular activation in recreational endurance runners than concurrent circuit and endurance



training. Improvements in strength, power, and muscle activation during the preparatory and strength training intervention periods appears to have contributed to enhanced endurance performance by improving  $v\text{VO}_{2\text{max}}$  and RE, and prepared subjects for increased endurance training volume which occurred during the final 14 week training period. Despite some detraining of strength that occurred during this period of reduced strength training and increased endurance training volume and intensity, overall strength and power improvements from strength training were maintained above pre-training values. Improvements in  $v\text{VO}_{2\text{max}}$  continued further in both MAX and EXP, while improvements RE, continued further only in MAX. We conclude that recreational endurance runners can benefit from including maximal or explosive strength training in their training program in order to improve overall endurance performance.

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**FIGURE LEGENDS:**

**Figure 1.** Study design including three training periods and five sets of measurements (denoted by arrows).

**Figure 2.** Absolute 1RM load (mean  $\pm$  SE) over 28 weeks of training \* = significant difference from -6 weeks, + = significant difference from week 0, § = significant difference from week 8 (\*, + =  $p < 0.05$ , \*\*, ++, §§ =  $p < 0.01$ , \*\*\* =  $p < 0.001$ ).

**Figure 3.** EMG of VL during 1RM over 28 weeks of training (mean  $\pm$  SE). \* = significant difference from -6 weeks, + = significant difference from week 0 § = significant difference from week 8 (\*, § =  $p < 0.05$ , \*\*, ++ =  $p < 0.01$ ).

**Figure 4.** CMJ jump height over 28 weeks of training (mean  $\pm$  SE). \* = significant difference from -6 weeks, + = significant difference from week 0 (\*, + =  $p < 0.05$ , \*\*\* =  $p < 0.001$ ).

**Figure 5.** Maximal oxygen uptake ( $VO_{2max}$ ) (mean  $\pm$  SE) measured at -6 weeks, weeks 0, 8 and +14. \* = significant difference from -6 weeks (\* =  $p < 0.05$ ).

**Figure 6.** Velocity of running at  $VO_{2max}$  ( $vVO_2$ ) (mean  $\pm$  SE) measured at -6 weeks, weeks 0, 8 and +14. \* = significant difference from -6 weeks, + = significant difference from week 0 and § = significant difference from week 8 (\*, +, § =  $p < 0.05$ , \*\*, §§ =  $p < 0.01$ , \*\*\* =  $p < 0.001$ ).

**Figure 7.** Running economy at  $10 \text{ km} \cdot \text{h}^{-1}$  over 28 weeks of training (mean  $\pm$  SE). \* = significant difference from -6 weeks and § = significant difference from week 8 (\* =  $p < 0.05$ , \*\* =  $p < 0.01$ , §§§ =  $p < 0.001$ ).

**Figure 8.** Ratio of serum testosterone to cortisol over 28 weeks of training. § = significant difference from week 8 (§ =  $p < 0.05$ ).

**TABLE LEGENDS:**

**Table 1.** Periodized strength training programs over 28 weeks of training.

**Table 2.** Average strength and endurance training sessions per week over 28 weeks of training (means  $\pm$  SE). \* = significant difference from -6 weeks, + = significant difference from strength training intervention week 0 - week 4, # = significant difference from strength training intervention week 4 - week 8 (# =  $p < 0.05$ , \*\* =  $p < 0.01$ , \*\*\* =  $p < 0.001$ , +++ =  $p < 0.001$ ).

**Table 3.** Average endurance training volume in kilometers per week over 28 weeks of training (means  $\pm$  SE). \* = significant difference from -6 weeks, + = significant difference from strength training intervention week 0 - week 4, # = significant difference from strength training intervention week 4 - week 8, § = significant difference from 8-week training intervention (\*, # =  $p < 0.05$ , \*\*, ++ =  $p < 0.01$ , \*\*\*, +++ =  $p < 0.001$ ).

**Table 4.** Basal levels of serum testosterone and cortisol (mean  $\pm$  SE) over 28 weeks of training.

FIGURES:

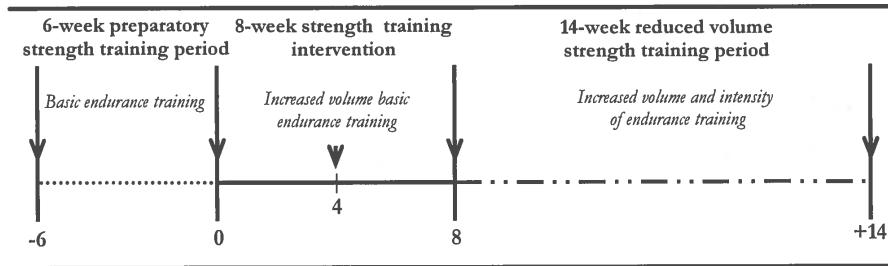


Figure 1.

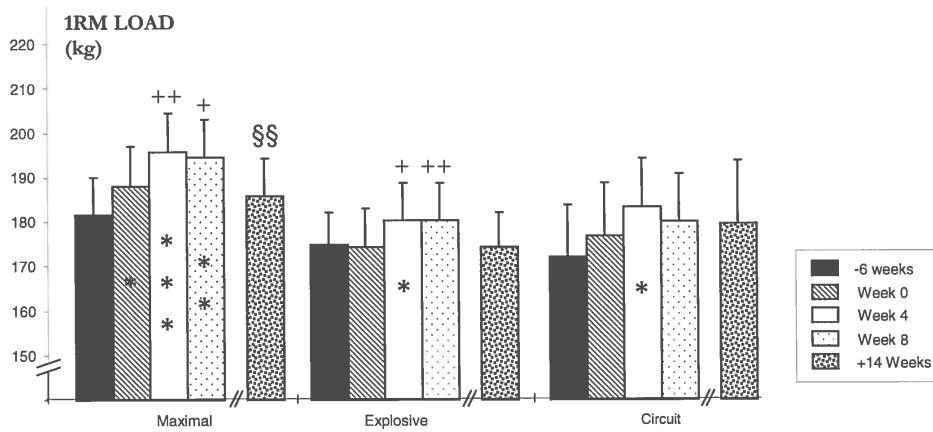


Figure 2.

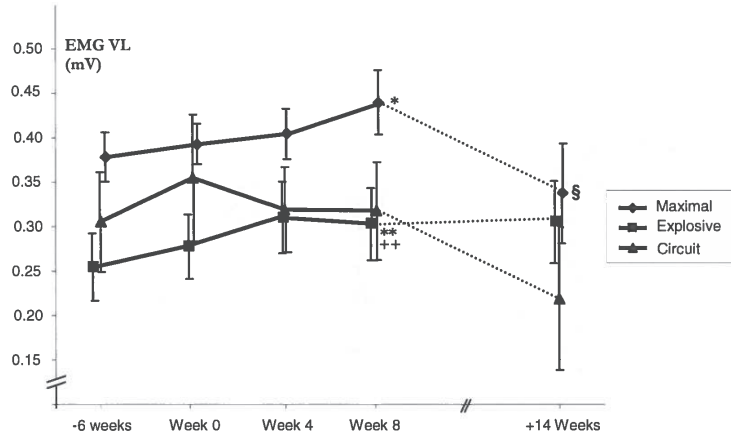


Figure 3.

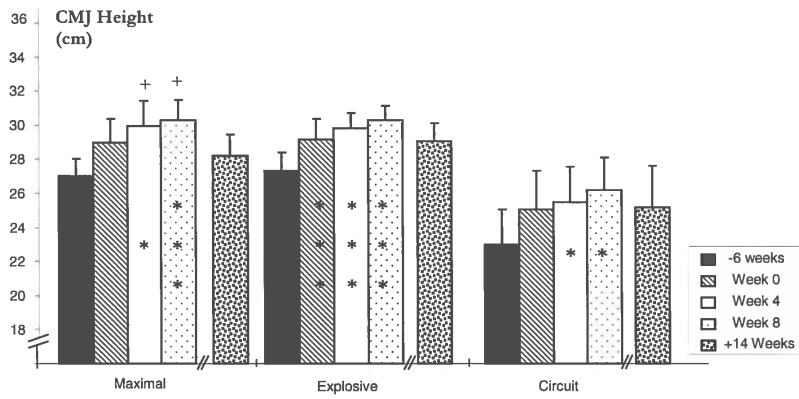


Figure 4.

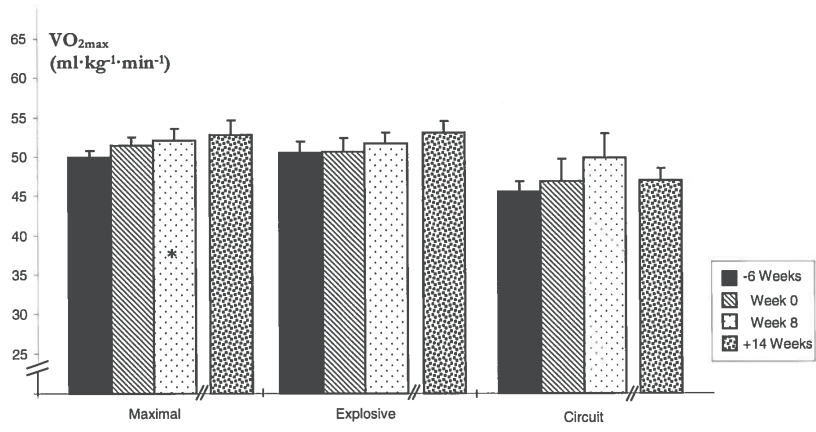


Figure 5.

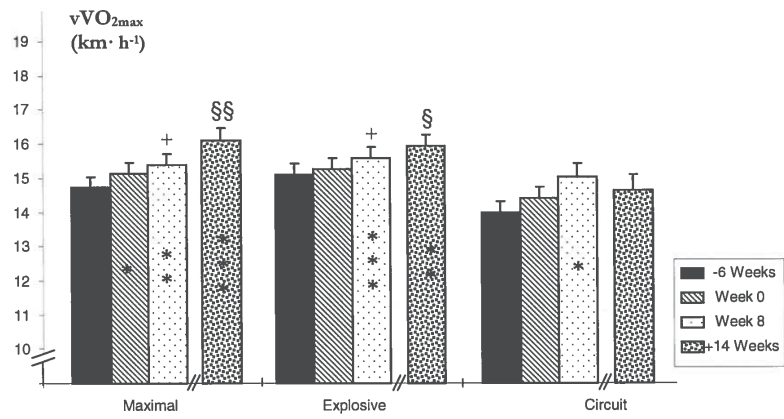


Figure 6.

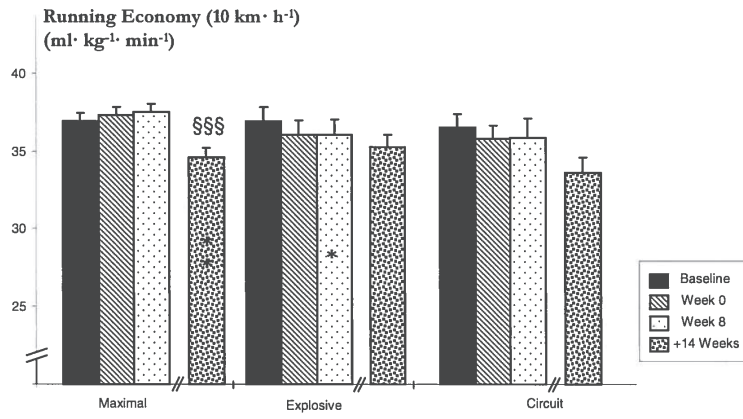


Figure 7.

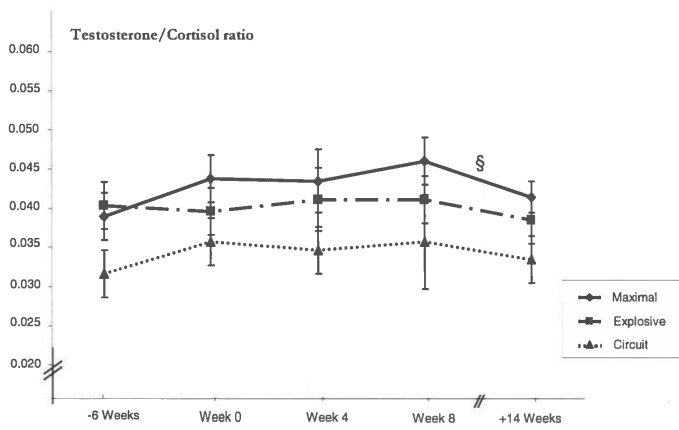


Figure 8.

## TABLES:

Table 1. Strength training programs

| 6-week preparatory strength training period   | 8-week strength training intervention   | 14-week reduced volume strength training period   |
|---|---|---|
| <b>Maximal Strength Training</b>  |   |   |
| <ul style="list-style-type: none"> <li>◆ 2-3 sets, 10-15 x 50-70% 1RM squat/leg press, knee extension, knee flexion, lat pull-down/ bench press, calf exercises and countermovement jump</li> </ul> | <ul style="list-style-type: none"> <li>◆ 3 sets, 4-6 x 80-85% 1RM squats (Smith) and leg press</li> <li>◆ 2 sets, 12-15 x 50-60% 1RM calf exercise</li> </ul>   | <ul style="list-style-type: none"> <li>◆ 3 sets, 6-8 x 75-80% 1RM squats (Smith)</li> <li>◆ 2 sets, 10-12 x 60-70% 1RM knee extension, knee flexion, lat pull-down, calf exercises and bench press</li> </ul>   |
| <b>Explosive Strength Training</b>  |   |   |
| <ul style="list-style-type: none"> <li>◆ 2-3 sets, 10-15 x 50-70% 1RM squat/leg press, knee extension, knee flexion, lat pull-down/ bench press, calf exercises and countermovement jump</li> </ul> | <ul style="list-style-type: none"> <li>◆ 3 sets, 6 x 30-40% 1RM explosive squats (Smith) and leg press</li> <li>◆ 2-3 sets, 10 second scissor jump with 20kg load</li> <li>◆ 2-3 sets 5 maximal individual squat jumps</li> <li>◆ 2-3 sets 5 maximal squat jumps (in series) (20kg load between 4 and 8)</li> </ul> | <ul style="list-style-type: none"> <li>◆ 3 sets, 6 x 30-40% 1RM explosive squats (Smith)</li> <li>◆ 3 sets, 10 second scissor jump with 20kg load</li> <li>◆ 3x5 maximal squat jumps (in series)</li> <li>◆ 2 sets, 10-12 x 60-70% 1RM lat pull-down, calf exercises and bench press</li> </ul> |
| <b>Circuit Training Group</b>   |   |   |
| <ul style="list-style-type: none"> <li>◆ 2-3 sets, 10-15 x 50-70% 1RM squat/leg press, knee extension, knee flexion, lat pull-down/ bench press, calf exercises and countermovement jump</li> </ul> | <ul style="list-style-type: none"> <li>◆ 3 sets, 40-50 seconds of: Squats, push-ups, lunges, sit-ups, calf-raises, back</li> </ul>  | <ul style="list-style-type: none"> <li>◆ 3 sets, 50 seconds of: squats, push-ups/ bench press, lunges, sit-ups, calf-raises, back extensions, planks and step-ups</li> </ul>  |



**Table 2.** Average strength and endurance training frequency (per week).

| Group     |           | 6-week preparatory strength training period | 8- week Strength Training Intervention |                  | 14-week reduced volume strength training period |
|-----------|-----------|---|--|------------------|---|
|           |           | -6  | Week 0 to Week 4                       | Week 4 to Week 8 | +14 Weeks                                       |
| Maximal   | Strength  | 1.4 ± 0.0                                   | 1.7 ± 0.1**                            | 1.6 ± 0.1**      | 0.6 ± 0.1***,†††,###                            |
|           | Endurance | 3.3 ± 0.2                                   | 3.1 ± 0.2                              | 2.9 ± 0.2        | 3.6 ± 0.2                                       |
| Explosive | Strength  | 1.4 ± 0.1                                   | 1.7 ± 0.1**                            | 1.7 ± 0.0***     | 0.5 ± 0.1***,†††,###                            |
|           | Endurance | 3.9 ± 0.2                                   | 3.7 ± 0.3                              | 3.1 ± 0.2**      | 3.7 ± 0.2#                                      |
| Circuit   | Strength  | 1.0 ± 0.7                                   | 1.3 ± 0.2                              | 1.3 ± 0.4        | 0.5 ± 0.1                                       |
|           | Endurance | 3.7 ± 0.4                                   | 3.1 ± 0.6                              | 3.0 ± 0.7        | 3.6 ± 0.5                                       |

**Table 3.** Average endurance training kilometers (per week).

| GROUP     |            | 6-week preparatory strength training period | 8- week Strength Training Intervention |                  | 14-week reduced volume strength training period |
|-----------|------------|---|--|------------------|---|
|           |            | -6  | Week 0 to Week 4                       | Week 4 to Week 8 | +14 Weeks                                       |
| Maximal   | Running km | 19.0 ± 2.8                                  | 22.2 ± 2.5                             | 30.3 ± 4.5*      | 38.1 ± 3.4***,†††,#                             |
| Explosive | Running km | 23.5 ± 5.6                                  | 26.0 ± 4.6                             | 38.3 ± 4.8**,*†† | 40.8 ± 5.6**,*††                                |
| Circuit   | Running km | 21.5 ± 4.1                                  | 20.0 ± 5.0                             | 29.8 ± 7.8       | 36.4 ± 5.3**,*††                                |

**Table 4.** Serum testosterone and cortisol concentrations.

|                       |           | 6-week preparatory strength training period | 8- week Strength Training Intervention |               |               | 14-week reduced volume strength training period |
|-----------------------|-----------|---|--|---------------|---------------|---|
|                       |           | -6 weeks                                    | Week 0                                 | Week 4        | Week 8        | +14 Weeks                                       |
| TESTOSTERONE (nmol/L) | Maximal   | 17.0 ± 4.3                                  | 18.7 ± 5.7                             | 17.4 ± 5.4    | 17.7 ± 3.3    | 17.2 ± 3.5                                      |
|                       | Explosive | 15.7 ± 2.0                                  | 15.9 ± 3.7                             | 17.1 ± 3.2    | 16.8 ± 3.0    | 16.3 ± 4.2                                      |
|                       | Circuit   | 14.6 ± 4.7                                  | 15.5 ± 4.8                             | 15.6 ± 2.6    | 13.7 ± 3.8    | 14.8 ± 4.8                                      |
| CORTISOL (nmol/L)     | Maximal   | 451.1 ± 65.0                                | 440.4 ± 98.1                           | 412.8 ± 79.9  | 401.9 ± 94.9  | 427.4 ± 79.1                                    |
|                       | Explosive | 414.8 ± 93.5                                | 414.6 ± 78.9                           | 452.5 ± 134.5 | 437.6 ± 120.2 | 442.3 ± 85.0                                    |
|                       | Circuit   | 462.2 ± 77.7                                | 435.5 ± 118.5                          | 471.7 ± 108.4 | 427.0 ± 124.7 | 458.3 ± 117.6                                   |

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## II

### **NEUROMUCULAR ADAPTATIONS DURING COMBINED STRENGTH AND ENDURANCE TRAINING IN ENDURANCE RUNNERS: MAXIMAL OR EXPLOSIVE STRENGTH TRAINING OR A MIX OF BOTH**

by

R.S. Taipale, J. Mikkola, V. Vesterinen, A. Nummela & K. Häkkinen. 2013.

