ACUTE EFFECTS OF HYPERTOPHIC- AND NEURAL STRENGTH EXERCISE ON ENDURANCE PERFORMANCE, RUNNING ECONOMY, RUNNING KINEMATICS AND LOWER LIMB MUSCLE ACTIVATION

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TIIVISTELMÄ

Auvinen, E-P. 2023. Hypertrofisen ja hermostollisen voimaharjoituksen akuutit vaikutukset kestävyyssuorituskykyyn, juoksun taloudellisuuteen, juoksun kinematiikkaan ja alaraajojen lihasaktiivisuuteen. Liikuntatieteellinen tiedekunta, Jyväskylän yliopisto, Biomekaniikan pro gradu -tutkielma, 77 s., 2 liitettä.

Johdanto. Voimaharjoittelulla tiedetään olevan positiivisia vaikutuksia juoksun taloudellisuuteen ja kestävyyssuorituskykyyn. Lihasta vaurioittava voimaharjoitus voi akuutista heikentää voimaharjoituksen jälkeen tehtävää kestävyysjuoksu harjoitusta, mikä voidaan havaita muutoksista juoksun taloudellisuudessa, kinematiikassa sekä lihasaktiivisuudessa. Kun voimaharjoitus toistetaan uudestaan nämä heikennykset saattavat olla pienentyneitä hermolihasjärjestelmä suojelumekanismin ansiosta. Tätä mekanismia kutsutaan toistoharjoitusvaikutukseksi (repeated bout effect). Tämä työn tarkoituksena on tutkia, miten hermostollinen ja hypertrofinen voimaharjoitus vaikuttaa akuutista juoksun taloudellisuuteen, kinematiikkaan ja lihasaktiivisuuteen 48 tuntia myöhemmin tehtävässä kestävyystestissä. Tarkoituksena on myös saada tietoa onko toistoharjoitusvaikutuksella merkitystä näissä muuttujissa, kun harjoitukset toistetaan kolme kertaa.
Menetelmät. 12 kestävyyskuntoilijaa jaettiin hermostolliseen (HER) ja hypertrofiseen (HYP) ryhmään. Tutkimus sisälsi 2 esimittausviikkoa ja 3 tutkimusviikkoa. Koehenkilöt suorittivat ensimmäisen (kontrolli) kestävyystestin ilman aiempaa voimaharjoitusta. Seuraavat 3 kestävyystestiä suoritettiin 48 tuntia joko hypertrofisen tai hermollisen voimaharjoituksen jälkeen, ryhmän mukaisesti. Maksimaalinen isometrinen jalkaprässi ja esikevennyshyppy suoritettiin ennen jokaista kestävyystestiä, kuten myös lihasarkuuden subjektiivinen arviointi visuaalisen skaalan (VAS) avulla. Kestävyystestin aikana mitattiin, laktaattia, hapenkulutusta, sykettä, koettua kuormittuneisuutta, uupumukseen kulunutta aikaa, juoksun 2D kinematiikkaa sekä lihasaktiivisuutta.
Tulokset. Hypertrofinen voimaharjoitus aiheutti suuren määrän lihaskipua kerralla 1, mutta kipu väheni kerroilla 2 ja 3. Juoksun taloudellisuus, juoksun kinematiikka ja lihasaktiivisuus eivät osoittaneet merkittäviä muutoksia ensimmäisen voimaharjoituksen jälkeen. VO2 tippui merkitsevästi anaerobisella kynnyksellä, kun verrataan ensimmäisen ja toisen voimaharjoituksen jälkeen suoritettuja testejä (45.6±6.9 vs. 44.3±6.5 ml/kg/min, $p < 0.05$). Samoin RER pieneni merkitsevästi, kun verrataan ensimmäisen ja kolmannen voimaharjoituksen jälkeen suoritettuja testejä (1.01±0.03 vs. 0.98±0.03, $p < 0.05$). HER-ryhmässä ei havaittu voimaharjoituksen aiheuttamia muutoksia juoksun taloudellisuudessa. Maksimaalinen kestävyyssuorituskyky parani merkittävästi molemmilla ryhmillä.
Johtopäätökset. Hypertrofinen voimaharjoittelu aiheutti merkittävää lihasvauriota, mikä johti pieniin muutoksiin juoksu taloudellisuudessa, juoksun kinematiikassa ja lihas aktiivisuudessa. Nämä muutokset vähenivät seuraavissa voimaharjoituskerroissa, mikä viittaa toistoharjoitusvaikutukseen. Toisaalta hermostollisella voimaharjoittelulla oli vain vähäinen vaikutus juoksun taloudellisuuteen. Tulosten perusteella näyttää, että hypertrofisen voimaharjoittelun suorittaminen voi vaikuttaa negatiivisesti 48 tuntia myöhemmin tehtävään kestävyysharjoituksen, kun taas hermostollinen voimaharjoitus ei näytä vaikuttavan 48 tuntia myöhemmin tehtävään kestävyyssuoritukseen.

Asiasanat: kestävyysjuoksu, yhdistelmäharjoittelu, lihasaktiivisuus, juoksun kinematiikka, juoksun taloudellisuus, toistoharjoitusvaikutus

ABSTRACT

Auvinen E-P. 2023. Acute Effects of Hypertrophic and Neural Strength Exercise on Endurance Performance, Running Economy, Running Kinematics and Lower Limb Muscle Activation. Faculty of Sport and Health Sciences, University of Jyväskylä, Master's thesis of Biomechanics, 77 pp. 2 appendices.

Introduction. It has been long established that strength training has beneficial effects on endurance performance by improving running economy. Strength exercise induced fatigue can cause acute decrement of the subsequent endurance running session, which can be seen in changes in running economy, kinematics and muscle activation. When endurance exercise is repeated these changes might attenuate due to protective mechanism of neuromuscular system, called repeated bout effect (RBE). This study examines how neural and hypertrophic strength exercises impact running economy, kinematics, and muscle activation during a running economy test performed 48 hours later. It also evaluates if the repeated bout effect affects these factors when the exercises and test are done three times.

Methods. 12 participants were divided to neural (NEU) and hypertrophic (HYP) group. Study concluded of 2 premeasurement weeks and 3 experimental weeks. Participants performed the first running economy (RE) test (control) without prior strength exercise. Then 3 next running economy tests (one per week) was performed 48 hours after either hypertrophic or neural strength exercise, based on assigned group. Maximal isometric leg press and countermovement jump performance was measured prior to all RE tests as well as subjective muscle soreness was assessed with visual analog scale (VAS). During RE test lactate, oxygen consumption, heart rate, rate of perceived exertion, time to exhaustion (TTE), 2D running kinematics and lower limb muscle activity was measured.

Results. Hypertrophic strength exercise induced great amount of muscle soreness after the bout 1 which then decreased to bouts 2 and 3. Running economy, running kinematics and muscle activation did not show any significant changes even after first strength exercise. VO2 significantly decreased at the anaerobic threshold when comparing bouts 1 and 2 (45.6±6.9 vs. 44.3±6.5 ml/kg/min, $p < 0.05$). Similarly, RER significantly decreased from bout 1 to bout 3 (1.01±0.03 vs. 0.98±0.03, $p < 0.05$). NEU group did not show any alterations caused by strength exercise in running economy test. Maximal endurance performance improved significantly for both groups.

Conclusion. It appeared that hypertrophic strength exercise resulted in a significant amount of muscle damage, which led to minor changes in running economy, running kinematics, and muscle activation during running. Nonetheless, these changes decreased during subsequent strength exercise bouts, indicating the occurrence of the repeated bout effect. In contrast, neural strength exercises had a negligible impact on running economy. Therefore, it is possible that performing hypertrophic strength exercises could negatively effect on the endurance workout conducted 48 hours later, whereas neural strength exercises may not.

