EFFECTS OF A 24-WEEK SAME-SESSION COMBINED ENDURANCE AND STRENGTH TRAINING PROGRAM ON PHYSICAL PERFORMANCE AND SERUM HORMONE LEVELS IN RECREATIONAL ENDURANCE RUNNERS

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ABSTRACT

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Combining endurance (E) and strength (S) loadings into the same training session might be an efficient time-saving strategy for endurance runners that want further develop performance thanks to the benefits obtained by adding strength training. However, performing strength training repeatedly after prolonged runs may generate a superior degree of stress on both neuromuscular and endocrine systems that, especially at high training frequencies, may compromise long-term strength training adaptations. This, in turn, might have important implications on endurance running performance. This study investigated the longitudinal changes in the acute responses to a same-session combined endurance and strength training and their influence on the long-term physical performance and serum hormone levels in recreational endurance runners.

Eleven male recreational endurance runners (32±5 years) completed a 24-week periodized combined training program consisting in 2 combined endurance and strength training sessions (E+S) and 3-4 endurance-only training sessions per week. Basal measurements of endurance performance (Vpeak, blood lactate at submaximal running speed), neuromuscular performance (MVC, 1RM, F500ms, CMJ) and endocrine function (testosterone, cortisol, GH, TSH and SHBG) were performed in the first week of training (week 0), after 12 weeks (week 12) and at the end of the training period (week 24) under controlled conditions. Acute neuromuscular and hormonal response to the combined training session and early recovery phase were also assessed in the same weeks of basal measurements with a specifically-designed training session, before E (PRE), after E (MID), after E+S (POST) and after 24 and 48 h of recovery.

The combined training session lead to significant ($p<0.05$) decreases at POST in neuromuscular performance (MVC, F500ms and CMJ) both at week 0 and 24 but not in power capacity (F500ms, CMJ) at week 12. Significant ($p<0.05$) increases occurred in testosterone, cortisol, GH at MID at week 0, 12 and 24, however, a longitudinal reduction was observed in the acute cortisol and TSH response at POST during the intervention period. Whereas MVC, F500ms and CMJ were recovered at 24 h, cortisol and TSH remained (although not always significantly) depressed at 24 and 48 h at week 0, 12 and 24. No long-term improvements in neuromuscular performance were detected during the study period. Significant increases in Vpeak ($p<0.01$) and blood lactate at 15 km h^{-1} ($p<0.05$) occurred in the last 12 weeks of training. Significant correlations were observed between F500ms at MID and Vpeak ($r=0.663$, $p<0.05$) and F500ms at MID and blood lactate at 15 km h^{-1} ($r=-0.673$, $p<0.05$) but only at week 12.

The present study confirmed that, training strength always after endurance may lead to an augmented stress to the endocrine system that may take several days to recover. Despite minor adaptations, this training design may impede strength and power development, counteracting the benefits of strength training on endurance performance.

Keywords: combined training, fatigue, recovery, chronic adaptations, strength, hormones, endurance running

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CONTENTS

1 INTRODUCTION

Distance running is one of the most common types of endurance activities as well as a recurring component in many sports. Maximal oxygen uptake (VO_{2max}) has been generally considered the best indicator of cardiovascular fitness and endurance running performance (Davies & Thompson 1979). In already conditioned runners, however, other aerobic variables have been demonstrated to be more sensible indexes of performance than VO_{2max} (Conley & Krahenbuhl 1980; Tokmakidis et al. 1998).

Growing evidence has revealed that neuromuscular characteristics also play a significant role in determining long distance running performance (Paavolainen et al. 1999a; Nummela et al. 2006). These results, powered by an increasing interest in optimizing performance, have kicked off a further series of studies based on the effects of strength training on running performance and on its physiological determinants. The most significant finding was that, when the strength training stimulus is adequate in volume, intensity and frequency, the performance of runners may benefit even without observable changes in VO_{2max} (Paavolainen et al. 1999b; Beattie et al. 2014).

However, endurance training has also been demonstrated to be capable to blunt long-term strength and power development, especially when high in volume and intensity (Hickson 1980; Wilson et al. 2012). Differences in training adaptations may partly explain this interference. Strength training stimulus produces, in fact, an increase in strength and power performance through improvements in both neural and muscular components (Folland & Williams 2007; ACSM 2009). On the contrary, endurance training does not induce significant improvements in these variables and, occasionally, it may also depress some aspects of them (Kraemer et al. 1995; Fitts & Widrick 1996).

A sort of interference may therefore exist in the concurrent adaptations to the two different training programs (Hickson 1980; Nader 2006). This incompatibility may be further aggravated by the superior stress induced by combined training program that, if too high in frequency, may increase the risk of overreaching or overtraining conditions in the long term (Kraemer et al. 1995; Bell et al. 2000).

Distance runners generally require higher endurance training volume, intensity and frequency than untrained subjects in order to maintain physical fitness and/or to achieve highly trained condition. Same-session combined training might then become a useful training approach in these subjects. Performing strength after endurance has shown to provide advantages in terms of endurance performance in previously untrained subjects, while this does not seem to apply to the inverse loading order (Chtara et al. 2005).

Despite the lower degree of interference and the superior tolerance to high volumes and intensities observed in endurance trained individuals, the implications that fatigue might have in determining long-term training outcomes in these subjects should not be ignored (Hunter et al. 1987; Leveritt et al. 1999). The large degree of stress imposed to the body by this combined training design might in fact reduce, if not cancel, the benefits provided by adding strength training on endurance performance. Today, knowledge of the effects of same-session combined training in endurance conditioned individuals is limited. A systematic study of sufficient duration examining both acute and chronic responses would permit to obtain important information about the effectiveness of this type of training on performance development in endurance runners.

2 PHYSIOLOGICAL DETERMINANTS OF ENDURANCE RUNNING PERFORMANCE

Endurance exercise can be defined as a cardiovascular activity in which muscles are exercised for an extended period of time (Joyner & Coyle 2008). This definition covers sports such as cross-country skiing, duathlon, long distance races, marathons, cycling, racewalking and rowing, triathlon and ultramarathons. Running is one of the most used endurance exercise modalities aside from being an integral part in many sports.

A basic principle of exercise physiology says that human body requires a certain amount of energy to achieve and maintain a specific work rate for a given duration (figure 1) (Joyner & Coyle 2008). In endurance running, this capacity is the resultant of a complicated interplay of many physiological functions including cardiorespiratory, neuromuscular and endocrine components.

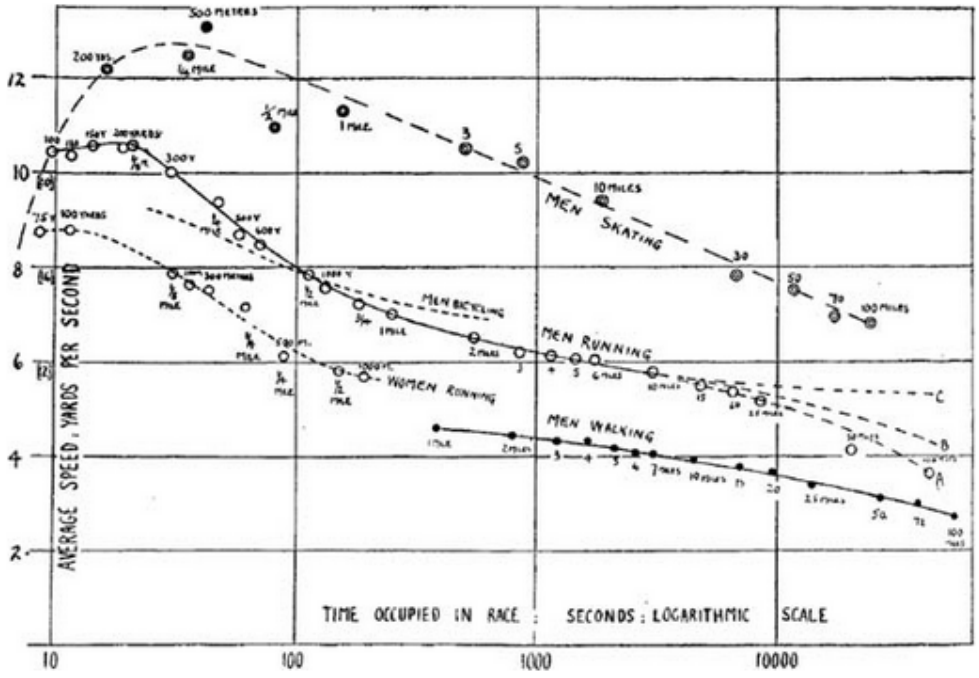

FIGURE 1. Hill's original plot. The horizontal axis represents the world record performance time while the vertical axis indicates the average performance speed. Men's running is reported in the middle tracing and women's running by the bottom tracing (Joyner & Coyle 2008).

2.1 Aerobic and anaerobic factors in endurance performance

The human body is a sophisticated machine capable of transforming chemical energy in mechanical energy, able to move muscles and joints. The energy to perform any muscle contraction is provided by the adenosine triphosphate (ATP) which is, in turn, continuously resynthesized by different energy pathways as fast as it is used (Gastin 2001). The phosphagen (ATP-PC) and the myokinase systems constitute the immediate energy system. These systems are based on simple chemical reactions that permit them to provide energy quickly. The main limitation of these systems seems to be related to the limited amount of supply of fuel and build up of by-products of metabolism causing a decrease in the recycling of energy (Gastin 2001). Glycolysis is defined as the breakdown of glucose to pyruvate, which in turn may be converted to lactate (anaerobic) or Acetyl-CoA (aerobic). A large amount of power is produced by glycolysis but not quite as much or as quickly as it is by the ATP-PC system. Dependently by the energy rate required, this system may be limited by either fuel supply or by-product accumulation that may impair performance before the depletion of the energetic substrate occurs (Gastin 2001; Cairns 2006). The oxidative system may be seen as an extension of glycolysis. In the presence of oxygen, in fact, pyruvate produced by glycolysis is converted in Acetyl-CoA and further metabolized through the Krebs cycle and the electron transport chain, similarly to that which occurs with the fatty acids. While the anaerobic systems are mainly involved during intense bouts of few seconds, oxidative system becomes predominant after little more than a minute (Gastin 2001). These energy systems work simultaneously and the predominance of one over the others is dictated by the characteristics of the physical activity performed (figure 2).

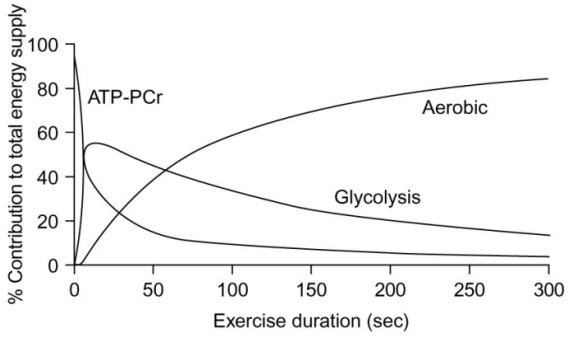

FIGURE 2. Relative contribution of the three major energy systems to the overall energy production required for any given duration of maximal exercise (Gastin 2001).

2.1.1 Oxygen's role: is VO_{2max} really so critical?

The larger contribution of the aerobic system in long distance races and the absence of waste products accumulation have led VO_{2max} to have a primary role in running performance (Davies & Thompson 1979). VO_{2max} represents the maximum aerobic power and depends on the cardiac output, total hemoglobin as well as its degree of saturation, the blood flow in the muscles and oxygen extraction from them (Joyner & Coyle 2008). Elite distance runners have generally values 50–100% greater than the ones observed in untrained individuals and increases in VO_{2max} generally occur in the first weeks of training (Daniels et al. 1978; Joyner & Coyle 2008).

However, VO_{2max} has been demonstrated not or just minimally related with performance in a group of well-trained endurance runners (Legaz-Arrese et al. 2005). Despite large initial gains, the long-term trainability of VO_{2max} is limited and several years of training have not produced any increase in this value in elite distance runners (Daniels et al. 1978; Legaz-Arrese et al. 2005). These limitations stress how, despite being a prerequisite for successful runners, VO_{2max} ceases to be a sensible parameter to assess performance development in the long term (Joyner & Coyle 2008).

2.1.2 The dual aspect of the anaerobic contribution

Despite a negligible amount of energy is provided by anaerobic pathways during marathon, it can supply up to 10–20% of total ATP production in shorter distances (i.e. 5/10-km) (Joyner & Coyle 2008). Anaerobic contribution may then become a determinant factor in those events where running pace cannot be maintained almost entirely by the use of aerobic metabolism (Bulbulian et al. 1986; Houmard et al. 1991).

A physiological consequence of the use of anaerobic glycolysis is the increased production of lactate (Cains 2006). Lactate formation depends mainly on exercise and muscle characteristics (Holloszy & Coyle 1984). Mitochondria concentration and oxidative enzymatic activity are the main determinants of lactate oxidation during and after exercise (Holloszy & Coyle 1984). A curvilinear relationship between blood lactate levels and running distance can be observed (figure 3) (Costill 1970).

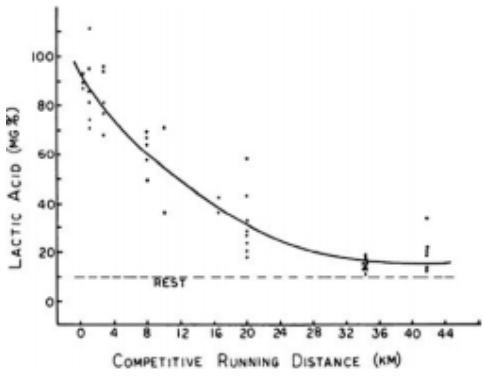

FIGURE 3. Changes in blood lactate at different race distances. Decreased levels reflect the diminished contribution of anaerobic glycolysis to the total energy supply (Costill 1970).

While lactate by itself may not be detrimental to the performance, by-product accumulation related to the anaerobic metabolism has been indicated as muscle fatigue inductor (Cairns 2006). The main line of action seems to be through impairment in the contractile properties of the muscles themselves (Cairns 2006). The latter are resulting from a reduction in Ca^{2+} sensitivity and release associated with a decrease in the ATP replenishment rate (Cairns 2006). It is clear, then, that a speed corresponding to VO_{2max} cannot be sustained for more than a few minutes (Joyner & Coyle 2008).

Because lactate production is strictly related with the extent of the use of the anaerobic metabolism, blood lactate level can be used as useful marker of exercise intensity and fatigue condition (Cairns 2006). By the term “lactate threshold”, we generally refer to a valuation of a breakpoint on the lactate-velocity curve with regard to the intensity of the exercise (Tokmakidis et al. 1998). This value approximates the maximal sustainable exercise intensity in function of the degree of lactate accumulation. Despite the variety of methods available to determine this parameter, the correlations between resulting lactate threshold and running performance remain strong (Tokmakidis et al. 1998). It is widely accepted that any rightward deviation of the blood lactate-running speed curve results in an increased speed at the lactate threshold (Tokmakidis et al. 1998).

The dual aspect of the anaerobic contribution makes clear how, to be successful runners, the goal is not limited to improve the maximum sustainable velocity before reaching the onset of fatigue. It is also important to be able to produce and maintain the required level of power in those conditions when glycolytic and oxidative pathways are highly activated and muscle contractility may be reduced (Paavolainen et al 1999c).

2.1.3 Best runners are those who are more economical

Another reason why VO_{2max} is not a sensitive predictor of running performance is due to the existing differences in running economy (RE) among long distance runners (Joyner & Coyle 2008). RE refers to the energy consumption at a given running pace and determines the energy demand required for any specific type of effort (Saunders et al. 2004). The variation in oxygen consumption at a given speed may reach 30% among runners with a similar aerobic capacity (Conley & Krahenbuhl 1980). This inter-individual variability depends on a plurality of factors, including anthropometric, physiologic and biomechanical aspects (Saunders et al. 2004).

Best runners are characterized by a better RE and this, in turn, permits them to run at a lower relative VO_{2max} (figure 4) (Saunders et al. 2004). The lower relative intensity reflects in a higher sustainable speed in those runs lasting more than 2 hours (h) (Costill 1970; Joyner & Coyle 2008). At the same time, the reduced use of anaerobic glycolisys for a given velocity may stress the importance of RE also in those performance where a large amount of energy is produced through the use of this metabolism. For these reasons, in well-trained distance runners, RE has been suggested as a better predictor of performance than VO_{2max} (Conley & Krahenbuhl 1980; Saunders et al. 2004).

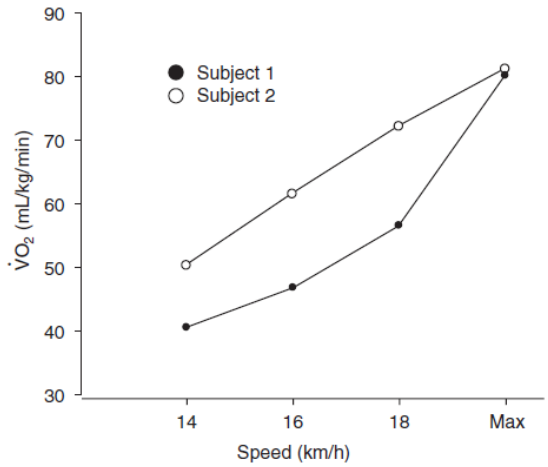

FIGURE 4. Comparison of two elite 10-km runners. The first subject has a better running economy than the second at any measured submaximal speed (Saunders et al. 2004).

2.2 Neuromuscular characteristics in distance running performance

There is a scientific consensus stating how VO_{2max}, lactate threshold and RE explain the vast majority of the variance in long distance running events (Joyner & Coyle 2008). Nonetheless, there is also growing evidence about the importance of neuromuscular characteristics in conditioning performance, especially in athletes with similar VO_{2max} (Paavolainen et al. 1999a; Nummela et al. 2006). This term 'neuromuscular characteristics' refers to the interaction between the neural and muscular system. It includes the degree of neural input to the muscles, motor unit recruitment pattern and synchronization, muscle stiffness regulation (Paavolainen et al. 1999b). All these variables are crucial in converting cardiorespiratory capacity into required movement.

2.2.1 Race pace: a question of power

The achievement of a proper race pace is a main factor to excel in any running event. Running velocity is strictly related to the neuromuscular capacity to counteract ground reaction forces (GRFs) generated during the stance phase (Kyröläinen et al. 2001; Weyand et al. 2010). In running, ground contact time (GCT) is short and this limits the time available to develop the maximum strength (figure 5) (Bosco 2002, 325-327; Weyand et al. 2010). Fast force production capacity becomes then an essential variable in defining successful runners (Paavolainen et al. 1999a; Nummela et al. 2008).

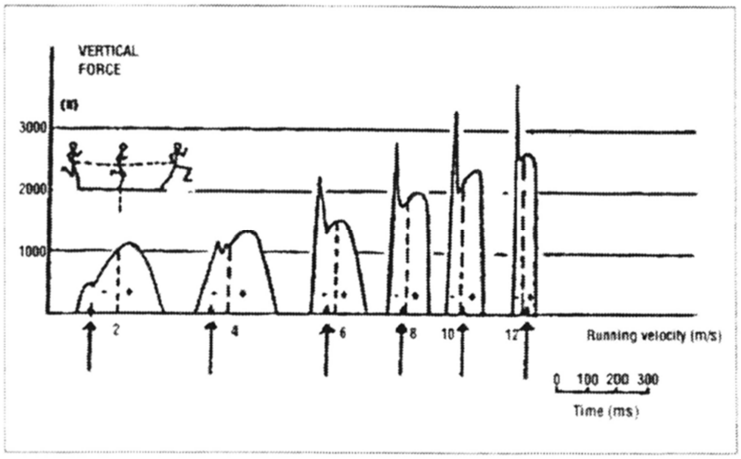

FIGURE 5. Vertical GRF at different running velocities. The magnitude of the GRF increases with the velocity while the time available to develop force decreases (Bosco 2002, 325-327).