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## Neuromuscular adaptations during combined strength and endurance training in endurance runners: maximal versus explosive strength training or a mix of both

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**Abstract** This study compared the effects of mixed maximal strength and explosive strength training with maximal strength training and explosive strength training combined with endurance training over an 8-week training intervention. Male subjects (age 21–45 years) were divided into three strength training groups, maximal (MAX,  $n = 11$ ), explosive (EXP, 10) and mixed maximal and explosive (MIX, 9), and a circuit training control group, (CON, 7). Strength training one to two times a week was performed concurrently with endurance training three to four times a week. Significant increases in maximal dynamic strength (1RM), countermovement jump (CMJ), maximal muscle activation during 1RM in MAX and during CMJ in EXP, peak running speed ( $S_{\text{peak}}$ ) and running speed at respiratory compensation threshold ( $RCT_{\text{speed}}$ ) were observed in MAX, EXP and MIX. Maximal isometric strength and muscle activation, rate of force development (RFD), maximal oxygen uptake ( $\dot{V}O_{2\text{max}}$ ) and running economy (RE) at 10 and 12 km hr<sup>-1</sup> did not change significantly. No significant changes were observed in CON in maximal isometric strength, RFD, CMJ or muscle activation, and a significant decrease in 1RM was observed in the final 4 weeks of training. RE in CON did not change significantly, but significant increases were observed in  $S_{\text{peak}}$ ,  $RCT_{\text{speed}}$  and ( $\dot{V}O_{2\text{max}}$ ). Low volume

MAX, EXP and MIX strength training combined with higher volume endurance training over an 8-week intervention produced significant gains in strength, power and endurance performance measures of  $S_{\text{peak}}$  and  $RCT_{\text{speed}}$ , but no significant changes were observed between groups.

**Keywords** Concurrent training · Neuromuscular performance · Endurance performance · Running economy · Strength

### Introduction

In recent years, the importance of neuromuscular performance in endurance sports has been examined by a number of researchers who have come to the general conclusion that strength training improves neuromuscular performance, while consequently improving sport-specific economy in endurance disciplines like running (Paavolainen et al. 1999; Taipale et al. 2010; Støren et al. 2008) and cross-country skiing (Paavolainen et al. 1991; Mikkola et al. 2007; Hoff et al. 2002). Improved neuromuscular performance includes, e.g., increases in voluntary muscle activation, which leads to increases in strength and power, associated with increased speed and decreased oxygen consumption at submaximal speeds (improved economy). Strength training, performed concurrently with endurance training has been reported to be beneficial to the neuromuscular characteristics of trained and untrained men and women of all ages (e.g., Hoff et al. 2002; Häkkinen et al. 2003; Mikkola et al. 2007; Paavolainen et al. 1991, 1999; Sillanpää et al. 2008; Støren et al. 2008; Taipale et al. 2010); however, an “interference effect” has also been reported when strength and endurance exercises are performed concurrently (Hickson 1980). The cause of the

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"interference effect" appears to be related to the divergent responses and adaptations to strength and endurance training, and is associated with high volume or intensity, long duration and mode of training (Wilson et al. 2011).

Within strength training, it is important to remember that there is a specificity effect of the training mode and its adaptations in the neuromuscular system. For example, maximal strength training with high loads (such as 70–90 % 1RM) and low repetitions per set generally result in neural adaptations followed by muscle hypertrophy over prolonged training periods. In contrast, explosive resistance training with low to medium loads (such as 30–60 % 1RM) but high action velocity movements improves the neuromuscular characteristics, especially rapid activation of the muscles due to increased motor unit recruitment. Both maximal and explosive strength training performed concurrently with endurance training have been shown to be more effective in improving strength, power and muscular activation in recreational endurance runners than concurrent circuit and endurance training (Mikkola et al. 2011; Taipale et al. 2010). A mix of maximal and explosive strength training in a single session and performed concurrently with endurance training may therefore be hypothesized to have an additive effect over maximal or explosive strength training performed concurrently with endurance training in recreational endurance runners.

It is commonly known that mode, intensity (Tremblay et al. 2005) and duration of training (Tremblay et al. 2005) also influence serum hormonal concentrations and that hormones play a significant role in adaptations to strength and endurance training. The typical acute serum hormonal response to a strength training session in men includes, e.g., an increase in serum testosterone (TESTO), while prolonged endurance exercise sessions cause significant increases in serum levels of cortisol (CORT) and/or a decrease in serum levels of TESTO. Acute changes in serum hormonal concentrations may lead to chronic changes in basal serum hormonal concentrations. In endurance trained men, Consitt et al. (2002) reported a decrease, or no change, in basal concentrations of hormones, while Hackney et al. (2003) reported decreased TESTO and increased CORT. Other hormones may also be affected by training and/or related stress. For example, decreased thyroid-stimulating hormone (TSH) has been linked to psychological stress (i.e., Nadolnik 2011) as well as physical stress (Pakarinen et al. 1991). Other research has suggested that prolonged strength training does not affect TSH levels (Pakarinen et al. 1988). Dehydroepiandrosterone sulfate (DHEAS) has also been shown to decrease with exercise stress (possibly as a result of oxidative stress) and aging (Aldred et al. 2009).

Based on our previous knowledge of the benefits of adding strength training to endurance training to improve

neuromuscular performance and sport-specific economy, the purpose of the present study was to compare the effects of adding three different low volume modes of strength training performed concurrently with a higher volume of endurance training relative to strength training. Neuro-muscular, cardiorespiratory and hormonal adaptations to training were examined in male recreational endurance runners over an 8-week strength training intervention. The strength training modes studied included mixed maximal and explosive strength training, maximal strength training and explosive strength training, while endurance training was similar across groups.

## Methods

### Subjects

Thirty-seven male recreational endurance runners (age 21–45 years) were recruited as part of a marathon training school and completed the study. The study design, including information on the possible risks and benefits of participation, were thoroughly explained prior to subjects signing an informed consent document. Ethical approval was granted by the university ethical committee and the study was conducted according to the most recent Declaration of Helsinki.

Subjects were divided into groups matched for age, anthropometrics, training experience, strength and endurance characteristics such as maximal oxygen uptake ( $\dot{V}O_{2\max}$ ), peak running speed ( $S_{\text{peak}}$ ), running speed at lactate threshold ( $S_{\text{LT}}$ ) and running speed at respiratory compensation threshold ( $S_{\text{RCT}}$ ) following baseline testing, which was performed approximately 6 weeks prior to the strength training intervention. During the actual strength training intervention, groups included a maximal strength training group (MAX,  $n = 11$ , age:  $35.5 \pm 6.1$  years, height:  $178.6 \pm 5.5$  cm (mean  $\pm$  SD)), an explosive strength training group (EXP,  $n = 10$ ,  $36.5 \pm 6.5$  years,  $180.5 \pm 6.4$  cm) a mixed maximal and explosive strength training group (MIX,  $n = 9$ ,  $31.3 \pm 8.9$  years,  $178.1 \pm 5.3$  cm) and a circuit training control group which used only their own body weight as a load (CON,  $n = 7$ ,  $33.7 \pm 8.8$  years,  $180.0 \pm 4.8$  cm). There were no significant differences between groups at baseline. Subjects were not using medications and had no injuries that would affect physical performance.

### Study design and training

This study is part of a large experimental design involving prolonged and periodized concurrent strength and endurance training. Some of the results obtained with the present

subjects have been previously reported (Mikkola et al. 2011; Taipale et al. 2010). The unique purpose of this study was to compare mixed maximal and explosive strength training combined with endurance training to maximal strength training, explosive strength training and circuit training combined with endurance training in men. While data from the preparatory strength training period are not reported in this manuscript, it should be noted that the subjects performed 6 weeks of preparatory training (prior to this 8-week intervention) in which submaximal loads of 50 % progressing up to 70 % 1RM were used. The purpose of the 6-week preparatory strength training period was for subjects to learn proper lifting technique prior to the intervention. As subjects were previously untrained in terms of strength, it was also important to include this period in their training to avoid injury while also reducing the effect of learning on strength gains that may have otherwise occurred during the intervention period. Exercises used for the lower extremities during the 6-week preparatory period were the same as those used during the 8-week strength training intervention.

The present 8-week strength training program was focused on the leg extensors as they are a major muscle group at work in human locomotion and running and thus

an important muscle group to study. The 8-week intervention period was used, because 8 weeks of strength training after a 6-week preparatory period of strength training should be long enough to show differences between training modes (e.g., Häkkinen and Komi 1981). Each strength training session was preceded by 20–30 min of low-intensity endurance exercise (below lactate threshold) and warm-up sets using lower resistance were performed prior to squat and leg press exercises. In MAX, EXP and MIX, 2–3 min of rest separated exercise sets throughout the study (Table 1). Using body weight as a load, CON completed typical circuit training including squats, push-ups, lunges, sit-ups, toe raises, back-ups, planks and step-ups in series. A work to rest ratio of 45/15 s and 50/10 s was used during weeks 0–4 and 4–8, respectively. This circuit training protocol is typical of one that an endurance runner may use. The purpose of this type of protocol is primarily to improve muscle endurance, and not maximal strength. Strength training was performed one to two times a week in all training groups and was monitored by experienced personnel so that training loads were progressively increased when necessary.

Subjects performed endurance training throughout the study. Endurance training was typically performed on

**Table 1** Eight-week strength training programs

| Strength training intervention programs         |      |             |  |                |
|---|------|-------------|--|----------------|
|   | Sets | Repetitions | Load   | Recovery (min) |
| <i>Maximal strength</i>                         |      |             |  |                |
| Squat   | 3    | 4–6         | 80–85 % 1RM                                      | 2              |
| Leg press                                       | 3    | 4–6         | 80–85 % 1RM                                      | 2              |
| Calf exercise                                   | 2    | 12–15       | 50–60 % 1RM                                      | 2              |
| Sit-ups, back-extension                         | 3    | 20–30       | Body weight                                      | 2              |
| <i>Explosive power</i>                          |      |             |  |                |
| Squat   | 3    | 6           | 30–40 % 1RM                                      | 2              |
| Leg press                                       | 3    | 6           | 30–40 % 1RM                                      | 2              |
| Scissor jump                                    | 2–3  | 10s         | 20 kg  | 2              |
| Maximal squat jump                              | 2–3  | 5 single    | Body weight                                      | 2              |
| Maximal squat jump                              | 2–3  | 5 in series | Body weight <sup>a</sup> (20 kg between 4 and 8) | 2              |
| Sit-ups, back-extension                         | 3    | 20–30       | Body weight <sup>a</sup>                         | 2              |
| <i>Mixed maximal strength + explosive power</i> |      |             |  |                |
| Squat and leg press (week 0–week 4):            |      |             |  |                |
| Warm-up   | 1–2  | 10          | 50–70 % 1RM                                      | 2              |
| Maximal strength training                       | 2    | 6           | 6RM  | 3              |
| <sup>a</sup> Squat/Leg press (week 4–week 8):   |      |             |  |                |
| Warm-up   | 2    | 10          | 50–70 % 1RM                                      | 2              |
| Maximal strength training                       | 3    | 4           | 4RM  | 3              |
| Box jumps                                       | 2–3  | 8–10        | Body weight                                      | 2–3            |
| Vertical jumps                                  | 2–3  | 8–10        | Body weight                                      | 2–3            |
| Sit-ups, back-extension                         | 3    | 20–30       | Body weight                                      | 2              |

The italics indicate the final four weeks of the training program (week 4–8)

<sup>a</sup> Modification in program during the final 4 weeks of the training intervention



non-strength training days with a training intensity below the lactate threshold (LT) throughout the study. Training intensities based on LT and respiratory compensation thresholds (RCT) were individually determined for each subject each time they were tested for maximal oxygen uptake. Subjects were informed of their individual heart rate ranges between which to train and used a heart rate monitor (Suunto t6, Vantaa, Finland) to monitor and record each training session. Training was individualized for subjects based on their training background and current fitness level; however, the relative volume and intensity were similar. Training volume (including running and "other" endurance activities) in terms of km week<sup>-1</sup> was 42.6 ± 30.5, 40.7 ± 30.5, 33.0 ± 28.5 and 25.5 ± 22.0 for MAX, EXP, MIX and CON, respectively. Total endurance training time per week was 5:38 ± 0:56 h. There were no statistically significant differences in training volume. The subjects kept a training diary throughout the study, recording strength training sessions, kilometers and duration of running and "other" endurance activity (cycling, cross-country skiing and Nordic walking). Training plans were personalized based on training ability and background. Measurements took place prior to the preparatory period (for stratification into groups), before, during and after the strength training intervention (weeks 0, 4 and 8).

## Measurements

### Body composition

In addition to standing height (measured using standard methods), body mass and body composition were measured using bioimpedance (InBody720 body composition analyzer, Biospace Co. Ltd, Seoul, South Korea). Measurements were always taken in conjunction with blood tests between 07.30 and 08.00. Thus, subjects always arrived for testing in a fasted and rested state, helping to keep the possible confounding variables of diet and hydration status to a minimum. Subjects were measured in their underwear and were instructed to remove their watches, shoes and socks prior to measurement.

### Performance measures

#### Aerobic capacity

Endurance performance characteristics were measured using a treadmill running protocol (Mikkola et al. 2007). The running velocity began at 8 km h<sup>-1</sup> and was increased by 1 km h<sup>-1</sup> every third minute until volitional exhaustion.

Treadmill incline remained a constant 0.5° throughout the test. Heart rate was recorded continuously using a heart rate monitor (Suunto t6, Vantaa, Finland). The mean heart rate values from the last minute of each stage were used for analysis. Oxygen consumption was measured breath by breath throughout the test, using a portable gas analyzer (Oxycon Mobile®, Jaeger, Hoechberg, Germany). Maximal oxygen uptake ( $\dot{V}O_{2\max}$ ) was determined to the highest average 60 s  $\dot{V}O_2$  value. Other factors such as heart rate,  $\dot{V}O_2$  and respiratory exchange ratio were monitored for determination of maximal effort. Fingertip blood samples were taken every 3rd min to measure blood lactate concentrations. For blood sampling, the treadmill was stopped for approximately 15–20 s. Blood lactates were analyzed using a Biosen S\_line Lab + lactate analyzer (EKF Diagnostic, Magdeburg, Germany). Running economy (RE) was evaluated by examining  $\dot{V}O_2$  at 10 and 12 km h<sup>-1</sup>, speeds comparable to the marathon running speeds of our subjects. Peak running speed ( $S_{\text{peak}}$ ) was calculated as follows  $S_{\text{peak}} = \text{speed of the last whole completed stage (km h}^{-1}\text{)} + (\text{running time (s) of the speed at exhaustion} - 30 \text{ s}) / (180 - 30 \text{ s}) \times 1 \text{ km h}^{-1}$  (Mikkola et al. 2011). Lactate threshold (LT) and respiratory compensation threshold (RCT) were determined using blood lactate, ventilation,  $\dot{V}O_2$  and  $\dot{V}CO_2$  (production of carbon dioxide) according to Meyer et al. (2005).

### Strength and power measurements

#### Isometric leg press

An electromechanical isometric leg extension device (designed and manufactured by the Department of Biology of Physical Activity, University of Jyväskylä, Finland) was used to measure maximal strength and rate of force development (RFD). The subjects' knee angle was 107°, while the hip angle was 110°. Subjects were instructed to produce force "as fast and as hard as possible" for approximately 3 s. Subjects performed at least three maximum voluntary contractions (Häkkinen et al. 1998). If the maximum force during the last trial was greater than 5 % compared to the previous trial, an additional trial was performed. The best performance trial, in terms of maximal force measured in newtons (N), was used for statistical analysis.

#### One repetition maximum

One repetition maximum (maximal dynamic bilateral horizontal leg press in a seated position) was measured using a David 210 dynamometer (David Sports Ltd., Helsinki, Finland) (Häkkinen et al. 1998). Prior to attempting

1RM, subjects completed a warm-up consisting of  $5 \times 70\%$  1RM,  $1 \times 80\text{--}85\%$  1RM and  $1 \times 90\text{--}95\%$  of estimated 1RM, with 1 min of rest between sets. Following this warm-up, no more than five attempts to reach 1RM were made. Leg extension action started from a knee angle of approximately  $65^\circ$ . Subjects were instructed to grasp handles located by the seat of the dynamometer and to keep constant contact with the seat and backrest during leg extension to a full extension of  $180^\circ$ . Verbal encouragement was given to promote maximal effort. The greatest weight that the subject could successfully lift (knees fully extended) to the accuracy of 2.5 kg was accepted as 1RM.

#### Countermovement jump

A force platform (Department of Biology of Physical Activity, Jyväskylä, Finland) was used to measure maximal dynamic explosive force in a countermovement jump (CMJ) (Bosco and Komi 1978). Subjects were instructed to stand with their feet approximately hip-width apart with their hands on their hips. Subjects were then instructed to perform a quick and explosive CMJ on verbal command, so that knee angle for the jump was no less than  $90^\circ$ . Force data was collected and analyzed by computer software (Signal 2.14, CED, Cambridge, UK), which used the equation  $h = I^2/2gm^2$  to calculate jump height from impulse ( $I =$  impulse,  $g =$  gravity and  $m =$  mass of subject).