Key words: endurance running, concurrent training, muscle activity, running kinematics, running economy, repeated bout effect

ABBREVIATIONS

ALB	Alternate leg-bouncing
BF	Biceps Femoris
CMJ	Countermovement jump
CT	Concurrent training
DLB	Double leg bounce
DOMS	Delayed onset of muscle soreness
EIMD	Exercise induced muscle damage
EMG	Electromyography
GA	Gastrocnemius
IC	Initial contact
MLSS	Maximal lactate steady state
MVC	Maximal voluntary contraction
OBLA	Onset of blood lactate accumulation
RBE	Repeated bout effect
RBS	Recovery between sets
RE	Running economy
RER	Respiratory exchange ratio
RF	Rectus Femoris
RM	Repetition maximun
RMS	Root mean square
ROM	Range of motion
RPE	Rate of perceived exertion
RT-SEP	Resistance training-induced sub-optimization of endurance performance
SE	Strength exercise
SSC	Stretch-shortening cycle
TO	Toe-off
VAS	Visual analog scale
vAnT	Velocity at anaerobic threshold
VL	Vastus Lateralis
VO_{2max}	Maximal oxygen uptake

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APPENDICES

Appendix 1: Muscle soreness questionnaire

Appendix 2: Health and physical activity questionnaire

1 INTRODUCTION

It has long been established that strength training can improve endurance performance (Hickson et al. 1988). The improvements in endurance are partly due to enhanced movement economy, which is achieved through improvements in neuromuscular performance (Paavolainen et al. 1999; Mikkola et al. 2011; Ronnestad & Mujika 2014). This allows athletes to perform exercises at the same submaximal intensity with lower energy expenditure (Fletcher et al. 2010; Beattie et al. 2017). Movement economy has been identified as one of the main differentiating factors among athletes with similar maximal oxygen uptake (Joyner & Coyle 2008; Barnes & Kilding 2015).

However, caution should be taken when designing concurrent training programs that combine endurance and strength training, as strength training-induced fatigue can acutely impair the quality of the subsequent endurance training session, and may, in the long run, cause sub-optimal endurance development (Doma et al. 2017). Maximal, explosive, and reactive strength training, which mainly elicit neural adaptations, appears to be more beneficial for endurance athletes than hypertrophic strength training, which interferes more with endurance adaptations (García-Pallarés & Izquierdo 2011). Interference from strength exercise may attenuate when strength training is performed regularly. The repeated bout effect (RBE) is a known phenomenon that relates to a built-in defense mechanism within the neuromuscular system, where one bout of strength exercise provides resistance to subsequent damage (Hyldahl et al. 2017).

Previous literature has mainly focused on the effects of either hypertrophic strength exercise (Burt et al. 2013; Doma et al. 2015) or exercise with a mainly eccentric component, such as downhill running (Dutto & Braun 2004; Chen et al. 2009), on the acute effects of strength exercise on endurance performance and the RBE. Acute fatigue from strength exercise has been shown to alter the kinematics of running, resulting in changes in the mechanics of the hip, knee, and ankle (Hamill et al. 1991; Paschalis et al. 2007; Chen et al. 2009), as well as in contact time, stride length, and frequency, although there are some conflicting results (Palmer & Sleivert 2001).

Numerous studies have investigated the acute effects of different types of strength exercise on muscle activation. It appears that there are significant differences between hypertrophic and

neural strength exercises. Neural strength exercise mainly induces adaptations and fatigue in the nervous system, leading to a decrease in EMG amplitude immediately after exercise. In contrast, hypertrophic strength exercise may not lead to a decrease in muscle activation due to the high amount of peripheral fatigue it induces. (Linnamo et al. 1998; McCaulley et al. 2009). However, there are only few studies examining the acute effects of strength exercise on muscle activation during running (Kellis & Liassou 2009). Kellis & Liassou (2009) investigated how knee or ankle fatiguing protocol impact the muscle activity during level running. They found that increases in muscle activity were most noticeably in the swing phase and pre-activation phase of the gait cycle, but more research is clearly needed on this subject.

The aim of this study is to investigate the acute effects of neural and hypertrophic strength exercises on running economy, running kinematics, and lower extremity muscle activation during a running economy test performed 48 hours after the strength exercise. Additionally, this study aims to determine whether the RBE influences these variables when the strength exercise and running economy test are performed three times (once per week).

2 STRENGTH- AND ENDURANCE PERFORMANCE AND ADAPTATIONS

Both strength and endurance performance are widely researched subjects. In sports that require exercise lasting longer than 2 minutes or shorter, powerful work cycles performed over extended periods, endurance performance is crucial. Endurance refers to the capacity of the muscles to sustain a certain power or speed without getting fatigued for as long as possible. The objective of aerobic training is to improve endurance performance, thereby enabling the exercise to be performed with greater power or speed for more extended periods. (Jones & Carter 2000)

Strength training is one of the most used training methods across all sports. Chronic strength training adaptations can be categorized into morphological and neural adaptations (Folland & Williams 2007). The effectiveness of strength training is greatly impacted by the level of intensity, volume, density, rest periods, exercise selection and movement speed within the strength exercise program. (Kraemer 1983). Strength training has been clearly shown to be beneficial for endurance performance by enhancing movement economy (Ronnestad & Mujika 2014; Lum et al. 2016).

2.1 Determinants of endurance performance

Endurance sport performance is commonly defined as an athlete's ability to maintain the highest possible speed throughout the entirety of a race, a metric that is widely applicable across nearly all endurance sports. It is widely recognized that endurance capacity is determined by three primary factors: maximal oxygen uptake ($VO2_{max}$), movement economy, and fractional utilization of $VO2_{max}$ ($\%VO2_{max}$) (Basset & Howley 2000; Beattie et al. 2014). As this study will primarily focus on running, the term running economy (RE) will be used interchangeably with movement economy.

2.1.1 Anaerobic threshold

Figure 1 presents an example of how endurance performance determinants can be categorized. One of these determinants, lactate threshold, is frequently used instead of fractional utilization of $VO2_{max}$. Although these two concepts partly describe the same phenomenon, they are not identical. Ghosh (2004) states that lactate threshold combines $VO2_{max}$, economy, and fractional

utilization of $VO2_{max}$, and is the best single physiological predictor of endurance performance. Lactate threshold or fractional utilization of $VO2_{max}$ becomes the most important determinant of endurance performance when race/exercise duration exceeds 10-15 minutes. After that time most or all of the exercise is performed at a pace lower than the maximal oxygen uptake. (Joyner & Coyle 2008)

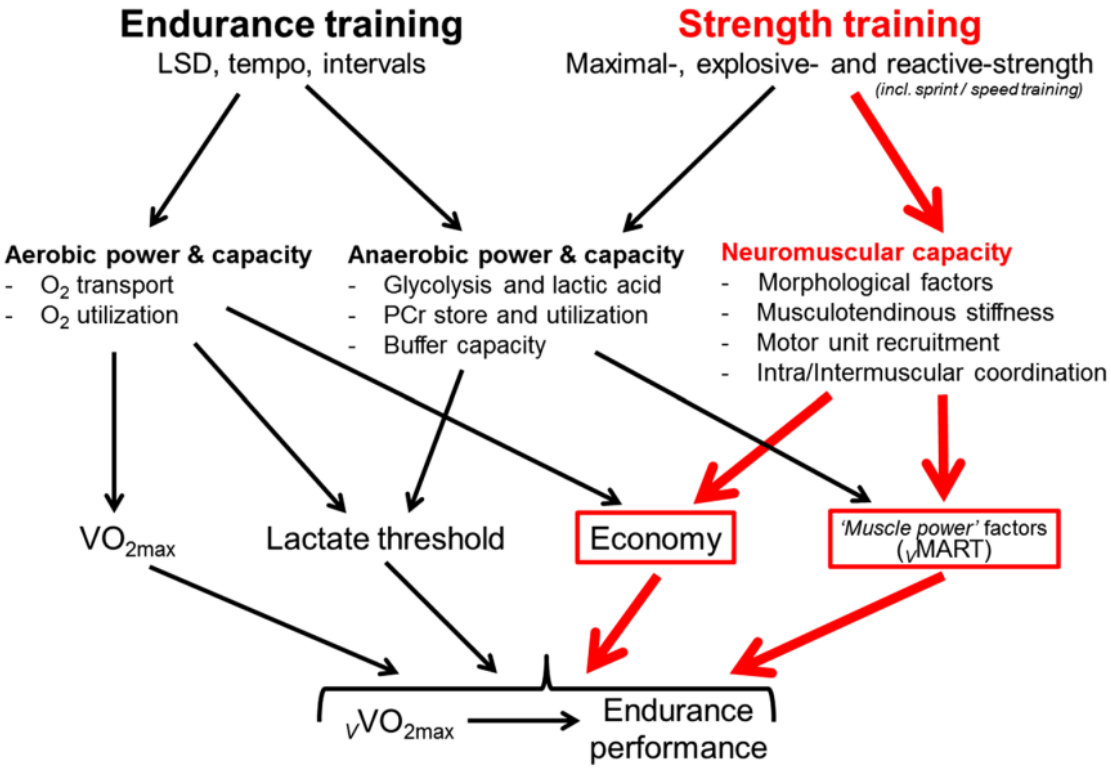

FIGURE 1. Model of determinants for elite endurance performance and potential benefits from strength training (red font). LSD=long slow distance training. PCr=phosphocreatine, vMART = peak velocity in maximal anaerobic running test. (Beattie et al. 2014).