These concepts find agreement in Noakes (1988) who suggested that muscle power factors, such as rate and force of cross-bridge activity, may strongly affect endurance running performance not allowing the optimum use of oxygen. The contribution of these factors has been described into a model that explains 5-km and 10-km run time, confirming how the limiting factors in these running events are not solely metabolic in nature (Paavolainen et al. 1999a; Paavolainen et al. 1999c; Nummela et al. 2008).

2.2.2 Are the most explosive long distance runners also the most economical?

Rapid muscle contractions are associated with a greater recruitment of fast twitch muscle fibers that may increase the metabolic cost of the run anticipating the onset of fatigue (Roberts et al. 1998). This evidence is somewhat conflicting with the importance of muscle power factors in endurance performance. However, one must keep in mind that other variables, than purely muscular, come into action in determining RE.

Among these aspects, stretch-shortening cycle (SSC) efficiency has proven to be an important determinant in RE and, more generally, endurance performance (Paavolainen et al. 1999b). During running, the elastic components of the musculotendinous complex deform under the load, storing potential energy during the eccentric phase to reuse it in the subsequent concentric phase, acting like a spring (Nicol et al. 2006). This allows muscle to operate at slower shortening velocities, producing greater mechanical force and power output while also using less metabolic energy (figure 6) (Komi 2000).

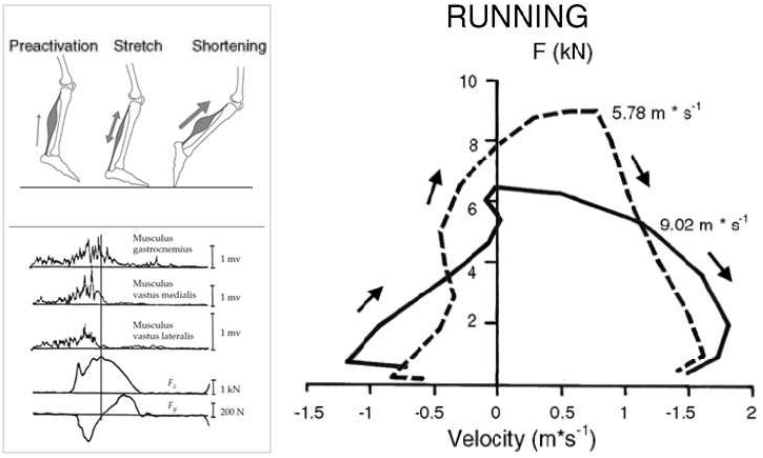

FIGURE 6. On the left: the 3 SSC phases, EMG activities and GRFs during running at moderate speed. On the right: force-velocity curves measured from both pure concentric actions in isolated muscle and during SSC at two different running velocities (modified from Komi 2000).

The central nervous system (CNS) is determinant in the modulation of SSC. Lower limb muscle preactivation enhances musculotendinous stiffness (MTS), optimizing the exploitation of stored elastic energy, thanks also to the contribution of the stretch reflex (figure 6) (Kyröläinen et al. 2001; Nicol et al. 2006). A higher muscle preactivation, shorter GCTs coupled with a lower electromyographic activity during the propulsion phase have been detected in the faster and most economical runners (Paavolainen et al. 1999a; Paavolainen et al. 1999c; Nummela et al. 2008). Kyröläinen et al. (2001) observed how these variables vary with running speed suggesting SSC as a mechanism to sustain higher GRFs. An earlier onset of fatigue has been observed in those runners whose neuromuscular function is dropping (Paavolainen et al. 1999c; Nummela et al. 2008). These evidences stress the importance for endurance runners of maintaining an efficient SSC mechanism in order to produce force rapidly and repeatedly throughout the duration of the race (Paavolainen et al. 1999c; Nummela et al. 2008).

2.3 The endocrine system in endurance and sport performance

Hormones are chemical messenger that transmit signals from a cell (or a group of cells) to another, regulating most of body functions via alteration in cell metabolism. Endocrine hormones are generally released in the bloodstream by their own host gland in response to nervous, chemical or hormonal stimuli (Kraemer & Rogol 2005, 1-3). They can circulate either free or bound with specific carrier proteins that may alter or inhibit their actions. Each hormone requires the interaction with a specific receptor to carry out its functions. Once the receptor is bound, this leads to a cascade of cellular events culminating in specific physiological responses (Kraemer & Rogol 2005, 3-4).

Blood hormonal levels do not exactly reflect the effect induced by hormones on metabolism. The magnitude and time course of hormonal response vary due to secretion, fluid volume shifts, degradation rates, tissue clearance, interactions with binding proteins and receptors (Kraemer & Ratamess 2005). Nevertheless, the extent of the metabolic effect of hormones remains principally related to the number of circulating hormone molecules that affects the likelihood for hormone-receptor interactions (Kraemer & Ratamess 2005). Finally, substrate and materials availability exerts a deep impact upon the optimization of training adaptations (Hawley 2009).

Hormones are of primary importance in sport performance, maintaining homeostasis during exercise and mediating training adaptations as well (Kraemer & Rogol 2005, 2; Kraemer & Ratamess 2005). Glucocorticoids, particularly cortisol, have an important function in the metabolic control during exercise through a permissive effect on lipid mobilization and amino acids metabolism (Kraemer & Rogol 2005, 194-195). Glucocorticoids may be also involved in the mitochondrial biogenesis process in skeletal muscle (Goffart & Wiesner 2003). At high concentrations, they may alter anabolic processes leading muscle hypertrophy and thus compromising strength training adaptations (Kraemer & Ratamess 2005). Differently, acute elevations may indicate a remodelling process occurring in skeletal muscle (Kraemer & Rogol 2005, 330-331).

Opposite to cortisol, testosterone promotes strength and power development through both neural and morphological adaptations, influencing nervous system and stimulating tissue repair and muscle growth (Kraemer & Rogol 2005, 331-334). Most of circulating testosterone is bound to sex hormone-binding globulin (SHBG) (Kraemer & Rogol 2005, 290-293). A decreased percentage of this hormone bound to SHBG may reflect an augmented effectiveness in maximizing strength gains (Häkkinen et al. 1988).

Among its functions, growth hormone (GH) (in the 22-kD isoform) promotes muscle tissue anabolism, in part through the mediation of insulin-like growth factor I (IGF-I) (Kraemer & Rogol 2005, 2; Vijayakumar et al. 2010). Acute GH action stimulates lipolysis in adipose tissue and skeletal muscle, supporting the glycogen-sparing action of cortisol during exercise (Kraemer & Rogol 2005, 602-603; Vijayakumar et al. 2010).

Similarly to cortisol and GH, thyroid-stimulating hormone (TSH) may affect energy metabolism, inducing a permissive action on lipid mobilization via stimulation of thyroid hormones (Kraemer & Rogol 2005, 2). Furthermore, TSH may also indirectly promote mitochondrial biogenesis in muscle tissue (Goffart & Wiesner 2003).

Biological factors as age, gender and circadian variations influence hormonal concentrations at rest (Kraemer & Ratamess 2005; Hackney & Viru 2008). While emotional and environmental stressors may also affect the endocrine system, training characteristics, recovery, nutrition and training experience remain the main determinants of hormonal responses to exercise (Kraemer & Ratamess 2005; Hackney & Viru 2008). The consideration of all these variables is of fundamental importance in order to safeguard the validity of the data in a research project (Hackney & Viru 2008).

3 ENDURANCE AND STRENGTH TRAINING RESPONSES

Training adaptations are the result of the long-term accumulation of particular proteins induced by specific exercise stimuli (Hawley 2009). Acute training responses provide the biological bases for the development of the specific training-induced phenotype (Kraemer & Ratamess 2005; Hawley 2009). Whereas the degree of stress produced by exercise loadings can be evaluated by the acute decrease in physical performance, the anabolic and catabolic processes occurring in response to the training session are mostly reflected in transient alterations in endocrine function (Hackney & Viru 2008). Although a single session of endurance or strength training does not generate stable physiological adaptations, it induces temporary cellular modifications that, when repeated several times, lead to the specific training-induced phenotype (Hawley 2009).

3.1 Chronic adaptations to endurance running

3.1.1 Cardiorespiratory and metabolic adaptations to endurance running

Long-term endurance training improves performance in several ways. Increases in cardiac output and blood volume are the most evident cardiovascular adaptations (Joyner & Coyle 2008). Moreover, increases in number and size of mitochondria, oxidative enzymes and capillary density are generally observed in the trained muscle fiber within few weeks by the beginning of an endurance training program (Holloszy & Coyle 1984; Joyner & Coyle 2008). These physiological changes determine the high VO_{2max} values observed in endurance runners (Joyner & Coyle 2008). The increased oxidative capacity and capillarization in the skeletal muscle also contributes to reduce lactate concentration at submaximal velocity (Holloszy & Coyle 1984; Joyner & Coyle 2008). A rightward shift of the whole blood lactate-running speed curve is a common adaptation observed in distance runners (figure 7) (Raczek 1989; Holloszy & Coyle 1984; Joyner & Coyle 2008).

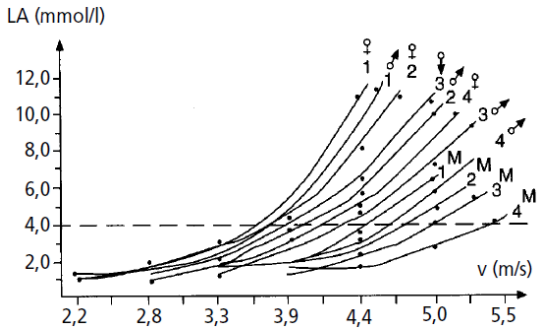

FIGURE 7. Shift in lactate-velocity curves in long-distance and marathon runners (M). On the top of each curve there is reported the gender of the runner and year of the study (Raczek 1989).

The positive effects of endurance training on VO_{2max} and lactate metabolism may be in part mediated by the improvements in RE observed after few months of training in previously untrained runners (Moore et al. 2012). These initial gains seem mainly due to a self-optimization process of running gait (Moore et al. 2012). Differently, long-term RE development appears to be related with training experience as a consequence of changes in a wide variety of factors (Saunders et al. 2004; Joyner & Coyle 2008).

Proper training stimulus, length of the training program and training status have been demonstrated to affect the magnitude of endurance training adaptations (Holloszy & Coyle 1984; Joyner & Coyle 2008). Few months of training are sufficient to induce significant training gains in previously untrained runners (Daniels et al. 1978; Moore et al. 2012). Longer periods are instead necessary to observe improvements in already conditioned runners with lack of progress that may occur in those characteristics that are already at or close to the maximal potential (Daniels et al. 1978; Legaz Arrese 2005).

The principle of training specificity indicates aerobic training as mean to improve in endurance performance. This may include long distances performed at constant pace (CT) or shorter distance but at high intensity interval work (HIIT). There is some debate regarding which type of training induces larger performance gains. HIIT has demonstrated to be similar or superior to CT in improving cardiovascular and metabolic parameters in the short term (Helgerud et al. 2007). However, prolonged periods characterized by many high intensity training sessions may also lead to overreaching condition (Seiler & Tønnessen 2009). Combine low and high intensity workouts in a periodized manner has been suggested as a good approach to optimize training effectiveness while limiting the risk of overtraining (Seiler & Tønnessen 2009).

Running activities do not evoke maximum activation in working muscles (Sloniger et al. 1997). Given the importance of the latter in stimulating anabolic processes associated with strength development, regular endurance training practice has no or little effect on this capacity or muscle size (Kraemer et al. 1995; Fitts & Widrick 1996). High-volume endurance running programs may instead induce atrophy in skeletal muscle fibers and impair strength and power performance (Fitts & Widrick 1996). These neuromuscular changes might be in part related to the long-term effect of endurance training on endocrine parameters (Hackney et al. 2008; Skoluda et al. 2012).

3.1.2 Hormonal adaptations to endurance running

Although moderate distance running has no or just minimal impact on resting testosterone, intensive and prolonged training regimes have been associated with a significant reduction of this hormone (Kraemer & Rogol 2005, 298-299; Hackney et al. 2008). Testosterone levels of highly trained distance runners can drop up to half of those observed in their untrained counterpart of similar age (Hackney et al. 2008). This exercise-hypogonadal condition seems to be a consequence of both peripheral (i.e. testicles) and central (alterations in luteinizing hormone and/or prolactin release) adaptations occurred in the hypothalamic–pituitary–gonadal axis (Hackney et al. 2008).

Prolonged strenuous endurance exercise and repeated physical stress have been also proposed as possible reasons of the elevated cortisol values observed in these subjects (Skoluda et al. 2012). A dose-response association between training volume and cortisol levels has been noticed in amateur endurance runners (figure 8) (Skoluda et al. 2012).

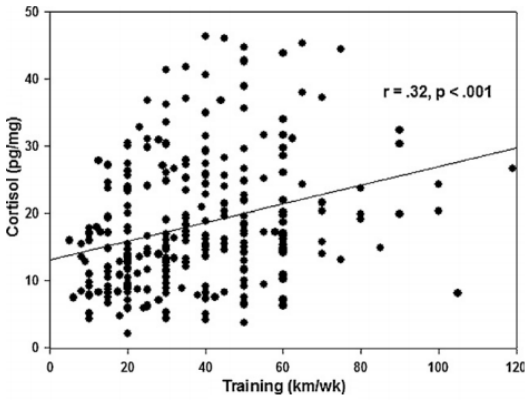

FIGURE 8. The plot shows the correlation observed between hair cortisol levels and kilometers run per week in the preceding three months of endurance training (Skoluda et al. 2012).

Three weeks of reduced training failed to normalize resting testosterone and cortisol concentration in male distance runners whilst normal testosterone levels have been observed after 36 weeks (Kraemer & Rogol 2005, 608; Safarinejad et al. 2009). These data suggest how these alterations in endocrine function may be a long-lasting component of the training adaptation process occurring in endurance runners.

3.2 Acute responses to endurance running

3.2.1 Acute neuromuscular and metabolic responses to endurance running

Endurance running is a physically demanding activity and acute neuromuscular impairments associated with strength and power loss in working muscles are commonly observed after prolonged events such as marathon (Nicol et al. 2006). Changes in GRFs and GCTs reflect deterioration in RE and exercise capacity occurring during this kind of performances (Komi 2000). These alterations appear to be mainly related to a reduced neural capacity to activate muscles and regulate MTS induced by repeated stretch loads (Nicol et al. 2006). A nonlinear relationship between the duration of exercise and the degree of neuromuscular fatigue generated has been noticed (Nicol et al. 2006).

Some subjects may recover quickly from prolonged runs but in other cases alterations in neuromuscular parameters may last up to one week (Avela et al. 1999; Nicol et al. 2006). Despite a large inter-individual variability, Komi (2000) has proposed a bimodal recovery pattern, with acute reduction induced by metabolic fatigue (e.g. glycogen depletion) and a second prolonged decrease associated with muscle damage (figure 9).

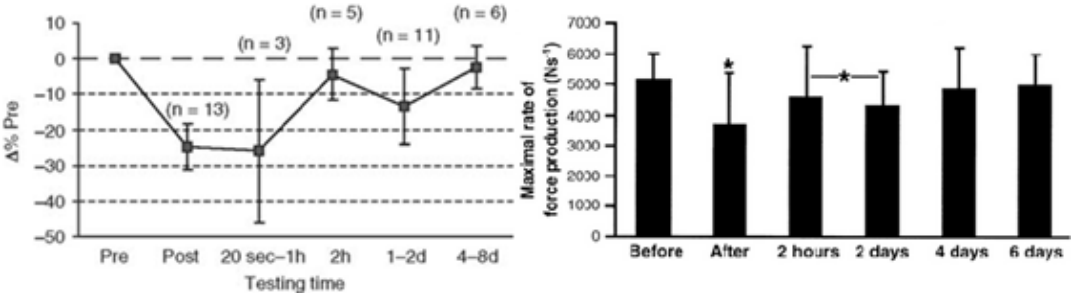

FIGURE 9. Changes in maximum voluntary muscle contraction (MVC) and maximal rate of force production in response to exhaustive SSC exercises (Avela et al. 1999; Nicol et al. 2006).

Changes in neuromuscular performance may also occur following relatively short endurance runs (i.e. 5/10-km), although being milder than those in longer events (Paavolainen et al. 1999c; Nicol et al. 2006; Nummela et al. 2008). The higher speed required to compete at these distances leads to a large use of anaerobic metabolism that becomes a major source of fatigue (Joyner & Coyle 2008). Even though a bimodal trend may be detected, the limited muscle damage generated by these performances generally makes a full recovery can be achieved within two days (Nicol et al. 2006).

Although long distance runs are characterized by a large degree of neuromuscular disturbance, high-level runners have proven to resist fatigue better than low-level ones (Paavolainen et al. 1999c; Nummela et al. 2008). Increase in neuromuscular parameters, especially those related to rapid force production capacity, may even be observed in these subjects after intense runs (Vuorimaa et al. 2006; Boullosa et al. 2011).

3.2.2 Acute hormonal responses to endurance running

The overall stress imposed to the body during exercise, expressed mainly as a function of intensity and duration, has proved to strongly affect the endocrine system (Bunt et al. 1986; Tremblay et al. 2005). The intensity required during the vast majority of long distance races (≥70-80% VO_{2max}) evokes significant increases in testosterone, cortisol, GH and TSH response already after 30-40 minutes of running (figure 10) (Galbo et al. 1977; Pritzlaff et al. 1999; Vuorimaa et al. 2008). Prolonged exhausting runs (e.g. marathon) may induce a 2- to 5-fold increase in cortisol (Kraemer & Rogol 2005, 602-603). A shift to a more catabolic environment has been detected after 80 minutes of running (Tremblay et al. 2005). The decrease in testosterone levels observed after 3-4 h of running may further accentuate this condition (Kraemer & Rogol 2005, 602-603).

Training status may modulate the magnitude of the acute hormonal response to exercise (Bunt et al. 1986; Hesse et al. 1989; Vuorimaa et al. 2008). Despite having lower baseline testosterone levels than untrained counterpart, endurance runners showed a greater acute response to strenuous exercise in a consistent manner with a better capacity to tolerate stress (Hackney et al. 1997). In a similar way, subjects who are already conditioned for endurance show a lower cortisol, similar or higher GH and TSH response at the same relative exercise intensity (Bunt et al. 1986; Hesse et al. 1989; Vuorimaa et al. 2008).

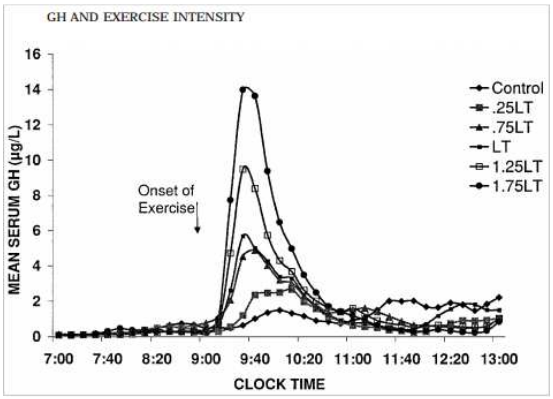

FIGURE 10. GH responses to 30 minutes running at different intensities (Pritzlaff et al. 1999).

Exercise characteristics influence also the recovery time course of endocrine function. While strenuous running bouts may depress testosterone, cortisol and TSH levels after 60-120 minutes of recovery for 24 to 48 h, moderate efforts may not necessary induce prolonged endocrine alterations (Tanaka et al. 1986; Jensen et al. 1991; Daly et al. 2005; Tremblay et al. 2005; Hackney & Dobridge 2009). Differently from what has been observed for the above mentioned hormones, 24-h integrated GH concentration does not result affected by a single endurance training session (Wideman et al. 2002).

3.3 Chronic adaptations to strength training

3.3.1 Neuromuscular and metabolic adaptations to strength training

It is proven that strength training induces important neuromuscular adaptations in both short and long term (Folland & Williams 2007; ACSM 2009). Improvements in neural function associated with a variable increase in muscle mass are the most common changes (Folland & Williams 2007; ACSM 2009). Alteration in myofibrillar protein isoforms, increases in anaerobic substrates and enzymes, buffer capacity may also be observed (MacDougall et al. 1977; Folland & Williams 2007; ACSM 2009). The magnitude of these adaptations depends mainly on the strength training characteristics, length of training program and training status of the subjects (table 1) (ACSM 2009).