#### Electromyographic activity

Electromyographic activity (EMG) was recorded from the vastus lateralis (VL) and vastus medialis (VM) of the right leg during maximal isometric leg extension, 1RM and CMJ. Electrode positions were marked with small ink tattoos (Häkkinen and Komi 1983) on the skin during the first testing session to ensure that electrode placement over the entire experimental period would be consistent. The guidelines published by Surface ElectroMyoGraphy for the Non-Invasive Assessment of Muscles (SENIAM <http://www.seniam.org/>) were followed for skin preparation, electrode placement and orientation. Inter-electrode distance was 20 mm (input impedance  $<10$  k $\Omega$ , common mode rejection ratio 80 dB, 1,000 gain). Raw signals were passed from a transmitter, positioned around the subjects' waist, to a receiver (Telemetry 2400R, Noraxon, Scottsdale, AZ, USA) from which the signal was relayed to the computer via an AD converter (Micro1401, CED, Cambridge, UK). Whole range EMG was recorded from the starting knee angle of approximately  $65^\circ$  to full leg extension of  $180^\circ$  and subsequently analyzed by computer software (Signal 2.14, CED, UK).

#### Serum hormones

Venous blood samples (10 ml) were collected from the antecubital vein using sterile needles into serum tubes (Venosafe, Terumo Medical Co., Leuven, Belgium) by a qualified laboratory technician. Subjects were tested after 12 h of fasting between 07.30 and 08.00 and were instructed to refrain from strenuous physical activity for 24 h prior to the tests. Whole blood was centrifuged at 2500g (Megafuge 1.0R, Heraeus, Germany) for 10 min after which serum was removed and stored at  $-80^\circ\text{C}$  until analysis. Samples were used for determination of serum testosterone TESTO, CORT, TSH, DHEAS and sex-hormone binding globulin (SHBG). Analyses were performed using chemical luminescence techniques (Immunlite 1000, DCP Diagnostics Corporation, Los Angeles, California, USA) and hormone-specific immunoassay kits (Siemens, New York, NY, USA). The sensitivity of TESTO, CORT, TSH, DHEAS and SHBG assays were  $0.7$  nmol  $\text{l}^{-1}$ ,  $5.5$  nmol  $\text{l}^{-1}$ ,  $0.004$  mIU  $\text{l}^{-1}$ ,  $0.08$   $\mu\text{mol}$   $\text{l}^{-1}$  and  $0.2$  nmol  $\text{l}^{-1}$ , respectively. The intra-assay coefficients of variation for TESTO, CORT, TSH, DHEAS and SHBG were: 5.7, 4.6, 3.9, 9.5 and 2.4 %, respectively.

#### Statistical methods

Standard statistical methods were used for calculation of means, standard deviation, standard error and Pearson product-moment correlation coefficients. Group differences were analyzed using a one-way analysis of variance (one-way ANOVA) and within group differences (group-by-training interaction) were analyzed using repeated measures ANOVA. ANCOVA (using baseline measurements as a covariate) was used in analyzing CMJ due to changes in jumping height following the 6-week preparatory period. In the presence of a significant  $F$  value, post hoc comparison of means was provided by Fisher's LSD test. The criterion for significance was set at  $* = p \leq 0.05$ ,  $** = p < 0.01$  and  $*** = p < 0.001$ . Statistical analysis was completed using SPSSWIN 15.0 (SPSS Inc., Chicago, IL, USA).

#### Results

Maximal strength increased significantly in all experimental training groups after the first 4 weeks of the strength training intervention, while the increases in maximal strength plateaued in the final 4 weeks (Fig. 1). In the control group, maximal strength increased only slightly (n.s.) during the first 4 weeks of the strength training intervention, but a significant decrease in maximal strength was observed in the final 4 weeks (Fig. 1). Muscle

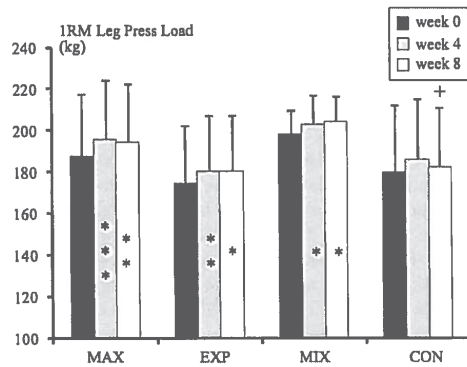


Fig. 1 Maximal strength as maximal bilateral dynamic leg press 1RM load (kg) during the 8-week strength training intervention (mean  $\pm$  SD). \* $p \leq 0.05$ , \*\* $p < 0.01$  and \*\*\* $p < 0.001$  from week 0 and <sup>+</sup> $p \leq 0.05$  from week 4

activation of VL increased significantly only in MAX from week 0 to 8 (11.8 %,  $p < 0.05$ ), while muscle activation as an average of VL + VM did not change significantly in any of the groups. No significant differences in maximal strength development or changes in muscle activation were observed between groups.

Maximal strength measured by maximal bilateral isometric leg press did not change significantly in any of the groups (Fig. 2). Rate of force development and muscle activation of VL and VM were also statistically unaltered during the 8-week strength training intervention. No significant differences in isometric strength and rate of force development or changes in muscle activation were observed between groups.

Significant increases were observed in CMJ height of all three experimental strength training groups by the end of the strength training intervention, while CON showed no

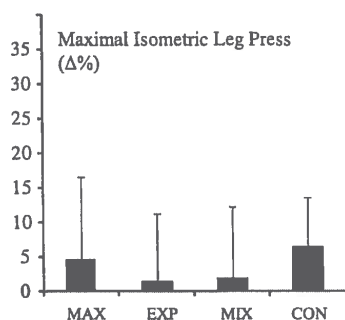


Fig. 2 Relative change (%) in maximal isometric leg press force after the 8-week strength training intervention

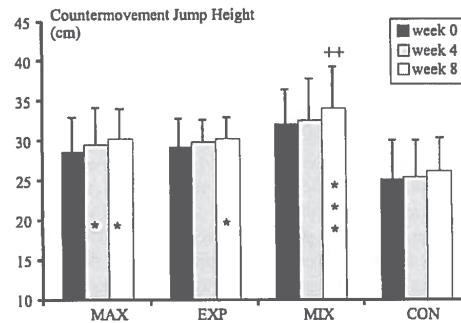


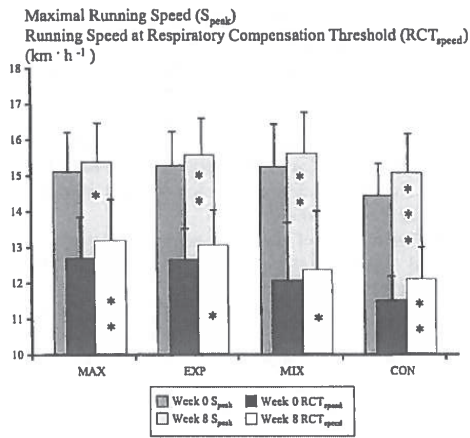
Fig. 3 Countermovement jump height (cm) during the 8-week strength training intervention (mean  $\pm$  SD). \* $p \leq 0.05$ , \*\* $p < 0.01$  and \*\*\* $p < 0.001$  from week 0 and, <sup>+</sup> $p \leq 0.05$  from week 4

changes (Fig. 3). In MAX, the significant increase in jumping height was observed already after 4 weeks of the strength training intervention, whereas in EXP and MIX, a significant increase was observed at the end of the strength training intervention (week 8). In MIX, the significant improvement in CMJ height occurred primarily in the final 4 weeks of the strength training intervention. Significant changes in muscle activation of VL and VM during CMJ were observed only in EXP (21.3 %,  $p < 0.05$  between weeks 0 and 8) over the entire strength training intervention. No significant differences in countermovement jump height development or changes in muscle activation were observed between groups.

Maximal oxygen uptake ( $\dot{V}O_{2max}$  in  $ml\ kg^{-1}\ min^{-1}$ ) did not change significantly in the experimental groups (MAX  $51.4 \pm 3.8$  to  $52.1 \pm 4.9$ , EXP  $50.6 \pm 5.2$  to  $51.7 \pm 4.0$  and MIX  $51.3 \pm 5.2$  to  $51.7 \pm 5.4$ ), while a significant increase in  $\dot{V}O_{2max}$  was observed in the control group ( $47.0 \pm 6.2$  to  $49.8 \pm 7.0$   $p < 0.01$ ). Running economy at  $10\ km\ hr^{-1}$  and  $12\ km\ hr^{-1}$  did not change in terms of  $ml\ kg^{-1}\ min^{-1}$  over the 8-week strength training intervention, while a significant increase in  $S_{peak}$  and  $RCT_{speed}$  was observed in all groups (Fig. 4). No significant differences in changes of  $\dot{V}O_{2max}$ ,  $S_{peak}$  or  $RCT_{speed}$  were observed between groups.

Body mass decreased significantly in MAX, EXP and CON, while body fat percentage did not change significantly during the 8-week strength training intervention in any group (Table 2). No significant changes were observed in changes of body mass or body fat percentage between the groups.

Serum hormone concentrations of TESTO, CORT, TSH and DHEAS did not change significantly over the 8-week strength training intervention. The serum concentration of SHBG decreased significantly in EXP from week 0 to week



**Fig. 4** Maximal running speed ( $S_{peak}$ , km hr<sup>-1</sup>) and running speed at respiratory compensation threshold ( $RCT_{speed}$ , km hr<sup>-1</sup>) before and after the 8-week strength training intervention (mean  $\pm$  SD). \* $p \leq 0.05$ , \*\* $p < 0.01$  and \*\*\* $p < 0.001$  from week 0 (lower values indicate  $RCT_{speed}$ ; upper values indicate  $S_{peak}$ )

**Table 2** Body mass and percent fat as measured by InBody during the 8-week strength training intervention (mean  $\pm$  SD)

|                  | Week 0         | Week 4         | Week 8                         |
|------------------|----------------|----------------|--------------------------------|
| <b>Mass (kg)</b> |                |                |                                |
| MAX              | 78.8 $\pm$ 5.9 | 78.3 $\pm$ 6.0 | 77.4 $\pm$ 5.9 <sup>+</sup>    |
| EXP              | 78.6 $\pm$ 6.3 | 78.4 $\pm$ 5.7 | 76.9 $\pm$ 5.4 <sup>**++</sup> |
| MIX              | 78.4 $\pm$ 6.8 | 78.6 $\pm$ 7.1 | 78.6 $\pm$ 7.0                 |
| CON              | 84.3 $\pm$ 9.5 | 84.0 $\pm$ 9.9 | 82.9 $\pm$ 9.6 <sup>**</sup>   |
| <b>Fat %</b>     |                |                |                                |
| MAX              | 17.2 $\pm$ 4.0 | 17.1 $\pm$ 4.2 | 16.7 $\pm$ 4.3                 |
| EXP              | 16.0 $\pm$ 6.1 | 16.0 $\pm$ 5.9 | 15.4 $\pm$ 5.7                 |
| MIX              | 16.2 $\pm$ 5.4 | 15.9 $\pm$ 5.5 | 15.8 $\pm$ 4.0                 |
| CON              | 20.8 $\pm$ 5.4 | 20.5 $\pm$ 5.7 | 19.8 $\pm$ 5.6                 |

\*  $p \leq 0.05$  from week 0, \*\*  $p \leq 0.01$  from week 0, <sup>+</sup>  $p \leq 0.05$  from week 4 and <sup>++</sup>  $p \leq 0.01$  from week 4

8, while in MAX, MIX and CON concentrations remained statistically unaltered (Table 3). No significant changes were observed in serum hormone concentrations between the groups.

**Discussion**

In this study, low volume mixed maximal and explosive strength training (MIX), maximal strength training (MAX) and explosive strength training (EXP) were performed concurrently with a higher volume of endurance training

**Table 3** Serum hormone and SHBG concentrations during the 8-week strength training intervention (mean  $\pm$  SD)

|   | WEEK 0            | WEEK 4            | WEEK 8            |
|---|-------------------|-------------------|-------------------|
| <b>TESTO (nmol l<sup>-1</sup>)</b>                |                   |                   |                   |
| MAX   | 18.7 $\pm$ 5.7    | 17.4 $\pm$ 5.4    | 17.8 $\pm$ 3.3    |
| EXP   | 15.9 $\pm$ 3.7    | 17.1 $\pm$ 3.2    | 16.8 $\pm$ 3.0    |
| MIX   | 23.2 $\pm$ 4.2    | 23.7 $\pm$ 5.0    | 20.1 $\pm$ 5.7*   |
| CON   | 15.5 $\pm$ 4.4    | 15.6 $\pm$ 2.7    | 13.7 $\pm$ 3.5    |
| <b>CORT (nmol l<sup>-1</sup>)</b>                 |                   |                   |                   |
| MAX   | 440.4 $\pm$ 98.1  | 412.8 $\pm$ 79.9  | 401.9 $\pm$ 94.9  |
| EXP   | 414.6 $\pm$ 78.9  | 452.5 $\pm$ 134.5 | 437.6 $\pm$ 120.2 |
| MIX   | 496.6 $\pm$ 148.0 | 542.9 $\pm$ 103.2 | 482.0 $\pm$ 119.2 |
| CON   | 468.3 $\pm$ 118.5 | 497.3 $\pm$ 108.4 | 456.7 $\pm$ 124.7 |
| <b>TSH (<math>\mu</math>IU ml<sup>-1</sup>)</b>   |                   |                   |                   |
| MAX   | 2.2 $\pm$ 0.8     | 2.2 $\pm$ 0.8     | 2.0 $\pm$ 0.8     |
| EXP   | 1.8 $\pm$ 0.4     | 2.0 $\pm$ 0.8     | 2.1 $\pm$ 1.0     |
| MIX   | 2.3 $\pm$ 1.1     | 2.5 $\pm$ 1.1     | 2.5 $\pm$ 1.1     |
| CON   | 1.9 $\pm$ 0.9     | 1.9 $\pm$ 0.8     | 1.7 $\pm$ 1.0     |
| <b>DHEAS (<math>\mu</math>mol l<sup>-1</sup>)</b> |                   |                   |                   |
| MIX   | 6.7 $\pm$ 1.4     | 6.6 $\pm$ 1.5     | 6.7 $\pm$ 1.3     |
| EXP   | 5.0 $\pm$ 1.8     | 5.0 $\pm$ 1.8     | 5.1 $\pm$ 1.4     |
| MK  | 7.4 $\pm$ 3.7     | 7.2 $\pm$ 3.6     | 6.7 $\pm$ 3.6     |
| CON   | 7.9 $\pm$ 4.8     | 6.4 $\pm$ 3.9     | 6.4 $\pm$ 4.0     |
| <b>SHBG (nmol l<sup>-1</sup>)</b>                 |                   |                   |                   |
| MAX   | 39.6 $\pm$ 11.2   | 37.9 $\pm$ 13.4   | 39.7 $\pm$ 12.9   |
| EXP   | 44.2 $\pm$ 14.2   | 41.3 $\pm$ 12.5   | 40.4 $\pm$ 11.3*  |
| MIX   | 39.1 $\pm$ 14.7   | 38.2 $\pm$ 15.5   | 39.2 $\pm$ 16.8   |
| CON   | 27.5 $\pm$ 7.4    | 29.0 $\pm$ 7.9    | 27.5 $\pm$ 7.3    |

\*  $p \leq 0.05$

over an 8-week training intervention in endurance runners. The primary findings from the intervention period showed significant increases in maximal dynamic strength (1RM), power (CMJ) and training specific maximal muscle activation in the experimental strength training groups. These neuromuscular changes were accompanied by increases in maximal running speed ( $S_{peak}$ ) and running speed at respiratory compensation threshold ( $RCT_{speed}$ ) without a significant change in  $\dot{V}O_{2max}$  or running economy. Interestingly, these significant increases in strength and power were primarily observed during the first 4 weeks of the strength training intervention, whereas a plateau in improvements was observed in the final 4 weeks. Maximal isometric strength, rate of force development (RFD) and muscle activation during the maximal isometric leg press action remained unchanged throughout the 8-week intervention. The circuit training control group (CON) did not show significant increases in any neuromuscular performance variables, and a significant decrease in 1RM was even observed in the final 4 weeks of training. This group

improved  $S_{peak}$ ,  $RCT_{specd}$  and  $\dot{V}O_{2max}$ , but did not improve running economy. Nevertheless, no significant differences in changes between groups were observed in any of the variables during the 8-week intervention.

The significant increases in maximal dynamic strength observed in MAX, EXP and MIX during the first 4 weeks of the strength training intervention were expected given that the subjects had no previous background in strength training aside from the 6-week period of preparatory training that they completed prior to the strength training intervention. Strength gains are known to occur relatively quickly in individuals previously untrained in strength in comparison to strength-trained individuals (Häkkinen 1985; Ahtiainen et al. 2003). It is, however, important to note that the average of 3.5 % gains in strength over the 8-week strength training intervention in the strength training groups were much smaller than those of 10–20 % (e.g., Ahtiainen et al. 2003) typically observed when heavy resistance training is performed alone. The average gains in maximal strength in the present study were also smaller than the gains of about 10 % observed after approximately 8 weeks of concurrent heavy resistance and bicycle endurance training in untrained men (e.g., Häkkinen et al. 2003). A plateau in maximal strength gains was observed in the final 4 weeks of the strength training intervention. While a plateau may typically be expected to occur later, the present 6-week preparatory strength training period did use relatively high loads of 50–70 % 1RM combined with endurance training, which induced small learning-related changes while preparing subjects for the strength training intervention. Therefore, by the end of the first 4 weeks of the actual strength training intervention, it is important to note that subjects had completed a total of 10 weeks of strength training. Previous studies of combined strength and endurance training (e.g., Hickson 1980; Hunter et al. 1987) have also shown plateaus in strength development after the initial weeks of training, while other studies have reported continued strength development over a longer periodized training period (e.g., Häkkinen et al. 2003; Karavirta et al. 2009). Most of the studies reporting a plateau in strength development have included a higher volume of progressive combined strength and endurance training (e.g., Hickson 1980) and a plateau could be observed already after 6–8 weeks of training. The strength training stimuli progressively of one to two times a week in the present study may not have been strong enough to stimulate continuous improvements in maximal strength in the groups performing endurance training concurrently at a relatively higher frequency of three to four times per week. It is of particular interest that the final 4 weeks of the strength training intervention showed a significant decrease in 1RM of CON. This decrease suggests that the circuit

training stimulus, combined with endurance training, was not enough to maintain maximal strength. It is important to note that all three experimental strength training modes, when combined with endurance training, were more effective in improving strength and power than combined circuit training and endurance training, though caution should be exercised with the interpretation of these results as no strength training only group was included in the present experimental design.