In Finland, anaerobic threshold (AnT) is typically used instead of lactate threshold. Anaerobic threshold is a combination of the second lactate threshold and the second ventilatory threshold (Nummela 2007). However, determining anaerobic threshold is more subjective than using lactate threshold alone. It is identified as the highest possible work rate at which lactate production and removal remain in balance (Nummela 2007). In addition to lactate threshold, there are several other concepts similar to anaerobic threshold, such as OBLA (onset of blood lactate accumulation) or MLSS (maximal lactate steady state). Aunola and Rusko (1991) showed that maximal lactate steady-state correlates well with AnT. OBLA refers to the point at

which blood lactate concentration reaches 4 mmol/l, which is often quite close to the anaerobic threshold (Ghosh 2004).

2.1.2 Maximal oxygen uptake

Maximal oxygen uptake ($VO2_{max}$) measures the highest possible amount of oxygen an individual can take up and utilize during exercise. It reflects the functional capacity of the cardiovascular and respiratory systems, as well as the ability of the skeletal muscles to extract and use oxygen. Further increases in work rate does not result in an increase in oxygen uptake, although a person can still exercise at a higher level than their $VO2_{max}$. (Jones & Carter 2000; Joyner & Coyle 2008) $VO2_{max}$ is affected by numerous factors, including genetics, age, sex, training status, altitude, muscle fiber type, and environmental conditions. Training-induced improvements in $VO2_{max}$ are primarily due to increases in cardiac output and oxygen extraction by the skeletal muscles. (McArdle et al. 2015, 461-497) The human muscles' ability to extract oxygen from the bloodstream is greater than the ability of the cardiovascular and respiratory systems to deliver oxygen and blood to the muscles. Therefore, the primary factor limiting maximal oxygen uptake during exercise is the capacity of the cardiovascular system, which includes the heart, blood vessels, and blood, to supply sufficient oxygen to meet the demands of the muscles. (Jones & Carter 2000)

2.1.3 Running economy

Running economy is defined as the oxygen uptake required to maintain a given running speed. Despite having similar $VO2_{max}$ values, individuals may differ in their oxygen uptake at the same submaximal running speed due to differences in running economy. (Joyner & Coyle 2008; Barnes & Kilding 2015) Running economy is a complex concept that is influenced by various factors, including metabolic, biomechanical, and neuromuscular factors, as well as factors related to cardiorespiratory efficiency as shown on figure 2. (Barnes & Kilding 2015) For instance, a study conducted by Daniels and Daniels (1992) reported that male runners could sustain the same running speed with significantly lower oxygen consumption than female runners, mainly attributed to differences in running economy between males and females.

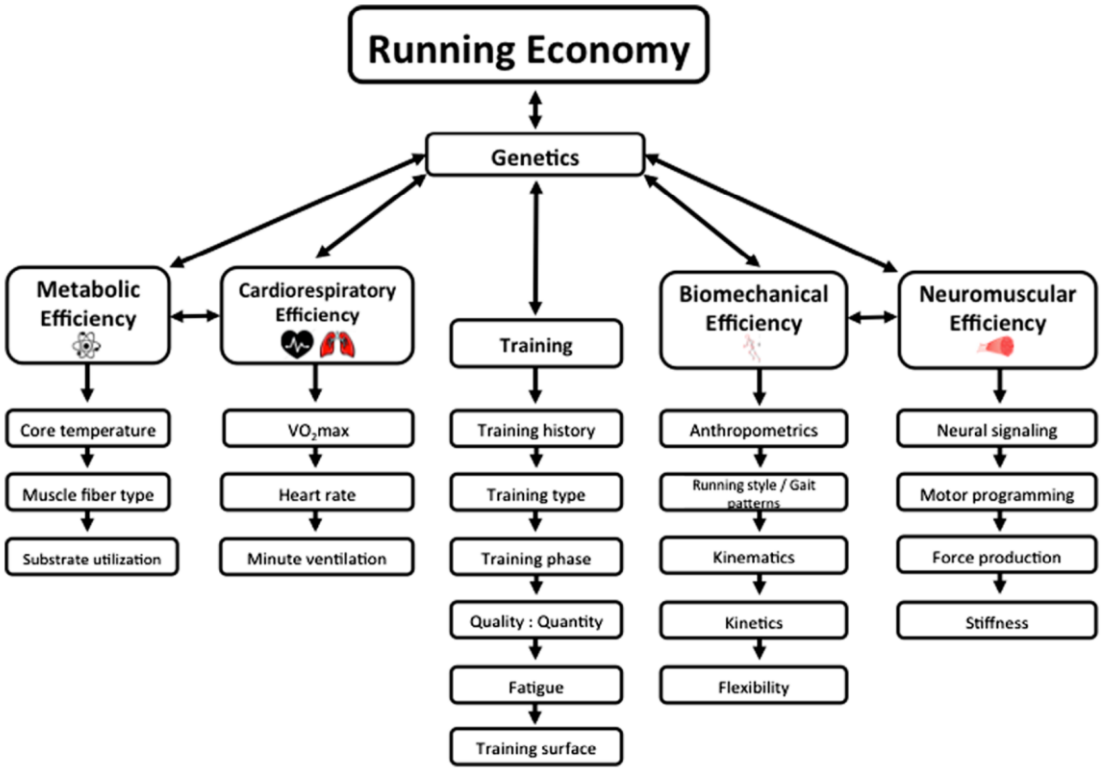

FIGURE 2. Factors affecting running economy (Barnes & Kilding 2015).

Metabolic factors affecting running economy are strongly related to adaptations induced by endurance training. For instance, increased cardiac output can result in decreased ventilation and oxygen consumption at a given absolute exercise intensity. Additionally, endurance training can promote the development of fatigue-resistant muscle fibers with improved oxidative capacity. (Barnes & Kilding 2015)

The capacity of the neuromuscular system is an important factor affecting running economy. Prior research suggests that there are changes in motor control and coordination and recruitment patterns of motor units for athletes with better movement economy (Jones & Carter 2000; Barnes & Kilding 2015). Greater muscle activity has been shown to relate to worse running economy due to the increased metabolic cost of active muscle (Kyröläinen et al. 2001; Moore 2016). However, changes in the stiffness of the muscle-tendon complex and the performance of the stretch-shortening cycle (SSC) can reduce muscle activity during running. Tendon stiffness has been linked to better running economy as it increases the storage of elastic energy and force production capacity (Fletcher et al. 2010), leading to less muscle work (and activity) required and a decrease in oxygen consumption. Similarly, improvements in the timing of the stretch-shortening cycle can increase the efficiency of energy storage during the eccentric

phase. This leads to more efficient use of energy during the concentric phase, resulting in better running economy (Barnes & Kilding 2015).

Anthropometrics is a factor that affects running economy, and even with training, not all runners can achieve the same level of efficiency as top-level athletes. South African distance runners have been shown to possess favorable anthropometric characteristics for running. Specifically, their achilles tendon has smaller torque arms compared to Caucasian runners, allowing for greater elongation and utilization of elastic energy. (Mooses & Hackney 2017) Additionally, South African runners have been found to have shorter ground contact time (Kong & Heer 2008), which may also be related to their anthropometric differences. Interestingly, there are significant variations in anthropometrics even among South African runners, as evidenced by the distinct somatotypes of Kenyan and Ethiopian runners. Kenyans typically exhibit an ectomorphic somatotype, characterized by long, slender legs, while Ethiopians tend to have a mesomorphic somatotype similar to those found in Northern Africa and the Middle East. (Mooses & Hackney 2017)

Spatiotemporal variables, kinematics, and kinetics all play important roles in running economy. Spatiotemporal variables, such as stride length, stride frequency, and ground contact time, are relevant to understanding the energetic demands of running. Typically, natural stride frequency and length are most economical for runners (Dugan & Bhat 2005; Barnes & Kilding 2015; Moore 2016). For experienced runners, there may be some variability in stride length without a significant impact on efficiency. However, novice runners or those who deviate 3 % or more from their natural stride may experience a decrease in efficiency and an increase in oxygen uptake (Dugan & Bhat 2005). For instance, Hoogkamer et al. (2016) demonstrated that adding small weight to running shoes can impair running economy and correspondingly slow 3 km time-trial performance. Similarly, Kyröläinen et al. (2001) showed that athletes with worse running economy have higher braking and mediolateral forces during running than athletes with better running economy.

Williams and Cavanagh (1986) found that longer ground contact times were associated with better running economy. However, the relationship between ground contact time and running economy remains unclear. Often, shorter ground contact times are linked to better running economy, as longer contact times may increase absorption (braking) during the stance phase, resulting in greater metabolic demands (Nummela et al. 2007; Moore 2016). Nonetheless, the

relationship between absorption and generation (acceleration) times during the stance phase may be more important factor for running economy than ground contact time alone.