TABLE 1. Strength training programs characteristics in relation to the training goal for novice and advance subjects. *=in a periodized manner, [1]=after main exercises, [2]=after complimentary exercises, [3]=after high-repetition sets, [4]=after low-repetition sets (adapted from ACSM 2009).

		INTYENSITY	VOLUME	REST	FREQUENCY
POWER	*NOVICE*	0-60% 1RM	3-5 sets/ex	≥2-3 min[1] / 1-2 min[2]	2-3 days/wk
	ADVANCED	0–100% 1RM*	multiple sets*	≥2-3 min[1] / 1-2 min[2]	4-5 days/wk
STRENGTH	*NOVICE*	60-70% 1RM / 8-12 reps	1-3 sets/ex	≥2-3 min[1] / 1-2 min[2]	2-3 days/wk
	ADVANCED	80-100% 1RM / 1-6 reps*	multiple sets*	≥2-3 min[1] / 1-2 min[2]	4-5 days/wk
HYPERTROPHY	*NOVICE*	70-85% 1RM / 8-12 reps	1-3 sets/ex	2-3 min[1] / 1-2 min[2]	2-3 days/wk
	ADVANCED	70–90% 1RM / 6-12 reps*	multiple sets*	2-3 min[1] / 1-2 min[2]	4-5 days/wk
MUSCULAR ENDURANCE	*NOVICE*	40–60% 1RM / 10-15 reps	>1-3 sets/ex	1–2 min[3] / <1 min[4]	2-3 days/wk
	ADVANCED	40-60% 1RM / 10-25 reps*	multiple sets*	1–2 min[3] / <1 min[4]	4-5 days/wk

Power training needs high contraction velocity to maximize training outcomes (ACSM 2009). Plyometrics can further contribute to power development due to its potent influence on the elastic and neural components of SSC (ACSM 2009). Although some improvements in maximal strength and muscle size may be observed, the low to moderate intensity generally used in this type of training are not suitable for optimal long-term gains in these variables (ACSM 2009; Saez Saez de Villareal et al. 2010).

High training loads are instead important to maximize strength gains (ACSM 2009). Power improvements may also occur with this training above all in previously no or low conditioned subjects (ACSM 2009; Cormie et al. 2010). The combination of both strength and power training stimuli in the same program may induce superior gains than the single training modality alone (ACSM 2009; Saez Saez de Villarreal et al. 2010).

The large metabolic stress due to the high training volume and short rest periods characterizing hypertrophy schemes may be critical to optimize muscle gains (ACSM 2009). The optimal milieu for hypertrophic development is also favored by the strong anabolic hormone response evoked by these programs (Kraemer & Ratamess 2005).

Strength training, above all when metabolically demanding, may even induce minor improvements in VO_{2max} and endurance capacity in previously untrained individuals (ACSM 2009; Lo et al. 2011). These changes may partly result from increases in mitochondrial enzyme activity, muscle fibre capillarization, shift in myosin isoforms and improvement in buffering capacity observed in these subjects after few weeks of training (Tang et al. 2006; ACSM 2009).

Previously untrained subjects generally achieve greater strength gains than already conditioned athletes showing also a minor degree of specificity in regard to the training program used (Ahtiainen et al. 2003; ACSM 2009; Cormie et al. 2010). One of the main reasons of this behaviour lies in the large degree of neural adaptations that occurs during the first weeks of training (Folland & Williams 2007; ACSM 2009). These adaptations include, but not limited to, a greater motor unit recruitment and synchronization, increased firing frequency and enhanced reflex activity (Folland & Williams 2007; ACSM 2009). A better intermuscular coordination, as a result of an improved co-activation of synergists associated with a reduced coactivation of the antagonists, has also been observed (Folland & Williams 2007). These changes allow increases in both strength and power without significant changes in muscle size. Nevertheless, a specific and progressive training stimulus remains critical in determining long-term training gains (figure 11) (Häkkinen et al. 1985a, b; ACSM 2009).

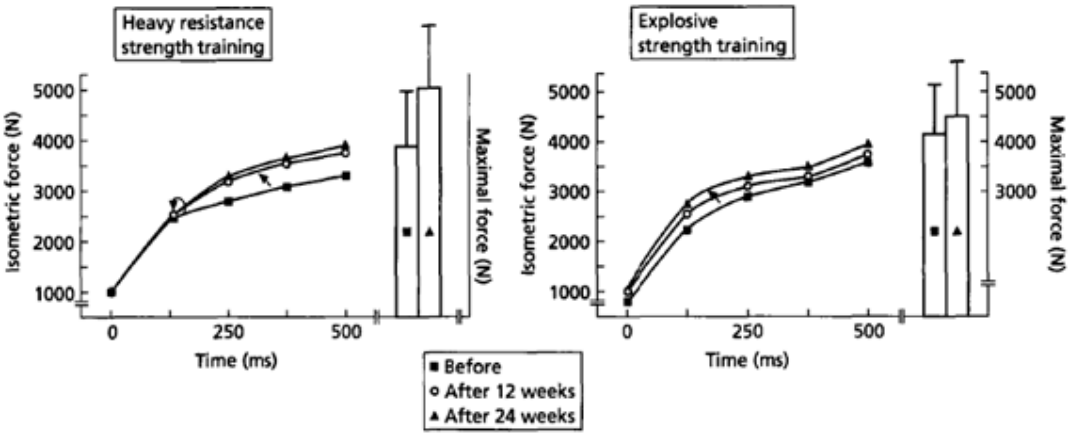

FIGURE 11. Effects of heavy resistance strength training and explosive strength training programs on maximal isometric force and force-time characteristics (Häkkinen et al. 1985a, b).

3.3.2 Hormonal adaptations to strength training

Some studies have observed a significant increase in basal testosterone levels and testosterone/SHBG (T/SHBG) ratio after strength training period in athletes and previously untrained subjects (Häkkinen et al. 1988; Staron et al. 1994). However, other studies suggest how these changes may not always occur or may be a transient response to changes in training characteristics (volume, intensity) rather than a chronic adaptation (Häkkinen et al. 1987; Ahtiainen et al. 2003; Kraemer & Ratamess 2005).

Changes in resting cortisol may indicate the degree of long-term training stress imposed to the body (Kraemer & Ratamess 2005). While increases have in fact been observed after very stressing training periods, chronic exposure to strength training generally reduces or does not affect resting levels of this hormone (Staron et al. 1994; Ahtiainen et al. 2003; Kraemer & Rogol 2005, 224; Kraemer & Ratamess 2005).

Strength training does not seem to affect significantly resting GH concentration, stressing the importance of repeated acute response as mediator of exercise-induced adaptations (Wideman et al. 2002; Ahtiainen et al. 2003; Kraemer & Ratamess 2005). Despite the lack of chronic changes in baseline TSH, transient alterations have been observed in athletes after one week of intense strength training, indicating a potential relation between stress and TSH (Alén et al 1993; Nadolnik 2011).

3.4 Acute responses to strength training

3.4.1 Acute neuromuscular and metabolic responses to strength training

Strength loadings have demonstrated to strongly affect the neuromuscular system (Häkkinen & Pakarinen 1993; Linnamo et al. 1998; McCaulley et al. 2009, Walker et al. 2012). Even though temporary increases in strength and power performance after repeated muscle contractions may be observed under some conditions, acute strength training response is generally characterized by significant decreases in muscle function (Häkkinen & Pakarinen 1993; Linnamo et al. 1998; McCaulley et al. 2009; Walker et al. 2012). The magnitude of these impairments depends mainly on the training characteristics that, in turn, determine the amount of the metabolic and neural demand (Häkkinen & Pakarinen 1993; Linnamo et al. 1998; McCaulley et al. 2009). Larger decreases in maximal strength and rapid force production capacities are related with “metabolic” strength exercises (e.g. hypertrophy schemes) rather than with “neural” loadings (e.g. explosive strength training sessions) (figure 12) (Häkkinen & Pakarinen 1993; Linnamo et al. 1998; McCaulley et al. 2009). However, the high degree of stress imposed on the CNS by particularly intense strength training sessions may lead to significant reductions in neuromuscular performance as well due principally to deterioration in neural drive to muscles (McCaulley et al. 2009; Walker et al. 2012).

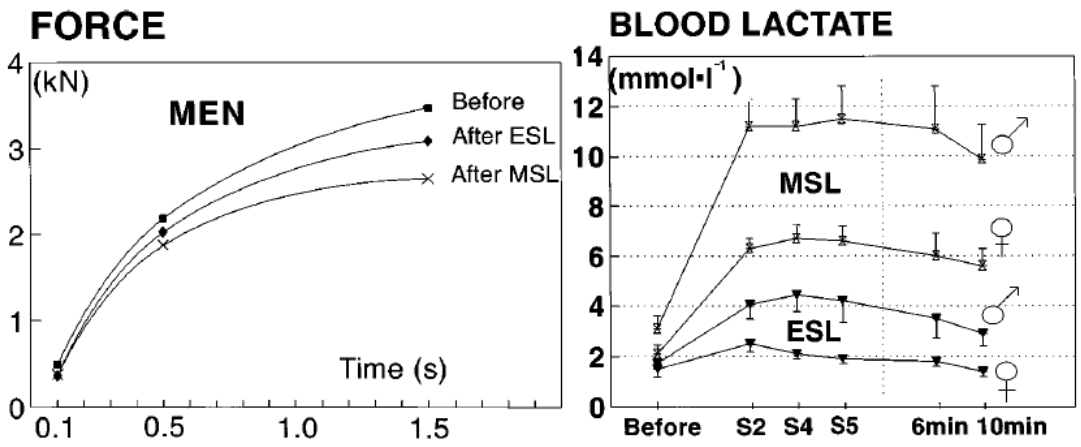

FIGURE 12. Force-time curves measured on isometric leg press dynamometer and blood lactate in response to maximal strength (MSL) and explosive (ESL) loadings (Linnamo et al. 1998).

A reduced acute lactate response to exhausting exercise may be observed after 10 weeks of high volume strength training program in previously untrained subjects (Kraemer et al. 1999). This result seems most likely related to the cardiovascular and metabolic adaptations induced by this type of strength training (Tang et al. 2006; ACSM 2009). However, not all studies confirm this finding (e.g. Ahitiainen et al. 2003). The high strength level, muscle mass and glycolytic enzyme activity generally characterizing strength athletes may also induce a greater lactate response in these subjects compared to untrained individuals, above all during exhausting strength training protocols (Brown et al. 1990). However, given the interplay of multiple factors in determining acute neuromuscular response to strength exercise, longitudinal increase in lactate response may not necessary exclude improvements in fatigue tolerance (Walker et al. 2013).

The magnitude of fatigue and the nature of the training stressor affects, in turn, the recovery time. Whereas two hours seem to be sufficient for a complete recovery after light and explosive resistance exercise, exhausting metabolically demanding strength loading may alter strength and power capacity for longer than 48 h (Linnamo et al. 1998; McCaulley et al. 2009). However, when high training loads are used and a sufficient volume is reached, neural strength training protocols may also lead to a high degree of fatigue due to central nervous origin and prolonged need for recovery (McCaulley et al. 2009).

3.4.2 Acute hormonal responses to strength training

Strength training stresses the endocrine system in a similar manner to endurance and parallels in the patterns of acute hormonal responses to the two different stimuli have been observed when equated for intensity and duration (figure 13) (Jensen et al. 1991; Wideman et al. 2002). The anaerobic glycolytic system strongly stimulates testosterone, cortisol and GH release (Kraemer & Ratamess et al. 2005). Highest acute hormonal concentrations have been observed in response to hypertrophic schemes while smaller or no changes occur after neural loadings (Häkkinen & Pakarinen 1993; Linnamo et al. 2005; McCaulley et al. 2009). The impact of metabolic stress on the endocrine system may also influence the length of recovery. Whereas no changes generally take place after neural strength training protocols, metabolically taxing workouts may lower testosterone levels during recovery longer than 48 h (Häkkinen & Pakarinen 1993).

Training experience has been in part related with acute testosterone, cortisol and, less clearly, GH response (Wideman et al. 2002; Kraemer & Ratamess 2005; Walker et al. 2013). Some, but not all, studies suggest that strength training practice may increase the testosterone response to subsequent acute exercise (Kraemer et al. 1998; Ahtiainen et al. 2004). Although the effects of strength training on the hypothalamic–pituitary–adrenal axis is still a topic of discussion, a recent study of Walker et al. (2013) found a decrease in cortisol response after 20 weeks in recreational active individuals. Differently, the magnitude of acute GH response may either increase or remain unchanged after strength training conditioning (Kraemer et al. 1998; Ahtiainen et al. 2003; Walker et al. 2013).

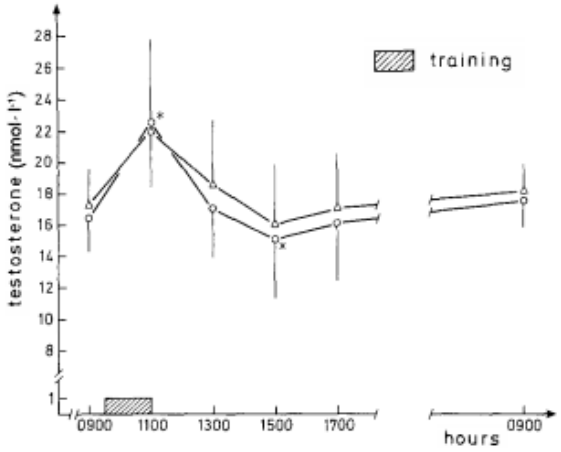

FIGURE 13. Time course of testosterone in response to 90 minutes of strength (Δ) and endurance (○) training equalized for intensity and duration. No significant differences were observed in testosterone levels between the two different training stimuli (Jensen et al. 1991).

4 COMBINED ENDURANCE AND STRENGTH TRAINING

Despite some similarities, endurance and strength training may bring to divergent and potentially competing neuromuscular, metabolic and hormonal adaptative responses (Chromiak & Mulvaney 1990; Leveritt et al. 1999). The decades of studying concurrent development of endurance and strength adaptations have resulted in conflicting evidence about the chronic effects of this type of cross-training. While no significant changes occurred in aerobic capacity in most of the studies, combined training has shown to be able to blunt the long-term hypertrophy, strength and power development (Hickson 1980; Dudley & Djamil 1985; Hunter et al. 1987; Craig 1991; Hennessy & Watson 1994; Kraemer et al. 1995; Bell et al. 2000; Häkkinen et al. 2003; Glowacki et al. 2004; Mikkola et al. 2012). This is especially true when the training volume and intensity are high and the duration of the combined training period is long (figure 14).

However, other studies found no or just minimal impairments in strength outcomes after combined training (Sale et al. 1990a; McCarthy et al. 1995; McCarthy et al. 2002; de Souza et al. 2013; Cantrell et al. 2014). These differences suggest that, even if the combination of the two different stimuli may limit optimal strength gains in some circumstances, this does not always imply a high degree of incompatibility between the two different programs.

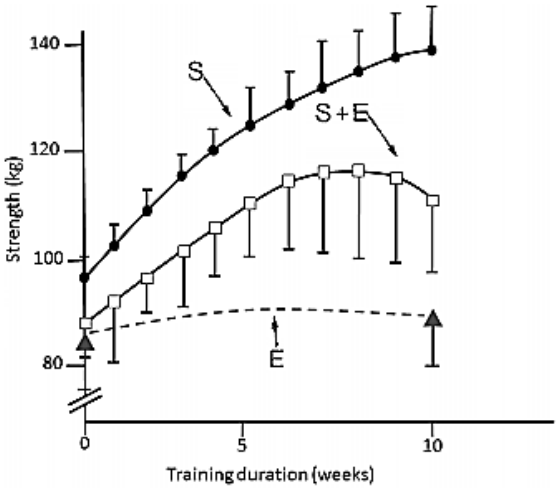

FIGURE 14. Maximum strength development (1RM) in response to strength (S), endurance (E) and high-volume combined (S+E) training periods of 10 weeks (adapted from Hickson 1980).

4.1 Underlying mechanisms of combined training incompatibility

4.1.1 The chronic hypothesis: from molecules to muscles

From a molecular standpoint, endurance and strength training lead to increase muscle protein synthesis through different intracellular signaling pathways, the activation of them is in turn dependent to the type of exercise performed (Nader 2006; Hawley 2009; Fyfe et al. 2014). The PI3K/AKT/mTOR pathway is considered a main mediator for contractile muscle accretion in response to strength training stimulus, by regulating both protein synthesis and degradation (Nader 2006; Hawley 2009; Fyfe et al. 2014). Adaptations to endurance training (i.e. mitochondrial biogenesis) have been instead associated with the activation of the 5'AMP-activated protein kinase (AMPK) signaling, a master regulator of cellular homeostasis (Nader 2006; Hawley 2009; Fyfe et al. 2014). Being regarded as an energy stress sensor, a crucial function of AMPK is to inhibit cellular processes that lead to energy consumption and stimulate those leading to energy production in response to decreased energy levels related to muscle contraction (Nader 2006; Hawley 2009; Fyfe et al. 2014).

In this regard, a sort of incompatibility between the different signaling networks has been proposed (Nader 2006; Hawley 2009). AMPK activation might in fact suppress via crosstalk at several steps in the PI3K/AKT/mTOR pathway, potentially resulting in a blunt muscle hypertrophy in the long term (figure 15) (Nader 2006; Hawley 2009). However, limited evidence supports the applicability of this model on humans. MTOR phosphorylation and myofibrillar protein synthesis may not necessarily be reduced after acute combined training session (Fyfe et al. 2014). Even if a sort of molecular interference occurred, the very short-lived AMPK activation would have a negligible effect on the net muscle protein accumulation between training sessions (Lundberg et al. 2014). Furthermore, long-term anabolic gene expression and muscle development seem not to be affected by 5 weeks of combined training regimen (Lundberg et al. 2014).

These discrepancies must be contextualized in light of the complex interplay existing between the different molecular mechanisms and multitude of potential training variables that condition both endurance and strength training adaptations. For these reasons, the “AMPK-PKB switch hypothesis” cannot be used as an ultimate explanation of the interference phenomenon (Fyfe et al. 2014).

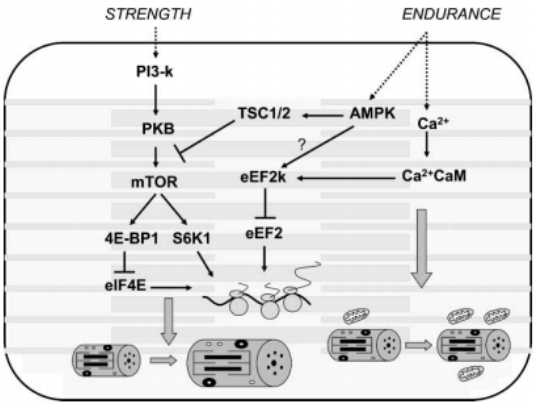

FIGURE 15. The proposed model of interference between the different signaling pathways that mediate skeletal muscle responses to strength and endurance training stimuli (Nader 2006).

Although endurance and strength training may demonstrate similar trends in muscle fiber type conversions, specific differences exist between the two different training-induced muscle phenotypes. The combination of both training stimuli into the same program could then alter the characteristics of contractile proteins respect to single training modes (Chromiak & Mulvaney 1990; Leveritt et al. 1999; Nader 2006). The majority of the studies found no or just a minimal alteration in the fiber distribution pattern between combined training and strength training only (Nelson et al. 1990; Sale et al. 1990a; Kraemer et al. 1995; McCarthy et al. 2002). Nevertheless, Putman et al. (2004) observed how a greater fast-to-slow MHC isoform transitions occurred when both types of training are performed in the same program. In the same study a 2- to 9-fold increase in the size of the type I muscle fibers after strength training only compared to combined training was also observed (figure 16). The lack of hypertrophy of slow-twitch muscle fibres (observed also in Kraemer et al. 1995; Bell et al. 2000; McCarthy et al. 2002) and, to a lesser extent, slower muscle fiber phenotype have been associated with the reduced long-term strength development observed after this type of training (Kraemer et al. 1995; Bell et al. 2000; Putman et al. 2004).