Although each of the three strength training groups showed significant strength gains, no significant differences were observed between the three experimental groups with regard to strength development. Eight weeks of low frequency strength training, when accompanied by a higher frequency of endurance training, may not be adequate to show the potential differences in absolute strength and power development that may be expected. Additionally, differences in adaptations to maximal or explosive strength training regimens may not be observed until after 8–12 weeks of specific strength training (Häkkinen 1994). The similar increases in maximal strength of MAX, EXP and MIX may, in this case, be explained by the same mechanisms, because gains in maximal strength are typically associated with increased muscle activation in early-phase adaptations that are followed by muscle hypertrophy (Häkkinen 1985, 1994). Naturally, one would expect maximal strength training to improve maximal strength, but explosive strength training can also cause some initial gains in maximal strength (Häkkinen et al. 1985). The improvements in muscle activation that were observed in MAX during dynamic 1RM and in EXP during CMJ may be indicators of these early-phase adaptations specific to the respective maximal and explosive strength training modes. It is worth noting that other muscles/groups of muscles may have influenced strength and power development and possibly contributed to the development of endurance performance parameters measured in this study. Muscle hypertrophy was not taken into account in this study, but our previous study (e.g., Taipale et al. 2010) indicates that small, but significant gains in the cross-sectional area of the trained muscles can be achieved even when maximal strength training is combined with endurance training.

It was interesting to observe that maximal isometric strength, RFD and muscle activation of the trained muscles during maximal isometric force production did not change in any of the groups. Percent increases in isometric strength are typically smaller than those observed in dynamic strength. Thus given that the percent increases in maximal dynamic strength in the present study were markedly smaller than those in studies involving strength training alone, we suspect that the isometric leg press measurement

was not sensitive enough to measure the smaller changes in force production induced by combined training. This seems to be true especially in the case of maximal isometric force, since Paavolainen et al. (1991, 1999) found that explosive strength training in endurance athletes positively influenced measures of performance including RFD (Paavolainen et al. 1991), but maximal isometric force did not change significantly. Many other studies examining combined strength and endurance training in non-athlete populations have used cycling as their endurance training method and seem to show greater increases in both dynamic and isometric strength of the lower extremities than the present study (e.g., Karavirta et al. 2009, Häkkinen et al. 2003). The greater increase in dynamic and isometric strength likely results, in part, from the differences in force production between the two training modes of running and bicycling, e.g., leg extensors produce a large amount of force during the concentric phase in cycling, whereas running is characterized by a rapid and repetitive stretch-shortening cycle action. It should also be noted that in cycling, the quadriceps femoris and gluteus muscles play a more significant role than in running where the biceps femoris also does significant work (Kyröläinen et al. 2001).

Countermovement jump height increased in MAX, EXP and MIX, but not in CON. The improvement in jumping height was particularly noticeable in MIX and occurred primarily during the final 4 weeks of the strength training intervention. It has previously been reported that maximal strength must be adequate for improvements in explosive power to occur (Newton and Kraemer 1994). Thus, it is possible that the combination of maximal strength training and explosive power training helped to develop maximal strength to a level that subsequently "allowed" for gains in explosive power, whereas maximal strength training and explosive power training alone did not provide enough training "diversity".

The lack of specific differences in the development of neuromuscular characteristics in the present study may be related to the fact that our subjects were untrained in terms of strength prior to this study. Cormie et al. (2010) found that improvements in athletic performance were similar over 10 weeks of training in untrained men performing either strength or ballistic power training, though it was noted that there were some differences in the mechanisms driving improvements. This appears to be in line with our findings as the magnitude of improvements in 1RM and CMJ are similar despite the different strength training regimens performed. Muscle activation during the 8-week training period improved significantly in MAX during dynamic 1RM, while muscle activation in EXP improved during CMJ which may suggest some specificity of training. Although no significant improvements in muscle

activation were observed in MIX, the improvement in activation of other leg muscles cannot be entirely ruled out.

Only minor (n.s.) changes were observed in  $\dot{V}O_{2max}$  of MAX, EXP and MIX, while a significant increase in  $\dot{V}O_{2max}$  was observed in CON. Large changes in  $\dot{V}O_{2max}$  were not expected in any group during this 8-week study period, as endurance training volume was relatively low and training intensity was primarily below LT. While intermittent high-intensity endurance training (such as interval training) can increase  $\dot{V}O_{2max}$  when performed three times a week over 8 weeks, low-intensity endurance training does not significantly influence endurance performance in terms of  $\dot{V}O_{2max}$  (Helgerud et al. 2007). The significant improvement observed in  $\dot{V}O_{2max}$  of CON may be attributed to circuit training, which essentially added an extra 1.5 training sessions per week of endurance training to the three to four times per week of endurance training. Changes in other endurance performance parameters such as  $S_{peak}$  and running economy, however, may be positively influenced by low-intensity endurance training (Billat and Koralsztein 1996). Although  $\dot{V}O_{2max}$  has long been reported to be the most important determinant of endurance performance, the value of  $S_{peak}$  combines  $\dot{V}O_{2max}$  and running economy into a single factor (Billat and Koralsztein 1996) which has been shown to predict performance in 10–90 km races (Noakes et al. 1990). The increase in  $S_{peak}$  and  $RCT_{speed}$  in all strength training groups suggests improved endurance performance despite the fact that there were no changes in  $\dot{V}O_{2max}$  or running economy during the 8-week training intervention. While endurance performance measured by  $S_{peak}$  and  $RCT_{speed}$  improved to a similar extent in all three strength training groups as well as the circuit training control group, we cannot exclude the possibility that a difference in adaptations might be observed in the weeks following the actual strength training intervention. Improved muscle strength and power may be beneficial in subsequent endurance training when athletes try to maximize their endurance performance characteristics. For example, when strength training volume is decreased and endurance volume is increased following 8 weeks of MAX or EXP training, we have previously found that MAX appears better prepared to tolerate the increased endurance volume and make use of neuromuscular adaptations to improve endurance performance including running economy and maximal running speed (Taipale et al. 2010).

Basal serum levels of CORT, TSH and DHEAS did not fluctuate significantly during the 8-week strength training intervention, while between weeks 0 and 8 serum TESTO decreased in MIX and serum SHBG decreased in EXP. The overall training stimuli in this investigation may not be "strong" enough to induce hormonal changes in all groups

in this relatively short period of time, though other studies from our laboratory have shown significant changes in the TESTO to CORT ratio over a more prolonged period of combined training (Taipale et al. 2010). The observed decreases in TESTO in MIX and SHBG in EXP may indicate some level of stress, overreaching or possibly interference. Untrained individuals may be more susceptible to training stress than trained individuals and this may be reflected in serum hormone concentrations. Previous research suggests that lesser improvements in strength in combined training may also be related to an elevated catabolic state which could influence, e.g., muscle hypertrophy (Kraemer et al. 1995; Bell et al. 2000).

In the present study, mixed maximal and explosive strength training, maximal strength training and explosive strength training were performed concurrently with endurance training over an 8-week training intervention and were "more effective" during this period than circuit training in increasing and maintaining maximal strength and explosive power. The three groups performing a low volume of strength training did not, however, differ from each other with regard to strength and power improvements while concurrently performing a higher volume of endurance training. While all groups made significant gains in the important performance measures of  $S_{peak}$  and  $RCT_{speed}$ , there may be additional benefits to increased maximal strength, power and muscle activation over a more prolonged period of time including, e.g., improved movement economy and  $S_{peak}$  (Taipale et al. 2010). Improvements in strength and power appear to be highly individual, thus performing the more diverse mixture of maximal and explosive strength training in a way that meets individual needs may be more effective than maximal or explosive strength training combined with endurance training alone.

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## IV

### **ACUTE HORMONAL AND FORCE RESPONSES TO COMBINED STRENGTH AND ENDURANCE LOADINGS IN MEN AND WOMEN: THE ORDER EFFECT**

by

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# Acute Hormonal and Force Responses to Combined Strength and Endurance Loadings in Men and Women: The “Order Effect”

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## Abstract

**Purpose:** To examine acute responses and recovery of serum hormones and muscle force following combined strength (S) and endurance (E) loading sessions in which the order of exercises is reversed (ES vs. SE).

**Methods:** This cross-over study design included recreationally endurance trained men and women (age 21–45 years, n = 12 men n = 10 women) who performed both loadings. Maximal bilateral isometric strength (MVC), isometric rate of force development (RFD) and serum concentrations of testosterone (T), cortisol (C), growth hormone (GH), insulin-like growth factor 1 (IGF-1), binding protein 3 (IGFBP3) and sex hormone binding globulin (SHBG) were measured during and after both loadings.

**Results:** Both of the present combined (ES and SE) loadings led to a greater acute decrease in MVC in men than in women, while RFD was slightly affected only in men. Recovery of MVC and RFD to baseline was complete at 24 h regardless of the order of exercises. In men, neuromuscular fatigue was accompanied by increased C concentrations observed post SE. This was followed by decreased concentrations of T at 24 h and 48 h that were significantly lower than those observed following ES. GH response in men also differed significantly post loadings. In women, only a significant difference in T between ES and SE loadings was observed at post.

**Conclusion:** These observed differences in hormonal responses despite similarities in neuromuscular fatigue in men indicate the presence of an order effect as the body was not fully recovered at 48 h following SE. These findings may be applicable in training prescription in order to optimize specific training adaptations.

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## Introduction

Hormones have an integral role in daily physiological function of humans. In large part, age, sex, and diurnal variations determine hormonal concentrations at rest, while physical and psychological stressors are known to stimulate unique responses. Physical stress, more specifically, exercise mode (strength training vs. endurance training), intensity [1,2] and duration [3,4] influence hormonal concentrations that initiate various physiological cascades that may contribute to muscle hypertrophy [5], increased capillary density [6] and initiation of mitochondrial biogenesis [7]. Rest/recovery after exercise [5] also plays a considerable role in maintaining homeostasis, while training status may augment or attenuate specific acute responses [1].

Testosterone is an important anabolic steroid that may increase remarkably as an acute response to both endurance [3] and heavy resistance exercise [5]. Endurance exercise [8] and submaximal or explosive type resistance exercise do not always induce as great a response [2], while endurance exercise of long duration (e.g. 2 hours) typically decreases testosterone levels, while cortisol levels

tend to increase [3]. Cortisol is a catabolic steroid that, in high concentrations, has been reported to interfere with the anabolic processes that promote muscle hypertrophy, which may in turn negatively affect strength development [9–11].

Growth hormone (GH) and insulin like growth factor-1 (IGF-1) work both synergistically and independently to stimulate many metabolic functions [12,13]. Growth hormone (GH) is considered to be an anabolic “family of related polypeptides” [14] that stimulates protein synthesis, cell reproduction and renewal. IGF-1 is positively associated with parameters of good health including cardiovascular fitness and body composition [15]. GH concentrations generally increase in response to both strength and endurance exercise thereby stimulating IGF-1 production.

Serum hormone concentrations are influenced by the availability of hormone transporters and receptors. Sex hormone binding globulin (SHBG) has a specific “high-affinity” binding site for transporting sex hormones like testosterone while IGF-1 binding protein 3 (IGFBP3) transports and helps to control the distribution of the majority of IGF-1 in circulation [16]. Binding of hormones

to proteins/globulins allows for an extended period of transport throughout the body, thus increasing the potential for action.

It is well established that both strength and endurance exercise can cause acute fatigue resulting in reduced strength and power of loaded muscles. Both maximal and explosive type strength exercises are able to induce acute fatigue; however, the mechanisms behind this fatigue may differ [17]. An endurance training session does not cause as much fatigue as a strength training session due to the differences in force production. Typical strength exercises require high levels of muscle activation, while endurance training demands lower levels of repetitive force production and even maximal uphill running cannot evoke maximal muscle activation [18].

The benefits of strength training for endurance performance has been well documented in recent years (e.g. [19–21]), though interference has been observed under certain conditions [22]. Studies on the acute responses to combined strength and endurance exercise and the specific role of order are limited. Endurance exercise followed by strength exercise has been shown to influence the magnitude of the testosterone, but not the cortisol response in strength trained men [23]; however, the acute hormone responses to strength and endurance training in a combined session and changes in concentrations during the subsequent recovery have not yet been illuminated. It may be hypothesized that the order of combined exercise will induce fatigue that is specific and may differ in magnitude affecting the subsequent exercise session in terms of training effectiveness [24].

The purpose of this study was to examine the acute exercise-induced serum hormone and neuromuscular responses and the time course of changes during recovery (24 and 48 hours post loading) following a loading of combined strength and endurance training session in recreationally endurance trained male and female runners. One loading session started with endurance exercise, which was immediately followed by strength exercise (ES) while the other loading session started with strength exercise, which was immediately followed by endurance exercise (SE). The possibility of an "order effect" on these responses was examined using this cross-sectional design.

## Methods

### Subjects

Subjects with a recreational endurance running background (age 21–45 years,  $n = 12$  men  $n = 10$  women) were recruited to participate in this study (Table 1). The target group included healthy men and women, exclusion criteria included: body mass index  $>28 \text{ kg} \cdot \text{m}^{-2}$ , illness, disease, injury or use of medications that would contraindicate participation in the study.

**Table 1. Subject Characteristics.**

|                    |             | Men (n = 12) | Women (n = 10) |
|--------------------|-------------|--------------|----------------|
| Height             | (cm)        | 177.4 ± 6.4  | 165.9 ± 7.6    |
| Weight             | (kg)        | 75.7 ± 3.6   | 59.8 ± 5.1     |
| Fat                | (%)         | 12.9 ± 3.6   | 22.0 ± 3.8     |
| BMI                |             | 24.1 ± 1.3   | 21.7 ± 1.8     |
| VO <sub>2max</sub> | (l/min)     | 4.1 ± 0.3    | 2.9 ± 0.4      |
|                    | (ml/kg/min) | 54.5 ± 4.0   | 48.5 ± 4.6     |

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### Ethics Statement

Ethical approval of methodology and consent procedures were granted by the University of Jyväskylä Ethical Committee and the study was conducted according to the provisions of the most recent Declaration of Helsinki. Subjects received written and oral information about the study design and measurement procedures. The possible risks and benefits of participation in the study were thoroughly explained prior to signing two copies of an informed consent document (one for the subjects' records and the other for our records). A resting electrocardiogram and health history questionnaires filled out by each subject were reviewed and approved by a physician prior to participation in the study.

Prior to the specific loading sessions, subjects completed a set of tests to familiarize themselves with the strength training equipment and to determine appropriated loads/intensities for the specific loadings. Measurements included: maximal isometric bilateral leg extension force and rate of force development as well as maximal oxygen uptake (VO<sub>2max</sub>). Body composition and baseline resting blood samples for serum hormones were also measured. Following completion of these baseline tests, subjects performed, in random order, two loadings of strength and endurance exercise (ES or SE) which were completed in a single session. Approximately one to three weeks separated pre-testing and loadings. Time of day variations in force and hormonal variables were controlled for by making sure that each subject performed loadings  $\pm 1$  hour from their pre-testing time and by taking blood samples in the morning (in a rested and fasted state) on each testing day.

**The strength loading.** focused primarily on the leg extensors and included both maximal and explosive strength exercises. Loads of 70–85% of 1RM were used for maximal strength exercises that included three sets of 5–8 repetitions. The final repetition of each set was near failure. Explosive strength exercises included three sets of 8–10 repetitions with maximal velocity of each repetition using 30–40% 1RM load. Exercises included: maximal bilateral leg press (3 sets maximal and 3 sets explosive), squat (3 sets maximal), loaded squat jump (3 sets explosive), and calf raises (2 sets maximal). Strength exercises were performed in a circuit such that leg press exercises were at the beginning, middle and end of the training session. There was 2 minutes of rest between sets. The total duration of strength training session was approximately 45 minutes.

**The endurance loading.** consisted of running on a 200 m indoor track. The intensity of running was at a steady-state at between each subject's previously determined individual lactate threshold (LT) and respiratory compensation threshold (RCT) for 60 minutes.

### Body Composition

In addition to standing height, body mass and body composition were measured using bioimpedance (InBody720 body composition analyzer, Biospace Co. Ltd, Seoul, South Korea). Measurements were always taken in conjunction with morning blood tests between 07.30–08.00 and subjects always arrived for testing in a fasted state, thus helping to keep the possible confounding variables of diet and hydration status to a minimum. Subjects were measured in their undergarments.

### Aerobic Capacity

Endurance performance characteristics were measured using a treadmill running protocol [25]. The running velocity began at  $8 \text{ km} \cdot \text{h}^{-1}$  and was increased by  $1 \text{ km} \cdot \text{h}^{-1}$  every third minute until volitional exhaustion. Treadmill incline remained a constant  $0.5^\circ$  degrees throughout the test. Heart rate was recorded continuously using a heart rate monitor (Suunto t6, Vantaa, Finland). Mean

heart rate values from the last minute of each stage were used for analysis. Oxygen consumption was measured breath-by-breath throughout the test using a portable gas analyzer (Oxycon Mobile®, Jaeger, Hoechberg, Germany). Maximal oxygen uptake ( $\dot{V}O_{2max}$ ) was determined to the highest average 60 s  $\dot{V}O_2$  value. Other factors such as a heart rate,  $\dot{V}O_2$ , and respiratory exchange ratio were monitored for determination of maximal effort. Fingertip blood samples were taken every 3rd minute to measure blood lactate concentrations. For blood sampling, the treadmill was stopped for approximately 15–20 seconds. Blood lactates were analyzed using a Biosen S\_line Lab+ lactate analyzer (EKF Diagnostic, Magdeburg, Germany). Lactate threshold (LT) and respiratory compensation threshold (RCT) were determined using blood lactate, ventilation,  $\dot{V}O_2$  and  $\dot{V}CO_2$  (production of carbon dioxide) according to [26].