There are two primary running styles: forefoot strike and rearfoot strike. There has been some debate regarding which style is more efficient. Forefoot strike runners tend to have shorter stride lengths, increased stride frequency, and shorter ground contact times compared to rearfoot strike runners (De Wit et al. 2000; Squadrone & Gallozi 2009; Ahn et al. 2014). During forefoot striking, the ankle is also more plantar flexed and the plantarflexor muscles are activated earlier and for a longer duration than in rearfoot striking (Ahn et al. 2014; Landreneau et al. 2014). This activation pattern may offer several benefits to the foot and ankle, such as increased capacity for storing elastic energy in the passive structures of the foot and ankle, as well as enhanced performance of the active muscle through greater storage of elastic strain energy in the cross-bridges and activated titin (Ahn et al. 2014). Despite these factors, there is no consensus in favor of either running style with regard to running economy (Barnes & Kilding, 2015).

Various kinematic parameters related to running have been found to impact running efficiency and energy cost. Williams and Cavanagh (1986) reported that increased knee flexion during stance phase, greater amplitude of the knee angle, and smaller plantarflexion at toe-off are associated with improved running efficiency. Other studies have found for example that smaller vertical oscillation and decreased peak hip flexion during braking associate with better running economy (Pizzuto et al. 2019). Running kinematics are discussed in detail in chapter 4.

2.2 Endurance training

Endurance training induces adaptations in the respiratory and cardiovascular systems as well as in the aerobic metabolism of muscles. Cardiovascular adaptations include an increase in heart volume, particularly in the dimensions of the left ventricle, resulting in an increase in stroke volume. Endurance training also leads to an increase in blood plasma volume and red blood cell mass, which enhances the capacity of blood to deliver oxygen to the working muscles. Moreover, both resting heart rate and blood pressure at rest and at submaximal exercise loads are reduced. (Tanaka & Svensen 1998; McArdle 2015, 461-497)

Respiratory adaptations include decrease in ventilation at submaximal exercise loads and increase in maximal ventilation. Even though, adaptions occur in respiratory system due to endurance training for majority of healthy adults the capacity of respiratory system is not a limiting factor for endurance performance. (Hynynen 2016; McArdle 2015, 461-497)

Adaptations to the muscle metabolism include the increase in the number and size of mitochondria, which are responsible for aerobic energy production in muscle cells. Additionally, endurance training leads to an increase in the activity of enzymes involved in aerobic energy production, thereby facilitating the ability of muscles to utilize oxygen more efficiently. Another important adaptation is the increase in muscle capillarization, or the growth of new capillaries in the muscle tissue. This increased capillarization enhances the ability of oxygen and nutrients to be delivered to the muscle tissue, thereby improving the muscles' ability to sustain prolonged exercise. Endurance training also leads to an increase in the amount of myoglobin, a molecule responsible for oxygen transport in muscles. The increased amount of myoglobin further enhances the muscles' ability to utilize oxygen for energy production. (Tanaka & Svensen 1998; McArdle 2015, 461-497) Endurance training causes also changes in muscle phenotypes with increase in type I cell dimensions and change in type II cells to become more fatigue resistant (McArdle 2015, 461-497).

Collectively, these adaptations enhance the capacity of endurance-trained muscles to utilize oxygen transported by the bloodstream, which leads to enhanced endurance performance. Additionally, the aforementioned adaptations improve running economy by reducing oxygen consumption during exercise at a given absolute intensity. Endurance training also improves running economy by enhancing the function of the neuromuscular system (Midgley et al. 2007).

2.3 Strength training

Strength training is one of the most popular forms of exercise around the world. It is used to improve the performance of the neuromuscular system and sports performance. The significance of muscular strength is great in all sports and is expected to become increasingly crucial in the future. (Häkkinen & Ahtiainen 2014, 250-258). Strength training also has many favorable effects on metabolism and health. Strength training induces both neural and morphological adaptations to the body. (Folland & Williams 2007)

The response to resistance training is greatly influenced by the manipulation of different strength training variables. The most crucial variables in strength training are intensity, the number of sets and repetitions, volume, rest intervals between sets, movement speed, and training frequency (Salles et al. 2009). This text will mainly focus on maximal strength training, which can be further categorized into hypertrophic and neural strength training (Häkkinen & Ahtiainen 2014, 250-258).

Hypertrophic strength training mainly induces morphological adaptations, referring to structural changes in the muscle. These changes include an increase in muscle cell size, known as hypertrophy, caused by the growth and proliferation of myofibrils, changes in the pennation angles of muscles, and possible hyperplasia, an increase in muscle cell count (Folland & Williams 2007). The main aim of hypertrophic strength training is to increase the cross-sectional area of the muscle. This is achieved by loading the muscle as much as possible to provide sufficient stimulus for protein synthesis. In hypertrophic strength training, repetitions are mostly between 6-12, and intensity is set at 70-85 % of one repetition maximum (1 RM). Rest periods between sets are typically brief, usually lasting between 1-2 minutes. Both multi-joint and single-joint exercises are utilized in typical hypertrophic strength training. In beginner strength trainees, hypertrophic strength training can also induce neural adaptations in the initial stages of training. During the initial weeks of strength training, neural adaptations are the primary contributor to strength gains. (Folland & Williams 2007)

Strength gains from neural strength exercise are attributed to several factors, including increased neural control through improvements in motor unit recruitment, synchronization, and firing frequency, as well as enhanced spinal reflexes with a greater degree of facilitation and less inhibition. Additionally, changes in agonist, synergist, and antagonist activations can lead to greater synchronization and, ultimately, increased power output. (Folland & Williams 2007) Neural strength training can be divided into two main categories: maximal and explosive strength. Maximal strength exercises involve lifting heavy loads at intensities between 85-100 % of one repetition maximum (1 RM) with low number of repetitions (1-3 reps per set). Adequate rest periods are required between sets, with recovery times of up to 5 minutes to ensure sufficient recovery. On the other hand, explosive (or plyometric) strength training involves using lighter loads at intensities between 30-80 % of 1 RM and performing all movements with the highest possible movement velocity. The rest periods between sets are similar to those in maximal strength training. (Häkkinen & Ahtiainen 2014, 250-258). As said

earlier, hypertrophic strength training can induce neural adaptations in beginners. Similarly, neural strength training can stimulate morphological adaptations in novices, such as increases in muscle fiber size and cross-sectional area (Campos et al. 2002).

Maximal strength training and explosive strength training with high contraction velocities both result in significant neural adaptations. This type of training is particularly useful for sports that require a small body weight, such as endurance running or skiing. In contrast, hypertrophic strength training focuses on increasing muscle fiber size and overall muscle mass. However, there is a limit to how much strength can increase solely through neural adaptations, and therefore some degree of muscle hypertrophy is necessary for long-term strength gains. (Häkkinen & Ahtiainen 2014, 250-258).

2.3.1 Acute responses to hypertrophic strength exercise

Hypertrophic strength exercise elicits various acute responses in the body, including metabolic responses such as increased energy expenditure and glycogen depletion. This exercise involves subjecting muscle tissue to significant mechanical tension, which can lead to muscle damage manifested as microscopic tears in muscle fibers, resulting in soreness, inflammation, and reduced muscle function. McCaulley et al. (2009) observed a significant decrease in muscle power production capacity following hypertrophic strength exercise, likely due to peripheral fatigue, which is referred to as neuromuscular inefficiency. (McCaulley et al. 2009) Inflammatory responses occur in response to the microscopic tears in muscle tissue caused by mechanical stress during exercise, and this can be measured through an increase in inflammatory markers such as C-reactive protein (Schoenfeld 2010).

Hypertrophic strength exercise induces a transient increase in lactate and anabolic hormones such as testosterone, cortisol, growth hormone, and sex hormone-binding globulin (SHBG). McCaulley et al. (2009) investigated the effects of different types of strength exercises (hypertrophic, maximal, and explosive) on hormonal responses, and their findings showed that only hypertrophic strength exercise resulted in significant changes in cortisol, testosterone, and SHBG concentrations acutely after exercise. This finding is consistent with previous research (Häkkinen & Pakarinen 1993; McCall et al. 1999). The clear hormonal response observed can be attributed to the high training volume and low recovery time associated with hypertrophic strength exercise (McCaulley et al. 2009).