In line with the principle of training specificity, some authors have suggested that skeletal muscle cannot adapt metabolically or morphologically to both strength and endurance training simultaneously (Kraemer et al. 1995; Leveritt et al. 1999). Despite some evidences may support this hypothesis, the high training volume and frequency characterizing most of the above mentioned research should be taken into account when studying muscle adaptations and, more generally, combined training incompatibility.

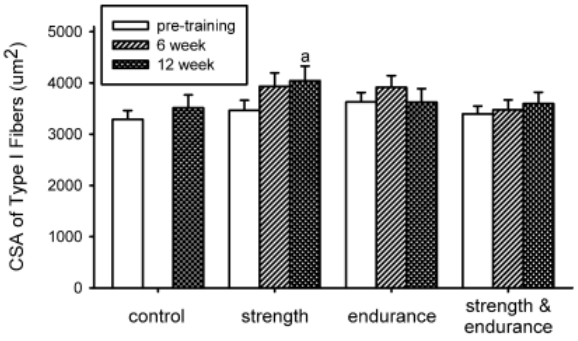

FIGURE 16. Change in CSA of type I fibers in response to different 12-week training programs. No increase occurred after combined strength and endurance training (Putman et al. 2004).

4.1.2 Combined training: a potential source of overtraining?

The augmented stress resulted from the summation of the two different training loads may lead to an interference effect due to the attainment of overtraining condition (Chromiak & Mulvaney 1990; Nelson et al. 1990; Kraemer et al. 1995; Leveritt et al. 1999; Nader 2006; Wilson et al. 2012). This hypothesis is supported by evidences from both neuromuscular and endocrine perspective.

Despite an initial normal development may be observed, strength performance may result impaired after 6-8 weeks of combined training programs when high training frequencies are used (Hickson 1980; Dudley & Djamil 1985; Hunter et al. 1987; Hennessy & Watson 1994; Kraemer et al. 1995; Bell et al. 2000). An increase in basal cortisol levels has been also observed after 6-8 weeks of high-frequency combined training (Kraemer et al. 1995; Bell et al. 2000). The larger catabolic environment has been related with the blunted long-term muscle strength and hypertrophy observed in these studies. Specifically, high cortisol levels seem to contribute enhancing the rate of catabolic events in slow twitch muscle fibers (Kraemer et al. 1995; Putman et al. 2004).

On the contrary, low-frequency periodized combined training programs (≤3 sessions per week) have shown to avoid significant impairments in strength and hypertrophic adaptations without increasing in catabolic hormones (McCarthy et al. 1995; McCarthy et al. 2002; Häkkinen et al. 2003; Glowacki et al. 2004; Shawn & Shawn 2009; de Souza et al. 2013; Mikkola et al. 2012; Cantrell et al. 2014). Furthermore, type I muscle fibers characteristics do not seem significantly altered at these frequencies compared to strength training performed in isolation (McCarthy et al. 2002; Häkkinen et al. 2003).

However, in most cases fast force production capacity results still significantly impaired similarly to what observed at higher frequencies (figure 17) (Häkkinen et al. 2003; Glowacki et al. 2004; Mikkola et al. 2012). A reduced neural capacity to rapidly activate the muscles as response to combined training has been proposed as primary cause of blunt power development (Leveritt et al. 1999; Häkkinen et al. 2003).

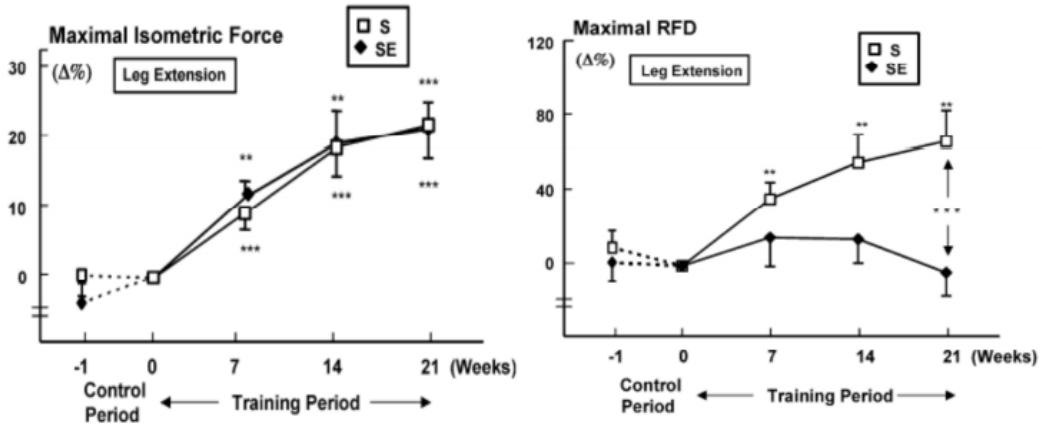

FIGURE 17. 21-week low-frequency combined endurance and strength training program versus strength training alone. No differences occur in MVC between the programs while long-term power performance results impaired in the combined training group (Häkkinen et al. 2003).

A recent meta-analysis has indicated that the magnitude of the interference response on neuromuscular adaptations is mostly influenced by the frequency and duration of the endurance component (Wilson et al. 2012). A subsequent study found how, when strength training component remains unchanged and overall frequency is maintained relatively low, the ratio between the endurance and strength training volumes is directly related with the degree of interference (Jones et al. 2013). These findings seem to provide more support to the hypothesis of physiological incompatibility than to the one of overtraining.

A solid opinion concerning the role of overtraining in determining training interference is missing due to the paucity of studies focusing on the contribution of the endocrine system in this phenomenon. Whereas a prolonged catabolic condition is undoubtedly harmful for strength gains and hypertrophy, the presence of some aspects of combined training incompatibility also at low training frequencies reinforces the idea that endurance training stimulus might be an independent factor in limiting long-term strength development (Häkkinen et al. 2003; Mikkola et al. 2012).

4.1.3 The role of fatigue and recovery in the “interference phenomenon”

Combined training-induced strength impairments have been observed to be specific for the muscles activated (Hunter et al. 1987; Craig et al. 1991; Hennessy & Watson 1994; Kraemer et al. 1995). These results point out the manly local action of combined training incompatibility suggesting a potential critical role of peripheral fatigue in the development of this phenomenon (Craig et al. 1991; Leveritt et al. 1999). Endurance loadings, especially when prolonged and/or intense, produce acute decrements in neuromuscular performance (de Souza et al. 2007). In a combined training program, these decreases, if not totally recovered, may lead to a reduction in the quality of subsequent strength training sessions that, if chronically repeated, may in turn affect optimal strength and power development (Leveritt et al. 1999). From an endocrine perspective, endurance loading may blunt the following strength-induced acute GH response when the two different loading are performed in the same training session (Goto et al. 2005; Schumann et al. 2013; Schumann et al. 2014a). Moreover, when endurance precedes strength (E+S), depressed levels of testosterone have been observed up to 48 h after combined training session (Schumann et al. 2013). These prolonged alterations have been speculated to reflect a superior stress imposed on the endocrine system by this loading order (Schumann et al. 2013).

Although E+S order may negatively affect muscle voluntary activation in the long term, no impairments in strength gains and muscle mass have been generally noticed in previously untrained subjects when moderate training intensity, volume and frequency were used (McCarthy et al. 2002; Shaw & Shaw 2009; Eklund et al. 2014). Under these conditions, relatively large adaptations seem to occur regardless of the timing of the two different stimuli or their order (Schumann et al. 2014a; Schumann et al. 2014b; Eklund et al. 2014). Moreover, the altered testosterone response observed during recovery from E+S was almost normalized after 24 weeks of training (Schumann et al. 2014a).

Differently, intense or exhausting loadings performed in the same training session, or even in the same day, may exacerbate the training interference even when the frequency is low, with the source of fatigue that may be both neural and metabolic in nature (Sale et al. 1990b; Craig et al. 1991; Chtara et al. 2008; Lundberg et al. 2014). However, the superior degree of residual fatigue related to intense or exhausting endurance training protocols may not necessary lead to impairments in neuromuscular performance.

No loadings order effect on strength development has been in fact detected at high training intensities if the frequency is kept low (Collins & Snow 1993; Chtara et al. 2008). It has to be noted that, differently from what has been observed at higher training frequencies, the hypertrophy development does not appear to be affected by these training protocols when the frequency is maintained low irrespective of the loading sequence (Sale et al. 1990a; Sale et al. 1990b; Chtara et al. 2008; de Souza et al. 2013; Lundberg et al. 2014). The resultant between the increase overall stress in response to the training session for the benefit of an augmented duration of recovery between the different training sessions has been proposed as a potential explanation of these findings (Fleck & Kraemer 2014, 152-154). The normal increase in muscle hypertrophy and its contribution on strength performance might in turn explain the physiological strength development observed in some studies despite the potential occurrence of neuronal and/or molecular interference (Folland & Williams 2007; de Souza et al. 2013).

Finally, careful considerations concern the different training modalities. The lower fatigue associated with a moderate and prolonged muscle stimulation occurring during cycling is compatible with the lower degree of neuromuscular impairment observed with this training modality (Gergley et al. 2009; Wilson et al. 2012). Differently, the superior neuromuscular fatigue and muscle damage characterizing prolonged runs seems to exacerbate the interference phenomenon (Gergley 2009; Wilson et al. 2012).

4.2 The effects of strength training on distance running performance

4.2.1 Underlying mechanisms of combined training performance enhancement

In the past, strength training was not common among distance runners because of concerns about possible side effects of muscle gains on capillary density, mitochondrial and enzymatic capacity (Yamamoto et al. 2008). Although earlier studies seemed to confirm this hypothesis, latest evidence has shown that strength training may even increase oxidative enzymatic function in previously untrained individuals (Tang et al. 2006). While the high level of endurance conditioning impedes further improvements in aerobic variables in endurance runners after combined training, it has to be noted that neither adverse effects have been found in these subjects (Hickson et al. 1988).

Similarly, the majority of studies conducted so far have not detected any significant effect induced by combined training on VO_{2max} development when compared to endurance training alone (Hickson et al. 1988; Paavolainen et al. 1999b; Millet et al. 2002; Spurrs et al. 2003; Turner et al. 2003; Saunders et al. 2006; Mikkola et al. 2007; Støren et al. 2008; Guglielmo et al. 2009; Berryman et al. 2010; Ferrauti et al. 2010; Barnes et al. 2013; Sedano et al. 2013).

The improvements observed in strength performance in response to combined training have been almost entirely attributed to neural adaptations to the strength training component and no significant increases in total body and muscle mass occurred in most of the studies (Hickson et al. 1988; Paavolainen et al. 1999b; Millet et al. 2002; Spurrs et al. 2003; Turner et al. 2003; Saunders et al. 2006; Støren et al. 2008; Mikkola et al. 2011). However, small increases in the size of the main trained muscle have been occasionally observed (Mikkola et al. 2007; Taipale et al. 2010). The lack of muscle gain may be the result of a combination of several factors. These include the relative short length of the studies (6-14 weeks), no hypertrophy-specific training design and potential interference effect related to the high training volume and frequency used in these studies (Millet et al. 2002; Taipale et al. 2010).

Despite being potentially lower than those generally observed during strength training isolated, the neuromuscular improvements observed after combined training may improve RE and endurance performance without necessarily affecting VO_{2max} (figure 18) (Hickson et al. 1988; Paavolainen et al. 1999b; Millet et al. 2002; Spurrs et al. 2003; Saunders et al. 2006; Støren et al. 2008; Guglielmo et al. 2009; Berryman et al. 2010; Taipale et al. 2010; Barnes et al. 2013; Sedano et al. 2013; Ramirez-Campillo et al. 2014). The enhanced neuromuscular characteristics may also indirectly affect the contribution of anaerobic glycolysis at submaximal velocities (Rønnestad & Mujika 2013). Some studies report no or little change in the lactate threshold in runners, above all when it is calculated as percentage of VO_{2max} (Hickson et al. 1988; Paavolainen et al. 1999b; Støren et al. 2008; Ferrauti et al. 2010). However, substantial increases in velocity at lactate threshold have been observed (Hamilton et al. 2006; Mikkola et al. 2007; Guglielmo et al. 2009). Improvements in anaerobic running power have been also reported after combined training, potentially providing additional benefits to endurance performance (Paavolainen et al. 1999b; Mikkola et al. 2007).

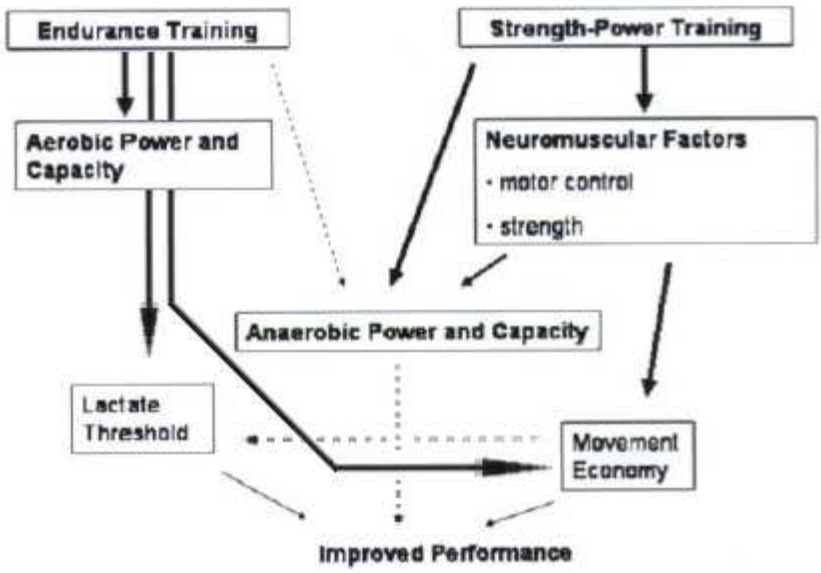

FIGURE 18. Probable interconnected mechanisms that affect endurance running performance during combined endurance and strength training regimens (modified from Beattie et al. 2014).

4.2.2 Optimal strength training design for long-distance runners

Given the positive effect of strength training on endurance performance, several studies have been designed in the attempt to determine the best strength training strategy to optimize these outcomes. Heavy strength training programs have been speculated to improve RE and delay the onset of fatigue mainly via alteration in neuromuscular recruitment pattern (Hickson et al. 1988; Tanaka & Swensen 1998; Millet et al. 2002; Bonacci et al. 2009; Rønnestad & Mujika 2013). An increase in strength and power of the slow twitch muscle fiber may in fact postpone the recruitment of the more fatiguing fast twitch fibers at submaximal velocities. This might in turn decrease the amount of muscle mass activated, permitting a “reserve” of high intensity that can be used for some short sprint phases at the right time during competition (Tanaka & Swensen 1998). Some researchers hypothesized how fatigue determinants may be further influenced by the myosin-isoform transition induced by strength training (Tanaka & Swensen 1998; Yamamoto et al. 2008; Rønnestad & Mujika 2013). However, to date, there is no evidence about this shift within muscle fibers isoforms in endurance runners who integrated strength training into their running routine.

The principal way on which explosive type of strength training may affect running performance seems to be via increased muscle activation rate and changes in MTS regulation (Spurrs et al. 2003; Mikkola et al. 2007). The first study using this kind of training in endurance runners pointed out the importance of high velocities and reaction forces to develop endurance performance (Paavolainen et al. 1999b). Subsequent studies confirmed this approach, also emphasizing the importance of plyometric component in the optimization of training outcomes (Spurrs et al. 2003; Turner et al. 2003; Saunders et al. 2006; Guglielmo et al. 2009; Berryman et al. 2010; Taipale et al. 2010; Barnes et al. 2013). However, not all studies found a correlation between the improvements in neuromuscular characteristics (e.g. changes in MTS) and performance variables investigated (Turner et al. 2003). Differences in training designs and methods should be taken into consideration when interpreting these conflicting results (Turner et al. 2003; Bonacci et al. 2009; Guglielmo et al. 2009; Berryman et al. 2010; Beattie et al. 2014).

Some studies have stressed the superiority of one over the other training modes for the optimization of running outcomes (Guglielmo et al. 2009; Berryman et al. 2010). However, the differences between maximal and explosive strength training programs (or a mix of both) are, in most cases, minimal. Although strength training adaptations have been shown to be specific for each exercise mode, a large inter-individual variability in the training response has been observed among endurance runners (Taipale et al. 2010; Mikkola et al. 2011; Barnes et al. 2013; Taipale et al. 2013). Generally, both types of resistance training appear to be more effective in improving strength and power in endurance runners than other programs (i.e. muscle endurance) untill a sufficient training stimulus is maintained (Taipale et al. 2010; Mikkola et al. 2011; Sedano et al. 2013; Taipale et al. 2013; Taipale et al. 2014a). The strength training frequency seems to be the most critical factor in optimizing performance. Even though a modest development in neuromuscular characteristics may be observed also at low training frequencies, a certain threshold may need to be reached for improvements in endurance performance related variables (Millet et al. 2002; Støren et al. 2008; Taipale et al. 2010; Mikkola et al. 2011; Taipale et al. 2013). A periodized strength training program with a frequency of, at least, 2 times per week is generally considered sufficient to achieve significant strength gains in a period compatible with the duration of the majority of the studies (Yamamoto et al. 2008; Rønnestad & Mujika 2013).

4.2.3 Is intra-session combined training design beneficial for runners?

The addition of the strength training component to the program of endurance runners that already perform a large volume of work may need to combine the two different loadings in the same day or, sometimes, even in the same training session. Intra-session combined training might also be a time-saving strategy for recreational runners or fitness enthusiasts who have a limited amount of time to dedicate to training. However, because of greater loads, volumes and frequencies are needed for further improvements in already trained individuals, this particular combined training design might expose endurance runners to a higher degree of interference than the untrained population.

When the two different loadings are performed close to each other with the aim of optimizing endurance performance, solid concern should be made about their order, especially in already conditioned distance runners. Previous literature has advocated that strenuous strength loadings negatively affect RE and running performance for 6-8 h after the bout, even in subjects already accustomed to strength training (Palmer & Sleivert 2001; Doma & Deakin 2014). Under these conditions, the running load might augment the physiological response induced by strength training (i.e. fatigue, muscle damage) leading to a cumulative stress that may interfere also with the quality of other endurance training sessions (Doma & Deakin 2013). A study of Chtara et al. (2005) has shown that the benefits of strength training on running performance may be canceled when endurance training sessions are repeatedly preceded by acute strength bouts.

The better outcomes in endurance performance variables seem to suggest E+S order as the preferred choice for endurance runners. This training sequence reflects recommendation for subjects competing under fatigued conditions to develop neuromuscular characteristics under similar conditions (Fleck & Kraemer 2014, 189-192). Moreover, recent research stated how train strength within few minutes after moderate to intense runs might also benefit from the augmented acute strength response observed at this time point in endurance trained individuals (Boullosa et al. 2011).

However, although endurance trained individuals seem to be less sensible to the interference phenomenon, the implications that fatigue generated with this training design at high training frequencies may have on strength development should not be underestimated (Hunter et al. 1987; Craig et al. 1991). At moderate to high running

volumes, intra-session combined training has shown to blunt strength training adaptations in trained distance runners similarly to what observed in previously untrained population (Taipale et al. 2010; Mikkola et al. 2011; Taipale et al. 2013). Whether the residual fatigue has played or not a determinant role in these results is difficult to say with the research design used in these studies. Nonetheless, it is worth nothing how, despite no long-lasting alterations in basal catabolic hormones have been observed in endurance runners undergoing this type of training, fluctuations in testosterone and SHBG have been noticed (Taipale et al. 2013; Taipale et al. 2014a). These endocrine alterations may suggest an increased overall training stress occurred despite the relatively low intensities used during these protocols (Taipale et al. 2013; Taipale et al. 2014a). When performed in the same training session endurance and strength training, alteration in force production capabilities have been observed up to 48 h after the session (figure 19) (Taipale & Häkkinen 2013; Taipale et al. 2014b). Then, cumulative fatigue resulting from the sum of the two different loadings might become an important aspect worthy of consideration also during this loading sequence.