#### Strength Measurements, Isometric Leg Press

An electromechanical isometric leg extension device (horizontal leg press, designed and manufactured by the Department of Biology of Physical Activity, University of Jyväskylä, Finland) was used to measure maximal bilateral strength and rate of force development (RFD). The subjects' knee angle was 107° measured using the greater trochanter, lateral tibiofemoral joint space and lateral malleolus as reference points, while the hip angle was 110°. Full extension of the leg was measured as 180°. Subjects were instructed to produce force "as fast and as hard as possible" for approximately 3 s. Force data was collected at a sampling frequency of 2000 Hz, and then filtered (20 Hz low pass filter). RFD was assessed over 20 ms ( $\pm 10$  s from maximal rate of force development). Force data was analyzed using customized scripts (Signal 4.04, CED, UK). Subjects performed at least three maximum voluntary contractions [27]. If the maximum force during the last trial was greater than 5% compared to the previous trial, and additional trial was performed. The best performance trial, in terms of maximal force measured in newtons (N), was used for statistical analysis. The reliability of these measurement techniques has been previously reported [28]. The intra-class coefficient of variation for strength measurements for the present study was 5.1% with an intra-class correlation of 0.98.

#### Blood Samples and Serum Hormones

Venous blood samples (10 ml) were collected after 12 hours of fasting between 07.30–08.00. Blood samples were collected using sterile needles into serum tubes (Venusafe, Terumo Medical Co., Leuven, Belgium) by a qualified lab technician who reviewed analyses of the basic blood count (Sysmex KX-21N, Kobe, Japan) to check for abnormalities prior to testing. Whole blood was centrifuged at 2500 g (Megafuge 1.0R, Heraeus, Germany) for 10 min after which serum was removed and stored at  $-80^\circ\text{C}$  until analysis. Blood samples were used for determination of serum testosterone, cortisol, growth hormone, insulin-like growth factor 1 and sex hormone binding globulin. Analyses were performed using chemical luminescence techniques (Immunlite 1000, DCP Diagnostics Corporation, Los Angeles, California, USA) and hormone specific immunoassay kits (Siemens, New York, NY, USA). The sensitivity of testosterone, cortisol, growth hormone, IGF-1, SHBG and IGF binding protein 3 assays were:  $0.5\text{ nmol}\cdot\text{l}^{-1}$ ,  $5.5\text{ nmol}\cdot\text{l}^{-1}$ ,  $0.026\text{ mIU}\cdot\text{l}^{-1}$ ,  $2.6\text{ nmol}\cdot\text{l}^{-1}$ ,  $5.5\text{ nmol}\cdot\text{l}^{-1}$ , and  $0.1\text{ }\mu\text{g}\cdot\text{ml}^{-1}$ , respectively. The intra-assay coefficients of variation for testosterone, cortisol, growth hormone, IGF-1, SHBG, and IGF-1 BP-3 were: 5.7, 4.6, 4.2, 3.1, 2.4, and 4.4% respectively.

#### Statistical Methods

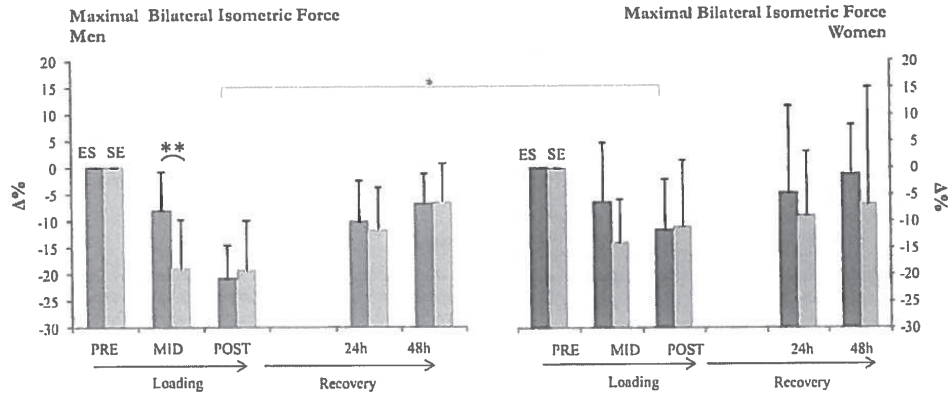
Standard statistical methods were used for calculation of means and standard deviation (SD). Group differences and group-by-loading interaction were analyzed by a repeated analysis using mixed models and an unstructured covariance matrix. Groups were compared with least significant difference (LSD) post hoc analysis in a mixed models analysis when appropriate. The criterion for significance was set at  $*=p=0.05$ ,  $**=p<0.01$  and  $***=p<0.001$ . Statistical analysis was completed with PASW Statistics 18 (SPSS Inc., Chicago, IL, USA).

#### Results

Both ES and SE loadings led to decreases in maximal and explosive strength. Absolute maximal bilateral isometric (MVC) strength in ES men decreased significantly following E ( $-8\pm 7\%$ ,  $p=0.002$  at MID) but not in women (Figure 1). After completion of both E and S, absolute strength decreased significantly in both men and women ( $-21\pm 7\%$ ,  $p<0.001$  and  $-12\pm 9\%$ ,  $p=0.007$ , respectively). In SE, absolute strength decreased significantly following S in both men and women ( $-19\pm 9\%$ ,  $p<0.001$  and  $-14\pm 8\%$ ,  $p=0.015$ ). After both S and E had been completed, absolute strength remained significantly reduced in men, while only a statistical trend was observed in women ( $-19\pm 9\%$ ,  $p<0.001$  and  $-12\pm 12\%$ ,  $p=0.074$ , respectively). The relative loading-induced decreases ( $\Delta\%$ ) in strength between ES and SE differed significantly at mid in men ( $p<0.001$ ) (Figure 1). The relative decreases ( $\Delta\%$ ) in strength in men and women were similar at MID; however, at post, a significant difference between ES men and ES women was observed. The absolute rate of force development (RFD) decreased during both loadings in ES and SE men (at post  $-19\pm 18\%$ ,  $p<0.001$  and  $-33\pm 28\%$ ,  $p=0.003$ , respectively, Figure 2). In women, RFD did not decrease significantly (at post  $-1\pm 19\%$  in ES and  $-22\pm 34\%$  in SE). The relative RFD was statistically different between men and women at post in ES. While RFD had returned to baseline in both men and women following ES and SE, MVC remained significantly decreased in SE and ES of men at 24 h ( $p<0.01$ ,  $-14\%$  and  $p<0.001$ ,  $-15\%$ , respectively) and in ES of men at 48 h ( $p<0.01$ ,  $-11\%$ ). No differences between loadings were observed.

The relative change in testosterone concentrations of men from pre were similar at mid and post of ES and SE, while at 24 h and 48 h of recovery the relative change from pre in SE was significantly lower than the relative change from pre in ES ( $p<0.05$  at 24 h and 48 h, Figure 3). The relative changes in serum testosterone concentrations in women were similar from pre to mid, but the change in SE was significantly greater than in ES at post ( $p<0.05$ ). At 24 h and 48 h of recovery in women, the relative changes from pre in both ES and SE were similar. Significant differences in the relative responses of testosterone between men and women were observed; testosterone increased more significantly in women in comparison to men at post SE ( $p<0.05$ ). ES men had significantly lowered testosterone in comparison to women ( $p<0.05$ ) at 24 h of recovery. Significant differences in absolute serum testosterone concentrations were also observed between ES and SE in men at 24 and 48 h.

The relative cortisol response from pre in men was significantly different between ES and SE at post ( $p<0.05$ ), but did not differ at mid (Figure 4). In women, no significant differences in changes were observed between loadings. The magnitude of cortisol response between men and women was significantly different at both mid and post in both ES and SE ( $p<0.05$ ). A significant



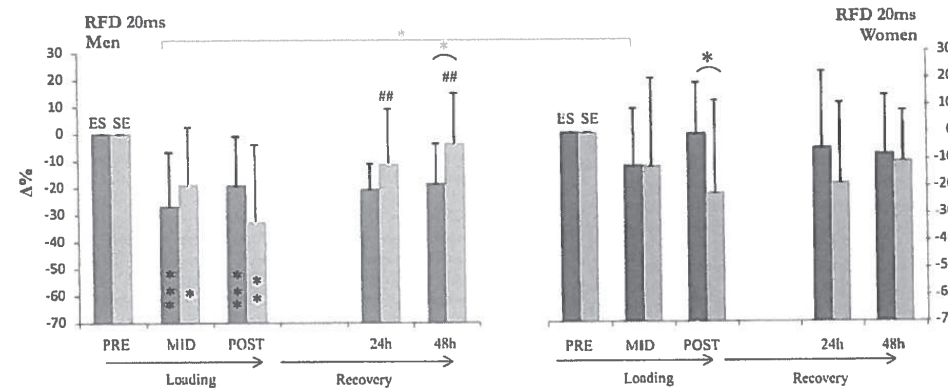
**Figure 1. Maximal isometric force.** \*\*, \*\*\* inside column = significant change ( $p < 0.01$ ,  $p < 0.001$ ) in absolute maximal isometric force from pre, # = significant change ( $p < 0.001$ ) in absolute maximal isometric force from mid. \* =  $p < 0.01$  significant differences between relative changes from pre in ES and SE loadings. doi:10.1371/journal.pone.0055051.g001

difference in absolute serum cortisol of men was observed between ES and SE at post ( $p < 0.05$ ).

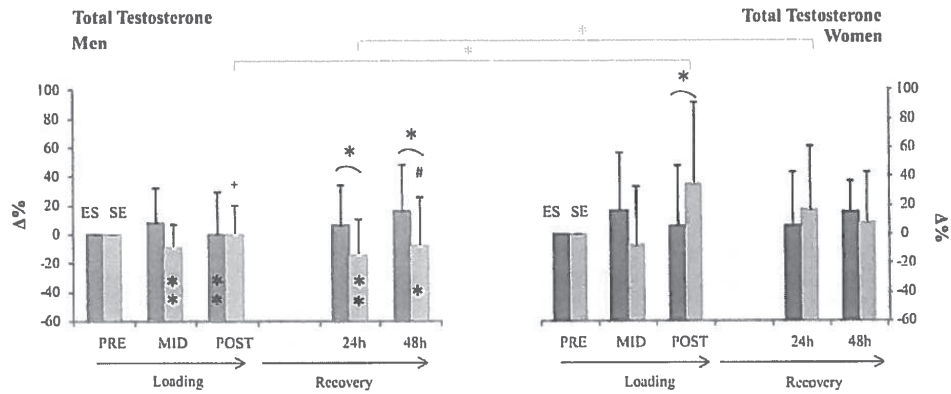
In men, the magnitude of growth hormone increase observed between pre and post was significantly greater following SE than ES ( $p < 0.001$ , Figure 5) while the magnitude of increase in GH from pre to mid of both loadings was similar. GH at 24 and 48 h had returned to baseline. In women, GH concentrations remained statistically unaltered. Difference in the magnitude of growth hormone responses between men and women at mid and post in both ES and SE were significant ( $p < 0.05$ ), while the difference between both ES and SE responses in men was greater ( $p < 0.01$ ) than in women. A significant difference in men of absolute GH concentrations was observed between ES and SE at post ( $p < 0.01$ ).

No statistically significant differences in relative changes of insulin-like growth factor I concentrations were observed between men or women during or between ES and SE loadings (Figure 6).

A significant increase in absolute concentrations of insulin-like growth factor binding protein 3 was observed in ES men at mid ( $p < 0.001$ ) and post ( $p < 0.05$ ) loading (Table 2). A significant decrease back to baseline was observed in IGFBP3 from post to 24 hours. Following the SE loading in men, a significant decrease in IGFBP3 from post to 48 hours was observed ( $p < 0.05$ ). In ES women, a significant decrease in IGFBP3 was observed from mid to post ( $p < 0.001$ ) while in SE women, a significant decrease from the peak in IGFBP3 levels between post and 24 h was observed. No differences were observed between men and women in absolute IGFBP3 response; however, the magnitude of change in



**Figure 2. Isometric rate of force development.** \*, \*\*, \*\*\* inside column = significant change ( $p < 0.05$ ,  $p < 0.01$ ,  $p < 0.001$ ) in absolute rate of force development from pre, ## = significant change ( $p < 0.01$ ) in absolute rate of force development from post. \* = significant difference ( $p < 0.05$ ) between changes in RFD from pre in men and women. \* = significant difference ( $p < 0.05$ ) between relative changes from pre in ES and SE loadings. doi:10.1371/journal.pone.0055051.g002



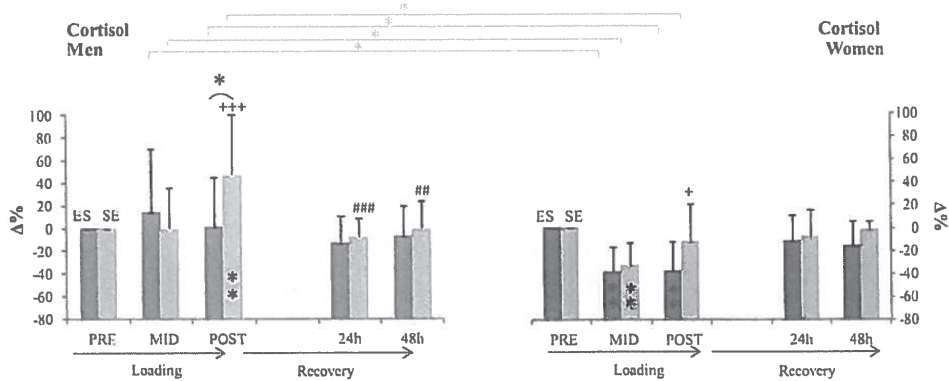
**Figure 3. Serum total testosterone.** \*\* (inside column=significant change ( $p < 0.01$ ) from pre in absolute serum total testosterone concentrations, + = significant change ( $p < 0.05$ ) in absolute serum total testosterone from mid. \* (=significant difference ( $p < 0.05$ ) between relative changes in serum testosterone concentrations from pre in men and women. # (=significant difference ( $p < 0.05$ ) between relative changes from pre in ES and SE loadings. doi:10.1371/journal.pone.0055051.g003

IGFBP3 was significantly different between ES men and women at post ( $p < 0.05$ ). Concentrations of serum SHBG remained statistically unaltered during the loading and recovery (Table 2).

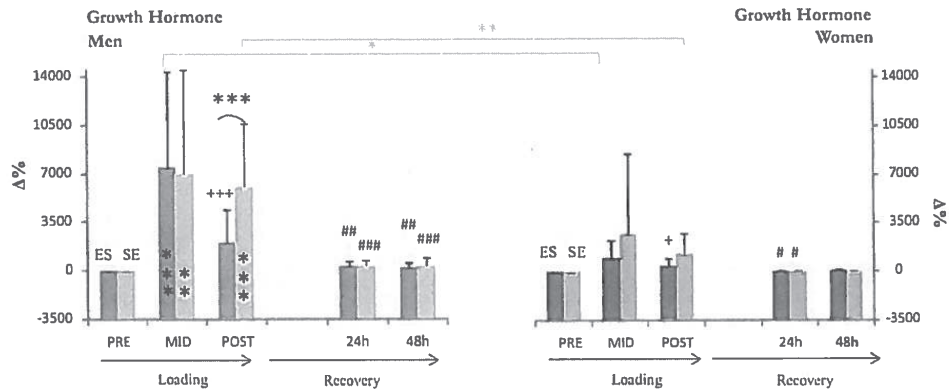
Small changes were observed in absolute serum concentrations of hormones measured in the morning over the 3-day intervention (Table 3). Serum testosterone differed between ES and SE at recovery of 24 h after loadings in men due to a significant decrease in morning serum testosterone observed in SE. At 48 hours, serum concentrations of testosterone in SE men had returned to baseline. No changes were observed in morning concentrations of testosterone in women. Serum cortisol remained unaltered in men, while a significant decrease was observed in ES of women from pre-AM to 48 h-AM ( $p < 0.01$ ). These decreases in cortisol

lead to a significant difference between loadings in ES and SE women.

No significant changes were observed between the loadings in GH of men or women. Insulin-like growth factor-1 concentrations did not significantly fluctuate, however a significant difference between concentrations of IGF-1 was observed between ES and SE men at 48 h ( $p < 0.01$ ). Morning IGFBP3 concentrations in men remained statistically unaltered throughout the experimental period in men, whereas a significant increase in IGFBP3 was observed in ES women between 24 h-AM and 48 h-AM. Sex hormone binding globulin concentrations remained statistically unaltered.