As mentioned previously, hypertrophic strength exercise induces morphological changes in the muscle, including an increase in muscle size. Protein synthesis is increased 24-72 hours after exercise (Drummond et al. 2009). The acute responses mentioned earlier, such as changes in hormones, metabolism, inflammation, and muscle damage, appear to activate the complex signaling systems that promotes protein synthesis following resistance exercise. In addition to these responses, other triggers for protein synthesis include metabolites such as hydrogen ions and creatine, muscle swelling, muscle cell hypoxia during exercise, and increased neural activity. (Schoenfeld 2010)

2.3.2 Acute responses to neural strength exercise

As discussed previously, studies have shown that strength training can induce chronic adaptations in motor unit synchronization and recruitment patterns, agonist/antagonist muscle activation patterns, and spinal reflexes, resulting in an increase in power output (Folland & Williams 2007). Such neural adaptations can occur within a few days of initiating a strength training program (Gabriel et al. 2006), but they should be distinguished from other acute changes that may occur immediately after exercise.

Neural strength exercise does not induce similar hormonal responses as hypertrophic strength exercise. McCaulley et al. (2009) showed that maximal or explosive type strength training did not induce any significant changes in testosterone, cortisol or SHBG levels immediately or 60 min after the exercise. Testosterone was increased in both conditions immediately after the exercise, but change was not statistically significant. Other studies have shown similar results (Häkkinen & Pakarinen 1993; Linnamo et al. 2005). This is probably due to higher recovery periods between sets and lower repetitions which are both required in order to maintain adequate recovery and intensity for neural strength exercise (McCaulley et al. 2009).

Neural strength exercise can lead to muscle soreness and damage. The extent of exercise-induced muscle damage (EIMD) is greater when the amount of eccentric contractions increases. For instance, plyometric exercise induces greater EIMD than concentric exercise, but not as much as complete eccentric exercise (Brockett et al. 1997). EIMD can reduce maximal force production and rate of force development (Sarabon et al. 2013). The decrease in rate of force

development after neural strength exercise has been shown to be more significant and prolonged (up to 48 hours) compared to hypertrophic strength exercise (McCaulley et al. 2009).

2.4 Repeated bout effect

Skeletal muscles have a built-in defense mechanism that responds to exercise-induced damage by initiating an adaptive response that provides resistance to subsequent damage. This phenomenon is referred to as the repeated bout effect (RBE) and has been extensively studied. Most studies on RBE have focused on mainly eccentric contractions that cause muscle damage. It is well established that performing a second bout of eccentric contractions within a few weeks after an initial bout, results in a significant and repeatable reduction in exercise induced muscle damage, muscle soreness, decrease in force production capacity and recovery time. (McHugh et al. 2003; Hyldahl et al. 2017) It should be noted that there may be some variations in these variables depending on the test subjects and strength exercise used. Repeated bout effect is reversible and the RBE effect attenuated after time period of 4-12 weeks post exercise (Nosaka et al. 2005).

According to Hyldahl et al. (2017), the protective effect of RBE increases with the intensity of the initial strength exercise. Higher intensity leads to a greater protective effect for subsequent exercises. Chen et al. (2010) discovered that performing exercises at 40% intensity every two weeks for four sessions created a similar protective effect to performing one maximal intensity exercise. Exercise volume may not be a factor that affects the amount of the RBE. One study found that maximal intensity exercise with 45 repetitions did not produce a better protective effect for subsequent exercises compared to maximal intensity exercise with only 10 repetitions. (Howatson et al. 2007)

According to Hyldalh et al. (2017) mechanisms behind repeated bout effect can be categorized into neural adaptations, muscle-tendon complex adaptations, inflammation and extra cellular matrix (ECM) remodeling (figure 3). Previous research has shown that motor unit synchronization can increase for up to 7 days after performing eccentric exercise. This increase in synchronization is believed to be a mechanism used by the central nervous system to coordinate the activity of muscles that work together. This helps to protect the muscle from further damage by spreading the mechanical load over a larger number of motor units when the

same submaximal eccentric exercise is repeated. (Dartnall et al. 2011) Other neural adaptations include changes in spinal level such as increased α-motorneuron excitability and increased inhibitory feedback.

Eccentric exercise imposes substantial mechanical stress on muscle fibers, leading to microscopic damage and disruption of the muscle's structural integrity. However, this initial damage triggers a cascade of repair and remodeling processes that promote adaptive changes in the muscle tissue, including hypertrophy, increased fiber size, and improved contractile strength. As a result of these adaptations, the muscle becomes better equipped to withstand future bouts of exercise and protect against further damage. (McHugh et al. 2003) Research has demonstrated that markers of inflammation decrease after the initial bout of eccentric exercise, suggesting a reduction in exercise-induced inflammation. McHugh et al. (2003) propose that this effect may be due to the structural adaptations discussed earlier, which result in fewer microtears within the muscle fibers.

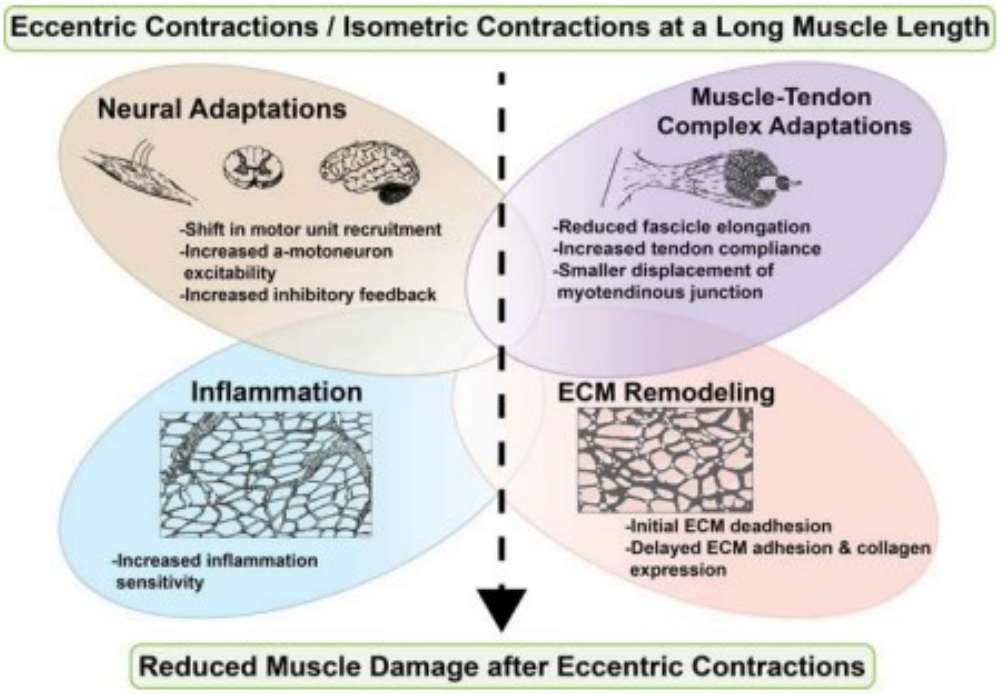

FIGURE 3. Mechanisms that are likely involved in RBE. ECM = extra cellular matrix. (Hyldahl et al. 2017).

The extracellular matrix serves multiple functions in skeletal muscle, including providing structural support, contributing to muscle stiffness, and facilitating mechanical force transfer. One of its primary roles is to protect muscle from injury by absorbing mechanical strain and increasing passive tension. Given this role, there is speculation that changes in ECM structure and composition may also contribute to the repeated bout effect. (Hyldahl et al. 2017)

3 COMBINING STRENGTH AND ENDURANCE TRAINING

When endurance- and strength training are combined into same training regime it is called concurrent training, irrespective of whether the training is performed on the dame day or separate days (Doma et al. 2017). It has been clearly proven that correct type of strength training enhances endurance capacity by improving movement economy (Hickson et al. 1988; Paavolainen et al. 1999; Ronnestad & Mujika 2014). When designing a concurrent training program, it is important to consider that fatigue caused by resistance training can negatively affect the quality of endurance training sessions, and potentially lead to less-than-optimal development of endurance. Therefore, caution should be taken when prescribing concurrent training to ensure the best possible outcomes. (Doma et al. 2017)

3.1 Concurrent training

Strength training and endurance training elicit distinct adaptations in skeletal muscle and the entire body. Endurance training, for instance, results in a decrease in glycolytic enzyme activity while increasing mitochondrial content and capillarity. Conversely, strength training increases myofibrillar protein synthesis while reducing mitochondrial content and capillarity. Myofibrillar protein synthesis remains elevated for up to 72 hours post-exercise following strength training. In contrast, moderate-intensity endurance exercise inhibits elongation factors, which are responsible for increasing myofibrillar protein synthesis, and this inhibition persists for the duration of endurance exercise. (Wilson et al. 2012) Endurance and strength training have varying effects on muscle phenotypes. For example, endurance training decreases type IIb muscle fiber size while strength training increases it (Tanaka & Swensen, 1998).