On the one hand, the cross-sectional design used in some of these studies has not provided information about the influence of prolonged training on fatigue and endocrine response. Moreover, the lack of longitudinal studies monitoring acute changes and recovery phase has limited the knowledge concerning the effects of neuromuscular and endocrine alterations on long-term strength development and, in turn, endurance performance. A systematic study incorporating both acute and chronic responses needs to determine the effectiveness of this type of combined training in endurance runners.

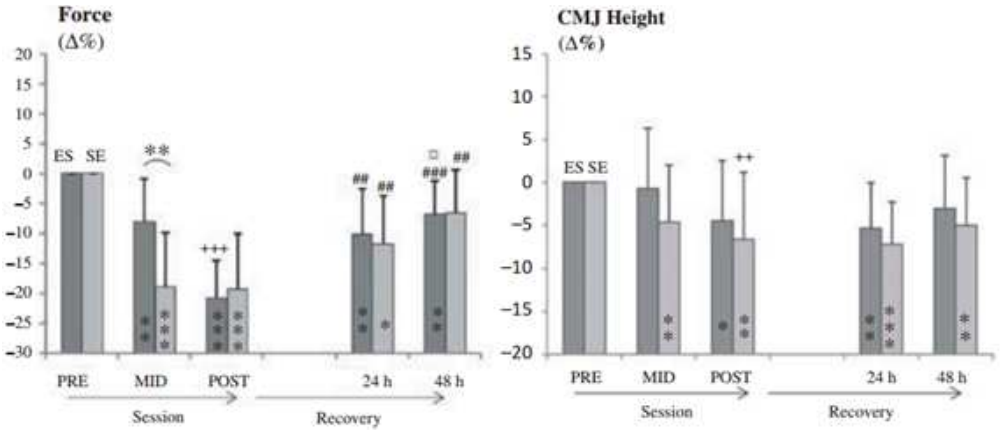

FIGURE 19. Relative changes in MVC and countermovement jump (CMJ) height in response to different loading sequences completed in the same session and recovery (Taipale et al. 2014b).

5 PURPOSE OF THE STUDY

The main purpose of the current study was to evaluate longitudinal changes in the acute response and recovery of neuromuscular performance and serum hormone levels to a prolonged same-session combined training period when strength training is repeatedly preceded by endurance loading (E+S). A further objective was to ascertain whether and to what extent these changes might influence long-term strength training adaptations and endurance performance in recreational endurance runners.

5.1 Research questions

1) Does/How a prolonged same-session combined endurance and strength training program affect the acute neuromuscular and hormonal response to the single training session and related recovery time course in recreational endurance runners?
2) Does/How these changes affect long-term strength training adaptations and endurance performance development?

5.2 Research hypotheses

The hypothesis to the proposed research questions are as follows:

1) The summation of the training loadings will result in acute decline in neuromuscular performance and/or alterations in endocrine function which may also be observed after 24 and 48 h of recovery (Schumann et al. 2013; Taipale et al. 2014b). In case of positive adaptations to the training program, the magnitude and duration of these responses will be reduced at week 12 and 24 (Schumann et al. 2014a). The time course of the endocrine function will not necessary coincide with neuromuscular performance (Taipale & Häkkinen 2013; Schumann et al. 2014a).

2) Limited improvement in neuromuscular characteristics are expected due to the specific loading order (E+S), intra-session combined training design and high training frequencies used in the present training program (Craig et al. 1991; Jones et al. 2013). Modest strength training adaptations will not significantly affect endurance running performance development.

6 METHODS

6.1 Subjects

16 recreationally endurance trained males from the Jyväskylä region participated in this study. The subjects were recruited by the following: newspaper advertisements, flyers were placed around campus and the city, advertisements were also seen on websites and the University of Jyväskylä websites, and e-mails were sent out to the University staff as well as students. The training level was assessed by a self-reported description concerning the endurance and strength training background. All the subjects had a minimum of 1 year of endurance running practice with 2-6 sessions (at both moderate and high intensity) per week and no strength training before the beginning of the study. A meeting was arranged with all the staff of the research project prior the start of the project where subjects were fully informed about the study purpose and measurement procedures. An informed consent was signed by each subject, stating the full knowledge of the subjects about the benefits and risks of participation in this research project. All subjects filled a questionnaire related to their health status, a 12-lead resting electrocardiogram (ECG) was recorded and blood pressure was checked by a cardiologist as part of a medical screening. All subjects reported not having acute and chronic illness, injury and not taking medications contraindicated intense physical efforts and not altering the endocrine function. Height was measured with a wall-mounted metric tape (accurate to 0.1 cm) and weight was measured with a digital scale (accuracy 0.1 kg) in a fasted state, without heavy clothing but with shoes. Not all subjects recruited were able to complete the study period successfully. Factors like minor injuries, medical reasons, motivational and personal issues reduced the number of subjects to 11 who completed the full study period. The demographic characteristics for all subjects who completed the study were as follows (mean±SD): age 32±5 years, body height 179±4 cm and body weight 79.8±5.9 kg. This study was approved by the University Ethical Committee and it was conducted according to the most recent Declaration of Helsinki.

6.2 Experimental design

This study was taken as a part of a bigger experimental design conducted between Autumn of 2011 and late Spring of 2013 by the Department of Biology of Physical Activity of the University of Jyväskylä. A longitudinal protocol, involving 24 weeks of combined training, was used to investigate the research hypotheses. Before the beginning of the training period, a familiarization period of 1 week, concerning training, equipment and measurement protocol, was performed by all subjects. Thenceforth, basal measurements of body composition and strength performance were conducted and serum hormone concentrations (testosterone, cortisol, GH, TSH) and SHBG were determined. Acute strength and endocrine responses to combined loadings were evaluated at PRE, MID and POST of a specifically designed combined training session and recovery capacity was assessed by follow-ups conducted at 24 and 48 h. Endurance performance and related blood lactate response were determined by an incremental exhaustive running test performed in the first part of the combined training session. In order to ensure adequate recovery, this combined training session was separated by baseline tests by at least 48 h of rest. All measurements were repeated after 12 and 24 weeks and were performed with regard to the time of day (within ±1 h of the testing time at week 0) in order to minimize the effect induced by circadian variation in the measured variables (Kraemer & Ratamess 2005; Hackney & Viru 2008). The overview of the study design is presented in figure 20.

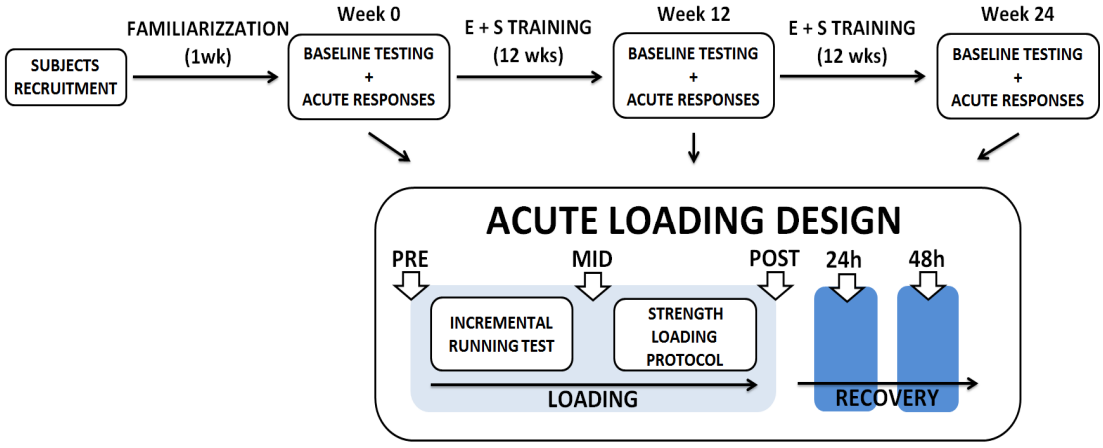

FIGURE 20. Overview of the study design.

6.3 Testing procedures

6.3.1 Basal strength measurements

Strength measurements involved both static and dynamic tests focusing mainly on the leg extensor muscles. In order to allow better insight of the effect of the training program on strength characteristics, different strength assessment methods have been used (Leveritt et al. 2003). All subjects were previously instructed about the correct way to perform tests and required not to drink alcohol for the 24 h and caffeine for the 12 h preceding the beginning of the measurements. Each test was performed at the least three times preceded by a warm up. In case of a difference higher than 5% between the last two trials up to two additional trials were performed. One minute was generally allowed between the trials. The best trial for each test was used for statistical analysis. As part of a larger experimental design only some measurements were used for the data analysis in the current study. The tests performed are listed below and their reliability has been previously reported (Viitasalo et al. 1980; Bosco & Viitasalo 1982).

Isometric horizontal leg press. A horizontal leg press dynamometer (designed and manufactured by the Department of Biology of Physical Activity, University of Jyväskylä, Finland) was used to measure MVC and explosive force production capacity. Hip and knee angle were set at 110 and 107 degrees, respectively and were determined by a hand-held goniometer during the familiarization period. The reference for this setting was the distance between seat and foot plate. The latter was obtained through a measuring tape fixed to the dynamometer also during the familiarization period. To minimize differences in knee angle through the study period, all subjects were required to use the same model of shoes during each measurement session. Subjects were instructed to grip the handles in both sides of the machine with their hands, keeping the back on the seat and pushing faster and strongest they can for at the least 3 seconds following a verbal command. Verbal encouragement was used to attain the maximal effort. The force signal was low-pass filtered at 20 Hz and subsequently analyzed by specific software (Signal 4.04, Cambridge Electronic Design Ltd, Cambridge, UK). MVC was defined as the highest value of force (N) recorded during the test. The average force (N) produced during the first 500ms (F500ms) from the start of the contraction was instead calculated in the the force-time analysis (Häkkinen et al. 2003).

Countermovement jump. A force platform (Department of Biology of Physical Activity, Jyväskylä, Finland) was used to measure maximal power of lower limbs. After a couple of warm ups, subjects performed three maximal CMJs bending the knee as close as possible to 90° with the hands on the hips. The force signal was low-pass filtered at 20Hz and subsequently analyzed by specific software (Signal 4.04, Cambridge Electronic Design Ltd, Cambridge, UK). The CMJ height was calculated from the flight time through the equation: $h=v^2/2g$, (h=jump height, v=take-off velocity) (Komi & Bosco 1978).

One repetition maximum. 1RM test was conducted on dynamic horizontal leg press device (David 210 dynamometer, David Sports Ltd, Helsinki, Finland) after a warm up (1 set of 5 repetitions at 70% of estimated 1RM, 1 set of 2 repetitions at 80-85% of estimated 1RM, 1 set of 1 repetition at 90-95% of estimated 1RM). Seat distance from the plate was set in the way that movement would have ranged from approximately 60° till full knee extension (180°). The same instructions concerning the position of the hands and the back given during the isometric leg press test were repeated in this test. The 1RM was defined as the greatest weight that the subject could successfully lift. An accuracy of 1.25 kg was accepted as 1RM.

6.3.2 Acute strength measurements and recovery

MVC, F500ms and CMJ have been evaluated at PRE and at 24 h and 48 h under the same methodological conditions used during the basal strength measurements. Only two series for each strength test were instead performed at MID (around 10 minutes after the endurance load) and at POST (immediately after the strength load) in the same order as during the basal strength measurements but with only 15 seconds of rest between. Capillary blood samples (20 µl) were taken from the fingertip through small capillaries at PRE, MID and POST immediately before acute strength measurements and put into capsules containing a hemolyzing and anticoagulant solution. Afterward, samples were analyzed with Biosen analyzer (C-Line Clinic, EKF, Magdeburg, Germany), with a sensitivity of 0.5 mmol l^{-1}. For avoiding diluting effects on bodily fluids that could interfere with the measured blood variables, only 2 dl of water were given to individuals who had requested at MID after blood sample was collected.

6.3.3 Endurance measures

Incremental running test. An incremental treadmill protocol was performed by all subjects under controlled conditions immediately after acute strength measurements at PRE. The test started without any warm up with a running velocity of 9 km h^{-1} increasing by 1 km h^{-1} each three minutes till exhaustion with a constant incline of 0.5% (Taipale et al. 2014a). The condition to stop the test was the inability to maintain the running velocity after a first verbal encourage. Five minutes of running on treadmill at the initial speed (9 km h^{-1}) were performed as cool down. Treadmill was stopped for the last 15-20 seconds of each step, when capillary blood samples (20 µl) were taken from the fingertip and stored into reaction capsules and subsequently analyzed. Blood lactate-running speed curve and maximal lactate accumulation were calculated. Peak treadmill velocity (Vpeak) was also calculated and used as predictors of endurance performance (Noakes et al. 1990). Vpeak was calculated as follows: Vpeak=speed of the last whole completed stage ($km \cdot h^{-1}$)+(running time (s) at exhaustion–30 seconds)/(180–30 seconds)*1 $km \cdot h^{-1}$ (Peserico et al. 2014).

6.3.4 Venous blood sampling and body composition

Venous blood samples (10 ml) were collected in the early morning (between 7.00 and 9.00 a.m.) immediately before baseline strength measurements. Additional blood samples were taken also at PRE, MID and POST during the combined training session at week 0, 12 and 24 and at the respective 24 h and 48 h follow-up. The specific instructions for baseline samples were fasting the 12 hours before the blood collection and sleeping at least 8 hours. The sampling was performed from the antecubital vein by a qualified laboratory technician using sterile needles. Collected whole blood was then divided in heparinized tubes (Venosafe, Terumo Medical Co., Leuven, Belgium) and centrifuged at 3500 rpm for 10 minutes (Megafuge 1.0R, Heraeus, Germany). Serum obtained by this process was subsequently stored at -80°C until analysis (approximately 4-8 weeks). Samples were used for determination of serum testosterone, cortisol, GH (22-kD), TSH and SHBG. Baseline testosterone/cortisol (T/C) and T/SHBG ratios were also calculated. Analyses were performed using chemical luminescence techniques (Immunlite 1000, Simens, New York, USA) and hormone-specific immunoassay kits (Siemens, New York, NY, USA). The sensitivity for serum hormones were: testosterone

0.5 nmol·l^{-1}, cortisol 5.5 nmol·l^{-1}, GH 0.03 mlU·l^{-1}, TSH 0.004 mlU·l^{-1} and SHBG 0.2 nmol·l^{-1}. The intra-assay coefficients of variation for testosterone, cortisol, GH, TSH and SHBG were 8.7±2.7%, 7.1±1.1%, 6.0±0.5%, 7.1±4.6% and 6.4±1.7% respectively. The inter-assay coefficients of variation for testosterone, cortisol, GH, TSH and SHBG were 10.6±3.2%, 7.9±1.2%, 5.8±0.3%, 11.1±4.3% and 7.6±1.4%, respectively.

A dual-energy X-ray absorptiometry (DXA) (LUNAR Prodigy, GE Healtcare, Madison, WI) was used to determine body composition of the subjects (Norcross & Van Loan 2004). The DXA machine was calibrated on the same morning of the measurements. Subjects were asked to wear only underwear with no metallic accessories and lay supine on a DXA scanner table. Lower extremities were secured with inelastic straps and the arms were positioned along the body with the palms facing the legs. Automatic analyses (enCORE, version 14.10.022) calculated total body fat mass and fat free mass values.

6.4 Training

All subjects participating in this study performed both single session combined endurance and strength trainings than endurance-only training sessions in the same training schedule. The first 12-weeks training period included two mandatory combined endurance and strength sessions (with strength load carried out within 10 minutes following endurance) plus other three mandatory endurance-only sessions with an optional forth training session per week. In the second 12-weeks training period all the four endurance-only training sessions became mandatory. To permit sufficient time for recovery, the two combined training sessions were separated by at least 48 h and either rest or perform just a light run (35-40 minutes, 60-65% of HR_{max}) was allowed to the subjects on the day before the combined E+S session. Combined training sessions were performed in laboratory and supervised by trained staff while the other endurance training sessions were performed outside the lab individually but constantly controlled and recorded by heart rate monitors (RS800cx, Polar Electro Oy, Kempele, Finland) through pre-programmed exercise routines. Missed workouts were signed and retrieved. All subjects were requested to continue their habitual physical activities throughout the study period. The main training characteristics were set as it to be followed.

Endurance training (at week 1-12/12-24). The aerobic conditioning was achieved through a periodized training design, including both continuous and interval training sessions. Most of training sessions were performed by running. Cycling or cross-country skiing were occasionally allowed in order to reduce the risk of injuries. The exercise intensity was set at target percentages of maximal heart rate (HR_{max}) measured during the incremental treadmill protocol (at week 0 and 12) and was monitored by a Polar® HR-monitor during each endurance training session. Exercise intensity ranged between 60% and 85% of HR_{max} in the first 12-weeks training period. Short interval training sessions of greater intensity (85-95% HR_{max}) were introduced during the second 12-week period (table 3).

Strength training (at week 1-12/12-24). The strength training program was mainly composed by maximal and explosive strength training sessions. Because the lower limbs include the major muscle groups at work in running, the choice of exercises focused mostly on that, though addiction arm and trunk exercises were also included. The intensity of the loads was set on individual 1RM calculated during the preceding baseline strength measurement. During the first 12-week period, training intensity was progressively increased while training volume decreased. During the last 12 weeks, instead, both training volume and intensity were increased in order to maximize maximal and explosive strength gains. Strength training program and exercises performed during the course of the training program are shown in table 4.

Acute strength loading protocol (at week 0, 12 and 24). A modified training protocol was used for the acute strength measurements (Schumann et al. 2013). After a maximal incremental running test, a three-strength loading model mixed with sets of explosive, maximal and hypertrophic strength was performed on the leg press (David 210, David Sports Ltd., Finland) (table 5). A detachable handle connected to a dynamometer was used to assist the repetitions when required. A maximum of 10 minute break was allowed in the transition between the two different training loads. The overall duration of this combined training session ranged between 70 and 80 minutes.

TABLE 3. Endurance training program. Duration and intensity (expressed as % of HR_{max}) are reported. [1]= performed only by running, [2]=not mandatory.