**Figure 4. Serum cortisol.** \*\* (inside column=significant change ( $p < 0.01$ ) from pre in absolute serum cortisol concentrations, +++ = significant change ( $p < 0.001$ ) in absolute serum cortisol from mid. \* (=significant difference ( $p < 0.05$ ) between relative changes in serum cortisol concentrations from pre in men and women, # (=  $p < 0.05$  significant differences between relative changes from pre in ES and SE loadings. doi:10.1371/journal.pone.0055051.g004

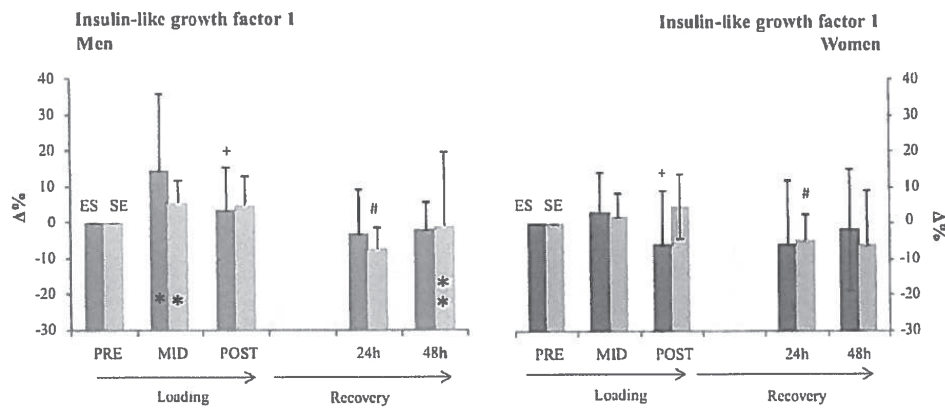


**Figure 5. Growth hormone.** \*\*\*\* inside column = significant change ( $p < 0.01$ ,  $p < 0.001$ ) from pre in absolute GH concentrations, +++ = significant change ( $p < 0.001$ ) in absolute GH from mid, ##, ### = significant change ( $p < 0.01$ ,  $p < 0.001$ ) in absolute GH from post, \*, \*\* (= significant difference ( $p < 0.05$ ,  $p < 0.01$ ) between changes in GH concentrations from pre in men and women, \*\*\* (=  $p < 0.001$  significant differences between relative changes from pre in ES and SE loadings. doi:10.1371/journal.pone.0055051.g005

**Discussion**

The order in which single-session combined strength and endurance training is performed, either endurance followed by strength (ES) or strength followed by endurance (SE), appears to affect hormonal and neuromuscular responses in recreationally endurance trained men and women. In men, serum cortisol (C) response was significantly greater in SE than ES at post loading while testosterone concentrations during the present loadings (pre-post) remained relatively stable. At 24 h and 48 h of recovery, however, serum testosterone (T) concentrations in SE men were significantly lower than at baseline while also being lower than those of ES men at 24 h and 48 h. Significant differences in serum

growth hormone (GH) responses of men were observed at post loading with concentrations remaining higher in SE despite the initial response to the first part of both ES and SE loadings being the same at mid. In women, a significant increase was observed in T at post SE whereas GH responses, though smaller, paralleled those of the men. Fatigue, as measured by maximal and explosive force, was the same in SE and ES post loading, but greater in men than in women. These responses suggest that performing strength training prior to endurance training is a more potent stimulus for hormonal responses, especially in men, than performing endurance training prior to strength training. The different responses to ES and SE loadings suggest that the order of exercises in single-session combined strength and endurance training should be



**Figure 6. Insulin like growth factor 1.** \* inside column = significant change ( $p < 0.05$ ) from pre in absolute GH concentrations, + = significant change ( $p < 0.05$ ) in absolute IGF-1 from mid, # = significant change ( $p < 0.05$ ) in absolute IGF-1 from post. doi:10.1371/journal.pone.0055051.g006

**Table 2.** Absolute concentrations of insulin-like growth factor 1 binding protein 3 and sex-hormone binding globulin.

| IGF BP 3 ( $\mu\text{g}\cdot\text{ml}^{-1}$ ) | PRE             | MID                          | POST                         | 24 h                       | 48 h                       |
|---|-----------------|------------------------------|------------------------------|----------------------------|----------------------------|
| Men ES  | 4.7 $\pm$ 1.2   | 5.1 $\pm$ 1.1 <sup>***</sup> | 5.1 $\pm$ 1.1 <sup>*</sup>   | 4.6 $\pm$ 0.8 <sup>#</sup> | 4.6 $\pm$ 0.7              |
| Men SE  | 4.7 $\pm$ 0.9   | 5.0 $\pm$ 1.01               | 5.1 $\pm$ 1.2                | 4.6 $\pm$ 0.8              | 4.6 $\pm$ 0.9 <sup>#</sup> |
| Women ES                                      | 4.9 $\pm$ 0.7   | 5.1 $\pm$ 0.5                | 4.7 $\pm$ 0.6 <sup>***</sup> | 4.7 $\pm$ 0.51             | 4.9 $\pm$ 0.4              |
| Women SE                                      | 4.8 $\pm$ 0.8   | 4.8 $\pm$ 0.7                | 5.1 $\pm$ 1.0                | 4.6 $\pm$ 0.6 <sup>#</sup> | 4.8 $\pm$ 0.5              |
| SHBG (nmol $\cdot$ l $^{-1}$ )                | PRE             | MID                          | POST                         | 24 h                       | 48 h                       |
| Men ES  | 35.9 $\pm$ 9.2  | 37.4 $\pm$ 9.1               | 37.2 $\pm$ 9.1               | 34.0 $\pm$ 8.5             | 35.7 $\pm$ 9.0             |
| Men SE  | 35.6 $\pm$ 7.7  | 37.1 $\pm$ 7.8               | 38.0 $\pm$ 9.1               | 34.9 $\pm$ 7.8             | 34.9 $\pm$ 8.8             |
| Women ES                                      | 73.9 $\pm$ 24.9 | 76.4 $\pm$ 28.9              | 73.5 $\pm$ 24.7              | 72.4 $\pm$ 27.1            | 74.2 $\pm$ 21.6            |
| Women SE                                      | 69.0 $\pm$ 16.8 | 72.2 $\pm$ 20.7              | 75.2 $\pm$ 17.9              | 69.7 $\pm$ 18.3            | 70.1 $\pm$ 11.2            |

Significant difference from pre = \*, \*\* (p<0.05, p<0.01). Significant difference from mid = \*\*\* (p<0.001). Significant difference from post = # (p<0.05).  
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considered even in recreational athletes because these responses may ultimately have physiological relevance in optimizing training.

The overall cumulative neuromuscular fatigue resulting from the present combined loadings was the same in SE and ES post loading, but fatigue was greater in men than in women. Both maximal isometric strength (MVC) and rate of force development (RFD) were significantly decreased at post, but appeared to recover at a similar rate following the loadings, despite remaining significantly decreased in men at 24 h and 48 h. Muscle activation was not examined in this study, thus it is unknown if, and to what extent, central fatigue along with the observed peripheral fatigue, may have been a factor. Nevertheless, based on previous research we can assume that maximal muscle activation decreased along with maximal strength [29]. While decreased force production is used as an indicator of fatigue, the time-course of these responses may be different from those observed in serum hormone concentrations that are used as biomarkers to assess physiological stress. In this investigation, primarily T, C, and GH seemed to be affected by order of exercise, whereas insulin-like growth factor 1 (IGF-1), insulin-like growth factor 1 binding protein 3 (IGF-BP3) and sex hormone binding globulin (SHBG) appeared to behave similarly regardless of the order of exercise.

An acute increase in T concentrations is a typical observation following a strength training session that has been linked to strength development and muscle growth after training (e.g. [5,30]). In the present study, the T response in men was similar from pre to post in ES and SE, while in women a difference in the magnitude of change in T concentrations was observed at post ES and SE with a greater increase occurring following SE. In men, the T responses in ES and SE differed significantly during recovery at 24 h and 48 h post loading with relative T concentrations being lower on both days in SE than in ES. In women, T concentrations were similar at 24 h and 48 h of recovery. It appears that while voluntary force returned to baseline, serum hormone concentrations continued to respond to the exercise stimulus. In some studies, T has fully recovered after 1–2 days of rest [31,32] whereas in other studies with very stressful resistance loading conditions, T levels remained decreased even after 2 days of rest [33]. This decreased T concentration may indicate that the hormone is being used for physiological processes or may be suggestive of the presence of a protein catabolic state [34], which may be undesirable should it persist for several days. It should be noted, however, that the decreased T observed in the present study

was not accompanied by significantly elevated cortisol during recovery. Serum C concentrations in men were similar at mid ES and SE loadings, but significantly different at post, with a greater concentration of C observed in SE. Like other studies, the changes in C concentrations paralleled those observed in GH concentrations [4]. In women, there were no differences between ES and SE in terms of C behavior, but C was observed to decrease during the loading, a finding similar to Copeland et al. 2002 [35] who examined separate strength and endurance loadings in women. Recovery of C at 24 h and 48 h was complete in both loadings in men and women. As C plays an important role in activation of cascades leading to mitochondrial biogenesis in skeletal muscle [7], the greater magnitude of increase in C observed in SE of men and similar increase in women could, if it occurs with repeated training, be of physiological significance.

Previous studies have indicated that the magnitude of GH response is greater in "more fatiguing" strength training sessions [36,37] and amplified with repeated exercise bouts of strength training [38]. This response is of interest because the initial GH response in this study following E and S at mid in men was the same, whereas GH concentrations decreased significantly when E was followed by S (ES) and remained elevated when S was followed by E (SE) at post. Strikingly similar responses were observed in GH response by Goto et al. 2005 [39] who examined the effects of a 60 minute low-intensity cycling protocol on a strength training session designed to induce pronounced hormonal responses [40]. In women GH responses were minimal, and recovery to baseline was complete in both men and women by 24 h. The reason behind the continued elevation in GH of men during the SE loading is difficult to explain, but may be linked to previous observations that GH concentrations begin to increase at the start of aerobic exercise with peak concentrations at or close to the end of aerobic exercise (e.g. [41]). The decrease in GH concentrations observed after mid in ES may be explained, in part, by the same phenomena because aerobic exercise was stopped and the overall intensity of the present strength loading was relatively low. It is important to remember that the present strength loading was made up of both maximal and explosive exercises, and included several periods of rest between each set of exercises. Lower volume strength and power loadings are known to induce a smaller hormone response than high volume loadings with short periods of rest [42]. As the GH variant examined in this study was the commonly examined and most abundant of GH variants (22 kDa variant), it is important to acknowledge that other GH



**Table 3.** Absolute morning concentrations of serum hormones.

|  | PRE - AM   | 24 H - AM               | 48 H - AM              |
|--|------------|-------------------------|------------------------|
| <b>Testosterone (nmol·l<sup>-1</sup>)</b>                                |            |                         |                        |
| Men ES   | 16.0±4.2   | 15.5±3.4 <sup>#</sup>   | 15.7±4.1               |
| Men SE   | 15.8±2.9   | 13.8±2.4 <sup>##</sup>  | 14.5±5.1               |
| Women ES   | 1.2±0.5    | 1.2±0.5                 | 1.2±0.5                |
| Women SE   | 1.1±0.4    | 1.0±0.2                 | 1.1±0.3                |
| <b>Cortisol (nmol·l<sup>-1</sup>)</b>                                    |            |                         |                        |
| Men ES   | 480±99     | 502±113                 | 485±109                |
| Men SE   | 505±121    | 479±83                  | 489±90                 |
| Women ES   | 478±70     | 453±89                  | 386±128 <sup>##</sup>  |
| Women SE   | 449±62     | 429±61                  | 477±64 <sup>#</sup>    |
| <b>Growth Hormone (mIU·l<sup>-1</sup>)</b>                               |            |                         |                        |
| Men ES   | 0.41±0.33  | 0.48±0.40               | 0.92±1.45              |
| Men SE   | 0.72±1.05  | 1.00±1.38               | 1.15±1.29 <sup>*</sup> |
| Women ES   | 8.84±13.21 | 3.04±6.55 <sup>##</sup> | 1.80±1.49              |
| Women SE   | 4.68±5.19  | 2.27±2.96 <sup>*</sup>  | 2.68±3.16              |
| <b>Insulin-like growth factor 1 (nmol·l<sup>-1</sup>)</b>                |            |                         |                        |
| Men ES   | 18.7±5.8   | 17.5±4.6                | 17.4±3.9 <sup>##</sup> |
| Men SE   | 20.0±4.7   | 20.2±6.5                | 21.9±6.5 <sup>##</sup> |
| Women ES   | 21.3±4.5   | 20.5±5.5                | 20.0±5.4               |
| Women SE   | 20.7±5.4   | 20.1±5.3                | 20.5±5.3               |
| <b>Insulin-like growth factor binding protein 3 (µg·ml<sup>-1</sup>)</b> |            |                         |                        |
| Men ES   | 4.9±1.7    | 4.6±0.8                 | 4.6±0.7                |
| Men SE   | 4.9±1.15   | 4.6±0.8                 | 4.6±0.9                |
| Women ES   | 4.9±0.5    | 4.7±0.5                 | 4.9±0.4 <sup>*</sup>   |
| Women SE   | 4.8±0.8    | 4.6±0.6                 | 4.8±0.5                |
| <b>Sex hormone binding globulin (nmol·l<sup>-1</sup>)</b>                |            |                         |                        |
| Men ES   | 34.7±10.1  | 34.0±8.5                | 35.7±9.0               |
| Men SE   | 35.1±8.1   | 34.9±7.8                | 34.9±8.8               |
| Women ES   | 72.9±26.4  | 72.4±27.1               | 74.2±21.6              |
| Women SE   | 69.1±16.1  | 69.7±18.3               | 70.11±11.2             |

Significant difference between loadings = # ( $p < 0.05$ ) and ## ( $p < 0.01$ ). Significant difference from Pre - AM measurements = \* ( $p < 0.05$ ) and \*\* ( $p < 0.01$ ). Significant difference from 24 h - AM measurements = + ( $p < 0.05$ ). Significant difference between men and women in T at all time-points, in GH at pre in SE  $p < 0.05$  and in SHBG at all time-points.

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variants may have responded differently and that it is necessary to exercise some caution when interpreting these results. It seems, however, reasonable to suggest that the extended period of GH elevation induced by SE may have physiological significance should it be present during repeated SE training sessions.

No differences were observed between ES and SE in IGF-1 responses and responses in men and women were not significantly different, these findings are parallel with Copeland et al. 2002 (women with 30 min recovery) [35]. IGF-1 and IGF-BP3 behaved similarly in ES and SE loadings and recovery in both men and women with a significant difference in magnitude of change observed only at post ES between men and women. SHBG was statistically unaltered during the loadings with no differences in responses observed between men and women. Our results parallel those of previous studies in which IGF-1 levels were not affected in the 2 days following the loading protocols despite an increase in GH in men [13].

The difference observed between the acute responses of GH and IGF-1 supports the idea that these hormones work independently in response to exercise stress [13]. As blood samples were only taken post loading and some of the hormones e.g. GH are secreted in a pulsatile manner, we were unable to determine if there were further responses in the hours immediately after a training session as has been observed in other studies (e.g. [36]). In previous studies, however, GH concentrations appear to stabilize 1-2 hours after fatiguing heavy-resistance protocols [33].

Concentrations of serum hormones measured in the morning also appeared to be affected by loadings. In men, a significant decrease in T from baseline was observed after SE at 24 h-AM. The difference between ES and SE concentrations at this time point was significant and similar to that observed when examining corresponding time of day hormonal concentrations. These parallel changes in morning and time of day concentrations of T indicate that the SE exercise stimulus produced a greater stress

response than ES and they correspond to findings that T may not fully return to resting values after training that induces physiological stress [33]. As testosterone levels typically increase overnight, this observation suggests that pituitary-testicular action was indeed suppressed. Following SE in women, a significant increase in morning C concentration was observed between 24 h-AM and 48 h-AM while a significant decrease in morning C concentration was observed in ES of women between 24 h-AM and 48 h-AM resulting in a significant difference between ES and SE, a finding that does not parallel time of day concentrations. This delayed C response could indicate that women did not recover completely from SE within the two days of rest examined.

The magnitudes of hormonal responses in women observed in the present study were somewhat smaller than those observed in men. This response was expected, as numerous studies have previously shown that hormonal responses in women are typically more subtle than in men [2,40,43,44]. When studying women it is natural to contemplate the potential influence of different phases of the menstrual cycle, however, it appears that e.g. pituitary-adrenal responses to short-term, moderate intensity exercise are not significantly influenced by the menstrual cycle phase [45]. When examining acute hormonal responses to exercise in both men and women, circadian rhythms should also be taken into account. Previous studies have shown a substantial decrease in e.g. testosterone concentrations in the morning between 04:00 and 08:00 and a more gradual decrease from 08:00 until 22:00 [46], while others have shown evidence that cortisol is affected by diurnal variations in both young men and women [40,47]. These diurnal variations, including nocturnal responses, may conceal some physiological responses as Häkkinen et al. [40] has postulated, on the other hand, e.g. the magnitude of growth hormone response to exercise appears to be independent of time of day [47]. In addition to diurnal variations, nutrition, overtraining/excessive fatigue and stress [5] as well as individual training status [48] can contribute to hormonal changes reported in studies. Taking this into consideration, the present study included a fairly homogenous group in terms of training status as well as specific instructions for subjects on how to prepare for each loading to minimize possible confounding factors. As the above factors have been taken into consideration, it may be suggested that the observed changes and differences in serum concentrations of hormones, though small, may have a dynamic role in subsequent physiological processes and training adaptations. In fact, there is some evidence that performing endurance training prior to strength training (ES) is more effective in improving endurance performance characteristics than SE training and E and S performed alone [49,50], whereas intrasession sequence of E and S training may not influence adaptations in muscle strength and power [51].

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## Conclusions

Both of the present combined (ES and SE) loadings led to more fatigue in men than in women in maximal bilateral isometric strength and isometric rate of force development was affected only in men, though to a smaller degree. In men, neuromuscular fatigue was accompanied by an increase in cortisol concentrations observed at post in SE, which was followed by decreased concentrations of testosterone in SE at 24 h and 48 h post loading. Despite the initial response to the first part of both ES and SE loadings being the same at mid, GH response in men was also observed to be different with concentrations remaining higher at post in SE. These observed differences in hormonal responses in men regardless of similarities in neuromuscular fatigue indicate the presence of an order effect. Acute hormonal responses in men in general were greater than in women. An order effect was also observed in women as a significant difference in testosterone between ES and SE loadings was observed at post, while no changes or order effect was observed during recovery. Although a great decrease in maximal strength was observed post loading in both SE and ES of men and women, there was no order effect observed in neuromuscular measures of either maximal or explosive strength. The present study does, however, demonstrate an order effect in hormone responses, particularly in T of men that indicated that the body may not have been fully recovered 24 h and 48 h following SE. The time-course of neuromuscular fatigue and recovery appears not to match the time-course of serum hormone responses to ES and SE loadings and an order effect appears to be present when combining strength and endurance training into a single session. These findings may be utilized in order to optimize training.

## Acknowledgments

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## Author Contributions

Conceived and designed the experiments: RT KH. Performed the experiments: RT KH. Analyzed the data: RT KH. Contributed reagents/materials/analysis tools: RT KH. Wrote the paper: RT KH.

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V

**ACUTE NEUROMUSCULAR AND METABOLIC RESPONSES  
TO COMBINED STRENGTH AND ENDURANCE LOADINGS:  
EXAMINATION OF THE “ORDER EFFECT”  
IN RECREATIONALLY ENDURANCE TRAINED RUNNERS**

by

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***Acute neuromuscular and metabolic responses to combined strength and endurance loadings:  
the “order effect” in recreationally endurance trained runners***

*Running title: The order effect in combined strength and endurance*

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**ABSTRACT**

The study examined the acute neuromuscular and metabolic responses and recovery (24 and 48 h) to combined strength and endurance sessions. Recreationally endurance trained men (n = 12) and women (n = 10) performed: endurance running followed immediately by a strength loading (ES) and the reverse order (SE). Maximal strength (MVC), countermovement jump height (CMJ), and creatine kinase activity (CK) were measured pre, mid, post loading and at 24 and 48 h of recovery. MVC and CMJ decreased ( $p < 0.05$ ) post both ES and SE sessions in men. Only MVC decreased in ES and SE women ( $p < 0.05$ ). During recovery no order differences in MVC were observed between sessions in men, but MVC and CMJ remained decreased. During recovery in women, a delayed decrease in CMJ was observed in ES but not SE ( $p < 0.01$ ), while MVC returned to baseline at 24 h. CK increased ( $p < 0.05$ ) during both ES and SE and peaked in all groups at 24 h. The present combined ES and SE sessions induced greater neuromuscular fatigue at post in men than in women. The delayed fatigue response in ES women may be an order effect related to muscle damage during recovery.