The existing literature suggests that concurrent training hinders strength development when compared to strength training alone (Häkkinen et al. 2003; Chtara et al. 2008; Izquierdo-Gabarren et al. 2010; Wilson et al. 2012). However, some studies have not demonstrated interference of concurrent training on strength adaptations (McCarthy et al. 2002; Sillanpää et al. 2009). It is generally agreed that combining strength and endurance training can enhance endurance performance adaptations more than endurance training alone (Paavolainen et al. 1999; Mikkola et al. 2011; Wilson et al. 2012). Trained individuals can benefit from strength exercise to improve running economy, which is an essential factor that distinguishes runners with similar maximal oxygen uptake (Beattie et al. 2017). The impact of strength training on

endurance performance development should be considered, and the frequency and volume of strength training should be carefully controlled, while concurrent training periods should not be too long (Leveritt et al. 1999; Alcaraz-Ibanez & Rodriquez-Perez, 2018). According to Alcaraz-Ibanez and Rodriquez-Perez (2018), trained runners can improve their running performance with two weekly strength training sessions, separated by at least 48 hours. The strength exercises should aim to improve maximum and explosive strength.

The adaptations induced by concurrent training on running economy for previously trained individuals are primarily associated with improved neuromuscular performance. Combined strength and endurance training enhances the stiffness of the muscle-tendon complex, increases the use of elastic energy during exercise, and promotes the development of fatigue-resistant type IIa muscle fibers. Strength training also increases the size of type I muscle fibers. This means that type I fibers can be activated for a longer time before they fatigue, which delays the recruitment of fast type II muscle fibers (Fletcher et al. 2010; Ronnestad & Mujika 2014). Furthermore, strength training enhances neural control by improving the recruitment and synchronization of motor units, which is also positively related to better running economy (Jones & Carter 2000).

Strength training for endurance running should focus on achieving physiological and neural adaptations that enhance running efficiency, maximum speed attainable at maximal aerobic capacity and maximum anaerobic speed (vMART) (Beattie et al. 2017). According to García-Pallarés and Izquierdo (2011), concurrent training should involve periodization of aerobic and anaerobic training with hypertrophic and strength endurance training. It is not recommended to conduct maximal endurance training concurrently with hypertrophic strength training due to the strongly opposing peripheral adaptations. However, maximum and explosive strength training can be conducted with aerobic, anaerobic, or maximal endurance training. It should be noted that endurance training intensity does not affect the interference effect of maximum or explosive strength training. (García-Pallarés & Izquierdo 2011) Docherty & Sporer (2000) come to similar conclusion as seen in figure 4. It shows that strength training with intensity more than 10 RM (hypertrophic and strength endurance) should not be performed simultaneously with maximal endurance training.

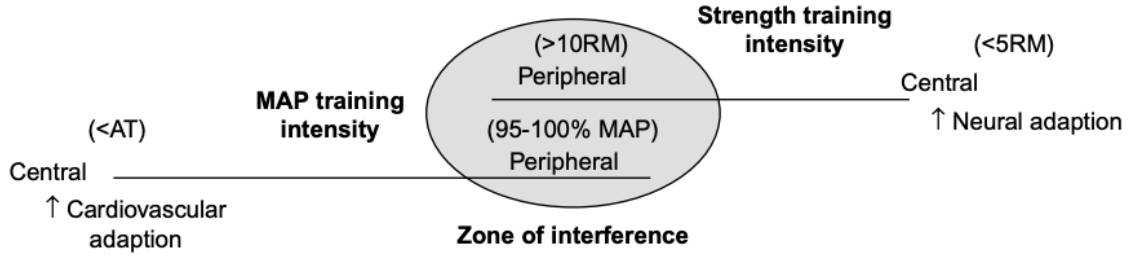

FIGURE 4. Primary location of adaptations for both endurance training intensity (MAP) and strength training intensity. Maximal aerobic training and hypertrophic/endurance strength training overlap. MAP = maximal aerobic power, AT = anaerobic threshold. (Docherty & Sporer 2000)

3.2 Acute responses of strength exercise on endurance performance

Previous literature has demonstrated that strength training can induce acute fatigue in the body, which may impair subsequent endurance exercise performance if inadequate recovery is allowed (Burt & Twist 2011; Doncaster & Twist 2012; Doma & Deakin 2013; Doma et al. 2015; Doma et al. 2017). Endurance performance has been shown to be decreased for up to 72 hours post-exercise. Figure 5 illustrates the possible mechanisms underlying this phenomenon, which Doma et al. (2017) have termed "resistance training-induced sub-optimization of endurance performance" (RT-SEP). These mechanisms include resistance exercise-induced muscle damage, muscle soreness, neural fatigue, and glycogen depletion. RT-SEP can be controlled by careful planning of a concurrent training program. Several strength training-related variables, such as intensity, volume, recovery time, exercise order, training volume, and previous training background, can affect the amount of strength exercise-induced residual fatigue. If these variables are taken into account correctly, concurrent training will be beneficial for endurance performance. However, if RT-SEP persists over a long period of time, it can lead to chronic effects and sub-optimal endurance development, as shown in figure 5. (Doma et al. 2017)

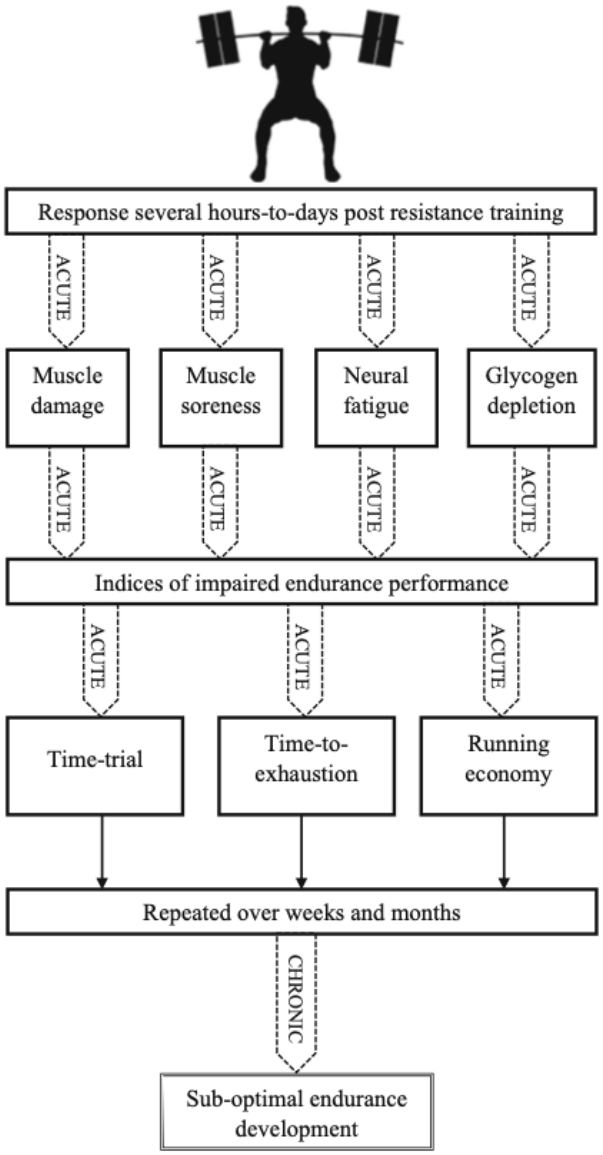

FIGURE 5. Resistant training-induced sub-optimisation of endurance performance (RT-SEP). (Doma et al. 2017)

Most of the mechanisms proposed by Doma et al. (2017) to explain the residual fatigue caused by resistance training leads to reductions in running economy. Although running economy is not a direct measure of endurance performance and does not solely dictate the adaptations of an endurance training session, research has shown that decreased running economy can lead to decreases in multiple indicators of endurance performance, such as time-trial performance (Burt & Twist 2012), time-to-exhaustion (Doma & Deakin 2013), oxygen consumption (Chen et al. 2007), and heart rate and RPE (Chen et al. 2009) at submaximal exercise intensities. This suggests that an endurance training session following a resistance training session may need to be performed at a lower intensity, which could result in sub-optimal endurance adaptations.