	Combined x2[1]	Long Endurance	Long Intervals[1]	Easy Endurance[2]
Week 1-4	35min/65-85%	70-110min/60-65%	20min/80%	35-40min/60-65%
Week 5-8	40min/65-85%	75-115min/60-65%	20min/85%	35-40min/60-65%
Week 9-12	45min/65-85%	80-120min/60-65%	25min/85%	35-40min/60-65%

	Combined x2[1]	Long Endurance	Long Intervals[1]	Easy Endurance	Short Intervals[1]
Week 13-16	40min/65-85%	85-115min/60-65%	20min/80%	40min/60-65%	12-15min/85-90%
Week 17-20	45min/65-85%	90-120min/60-65%	25min/85%	40min/60-65%	15-20min/90-95%
Week 21-24	50min/65-85%	95-130min/60-65%	30min/85%	40min/60-65%	20-25min/90-95%

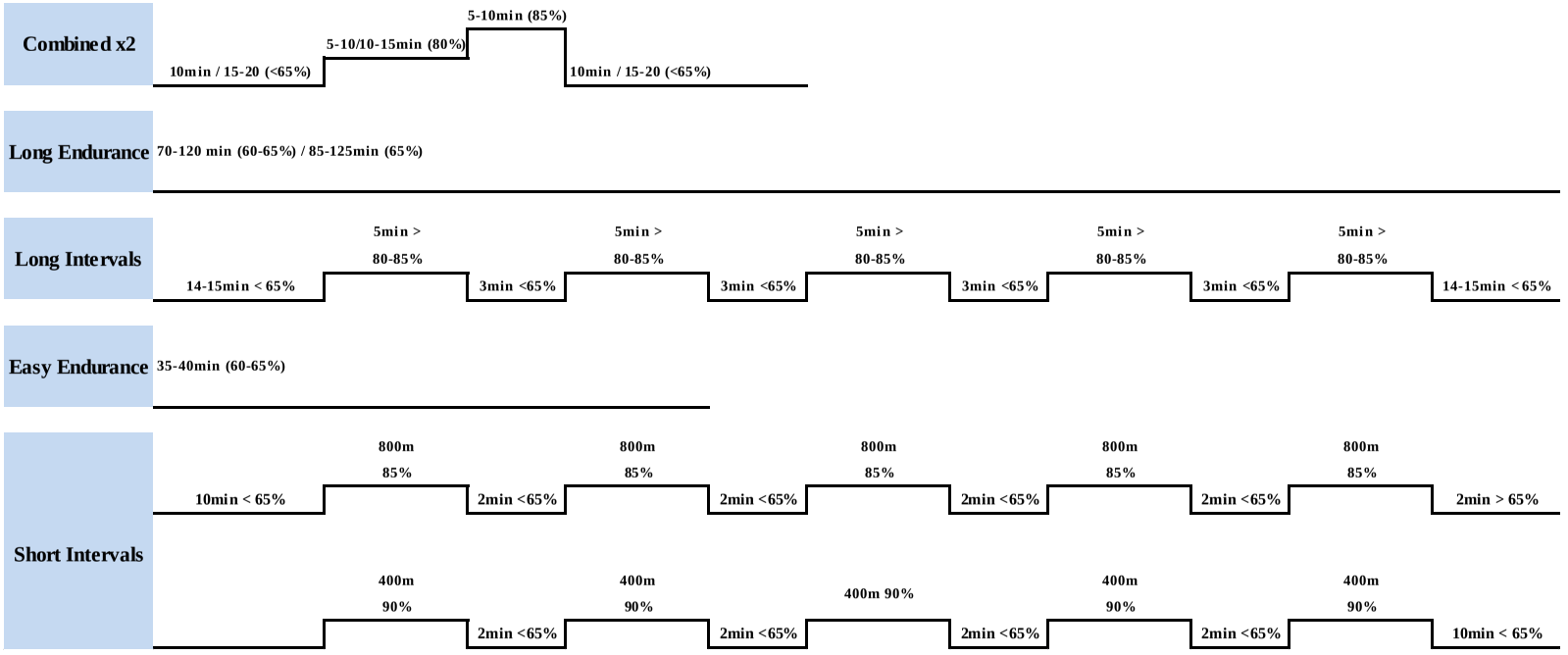

TABLE 4. Strength training program. Volume (number of series per repetitions), intensity (% of 1RM) and duration of rest periods are reported.

[1,2,3,4]=performed alternately during the microcycle.

	Week 1-4	Week 5-8	Week 9-12
Leg press	2-3x20-25/40-50%/1-2 min	2-3x8-12/60-80%/60-90 sec	2-3x4-5/80-85%~3-4x8-10/40-50%/150-180 sec
Lunges			
Step Up with Leg Lift	2-3x10-15/BW/1-2 min	2-3x5-7/BW/150-180 sec[1]	2-3x4-8/BW/150-180 sec
Leg extension			2-3x4-5/75-85%/150-180 sec
Leg curl	2-3x20-25/40-50%/1-2 min	2-3x8-12/60-80%/90-120 sec	
Standing calf raise	2x15-30/BW-20 kg/1-2 min	2x5-6/20-30%/90-120 sec[1]	
Abdominal crunches	2-3x15-25/BW/1-2 min	2-3x10-12/1,25-5 kg/90-120 sec	2-3x12-16/1,25-5kg/90-120 sec
Hip extension	2-3x15-25/BW/1-2 min	2-3x10-12/2,5-7,5 kg/90-120 sec	2-3x12-16/2,5-7,5kg/90-120 sec
Vertical jumps	2-3x10-15/BW/1-2 min	2-3x8-12/20 kg/150-180 sec[2]	
Drop jumps		2x10-12/BW/150-180 sec[2]	2-3x8-14/BW/150-180 sec[4]
Box jumps			2-3x16-20/BW/150-180 sec[4]
Hurdle hops			
Hurdle drills			
Military press		2x8-12/60-80%/90-120 sec[3]	2x10-14/60-75%/90-120 sec[3]
Biceps curl	2x20-25/40-50%/1-2 min	2x8-12/60-80%/90-120 sec[3]	3x12-14/60-75%/90-120 sec[3]
Resistance running with bands			
Ball chest throw			
Overhead ball throw			

	Week 13-14	Week 15-16	Week 17-20	Week 21-24
Leg press	2-3x20-25/40-60%/2-3 min	3x5-6/75-80%/2-3 min	3-4x3-4/80-85%~2-4x8-10/40-50%/2-3 min	2-3x5-8/40-50%~3x3-4/75-80%/2-3 min
Lunges	2-3x10-15/BW/2-3 min			
Step Up with Leg Lift		2-3x5-10/BW-12,5kg/2-3 min	2-4x6-7/15-25kg~2-4 x6-7/12,5kg/2-3 min	3x12-14/15-25kg/2-3 min
Leg extension	2-3x20-25/40-50%/2-3 min			3x3-4/75-80%/2-3 min
Leg curl		2-3x5-6/75-80%/2-3 min	2-4x5-6/75-80%/2-3 min	
Standing calf raise	3x15-25/BW–12,5 kg/2-3 min	2-3x15-20/12,5 kg - 17,5kg/2-3 min	2-3x15-20/12,5-17,5kg/2-3 min	
Abdominal crunches	3x15-25/BW/2-3 min	2-3x12-15/1,25 kg-2,5kg/2-3 min	2-3x12-15/75-80%/2-3 min	2-3x12-15/1,25-2,5kg/2-3 min
Hip extension	3x15-25/BW/2-3 min	2-3x12-15/2,5 kg - 5kg/2-3 min	2-4x12-15/2,5-5kg/2-3 min	2-3x10-12/2,5-5kg/2-3 min
Vertical jumps	2-3x10-15/BW–12,5 kg/2-3 min	2-3x8-10/12,5kg/2-3 min		
Drop jumps			2-3x10-14/BW/2-3 min	
Box jumps				
Hurdle hops				2-3x5/BW/2-3 min
Hurdle drills				2-4x10/BW/2-3 min
Military press		2-3x6/75%/2-3 min[3]	2-3x12-25/40-65%2-3 min[3]	3x10-12/70-75%/2-3 min
Biceps curl	2x20-25/40-50%/2-3 min	2-3x25/40%/2-3 min[3]	2-3x12-25/40-65%2-3 min[3]	
Resistance running with bands				1-3x10-20/BW/2-3 min
Ball chest throw				1-3x5-10/BW/2-3 min
Overhead ball throw				1-3x5-10/BW/2-3 min

TABLE 5. Strength training acute loading. Number of series, repetitions, intensity (% of 1RM) and rest period duration are reported. [1]=additional weight was added if the previous set appeared to be too light for the subject in order to achieve at least one set of repetition maximum.

Explosive	**10x40% Leg press explosive**
	3min rest
	10x40% Leg press explosive
	3min rest
	10x40% Leg press explosive
	3min rest
Maximal	**3x75% Leg press**
	3min rest
	3x90% Leg press
	3min rest
	3x90% Leg press[1]
	3min rest
	3x90% Leg press[1]
	2min rest
Hypertropic	**10x75% Leg press**
	2min rest
	10x80% Leg press[1]
	2min rest
	10x80% Leg press[1]
	2min rest
	10x75% Leg press

6.5 Statistical analysis

Means, standard deviation (SD) and percent changes (%) within group were calculated with standard statistical methods. Shapiro-Wilk and Levene's Test were used to verify the normal distribution and homogeneity of the parameters, respectively. Logarithmic transformation was performed when necessary. In normally distributed parameters, within-group differences were assessed by repeated measures ANOVA using Bonferroni adjustments by multiplying all pairwise p values with the number of comparisons. Bivariate correlations were computed using Pearson's product moment correlation coefficients (r). In non-normally distributed parameters after log transformation, within-group differences were analyzed by Friedman test using Wilcoxon signed-rank post-hoc test with Bonferroni adjusted significance levels. Bivariate correlations were instead computed using Spearman's rho (r). The level of significance for all tests was set at *, §, #=$p \leq 0.05$, **, §§, #=$p \leq 0.01$, and ***, §§§, ###=$p \leq 0.001$. Any observed trend ($p < 0.08$) was also reported [(*), (§), (#)]. Statistical analysis was performed using IBM SPSS 20.0 (SPSS, Inc., Chicago, IL, USA).

7 RESULTS

7.1 Anthropometry

No significant changes were observed in body weight in the group of subjects during the overall combined training period (79.8±5.9 kg at week 0). A small but significant decrease in fat free mass (-2.3±0.8%, p=0.044) instead occurred after 24 weeks of training (66.9±6.2 kg) counterbalanced by a significant increase in total body fat mass.

7.2 Treadmill running test

Peak treadmill velocity. Vpeak (17.0±1.0 $km \cdot h^{-1}$ at week 0) increased nonlinearly during the 24-week combined training period (figure 21). A significant improvement in Vpeak was observed at the end of the intervention period (+4±4%, p=0.009) with the greatest increases that took place in the last 12 weeks of training (+3±2%, p=0.008).

Lactate kinetics. A significant reduction in blood lactate levels at 15 $km \cdot h^{-1}$ and a decreasing trend in blood lactate levels at 14 $km \cdot h^{-1}$ and were observed in the last 12 weeks of training (14 $km \cdot h^{-1}$: -15±19%, p=0.07; 15 $km \cdot h^{-1}$: -20±17%, p=0.02) as well as after the overall training period (14 $km \cdot h^{-1}$: -18±28%, p=0.084; 15 $km \cdot h^{-1}$: -23±19%, p=0.035) (figure 22). Also a significant reduction in blood lactate concentration at 16 $km \cdot h^{-1}$ occurred after 24 weeks of training (-19±21%, p=0.036). No significant changes in maximal lactate accumulation were observed in any measurement point.

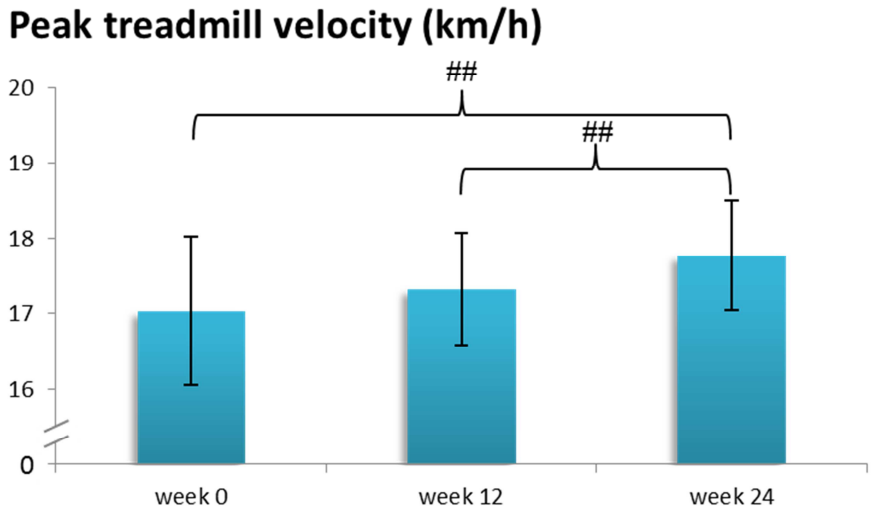

FIGURE 21. Peak treadmill velocity (mean±SD) at the beginning, after 12 weeks and after the whole 24-week training period. #=significant difference between time points (##=p<0.01).

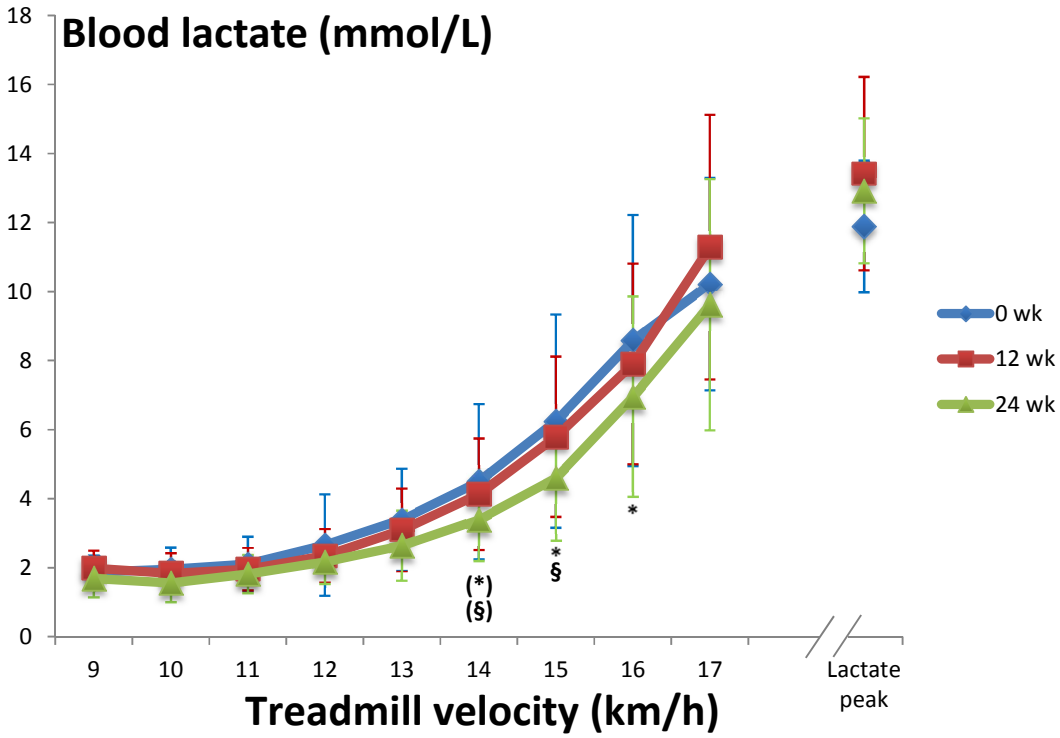

FIGURE 22. Blood lactate-running speed curve and maximal lactate accumulation during the incremental treadmill test (mean±SD) at the week 0, at week 12 and at week 24. *=significant difference from week 0 to 24, §=significant difference from week 12 to 24 (*, §=p<0.05). (*)=trend from week 0 to 24, (§)=trend from week 12 and 24 [(*), (§)=p<0.08].

7.3 Chronic strength adaptation

The present 24 weeks of combined endurance and strength training program did not produce any measurable change in MVC (2667±455 N at week 0) or in F500ms (1769±350 N at week 0). Neither 1RM (164.5±22 kg at week 0) nor CMJ (32±3 cm at week 0) demonstrated significant changes after 12 or 24 weeks of training (figure 23).

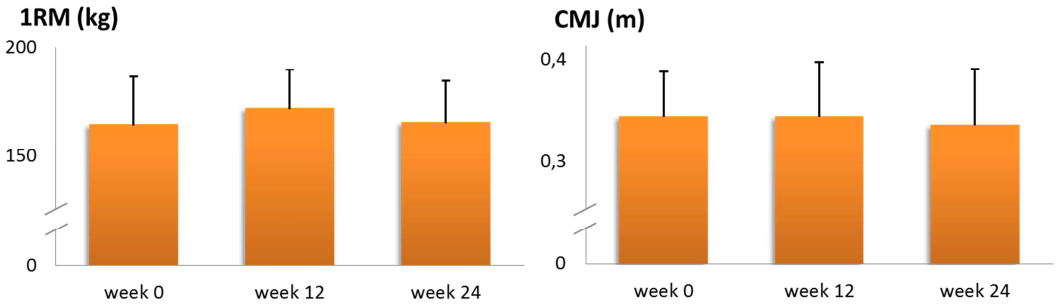

FIGURE 23. Changes in one repetition maximum and countermovement jump performance (mean±SD) occurred during the 24-week combined endurance and strength training period.

7.4 Acute strength response

Maximum force produced on the isometric horizontal leg press. At week 0, significant decreases in MVC occurred both after E at MID (-11±10%, p=0.03) and after E+S at POST (-22±11%, p=0.014) and a decreasing trend was observed from MID to POST (-12±14%, p=0.079) (figure 24). No significant decreases in MVC were longer observed after E at MID at week 12 and 24 while significant reductions were still observed after E+S at POST (week 12: -14±7%, p=0.01; week 24: -20±8%, p=0.01) and also detected from MID to POST (week 12: -12±9%, p=0.03; week 24: -18±17%, p=0.018). MVC was completely recovered by 24 h at all time points.

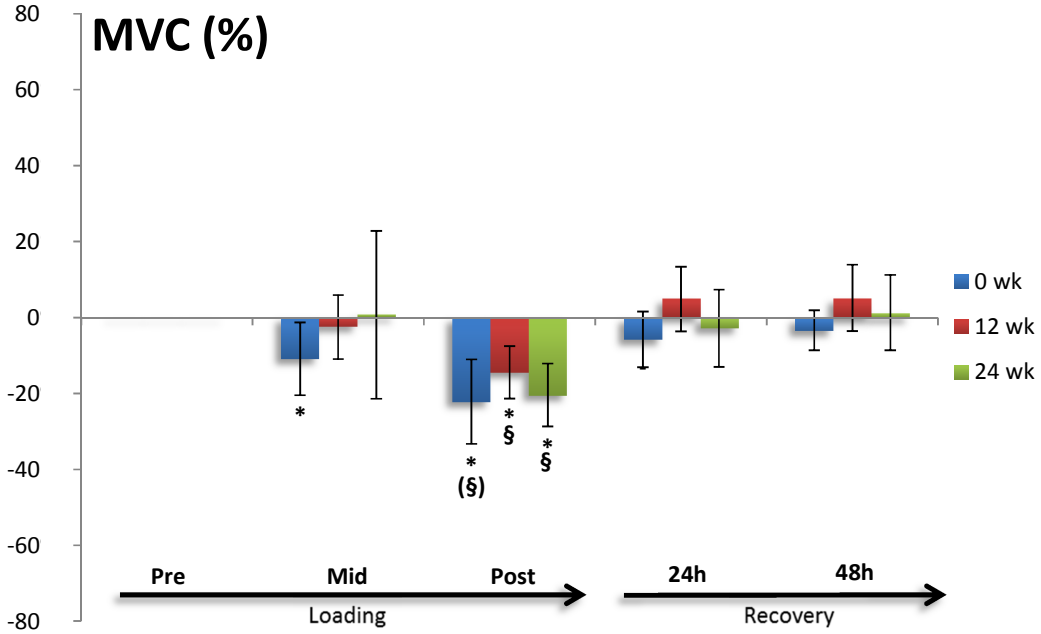

FIGURE 24. Relative changes (mean±SD) in maximal voluntary isometric contraction during the acute loadings and recovery over the 24-week training period. *=significant difference from PRE to MID and from PRE to POST, §=significant difference from MID to POST (***, §** =p<0.05). (§)=trend observed from MID and POST [(§)=p<0.08)].

Explosive force produced on the isometric horizontal leg press. At week 0, a moderate downward trend was observed in F500ms after E at MID (-12±3%, p=0.063) and significant decreases occurred after E+S at POST (-24±13%, p=0.01) and from MID to POST (-13±17%, p=0.049) (figure 25). No acute decreases in F500ms were longer observed after E at MID and after E+S at POST after 12 weeks of training and the F500ms measured after E+S at POST was significantly higher than the corresponding value at week 0 (+6±19%, p=0.017). However, at week 24, significant decreases in F500ms were still observed after E+S at POST (-18±12%, p=0.01) and from MID to POST (-15±16%, p=0.038) with the magnitude of the decrease at POST that returned to what observed at week 0 (-7±8%, p=0.045). No significant reductions in F500ms were detected at 24 h or 48 h during the study period.