**Keywords:** combined strength and endurance, order effect, muscle damage, recreational runners

## INTRODUCTION

The recreational endurance athlete might perform both strength and endurance training in the same session in order to save time or to fulfil general exercise recommendations in our busy society. Previous studies have shown that maximal and explosive strength training are effective in improving neuromuscular characteristics of running performance such as running economy and maximal running speed (Mikkola et al. 2007, Taipale et al. 2010). Maximal strength training is characterized by use of high load (% 1RM) and low number of repetitions per set (Häkkinen, Komi 1983, Ahtiainen et al. 2003), while explosive strength training is characterized by use of lower loads (either %1RM or body weight only) and movements with high action velocity (Häkkinen, Komi & Alén 1985, Häkkinen, Komi 1985, Kyröläinen et al. 2005, Newton, Kraemer 1994). Both maximal and explosive strength training sessions can induce a significant amount of acute fatigue, however, the mechanisms (central or peripheral) behind fatigue may not be the same (Linnamo, Häkkinen & Komi 1998). Endurance exercise, such as running, also induces fatigue, however, the magnitude of fatigue is may be less than that induced by strength exercise due to fundamental differences in force production demands. A longer intensity and/or duration of running may nevertheless induce a significant amount of fatigue. In addition, the repetitive force production using the stretch-shortening cycle (SSC) that is required for running can be very demanding for the neuromuscular system (Komi 2000), although maximal muscle activation is not typically observed in running (Sloniger et al. 1997).

In general, fatigue results from either central factors including decreased recruitment of motor units and reduced firing frequency of active units (Gandevia 2001) or peripheral factors including lactic acid accumulation, decreased muscle pH, and decreased  $Ca^{2+}$  transport within the muscle that may contribute to the inhibition of muscle contractile characteristics (Sahlin 1992). Mechanical stress in the form of eccentric motions that may result in muscle damage is higher in running than cycling (Bell et al. 2000) and may be a contributing factor to fatigue and decreases in force-production capabilities. Decreased force production capabilities resulting from muscle damage may be particularly significant the days following a training session, in which the resulting muscle soreness may contribute to impaired force production and/or running capabilities (Komi 2000). Creatine kinase is an enzyme and indirect marker for muscle damage that is related to this delayed onset of muscle soreness (DOMS) (Cheung, Hume & Maxwell 2003), which has been shown to peak within 2 days following e.g. running a marathon (Kyröläinen et al. 2000).

While combining strength and endurance into a single session is relatively common, Nelson et al. (1990) suggested that strength training inhibits gains in aerobic development and Bell et al. (1998) found that strength gains may be inhibited. This inhibition in the development of either strength or endurance characteristics when strength and endurance training are performed concurrently is commonly referred to as the “interference effect” (Hickson 1980). This is not always present when, for example, a lower volume and intensity of strength and endurance training are combined e.g. (McCarthy, Pozniak & Agre 2002, Häkkinen et al. 2003). The “interference effect” has primarily been examined over various periods of training. It is, however, logical to reason that interference begins with the acute responses to combined training as might be deduced from the differences in intracellular signalling networks stimulated by each strength and endurance training (Hawley 2009, Nader 2006) and due to the previously mentioned fundamental differences in force production demanded by strength exercises and running. In addition to the differences in intracellular signalling networks, fatigue and muscle damage may play a role in the acute interference effect, while the order of the exercises performed may either attenuate or intensify subsequent responses.

The effect of the order of exercise in single session on various physiological responses has received some attention e.g. (Bell et al. 1988, Chtara et al. 2005, Chtara et al. 2008), however, in recreational endurance runners this phenomenon has been studied to a lesser degree. The purpose of this study was to examine the acute exercise-induced neuromuscular and metabolic responses and time-course of changes during recovery (24 and 48 hours post loading) following strength and endurance loadings combined into a single session in recreationally endurance trained runners. One session started with endurance loading that included running for 60 minutes on an indoor track at a moderate intensity, which was immediately followed by a strength loading that included a mixture of maximal and explosive exercises for the lower extremities (ES), while the other session started with the strength loading, which was immediately followed by the endurance loading (SE). Using this cross-sectional design, the possibility of an “order effect” on neuromuscular and metabolic responses was examined.

## **METHODS**

### **Subjects**

Recreationally endurance trained subjects (age 21-45 years, n = 12 men n = 10 women) were recruited to participate in this study (Table 1). Exclusion criteria included: body mass index > 28 kg · m<sup>-2</sup>, illness, disease, injury or use of medications that would contraindicate participation in the study.



Subjects received specific written and oral information about the study design and measurement procedures. The possible risks and benefits of participation in the study were explained prior to signing an informed consent document. Each subject had a resting electrocardiogram and health history questionnaire reviewed and approved by a qualified physician prior to participation in the study. Ethical approval was granted by the University Ethical Committee and the study was conducted according to the provisions of the most recent Declaration of Helsinki.

**\*\*Table 1 near here\*\***

Prior to the combined experimental sessions of ES and SE, subjects completed a set of tests to familiarize themselves with the strength training equipment and to determine appropriated loads/intensities for the specific loadings (this data is not reported). Measurements included: maximal bilateral isometric leg press force, rate of force development and maximal oxygen uptake ( $VO_{2max}$ ). Body composition was also measured. Following completion of these tests, subjects performed, in random order, two sessions of combined strength and endurance sessions (ES or SE). Time of day variations in force were controlled for by making sure that each subject performed sessions  $\pm 1$  hour from their pre-testing time. Maximal isometric force (MVC), average force 0-500 ms ( $Av_{500}$ ), countermovement jump height (CMJ) and creatine kinase activity (CK) were measured pre, mid and post each experimental session as well as during recovery at 24 and 48 hours post experimental session. Muscle activation during MVC was only measured at pre, mid and post for each experimental session. Blood lactates were measured 3 times during the strength loading and 4 times during the endurance loading.

***The strength loading*** focused primarily on the leg extensors and included both maximal and explosive strength exercises. Loads of 70-85% of 1RM were used for maximal strength exercises that included three sets of 5-8 repetitions. The final repetition of each set was near failure. Explosive strength exercises included three sets of 8-10 repetitions using 30-40% 1RM load. Exercises included: maximal bilateral leg press (3 sets maximal and 3 sets explosive), squat (3 sets maximal), loaded squat jump (3 sets explosive), and calf raises (2 sets maximal). Strength exercises were performed in a circuit such that leg press exercises were at the beginning, middle and end of the exercise session. There was 2 minutes of rest between sets. The total duration of strength exercise session was approximately 45 minutes.

*The endurance loading* included running on a 200m indoor track. The intensity of running was at a steady-state between each subject's previously determined individual lactate threshold (LT) and respiratory compensation threshold (RCT) for 60 minutes. The average intensity calculated from  $\dot{V}O_{2\max}$  measured on the treadmill and  $\dot{V}O_2$  measured during the first and last 10 minutes of the endurance loading was ~80% of  $\dot{V}O_{2\max}$ .

## MEASUREMENTS

### Body composition

In addition to standing height, body mass and body composition were measured using bioimpedance (InBody720 body composition analyser, Biospace Co. Ltd, Seoul, South Korea). Measurements were always taken in the morning between 07.30-08.00, and the subjects always arrived for testing in a fasted state, thus helping to keep the possible confounding variables of diet and hydration status to a minimum. Subjects were measured in their undergarments.

## PERFORMANCE MEASURES

### Aerobic capacity

Endurance performance characteristics were measured using a treadmill running protocol (Mikkola et al. 2007). Running velocity was 8 km·h<sup>-1</sup> for women and 9 km·h<sup>-1</sup> for men and was increased by 1 km·h<sup>-1</sup> every third minute until volitional exhaustion. Treadmill incline remained a constant 0.5 degrees throughout the test. Heart rate was recorded continuously using a heart rate monitor (Suunto t6, Vantaa, Finland). Mean heart rate values from the last minute of each stage were used for analysis. Oxygen consumption was measured breath-by-breath throughout the test using a portable gas analyzer (Oxycon Mobile®, Jaeger, Hoechberg, Germany). Maximal oxygen uptake ( $\dot{V}O_{2\max}$ ) was calculated from the highest average 60 s  $\dot{V}O_2$ . Other factors such as a heart rate,  $\dot{V}O_2$ , respiratory exchange ratio and volitional fatigue were monitored for determination of maximal effort. Fingertip blood samples were taken every 3rd minute to measure blood lactate concentrations. For blood sampling, the treadmill was stopped for approximately 15-20 seconds. Blood lactates were analysed using a Biosen S\_line Lab+ lactate analyser (EKF Diagnostic, Magdeburg, Germany). Lactate threshold (LT) and respiratory compensation threshold (RCT) were determined using blood lactate, ventilation,  $\dot{V}O_2$  and  $\dot{V}CO_2$  (production of carbon dioxide) according to (Meyer et al. 2005).

**Strength and power measurements:****Isometric leg press**

An electromechanical isometric leg press extension device (designed and manufactured by the Department of Biology of Physical Activity, University of Jyväskylä, Finland) was used to measure maximal bilateral isometric strength (MVC) and average force over the first 500 ms of the MVC in a horizontal leg press position. The subjects' knee angle was 107° while the hip angle was 110°. Subjects were instructed to produce force "as fast and as hard as possible" for approximately 3 seconds. Subjects performed at least three maximum voluntary contractions (Häkkinen et al. 1998). If the maximum force during the last trial differed more than 5% from the previous trial, and additional trial was performed. The best performance measured in Newtons (N), was used for statistical analysis. During the experimental sessions, maximal force was measured only two times with a 15 second interval of rest to record acute fatigue.

**Countermovement jump**

A force platform (Department of Biology of Physical Activity, Jyväskylä, Finland) was used to measure maximal dynamic explosive force by countermovement jump (CMJ) height (Komi, Bosco 1978). Subjects were instructed to stand with their feet approximately hip-width apart with their hands on their hips. Subjects were then instructed to perform a quick and explosive countermovement jump on verbal command so that knee angle for the jump was no less than 90 degrees. Force data was collected and analysed by computer software (Signal 2.14, CED, Cambridge, UK), which used the equation  $h = I^2 \cdot 2gm^{-2}$  to calculate jump height from impulse ( $I$  = impulse,  $g$  = gravity and  $m$  = mass of subject).

**Electromyographic activity**

Electromyographic activity (EMG) was recorded from the vastus medialis (VM) muscles of the right leg during maximal isometric leg extension and CMJ. Electrode positions were marked with small ink tattoos (Häkkinen, Komi 1983) on the skin during the first testing session to ensure that electrode placement over the entire experimental period would be consistent. The guidelines published by Surface ElectroMyoGraphy for the Non-Invasive Assessment of Muscles were followed for skin preparation, electrode placement and orientation. Inter-electrode distance was 20 mm (input impedance < 10 k $\Omega$ , common mode rejection ratio 80 dB, 1000 gain). Raw signals were passed from a transmitter, positioned around the subjects' waist, to a receiver (Telemetry 2400R, Noraxon, Scottsdale, AZ, USA)

from which the signal was relayed to the computer via an AD converter (Micro1401, CED, Cambridge, UK).

### **Blood lactate and creatine kinase**

Blood lactates were measured during both strength and endurance loadings and were analysed using a Biosen S\_line Lab+ lactate analyser (EKF Diagnostic, Magdeburg, Germany) with a sensitivity of  $0.5 \text{ mmol} \cdot \text{L}^{-1}$  and serum creatine kinase activity was analysed using a Konelab 20XTi (Thermo Fisher Scientific Oy, Finland) with a sensitivity of  $15 \text{ U} \cdot \text{L}^{-1}$ .

### **Statistical methods**

Standard statistical methods were used for calculation of means and standard deviation (SD). Group differences and group-by-loading interaction were analysed by a repeated analysis using mixed models and an unstructured covariance matrix. Groups were compared with least significant difference (LSD) post hoc analysis in a mixed models analysis when appropriate. The criterion for significance was set at  $* = p = 0.05$ ,  $** = p < 0.01$  and  $*** = p < 0.001$ . Statistical analysis was completed with PASW Statistics 18 (SPSS Inc., Chicago, IL, USA).

## **RESULTS**

Maximal bilateral isometric force decreased significantly by 8% ( $p < 0.01$ ) in men following E of ES and 19% ( $p < 0.001$ ) following S of SE creating a significant difference in the decrease in maximal force between ES and SE at mid ( $p < 0.01$ ). At post, the cumulative decrease in force was 21% ( $p < 0.001$ ) and 19% ( $p < 0.001$ ) in ES and SE, respectively (Figure 1). Force production in men began to return to baseline following both sessions for men during recovery at 24 hours and 48 hours, however, maximal force returned to baseline only in SE at 48 hours. Nevertheless, no significant differences between sessions were observed during recovery. Decreases in force production in women were somewhat more modest with a significant decrease in maximal force production observed at mid only following S of SE 12% ( $p < 0.01$ ). At post, force had decreased by 6% ( $p < 0.01$ ) and 9% ( $p < 0.05$ ) in ES and SE, respectively. Force production in women returned to baseline at 24 hours of recovery. No significant group differences were observed between the ES and SE sessions during recovery.

**\*\*Figure 1 near here\*\***

Muscle activation of VM during maximal bilateral isometric force production was only measured during ES and SE sessions at pre, mid and post. Muscle activation of VM decreased by 25% ( $p < 0.05$ ) in SE of men at post and 12% (n.s.) in ES at post (Figure 1). A difference between ES and SE sessions was observed in women at post ( $p < 0.05$ ), but no significant changes in muscle activation of VM were observed.

A decrease in the average force from 0-500 ms ( $Av_{500}$ ) during maximal bilateral isometric leg press was progressive and significant in ES and SE men (20% and 26%,  $p < 0.001$ ) at post (Table 2). At 24 hours of recovery  $Av_{500}$  had returned to baseline in ES, but was significantly below baseline in SE ( $p < 0.001$ ). At 48 hours of recovery,  $Av_{500}$  had returned to baseline in SE, but not ES ( $p < 0.01$ ). In women,  $Av_{500}$  did not change significantly in response to the ES or SE sessions until recovery at 24 hours when  $Av_{500}$  was significantly decreased from baseline in ES ( $p < 0.05$ ). No differences were observed between the sessions.

**\*\*Table 1 near here\*\***

Countermovement jump height decreased significantly from baseline ( $32.3 \pm 4.8$  cm) in men at mid of SE, but not ES (Figure 2). The cumulative decrease in jumping height following the ES and SE loadings, however, was similar by 5% ( $p < 0.05$ ) and 7% ( $p < 0.01$ ) in ES and SE, respectively. Jumping height remained significantly decreased in ES and SE men at 24 hours of recovery ( $p < 0.01$  and  $p < 0.001$ , respectively), whereas jumping height returned to baseline in ES at 48 hours but not in SE ( $p < 0.001$ ). No differences between sessions were observed. During both the ES and SE sessions in women, no changes were observed in jumping height from baseline ( $24.0 \pm 4.2$  cm), but at 24 hours of recovery, jumping height in ES was significantly reduced (9%,  $p < 0.01$ ). A significant difference in jumping height was observed at 24 hours between ES and SE ( $p < 0.05$ ) that was maintained at 48 hours of recovery despite the statistical return to baseline in jumping height.

**\*\*Figure 2 near here\*\***

Blood lactate during both strength and endurance loadings were primarily below  $4 \text{ mmol} \cdot \text{L}^{-1}$  (Figure 3). Significant increases in blood lactate were observed from the beginning of each loading. Differences between sessions ( $p < 0.05$ ) were observed in men at the beginning of the strength loading

(PreS) and at the end of the endurance loading (PostE). No differences between sessions were observed in women.

**\*\*Figure 3 near here\*\***

Creatine kinase activity increased significantly in men ( $p < 0.001$ , Figure 4) from resting values (241 and 187  $U \cdot L^{-1}$  in ES and SE, respectively) immediately following the onset of both strength (23%) and endurance (35%) loadings and remained elevated at post (45% and 65% in ES and SE). During recovery, CK activity in men remained above resting levels at 24 hours (705 and 525  $U \cdot L^{-1}$  in ES,  $p < 0.01$  and SE,  $p < 0.05$ ) 48 hours (456 and 384 in ES,  $p < 0.05$  and SE,  $p < 0.05$ ). In women, CK increased significantly from resting values (97 and 169  $U \cdot L^{-1}$  in ES and SE) slightly later than in men following the combined sessions at post ( $p < 0.01$ , 55% and  $p < 0.05$ , 51% in ES and SE). During recovery, CK remained elevated (271 and 255  $U \cdot L^{-1}$  at 24 h and 198 and 128  $U \cdot L^{-1}$  at 48h in ES and SE), however, this elevation in CK was not statistically significant.

**\*\*Figure 4 near here\*\***

## DISCUSSION

The present strength and endurance loadings, when performed in a single combined session, appeared to induce a similar amount of accumulated neuromuscular fatigue post session regardless of exercise order. Maximal and explosive strength were greatly decreased after the combined ES and SE sessions at post in men, while only maximal force decreased in women. In general, a greater amount of fatigue in terms of decreased maximal and explosive force production capabilities was observed in men than in women. No differences in force production capabilities were observed between combined sessions during 24 and 48 hours of recovery in men, but maximal and explosive force production characteristics remained decreased. In women, maximal strength returned to baseline already at 24 hours of recovery, while a delayed decrease in explosive force production capabilities was observed in the ES women at 24 and 48 hours of recovery. Both combined sessions induced significant increases in blood lactate, although concentrations remained primarily under 4  $mmol \cdot L^{-1}$ . Creatine kinase (CK) activity increased significantly during the both combined sessions and peaked well above reference range in both combined sessions in men and women at 24 hours of recovery.

Maximal and explosive force production capabilities decreased as an acute result of the present combined strength and endurance training sessions. Interestingly, recovery was not complete at 24 and 48 hours in men and, furthermore, a delayed fatigue response was observed in countermovement jump height of the ES women. As might be expected because of the fundamental differences in force-production demands between strength and endurance loadings, a somewhat greater amount of neuromuscular fatigue was produced following the strength loading than the endurance loading. This difference, however, was only statistically significant in maximal force production of men at mid. Accumulated fatigue at post was similar between sessions for both men and women. In agreement with previous studies, women appeared to be less fatigable than men (Linnamo, Häkkinen & Komi 1998, Bosco et al. 2000, Bosco et al. 2000, Häkkinen, Pakarinen 1995), although in the present study the precise mechanism explaining the difference in fatigability between men and women remains somewhat unclear.