Exercise-induced muscle damage and muscle soreness have been shown to impair running economy for multiple days after exercise. Chen et al. (2007, 2009) demonstrated that EIMD caused by downhill running reduced maximal voluntary contraction (MVC) and increased oxygen consumption during running for up to five days following the exercise. Notably, some studies have reported no impairment in running economy at submaximal exercise intensities despite clear indications of delayed-onset muscle soreness (DOMS) and muscle damage (Doma & Deakin, 2014). However, Doma & Deakin (2014) did observe impairments at maximal exercise intensity. It is possible that the EIMD and DOMS has a greater impact on high-intensity endurance performance. One reason behind this might be that type II muscle fibers experience more damage from strength exercise than type I fibers. During endurance exercise type II fibers are primarily used in high-intensity activities that exceed the anaerobic threshold. Therefore, the effect of muscle damage on performance will be most noticeable in activities that require high-intensity level. (Doma et al. 2017)

Neural fatigue may represent one of the primary mechanisms underlying the reduction in endurance performance following strength exercise. As previously discussed, strength training leads to numerous adaptations in both the central and peripheral nervous systems that can enhance running economy (Jones & Carter 2000; Fletcher et al. 2010). However, many of these neural pathways become highly activated and fatigued for up to 48 hours following strength exercise, potentially compromising optimal endurance adaptations without adequate recovery. It should be noted that the extent to which strength exercise impairs muscle force production depends heavily on the training variables and individual training background (Doma et al. 2017).

There have been some studies showing the effects of resistance training session on muscle glycogen content and the reduction of muscle glycogen levels can persist for several hours (Tesch et al. 1986; MacDougall et al. 1999). However, it is worth noting that the extent to which this depletion occurs might vary depending on the type and amount of nutrients consumed after exercise and the subsequent physical activity levels. According to Doma et al. (2017) there is limited evidence on effects of glycogen on endurance performance. Few studies have shown that VO^2 kinetics might decrease because of glycogen depletion. On the other hand, endurance training in glycogen depleted state has been shown to increase mitogen-activated protein kinase (MAPK) activity which may enhance the endurance adaptations. All in all, glycogen depletion

is one mechanism that might affect RT-SEP and long-term endurance training at glycogen depleted state might lead to increased risk of overtraining. (Doma et al. 2017)

Repeated bout effect might influence the amount of residual fatigue from strength exercise. As discussed earlier RBE causes several adaptations when strength training is repeated multiple times. The decrease in endurance performance caused by strength training may also become less significant with prolonged training. Several studies have shown that repeating strength exercise multiple times or having a flush-out period in order to remove the effect of RBE, did decrease the levels of muscle damage markers. (Chen et al. 2007; Chen et al. 2009; Doma & Deakin 2013; Burt el al. 2013; Doma et al. 2015) However, many of these studies (Chen et al. 2007; Chen et al. 2009; Doma & Deakin 2013; Doma et al. 2015) showed that indirect muscle damage markers following strength exercise decreased more compared to running economy and endurance performance at both maximal and submaximal exercise intensities. Although muscle damage was highly attenuated, endurance performance was still impaired. Doma et al. (2017) suggest that this may be due to the complex neurophysiological factors required for running, and that exercise-induced muscle damage explains only a portion of the impairments observed. It is possible that the resistance training-induced sub-optimal endurance performance may be present regardless of the number of strength exercise sessions (Doma et al. 2017).

4 KINEMATICS OF RUNNING

Kinematics is a branch of mechanics that deals with the movement of points and structures in space, without considering the forces that cause the movement. Motion analysis is a similar approach that provides a quantitative description of body segments during gait, without measuring the forces involved (Dugan & Bhat 2005). In running and walking, kinematics is often presented graphically as a function of the total gait cycle or time. Kinematics focus on variables such as the positions of body segments, joint angles, and ranges of motion. (Novacheck 1998)

There are numerous kinematic variables that can be evaluated or measured during the gait cycle. Studying these variables can help us understand whether a particular movement is beneficial or detrimental to performance. Kinematic variables typically refer to the patterns of movement, such as body segment positions, joint angles, body position, and velocity. (Ceri 2001) In running, the ankle, knee, and hip joint angles are the most important joint angles to consider. The range of motion (ROM) describes the maximum change in joint angles during movement. Kinematic researchers usually analyze joint angles during specific points in the running cycle, such as initial contact (=the moment when the foot first touches the ground) and toe-off (=moment when the foot of stance leg is lifted off the ground). (Novacheck 1998)

Proper running biomechanics involves synchronous movements of all the components of kinematic (or kinetic) chain. Kinematic chain describes the interrelated groups of body segments, connecting joints and muscles working together to perform movements. The lower kinematic chain includes toes, feet, ankle, lower legs, knees upper legs, hips, pelvis and spine. (Dugan & Bhat 2005) In this text mainly sagittal plane kinematics are discussed. In running there are also movements in frontal and transversal planes but those are much more subtle compared to sagittal plane kinematics (Novacheck 1998).

4.1 Gait cycle

The gait cycle refers to the time from the initial contact of one foot with the ground to the next contact of the same foot. The initial contact is the moment when the foot first touches the ground. In running, the gait cycle consists of stance, swing, and float phases, while in walking, it only has stance and swing phases since one foot always touches the ground. The stance phase

represents 60% of the walking gait cycle, and the swing phase represents 40%. Walking also has two periods of double support, where both feet are in contact with the ground. In running, there are no periods of double support, but there are two points when neither foot is in contact with the ground, known as double float. (Novacheck 1998; Dugan & Bhat 2005)

As seen in Figure 6, in running gait cycle, the stance, float, and swing phases account for 40%, 30%, and 30% of the cycle, respectively. However, these percentages vary according to running speed. For instance, in elite sprinters, toe-off (end of stance phase) occurs as early as 22% of the gait cycle, while in distance running, toe-off occurs between 35-40% of the cycle, implying that the swing phase is longer when velocity increases (Novacheck 1998). The float phase is divided into two equal parts, one after the stance phase and the other after the swing phase. Toe-off marks the point when the stance phase ends, and the float phase begins, indicating that the foot is no longer in contact with the ground (Adelaar 1986). Float is often considered as subphase of swing and in this study swing phase is considered to include float phases, so those are not discussed separately.

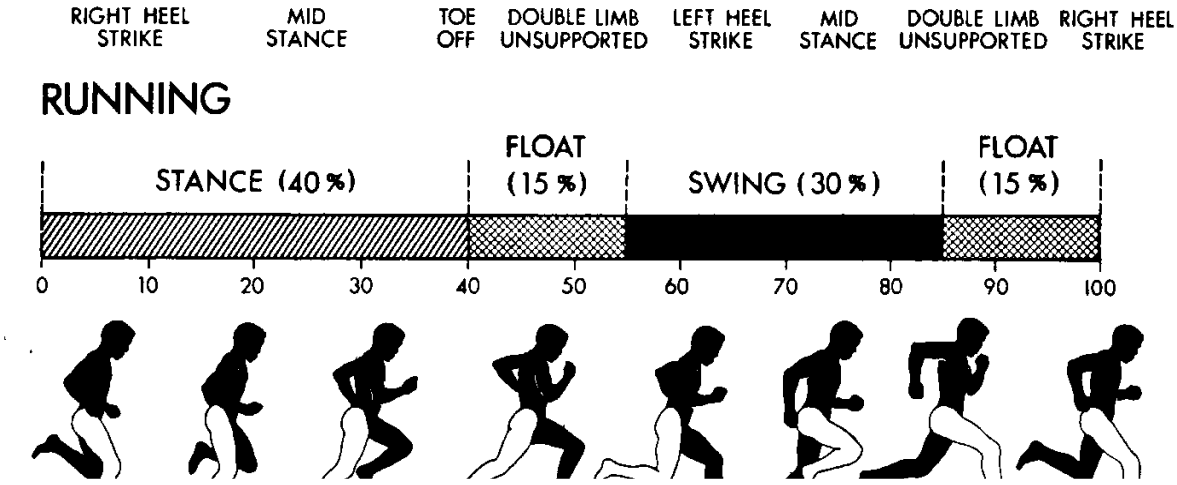

FIGURE 6. Gait cycle of running (Modified Adelaar 1986).