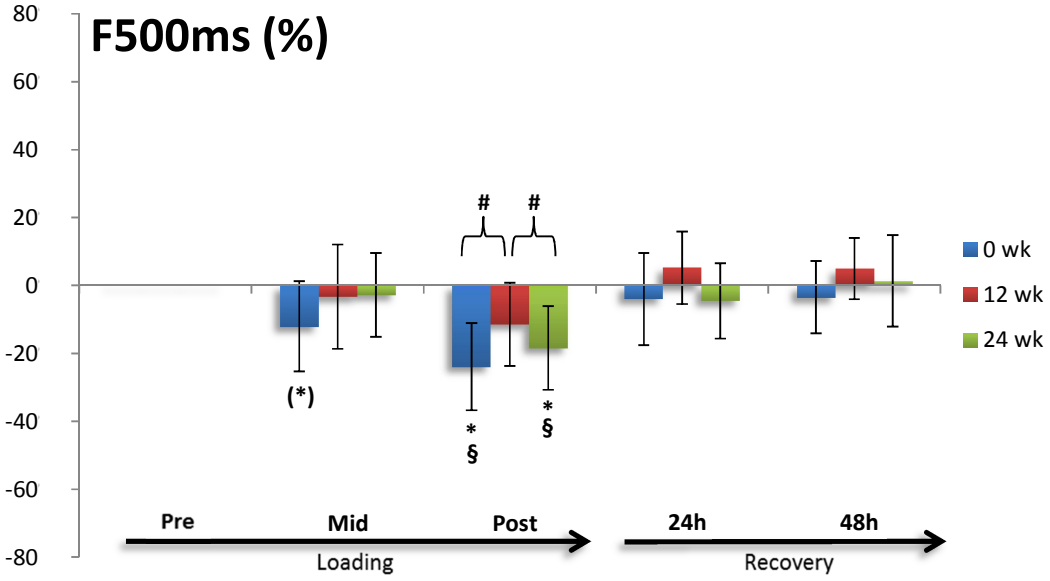

FIGURE 25. Relative changes (mean±SD) in force produced in the first 500ms during the acute loadings and recovery over 24 weeks of training. *=significant difference from PRE to POST, §=significant difference from MID to POST, #=significant change at POST at different time points (***, §, #**=p<0.05). (*)=trend observed from PRE to MID, [(*)=p<0.08].

Countermovement jump performance. CMJ remained statistically unaltered after E at MID at week 0 while significant decreases were observed after E+S at POST (-12±9%, p=0.023) and from MID to POST (-14±6%, p=0.01) (figure 26). An upward trend in CMJ was detected after E at MID after 12 weeks of training (+6±7%, p=0.063). Despite CMJ decreased significantly after the strength loading from MID to POST (-9±7%, p=0.028), no significant reduction was observed in this variable after E+S at POST compared to PRE at week 12 and a significant improvement occurred also compared to the corresponding measurement point at week 0 (+9±10%, p=0.033). The increasing trend in CMJ performance after E at MID was no longer observed at week 24 and, similarly to what has been observed at week 0, significantly decreases were detected after E+S at POST (-6±7%, p=0.049) and from MID to POST (-10±6%, p=0.01). No significant changes occurred in CMJ at 24 h or 48 h during the training intervention.

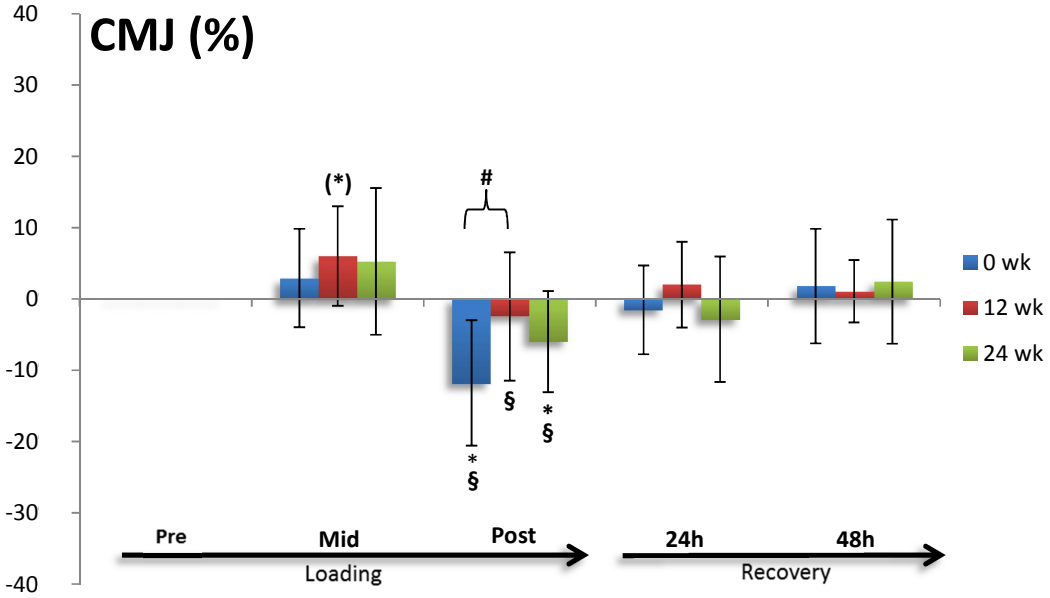

FIGURE 26. Relative changes (mean±SD) in countermovement jump performance during the acute loadings and recovery over the 24-week training period. *=significant difference from PRE to POST, §=significant difference from MID to POST, #=significant change at POST at different time points (***, §, #**=p<0.05). (*)=trend observed from PRE to MID, [(*)=p<0.08].

7.5 Chronic hormonal adaptation

No significant changes in the basal serum levels of cortisol (495±147 nmol l^{-1} at week 0), GH (1.0±1.7 mlU l^{-1} at week 0) and in the T/C ratio (4±1% at week 0) were observed during the 24-week combined training period (figure 27). Baseline serum TSH level (1.94±0.82 mlU l^{-1} at week 0) increased significantly during the first 12 weeks of training (+25±24%, p=0.028) but it was no longer elevated at week 24. A large but not significant decrease in basal serum testosterone level (18.8±15 nmol l^{-1} at week 0) was observed at week 12 (-13±35%) while a significant increase occurred during the last 12 weeks (+46±42%, p=0.021). T/SHBG ratio (0.6±0.3 at week 0) followed a similar time course to testosterone with a significant reduction observed at week 12 (-16±18%, p=0.004) and a subsequent significant increase during the last 12 weeks of training (+25±23%, p=0.001) which reported T/SHBG ratio to pretraining levels (figure 28).

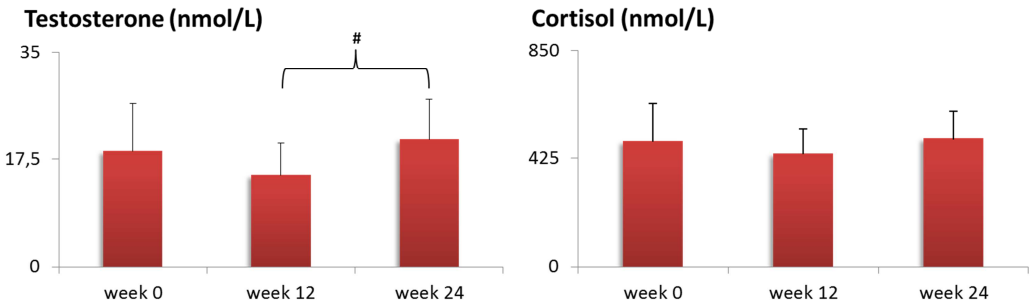

FIGURE 27. Changes occurred in baseline serum testosterone and cortisol levels (mean±SD) during the course of the 24-week combined endurance and strength training period (#=p<0.05).

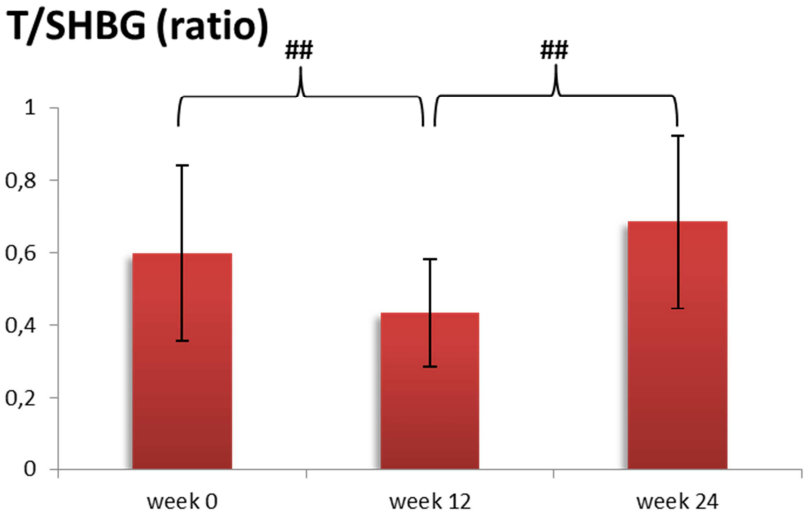

FIGURE 28. Changes occurred in testosterone/SHBG ratio (mean±SD) during the course of 24-week combined endurance and strength training period (##=p<0.01).

7.6 Acute hormonal response

Serum testosterone. A significant increase occurred in acute testosterone response after E at MID at week 0 (+30±28%, p=0.049) and 24 (+32±35%, p=0.013) (figure 29). An upward trend was also observed in acute testosterone response after E+S at POST at week 0 (+23±29%, p=0.079) whereas a significant decrease occurred from MID to POST at week 24 (-15±13%, p=0.01). No significant alterations were observed in the acute testosterone response at MID and POST at week 12. No significant changes were detected in serum testosterone levels at 24 h and 48 h of recovery in the study period.

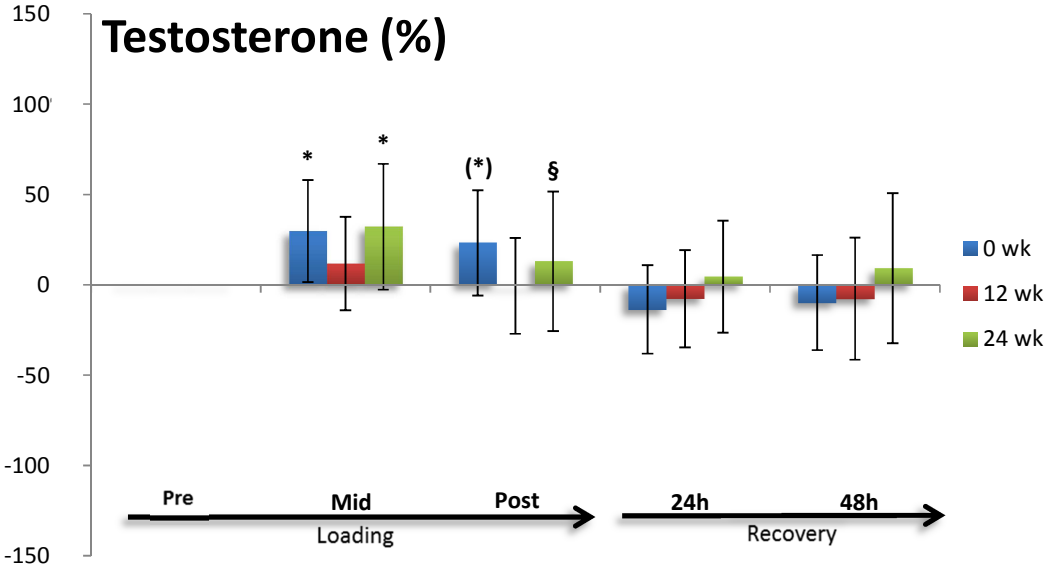

FIGURE 29. Relative changes (mean±SD) in serum testosterone levels during the acute loadings and recovery over the 24-week training period. *=significant difference from PRE to MID, §=significant difference from MID to POST (***, §**=p<0.05). (*)=trend observed from PRE to POST, [(*)=p<0.08].

Serum cortisol. Significant increases in acute cortisol response occurred after E at MID at week 0 (+41±28%, p=0.01), 12 (+51±34%, p=0.013) and 24 (+38±30%, p=0.023) (figure 30). Acute cortisol response remained statistically elevated after E+S at POST at week 0 (+39±36%, p=0.012) but not at week 12 and 24. A decreasing trend in the acute cortisol response was observed from MID to POST after 12 weeks of training (-14±16%, p=0.079), while a significant decline occurred from MID to POST at week 24 (-15±9%, p=0.013). A decreasing trend in serum cortisol levels was observed after 24 h of recovery at week 0 (-24±31%, p=0.057) while significant decreases were detected at this time point at week 12 (-28±27%, p=0.026) and 24 (-27±16%, p=0.007). Depressed serum cortisol levels were also observed after 48 h of recovery during the entire duration of the training period (week 0: -31±21%, p=0.015; week 12: -29±25%, p=0.02; week 24: -26±26%, p=0.026).

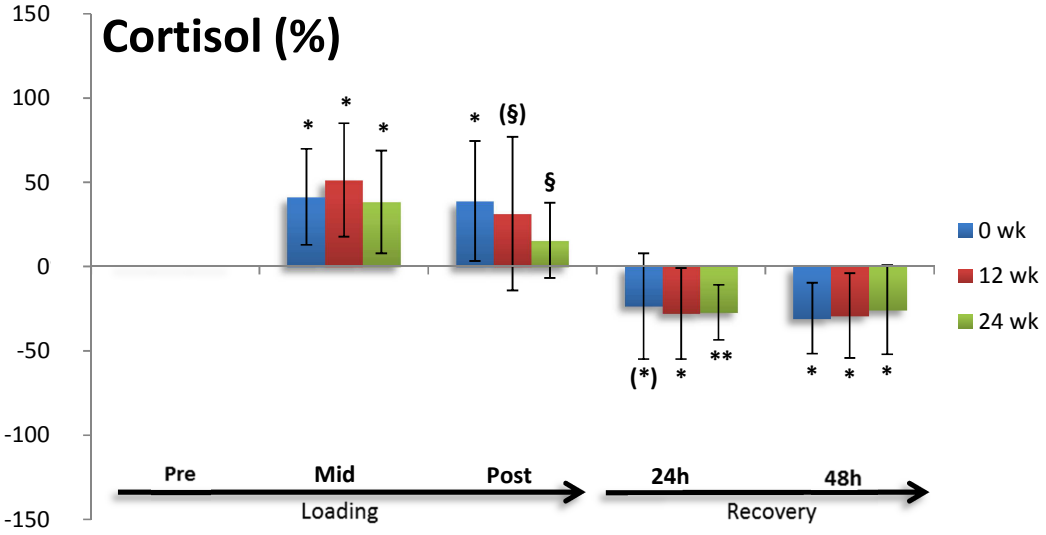

FIGURE 30. Relative changes (mean±SD) in serum cortisol levels during the acute loadings and recovery over the 24-week training period. *=significant difference from PRE to MID, from PRE to POST, from PRE to 24 h and from PRE to 48 h, §=significant difference from MID to POST (***, §**=p<0.05, **=p<0.01). (*)=trend observed from PRE to 24 h, (§)=trend observed from MID to POST [(*), (§)=p<0.08].

Serum growth hormone. Significant increases in acute GH response occurred after E at MID at week 0 (215±206 fold, p=0.01), 12 (227±190 fold, p=0.01) and 24 (225±275 fold, p=0.01) (figure 31). The acute GH response was also significantly elevated after E+S at POST at week 0 (59±50 fold, p=0.01), 12 (55±51 fold, p=0.018) and 24 (43±61 fold, p=0.013) but with a significant reduction occurred from MID to POST (week 0: -61±24%, p=0.01; week 12: -72±14%, p=0.01; week 24: -79±11%, p=0.01). No significant changes in serum GH levels were detected after 24 h or 48 h of recovery during the study period.

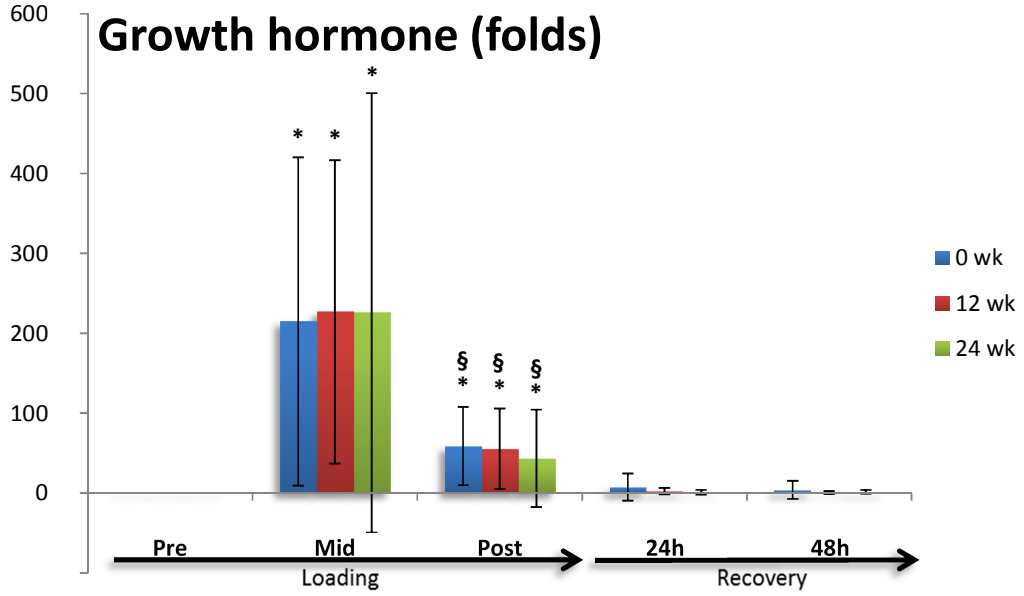

FIGURE 31. Changes (mean±SD) in serum growth hormone levels during the acute loadings and recovery over the 24-week training period. *=significant difference from PRE to MID and from PRE to POST, §=significant difference from MID to POST (***, §**=p<0.05).

Serum thyroid-stimulating hormone. No significant changes occurred in acute TSH response after E at MID at week 0 and 24 but an upward trend was observed at week 12 (+22±24%, p=0.066) (figure 32). Significant increases in acute TSH response were observed after E+S at POST (+46±45%, p=0.028) and also from MID to POST (+25±19%, p=0.021) at week 0. Only an increasing trend was detected in acute TSH response after E+S at POST after 12 weeks of training (40±37%, p=0.066) whereas no changes occurred at week 24. No significant changes in serum TSH levels were oberved after 24 h of recovery at any time point but a significant reduction was observed after 48 h of recovery at week 0 (-23±24%, p=0.044).

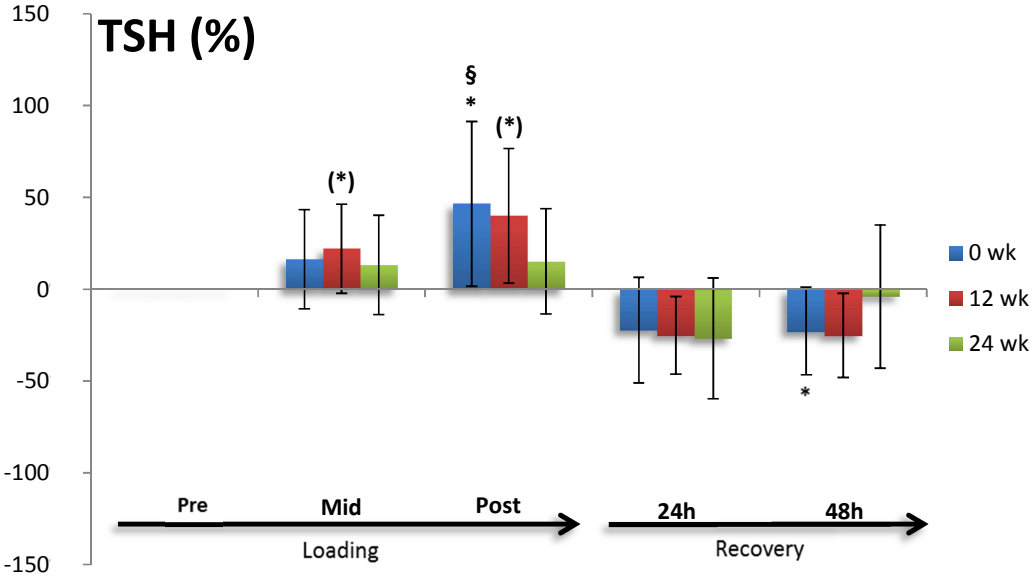

FIGURE 32. Relative changes (mean±SD) in serum TSH concentrations during the acute loadings and recovery over the 24-week training period. *=significant difference from PRE to POST and from PRE to 48h, §=significant difference from MID to POST (***, §**=p<0.05). (*)=trend observed significant difference from PRE to MID and from PRE to POST, [(*)=p<0.08].