The present study used surface electromyography during the experimental sessions to measure changes in muscle activation. The decrease in muscle activation of VM in men was observed to parallel decreases in maximal force production, although the decrease reached statistical significance only in SE. No significant changes in muscle activation were observed in women. The reason for maintained muscle activation in the ES men and both groups of women may be related to recruitment of higher threshold motor units or an increase in motor unit discharge rate (De Ruyter et al. 2005). The decrease that occurred concomitantly in maximal force production capabilities in the ES men and ES and SE women may then be attributed to mechanisms of peripheral fatigue. It is also possible that muscle activation was affected by the differences in movement patterns used during the combined sessions and testing. While the mixed maximal and explosive loading, as well as the endurance running were performed dynamically, our testing method for maximal force production and muscle activation was isometric. The differences observed muscle activation may be influenced, in part, the difference in modes used for the loadings and measurements (Blazevich et al. 2012).

Measuring muscle activation using surface electromyography is common and reliable, but gives somewhat limited information on the mechanisms behind the present muscular fatigue as it measures activity only at the level of the muscle (Millet et al. 2012). Had it been possible to use muscle stimulation and the superimposed twitch technique during maximum isometric contraction (Millet et al. 2012, Harridge, Kryger & Stensgaard 1999) in the present study design, we could have provided

further information regarding the mechanisms of fatigue that are demonstrated by significantly decreased forced production. Unfortunately, due to the volume of other variables measured in the present design, we were unable to use this technique (e.g. (Taipale, Häkkinen 2013)). Nevertheless, it is possible that central factors including decreased recruitment of motor units and reduced firing frequency of active motor units (Gandevia 2001) may have contributed to fatigue in the SE men, while peripheral mechanisms may have played a greater role in the ES men and both groups of women.

Concentrations of blood lactate were statistically elevated in both ES and SE sessions for both men and women. The concentration of blood lactate, however, only exceeded  $4 \text{ mmol} \cdot \text{L}^{-1}$  in the ES men during the strength loading. The increase in blood lactate is an indicator of exercise intensity and suggests that a brief shift in sources of energy metabolism may have occurred from aerobic to anaerobic. Blood lactate concentrations during the present strength loading were similar to those observed in the maximal strength loading of (Walker et al. 2012) in which central drive was determined to be the main contributor to fatigue. The levels of CK were significantly elevated in both men and women by the end of the combined loadings with peak concentrations observed at 24 hours of recovery. In contrast, after marathon running, CK has been shown to peak within 2 days (Kyröläinen et al. 2000). The eccentric component of running may play a marked role in muscle damage (Proske, Allen 2005). Absolute values of CK during recovery were above the reference values for CK in adult males ( $52\text{-}336 \text{ U} \cdot \text{L}^{-1}$ ) and females ( $38\text{-}176 \text{ U} \cdot \text{L}^{-1}$ ), although CK levels were not considered to be statistically elevated during recovery in women. The delayed fatigue response in explosive strength of women appears to be reflected, to some extent, in the CK response, although statistically significant differences between ES and SE were not observed. The standard deviations for CK in both men and women during recovery were quite substantial suggesting that the CK response to the present loading was largely individual.

Suppressed testosterone has been observed in endurance trained men (Hackney, Szczepanowska & Viru 2003), while a decreased anabolic status has been linked to a greater propensity for muscle damage (Hackney, Machado 2012). These observations suggest that the endurance training background of our subjects may influence the acute metabolic responses to the present combined training. In addition, some of the difference between men and women observed in CK may be related to the innate differences in hormonal milieu between men and women. Greater concentrations of estrogen are naturally observed in women than in men, thus it is interesting to note that estrogen has been reported



to have an effect on muscle contractile properties and may even have “protective” qualities due to positively influencing muscle repair processes including reduction of muscle damage and inflammation (Enns, Tiidus 2010).

It appears that both central (Gandevia 2001) and peripheral (Sahlin 1992) mechanisms contributed to the accumulated fatigue during the present combined sessions and subsequent recovery. Central fatigue may have contributed more to the decrease in force production, however, because the strength loading included movements using maximal force and velocity (De Ruiter et al. 2005), while blood lactate concentrations remained relatively low. Peripheral factors of fatigue including the previously mentioned increased blood lactate, decreased muscle pH, and decreased  $\text{Ca}^{2+}$  transport within the muscle may have also contributed to the inhibition of muscle contractile characteristics (Sahlin 1992). In addition, elevated concentrations of CK suggest that muscle damage may have played a role in decreased force production capabilities during recovery.

While the neuromuscular and metabolic responses described in the present study reveal some important information about the time-course of fatigue and recovery to the present loadings, these results, when combined with hormonal responses, reveal a more interesting overall picture. Our previous publication (Taipale, Häkkinen 2013) demonstrated that neuromuscular fatigue was accompanied by an increase in cortisol concentrations at post in the SE men. This was followed by decreased concentrations of testosterone during recovery of SE at 24 and 48 hours. The time-course of neuromuscular fatigue and recovery appeared to match the time-course of serum hormone responses to the ES and SE sessions, which could partly be explained by blood lactate exceeding threshold in men. The CK response may be linked to muscle soreness and could play a role in decreased force production capabilities. This difference in hormonal responses in men suggests that an order effect exists (Taipale, Häkkinen 2013) and that neuromuscular, hormonal and metabolic recovery is not complete following the present combined strength and endurance training session. The present study shows some evidence of an order effect in the explosive force production abilities of women during recovery and that the order of exercises may influence fatigue, although responses do appear to be highly individual. The present findings may be useful in optimizing training session and recovery planning. For example, endurance before strength should be avoided by women if explosive capabilities are needed in the days following this kind of combined session. In men, the order of the combined session does not induce differences in

decreases of force production capabilities, although, an order effect may be observed in hormonal balance of men especially during recovery when strength precedes endurance.

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**TABLES AND FIGURES**

**Table 1. Subject characteristics.**

|  | <b>Men (n = 12)</b> | <b>Women (n =10)</b> |
|--|---------------------|----------------------|
| <b>Age (years)</b>                               | 38.8 ± 7.1          | 33.5 ± 8.3           |
| <b>Height (cm)</b>                               | 177.4 ± 6.4         | 165.9 ± 7.6          |
| <b>Body mass (kg)</b>                            | 75.7 ± 3.6          | 59.8 ± 5.1           |
| <b>Body fat (%)</b>                              | 12.9 ± 3.6          | 22.0 ± 3.8           |
| <b>VO<sub>2max</sub> (L · min<sup>-1</sup>)</b>  | 4.1 ± 0.3           | 2.9 ± 0.4            |
| <b>(ml · kg<sup>-1</sup> · min<sup>-1</sup>)</b> | 54.5 ± 4.0          | 48.5 ± 4.6           |
| <b>Max Blood lactate (mmol · L<sup>-1</sup>)</b> | 10.3 ± 1.9          | 8.8 ± 1.7            |

Table 2. **Average force 0-500ms (N)**. \*\*, \*\*\* = significant change ( $p < 0.01$ ,  $p < 0.001$ ) in average force from pre, + = significant change in average force ( $p < 0.05$ ) from mid, ###,#### = significant change ( $p < 0.01$ ,  $p < 0.001$ ) in average force from post, † = significant change ( $p < 0.05$ ) in average force from 24h. No significant differences were observed between sessions or between men and women.

| Average force 0-500 ms | MEN                 |                   | WOMEN       |            |
|------------------------|---------------------|-------------------|-------------|------------|
|                        | ES                  | SE                | ES          | SE         |
| <b>PRE</b>             | 1836 ± 324          | 1813 ± 421        | 1063 ± 137  | 1132 ± 269 |
| <b>MID</b>             | 1594 ± 359***       | 1500 ± 280**      | 1036 ± 229  | 986 ± 233  |
| <b>POST</b>            | 1467 ± 335***,+     | 1298 ± 306***,+   | 1034 ± 174  | 952 ± 354  |
| <b>24h</b>             | 1561 ± 331***       | 1600 ± 385**.,### | 1022 ± 309# | 969 ± 356  |
| <b>48h</b>             | 1641 ± 305**.,###,† | 1679 ± 424###     | 1024 ± 330  | 970 ± 318  |

Figure 1 **A) Maximal bilateral isometric force and B) muscle activation of vastus medialis ( $\Delta\%$ )**. \*,\*\*,\*\*\* inside column = significant change ( $p < 0.05$ ,  $p < 0.01$  and  $p < 0.001$ ) in absolute maximal isometric force from pre, +++ = significant change ( $p < 0.001$ ) in absolute maximal isometric force from mid, ## = significant change ( $p < 0.01$ ) in absolute maximal isometric force from post, ⚡ = significant change ( $p < 0.05$ ) in absolute maximal isometric force from 24h. \*(,\*\*( =  $p < 0.05$ ,  $p < 0.01$  significant differences between relative changes from pre in ES and SE sessions. \*[] = significant difference between men and women ( $p < 0.05$ ). \* = significant change ( $p < 0.05$ ) in muscle activation of vastus medialis from pre, + = significant change ( $p < 0.05$ ) in muscle activation of vastus medialis from mid.

Figure 2. **Countermovement jump height ( $\Delta\%$ )**. \*,\*\*,\*\*\* inside column = significant change ( $p < 0.05$ ,  $p < 0.01$  and  $p < 0.001$ ) in  $\Delta\%$  countermovement jump height from pre, ++ = significant change ( $p < 0.01$ ) in  $\Delta\%$  countermovement jump height from mid, ## = significant change ( $p < 0.01$ ) in  $\Delta\%$  countermovement jump height from post, \*( =  $p < 0.05$  significant differences between relative changes from pre in ES and SE sessions. \*[] = significant difference between men and women ( $p < 0.05$ ).

Figure 3. **Blood lactate**. \*,\*\*,\*\*\* = significant change ( $p < 0.05$ ,  $p < 0.01$  and  $p < 0.001$ ) in blood lactate from pre strength loading (PreS). \*( = significant difference between ES and SE sessions. Arrows indicate the loading that was performed first in the session. Significant differences between men and women are noted in the text.

Figure 4. **Creatine kinase ( $\Delta\%$ )**. \*,\*\*,\*\*\* = significant change ( $p < 0.05$ ,  $p < 0.01$  and  $p < 0.001$ ) in  $\Delta\%$  creatine kinase from pre, +, +++ = significant change ( $p < 0.05$  and  $p < 0.001$ ) in  $\Delta\%$  creatine kinase from mid, # = significant change ( $p < 0.05$ ) in  $\Delta\%$  countermovement jump height from post, ⚡ = significant change ( $p < 0.01$ ) in  $\Delta\%$  creatine kinase from 24h. No differences between sessions or between men and women.



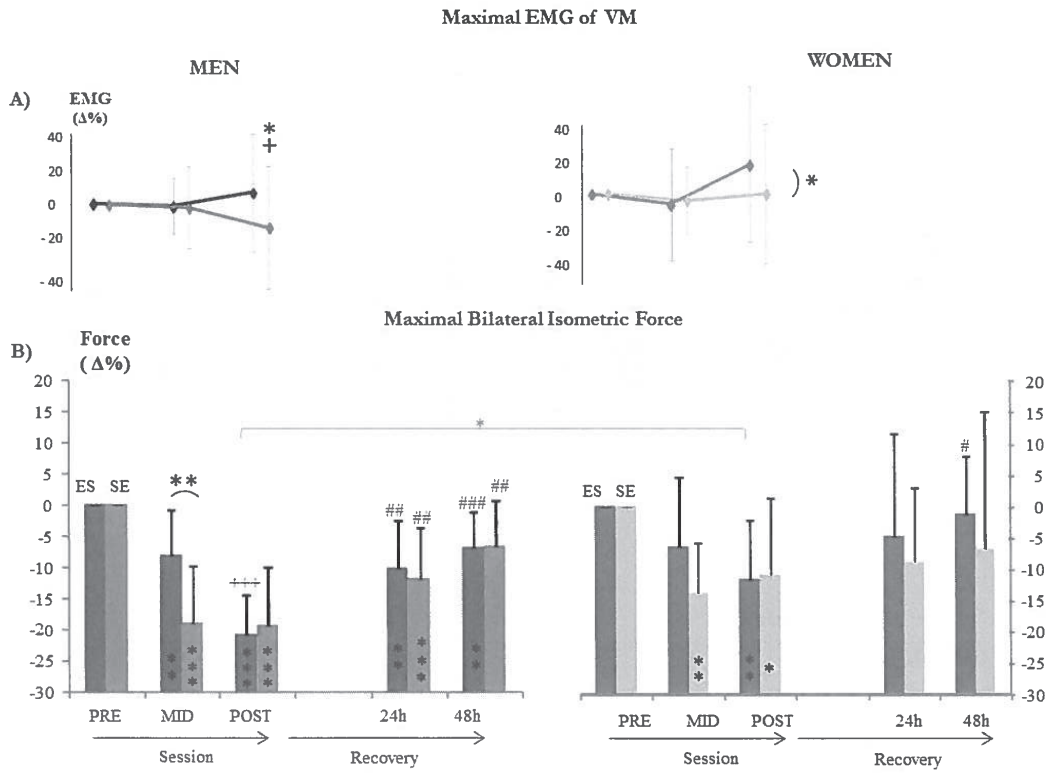


Figure 1.

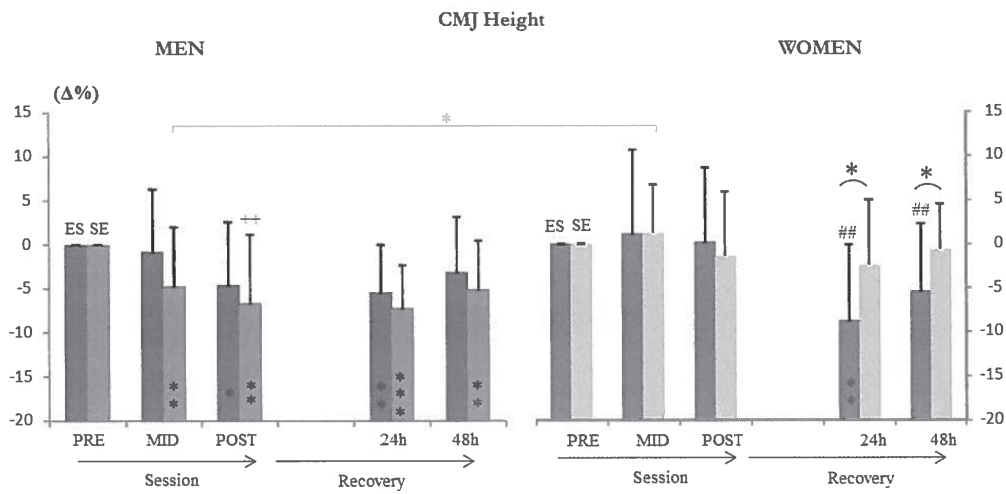


Figure 2.

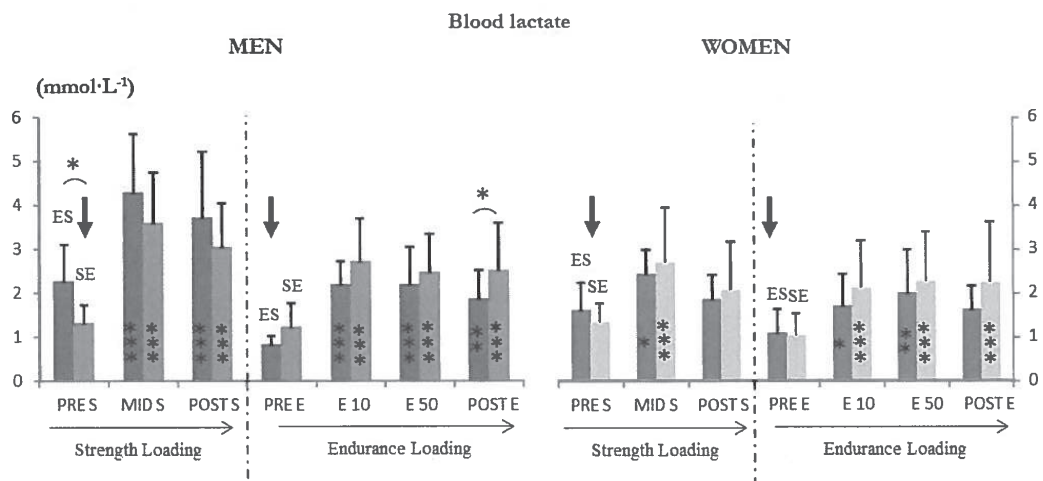


Figure 3.

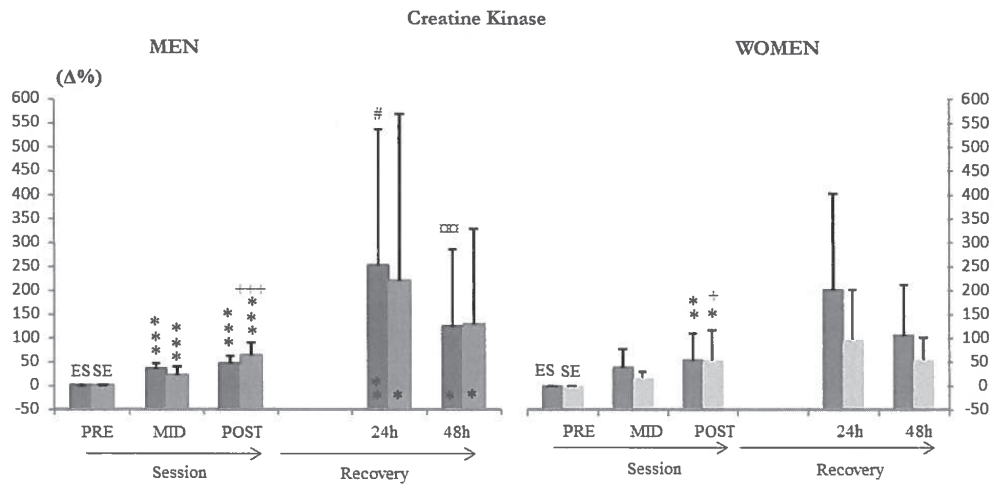


Figure 4.