7.7 Acute blood lactate response

Blood lactate concentration significantly increased after E at MID during the training period (week 0: 3.83±1.6 fold, p=0.01; week 12: 1.98±1.16 fold, p=0.01; week 24: 3.71±1.82 fold, p=0.01) (figure 33). After 12 weeks of training, a downward trend in blood lactate levels was observed after E at MID compared to week 0 (-33±51%, p=0.075) and a significant increase in blood lactate values was observed from MID to POST (+75±94%, p=0.038). No longitudinal changes were detected in blood lactate values after E+S at POST that remained significantly evelated compared to PRE at any measurement point (week 0: 8.87±2.7 mmol l^{-1}, p=0.01; week 12: 7.17±2.03 mmol l^{-1}, p=0.01; week 24: 7.84±2.13 mmol l^{-1}, p=0.01).

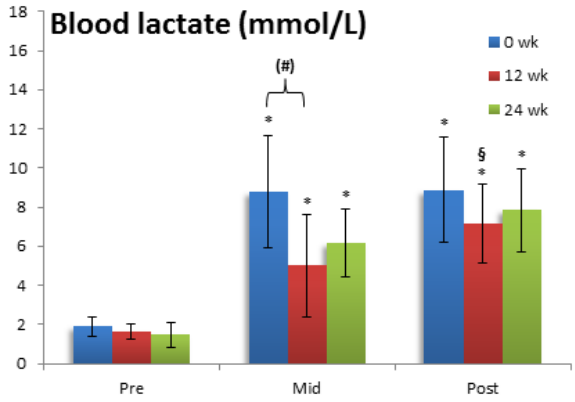

FIGURE 33. Acute blood lactate levels (mean±SD) during the acute loadings. *=significant difference from PRE to MID and from PRE to POST, §=significant difference from MID to POST (***, §**=p<0.05). (#)=trend observed at MID at different time points, [(#)=p<0.08].

7.8 Correlations between the variables

Significant positive correlations were observed between the blood lactate values measured after E at MID and after E+S at POST at week 0 and 24 (week 0: r=0.607, p=0.048; week 24: r=0.739, p=0.009). In the same weeks, significant correlations were also detected between CMJ measured at MID and POST (week 0: r=0.654, p=0.029; week 24: r=0.685, p=0.020). At week 12, CMJ after E at MID was negatively correlated with acute blood lactate response observed after E+S at POST (r=-0.771, p=0.009).

Significant correlations were observed between Vpeak and blood lactate at submaximal speed at week 0 (14 km·h^{-1}: r=-0.635, p=0.036; 15 km·h^{-1}: r=-0.695, p=0.018; 16 km·h^{-1}: r=-693, p=0.018), 12 (14 km·h^{-1}: r=-0.802, p=0.003; 15 km·h^{-1}: r=-0.717, p=0.013, 16 km·h^{-1}: r=-732, p=0.010) and 24 (14 km·h^{-1}: r=-0.808, p=0.003; 15 km·h^{-1}: r=-0.833, p=0.001, 16 km·h^{-1}: r=-871, p<0.001). Significant correlations were also observed between blood lactate at submaximal speed and F500ms at MID at week 12 (14 km·h^{-1}: r=-0.768, p=0.006; 15 km·h^{-1}: r=-0.673, p=0.023, 16 km·h^{-1}: r=-670, p=0.024) and 24 (14 km·h^{-1}: r=-0.624, p=0.040; 15 km·h^{-1}: r=-0.634, p=0.036, 16 km·h^{-1}: r=-637, p=0.035) and between Vpeak and F500ms value at MID in the same weeks (week 12: r=0.663, p=0.026; week 24: r=0.689, p=0.019). Finally, a strong correlation was observed between the low basal levels of testosterone and the related reduced acute responses observed at week 12 (MID: r=0.911, p<0.001; POST: r=0.947, p<0.001).

8 DISCUSSION

The main purpose of the current study was to examine changes in the acute response and recovery during a 24-week intra-session combined endurance and strength training program in neuromuscular performance and serum hormone concentrations. The impact of these changes on long-term strength training adaptations and endurance performance was further investigated. The main findings of this study were: (1) The current same-session combined endurance and strength training design induced moderate but significant acute loss in force production capabilities observed both at week 0 and 24, with the larger decreases observed after the strength load. At week 12, however, power performance was not significantly reduced at the end of the training session. (2) A large overall acute endocrine response was observed during the combined training session at week 0. Although no longitudinal change took place in the hormones investigated after the running load, progressive acute decreases in cortisol and TSH responses were observed at the end of the training session along the course of the study (3). While recovery of force production after the combined loadings was already completed at 24 h, prolonged endocrine alterations (cortisol and TSH) were observed at week 0, 12 and 24. (4) No long-term improvements in neuromuscular performance were detected during the study period. (5) Significant increases occurred in endurance performance during the course of the combined training period. These improvements were correlated with power performance only after the first 12 weeks.

8.1 Changes in acute neuromuscular and hormonal response

As expected, the present same-session combined training sessions design resulted in significant decreases in neuromuscular performance at week 0 and 24. Despite the different loading characteristics, the magnitude of these declines was comparable to that observed in recreational endurance runners after similar combined training sessions (Taipale & Hakkinen 2013; Taipale et al. 2014b). Interestingly, an upward trend in CMJ performance was observed at MID after 12 weeks of training and the same variable was no longer reduced at POST compared to PRE.

Both resistance exercises and prolonged runs have been shown to lead to acute decrease in force production capabilities (Linnamo et al. 1998; Nicol et al. 2006). Specifically, high-intensity running have shown to induce acute elevated neuromuscular fatigue in large part due to the extensive use of anaerobic metabolism (Joyner & Coyle 2008). However, an acute increase in power performance (CMJ) has been also noticed following intense running protocols in endurance trained subjects (Vuorimaa et al. 2006; Boullosa et al. 2011). An explanation for these differences may come from Tillin & Bishop (2009) stating how the acute strength response to exercise is the result of opposing fatigue and potentiation effects on the neuromuscular system induced by muscle contraction. Despite the exact mechanisms of this acute potentiation have not yet been elucidated, changes in muscle activation due to increased Ca^{2+} sensitivity and enhanced stretch reflex activity have been proposed as mediating factors (Tillin & Bishop 2009; Boullosa et al. 2011). Because these two physiological mechanisms are also among those affected by metabolic fatigue, both increased potentiation and reduced by-product accumulation may translate into an enhanced neuromuscular response (Cairns 2006; Tillin & Bishop 2009). Since the longitudinal decrease in blood lactate levels observed at MID after 12 weeks does not fully explain the improved CMJ performance occurred at the same time point, a key role played by neuromuscular potentiation in determining these results should not be excluded (Tillin & Bishop 2009).

One of the main issues to consider when planning same-session combined endurance and strength training is the impact of residual fatigue on the quality of the strength load (Craig et al. 1991). Whereas no deep analysis of neuromuscular function was performed in the present study, significant correlations in lactate values between MID and POST were observed at week 0 and 24. These correlations go in parallel with those occurred in CMJ performance at the same time points. Despite the neural component characterizing explosive and maximal strength sets, the acute strength load used in the current study has been previously observed to evoke an elevated lactate response associated with a significant decrease in neuromuscular performance (Schumann et al. 2013). The aforementioned correlations may reflect a cumulative metabolic stress occurred during the combined training session. However, at week 12, none of these correlations was detected but a negative relation has been established between the increase in CMJ performance at MID and blood lactate at POST. These data seem to confirm how, when an intense endurance load leads to a reduction in strength production capabilities, the quality of the subsequent strength training session may be affected if performed before a

complete recovery has occurred (Craig et al. 1991). Differently, when potentiation overcomes fatigue, the enhanced neuromuscular response might reduce the effort required to sustain the subsequent strength load and, consequently, the use of anaerobic metabolism. This, in turn, could permit to perform more work before the onset of metabolic fatigue impairs physical performance, as suggested by the significant increase in lactate levels occurred after strength loading at week 12 (Gorostiaga et al. 2014). Despite only CMJ improved at MID and POST after 12 weeks, also F500ms exhibited a similar time course, while MVC did not. The brief duration of the effect of potentiation on subsequent muscle contractions (100-150 ms) and differences in muscle action (SSC vs isometric) may explain these results (Tillin & Bishop 2009).

Differently from what has been previously observed in endurance runners undergoing intra-session combined training, in the current study, recovery in strength related variables was already achieved at 24 h (Taipale & Häkkinen 2013; Taipale et al. 2014b). Among the several factors that may affect the time course of recovery in neuromuscular characteristics, muscle damage has been suggested to have a main role in deterioration of strength and power capacities, especially after prolonged runs (Komi 2000; Nicol et al. 2006). Although the absence of markers of muscle damage makes it difficult to draw definitive conclusions, it can be speculated that the relatively short length of the running load has resulted in a limited degree of damage to muscle tissue compared to what has been observed in other studies (Taipale & Häkkinen 2013; Taipale et al. 2014b). The low training volume characterizing the acute strength loading and the prolonged rest periods used in explosive and maximal strength sets could have further contributed to limit the duration of recovery (Linnamo et al. 1998; McCaulley et al. 2009).

It is worth noting that, in the current study, the magnitude of the acute hormonal response to the present combined training and related recovery time course did not follow the same time course of neuromuscular performance. This finding has been already observed in both endurance runners and previously untrained individuals performing different combined training session designs (Schumann et al. 2013; Taipale & Häkkinen 2013; Schumann et al. 2014a). While testosterone levels were already recovered at 24 h, cortisol and TSH levels dropped during the early recovery, remaining suppressed both at 24 and 48 h (even though they did not always reached the statistical significance). Because the hormone receptor status has not been assessed in the present study, these low levels may reflect either impairments in the hypothalamic-pituitary-

adrenal axis or an increased utilization of these hormones for remodeling processes. Decreases in cortisol have been observed after prolonged and exhaustive endurance runs (Hackney & Dobridge 2009). TSH depression has been instead detected after prolonged and intense resistance training (Alén et al. 1993). However, a similar time course in cortisol and TSH response during recovery has been also observed after combined endurance and strength training session of moderate intensity and duration in previously untrained subjects (Schumann et al. 2013, 2014a). One can speculate how the combination of the two different loadings has resulted in a superior stress on the endocrine system that might have in turn prolonged the need of recovery independently by the timing of neuromuscular response. Because both hormones are also involved in the modulation of endurance training adaptation processes, these changes might also indicate a metabolic priority towards mitochondrial than myofibrillar protein synthesis during the early recovery phase (Goffart & Wiesner 2003; Taipale & Häkkinen 2013).

The greater increase in the overall hormonal response observed at both MID and POST reveals that the acute combined training protocol used in the current study was more stressing for the endocrine system than the one used by Taipale & Colleagues, which not resulted in prolonged alterations in hormonal levels (Taipale & Häkkinen 2013; Taipale et al. 2014b). Differently from other hormones, GH resulted in a blunted response at POST at all time points as already observed with this loading sequence (Goto et al. 2005; Schumann et al. 2013; Taipale & Häkkinen 2013; Schumann et al. 2014a). No important longitudinal changes occurred in the altered hormonal levels during recovery in the present study. However, although the degree of the endocrine response at MID remained pretty constant for the entire length of the study, the magnitude of the cortisol and TSH response at POST decreased progressively at week 12 and 24. These changes might indicate that an adaptation process occurred in response to either the overall combined training session or just the strength loading (Walker et al. 2013). Despite the current research design cannot provide an unequivocal answer, the endurance training background of the subjects participated in this study gives an indication for the latter one. The lack of changes in the time course of recovery of the endocrine function might then suggest that the superior stress related to this acute combined training design has overcome the endocrine system adaptability. Before drawing conclusions, however, it should be stressed that the follow-up measurements were performed only at 24 and 48 h. Therefore, longitudinal adaptations occurred in the endocrine response over this time period (48 h) may not be excluded.

8.2 Chronic adaptations in physical performance and endocrine function

No chronic improvements in neuromuscular performance were observed after 12 and 24 weeks of combined training in the present research. This is a surprising finding considering the length and characteristics of the strength training program as well as the absence of a previous strength training background in the subjects participating in this study (ACSM 2009; Rønnestad & Mujika 2013). The lack of strength development might have occurred due to the particular characteristics of the combined training design used in the present study. A similar lack in lower body strength gains was observed only by Craig et al. (1991) in previously untrained subjects undergoing strength training immediately after endurance running three times per week. Despite the periodized approach used in the current study, the intra-session design and the relatively high training intensity and frequency might have lead to a large degree of interference impeding any strength development (Craig et al. 1991; Jones et al. 2013). However, great caution should be exercised when interpreting the results since no experimental group performing only endurance training was included in the present research design. In fact, endurance conditioning may have a negative impact on neuromuscular characteristics in the long term lowering strength production capabilities in endurance trained individuals (Fitts & Widrick 1996). Therefore, the contribution from the strength training component in maintaining strength capacity during the course of the combined training program cannot be excluded.

Previous research suggests that the summation of the two different types of training modes may overcome the recovery capacity of the body leading to an elevated catabolic state which could, in turn, affect muscle mass and strength development (Kraemer et al. 1995; Bell et al. 2000). Despite the high training volume related to this training design, the baseline cortisol levels and T/C ratio did not change significantly at any time point and just transient alterations were observed in testosterone, TSH and T/SHBG ratio at week 12. These fluctuations may result from seasonal variations in the endocrine function (Leppäluoto et al. 1998; Smith et al. 2013). However, they may also be compatible with an augmented stress imposed to the body by the new training program (Häkkinen et al. 1987; Taipale et al. 2013; Taipale et al. 2014a). Significant correlations have been noted between the low baseline testosterone level and the reduced acute response for the same hormone observed both at MID and POST at week 12. These

relationships stress about the possibility that the transient endocrine alteration observed in basal testosterone after 12 weeks may have conditioned training adaptability at that specific time point. Nonetheless, these alterations were no longer observed at week 24 and just minor changes occurred in muscle mass during the course of the program. These data suggest that, even if the endocrine homeostasis was initially perturbed, subjects were capable to adapt to the new training stressor. When interpreting these data, one must keep in mind that the characteristics of the strength loading used during the acute measurements were intentionally selected to reproduce the overall periodized strength training stimulus through the combination of explosive, maximal and hypertrophic leg press protocols. Differently, the running protocol used in the combined training sessions performed during the normal training schedule was less demanding compared to the one used during the acute loading measurements. Then, a lower strain may have been put on the endocrine system compared to what has been detected during the acute measurements (Galbo et al. 1977; Vuorimaa et al. 2008).

While the lack of strength gains might discourage runners from undergoing this combined endurance and strength training plan, an analysis of longitudinal changes occurred during the training period may still reveal important information about the impact of this program on running performance-related variables. Increases in CMJ performance after intensive runs have been mostly observed in well-trained endurance athletes being strongly related with running experience (Vuorimaa et al. 2006). The increase in CMJ observed at MID after only 12 weeks of training might then reflect an adjuvant effect provided by the strength training component in improving this aspect of running conditioning. Similarly, the improvements occurred in F500ms at the same time point may indicate an enhanced capacity to maintain muscles activated under fatigue conditions which generally characterize the fastest and most economical runners (Paavolainen et al. 1999c; Nummela et al. 2008). The positive correlations observed between acute F500ms response at MID, blood lactate at submaximal speed and Vpeak observed at week 12 further reinforce the idea about an initial contribution of strength training adaptations in the development of endurance running performance. Although RE was not directly assessed in this study, these correlations suggest how endurance performance may have benefited from the positive effects that enhanced neuromuscular characteristics exert on mechanisms that determine metabolic fatigue, in accordance with the current state of knowledge (Rønnestad & Mujika 2013; Beattie et al. 2014).

Notwithstanding the potential implications of these early adaptations in conditioning running performance, their importance has been largely reconsidered in the second part of the training period. At the end of the 24 weeks, in fact, CMJ performance measured at MID was returned to its pre-training level, emphasizing a discontinuity in the potentiation phenomenon observed at week 12. Furthermore, although no further improvement occurred in acute F500ms response in the last 12 weeks and, both Vpeak and related submaximal blood lactate response improved significantly. The correlation between these two variables became also stronger while that between Vpeak and F500ms did not change. Importantly, once submaximal lactate response had been treated as covariate, no correlation was anymore observed between acute F500ms response and Vpeak after 24 weeks. These results suggest that the improvements occurred in submaximal lactate response and Vpeak during the last 12 weeks, that were also the largest ones occurred during the training intervention, have been probably caused by other factors rather than by strength training (i.e. increased endurance training volume and intensity). For this reason, it can be concluded that, despite the strength training component may have in part contributed to the running performance development during the first 12 weeks of training, its overall impact on these variable during the present study has been modest, if any.

Using the same single session loadings order, Chtara et al. (2005, 2008) observed significant improvements in both endurance and strength performance. The gains in endurance performance were larger than those occurred in the group performing only endurance training. It should be stressed, however, how the subjects participating in that study were moderately active but systematically untrained for both loadings. Since the strength training part was more “aerobic” than the one used in the current study, this may have favored the improvements observed in VO_{2max} and, in turn, endurance performance. The reduced training frequency used in that study (2 sessions per week) may have also minimized the interference between the two different training components and this may explain the occurrence of strength gains despite the use of a low-specific strength training protocol. No large changes in VO_{2max} were expected in the present study, due to the endurance training background of the subjects. Moreover, the high training frequency and intensity required to endurance runners to further develop their condition might have amplified the problem of training interference, above all with the same-session design used in this study. This may have counteracted the benefits generally achieved by endurance runners when adding strength training.

It is worth noting that with a similar combined training design but a slightly lower training intensity than used in the current study, Taipale et al. (2010) observed significant increases in neuromuscular performance after 14 weeks. Importantly, the largest significant improvements in endurance performance observed in that study occurred after a subsequent period of reduced strength training and increased endurance training volume and intensity. According with the authors, the neuromuscular improvements occurred during the first 14 weeks may have contributed to enhanced endurance performance by preparing subjects for the subsequent increases in endurance training volume and intensity. These results suggest that different periodization schemes might bring different training outcomes from the same combined training program.

8.3 Strengths and limitations of the current study

The strengths of the current study are related to the well-planned, scientifically based, longitudinal design. The large number of variables assessed at both acute and basal level throughout the 24 weeks of the study duration has permitted to obtain a wide and comprehensive overview of changes occurred during this period. However, the absence of a group performing endurance training alone has in part limited the interpretation of these findings. Because the nutrition status has been proven to affect hormonal response to exercise, the lack of fasting blood samples during the follow-ups may have affected the information obtained (Hackney & Viru 2008). However, given that most of the confounding factors were controlled, the impact of nutrition on the endocrine response has been minimized. Despite all the combined training sessions have been carried out in the lab, the large part of endurance training has been performed by subjects on their own. Although careful instructions and a HR-monitor recording every training session have been given to each subject, the adherence to the training program cannot be fully guaranteed. Lastly, the findings of this study are specific for endurance runners and they may not be applicable to populations from endurance disciplines other than running.

8.4 Conclusions and practical applications

The present study provides important information concerning the effectiveness of an intra-session combined endurance and strength training design in recreational endurance runners through the analysis of the physiological responses occurred during the training period. These results suggest that, although the quality of strength training can get benefits when preceded by an intense running protocol, this phenomenon does not necessarily occur in every training session and its impact on the long-term running performance development may be insignificant. Differently, the combination of the two different training loads in the same session leads to an augmented stress to the endocrine system that may take several days to recover. From a long-term perspective, performing strength training always after endurance at high frequencies can impede strength gains, offsetting the benefits that improved neuromuscular characteristics bring to endurance runners. For all these reasons, endurance runners should perform endurance and strength training sessions separately, or using periodically also strength and endurance orders.

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