During the running and sprinting gait cycle, there are alternating periods of acceleration and deceleration, which are known as absorption and generation phases. As seen in figure 7, these phases do not occur at same time points as initial contact and toe-off. Absorption phase consists of both stance phase absorption and swing phase absorption. (Novacheck 1998) During absorption, the horizontal velocity of the body's center of mass decelerates. After the initial contact, the vertical ground reaction force can be up to 2.2 times the body weight. Proper joint

motion, eccentric muscle contraction, and compression of articular cartilage are essential to absorb the impact. Dorsiflexion at the ankle joint and hip and knee flexion help to dissipate the force on impact. (Dickinson et al. 1985) The stance phase reversal (StR) marks the end of the absorption phase and the start of the generation phase. The swing phase reversal (SwR) indicates the end of the generation phase and the start of the next absorption phase. (Novacheck 1998)

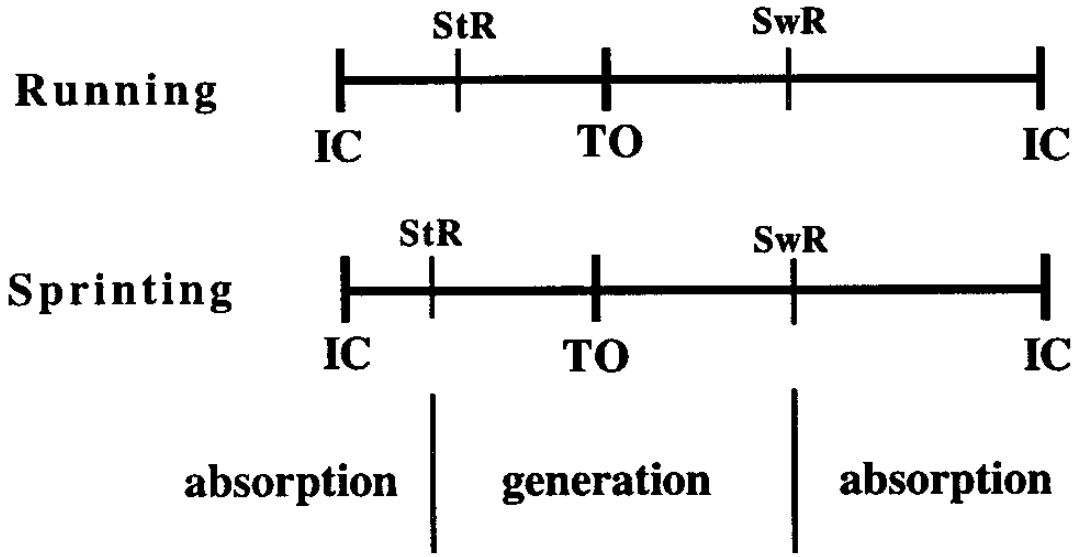

FIGURE 7. Running and sprinting gait cycle. IC = initial contact, TO = toe-off, StR = stance phase reversal, SwR = swing phase reversal. (modified Novacheck 1998)

The swing phase of the running and sprinting gait cycle can also be divided into two phases, which are the initial swing and terminal swing phases. The whole swing phase, including the float phases, is equally divided between these two phases. During the initial swing phase, after the toe-off, the body starts to accelerate forward while the hip, knee, and ankle are flexed in order to advance the limb forward. The terminal swing phase starts when the opposite limb has undergone toe-off, and the swinging limb gets ready to contact the ground. When the limb finally touches the ground, one full gait cycle is completed. (Ounpuu 1994)

4.2 Stride length, stride frequency and contact time

The most commonly used kinematic variables in studying running are stride length, stride frequency, and contact time. These are called as spatiotemporal variables and are often discussed alongside kinematics in scientific research. Stride length is the distance between the

initial contacts of the same foot, while step length refers to the distance between the initial contacts of opposite feet. Stride frequency (often called also 'cadence') is the number of steps per unit of time (usually measured in steps/min or steps/second), and contact time is the duration of the stance phase (between initial contact and toe-off). These variables are often measured using force plates, pressure insoles or calculated from motion analysis. (Cavanagh & Kram 1990) Runners typically unconsciously choose the optimal stride length and cadence at a given speed, although there are significant intra-individual differences in these variables due to factors such as body dimensions, muscle fiber type distribution, and fatigue. When increasing running speed, runners usually first increase their stride length and then increase their stride frequency. (Dugan & Bhat 2005)

4.3 Ankle angle

The ankle angle refers to the position of the foot in relation to the tibia (Novacheck 1998). When the foot makes the first contact with the ground, the ankle angle is typically around 90 degrees, but there may be a slight downward movement afterward. As the foot continues through the stance phase, the ankle reaches its highest upward position before gradually extending downward, with the maximum downward position reached just after the toe-off. During the swing phase, the ankle angle returns to around 90 degrees. (Milliron & Cavanagh 1990, 72-73) A graph of the ankle angle during a running at different velocities is shown in figure 8.

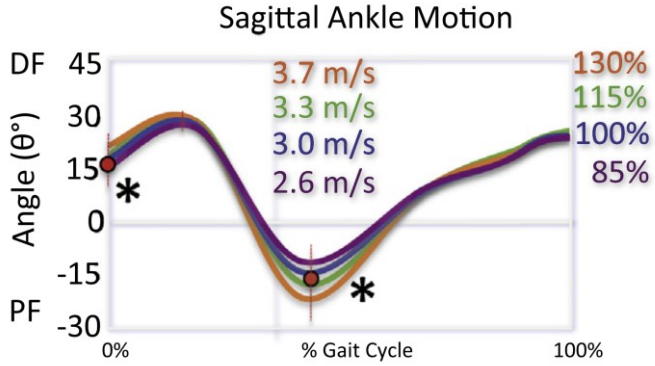

FIGURE 8. Ankle angle over one gait cycle at four different running velocities in recreational runners. 90° angle being plotted as 0°. Horizontal line represents the point of toe-off. DF= Dorsiflexion, PF=plantarflexion. (Orendurff et al. 2018).

During the gait cycle, the foot undergoes pronation and supination as the ankle moves through different phases. During the absorption phase, ankle dorsiflexion causes internal rotation of the tibia, resulting in foot pronation. The highest pronation occurs at around 40% of the stance phase (Novacheck 1998). As the swinging foot moves forward, it causes rotation of the hip, leading to external rotation of the supporting leg. This external rotation of the tibia causes the foot to supinate, and it reaches a neutral position at around 70 % of the stance phase. At this point, the transverse tarsal joint of the foot is "locked", making the foot stiffer and allowing for better power production during the push-off phase (Novacheck 1998). In runners with overpronation, there is a delay in the pronation and supination phases, leading to suboptimal power production during the push-off phase (Dugan & Bhat 2005).

4.4 Knee angle

The knee angle is defined as angle between femur and tibia. A knee angle of 0° indicates that the knee is fully extended, so tibia and femur are parallel to each other (Novacheck 1998). When the foot makes initial contact with the ground, the knee angle is usually between 10-20 degrees. The knee continues to flex until the middle of the stance phase, after which it starts to extend. The extension continues until toe-off or shortly after it. During the swing phase, the knee rapidly flexes and reaches its maximum flexion at the middle of the swing phase. After this point, the knee starts to extend almost as rapidly as it flexed. By the end of the swing phase, the knee is almost fully extended at around 0°. Just before initial contact, the knee usually undergoes a few degrees of flexion to be prepared for the impact. (Milliron & Cavanagh 1990, 71-72.) An example graph of knee motion during the running gait cycle can be seen in Figure 9.

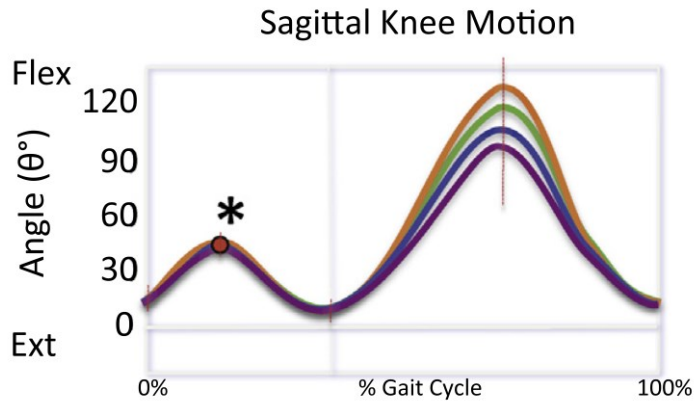

FIGURE 9. Knee angle over one gait cycle at four different running velocities in recreational runners. Horizontal line represents the point of toe-off. (Orendurff et al. 2018).

4.5 Hip angle

In this text the hip angle is defined as the position of the thigh in relation to the position of the pelvis. When the thigh and pelvis are in the same line, the hip angle is 0°. For instance, when standing still, the hip angle is almost 0°. (Novacheck 1998) One other definition used in literature is the position of the thigh in relation to the vertical line (Milliron & Cavanagh 1990, 69).

In the running gait, during the initial contact, the hip joint is slightly flexed, and the hip angle is between 20-25 degrees. At the beginning of the stance phase, there are only small changes in the hip angle until the knee is maximally flexed. Once the knee is at its maximum flexion, the hip starts to extend simultaneously with the knee until the hip angle reaches its maximal extension right before or right after the toe-off. During the swing phase, the hip begins to flex almost instantly after toe-off, but hip flexion occurs slower than simultaneous knee flexion. Hip flexion continues after the knee flexion reaches its maximum value, and the knee starts to extend while hip flexion still continues. Once the hip reaches its maximal flexion, it begins to extend until the new initial contact starts the gait cycle again (figure 10). (Milliron & Cavanagh 1990., 69-71)

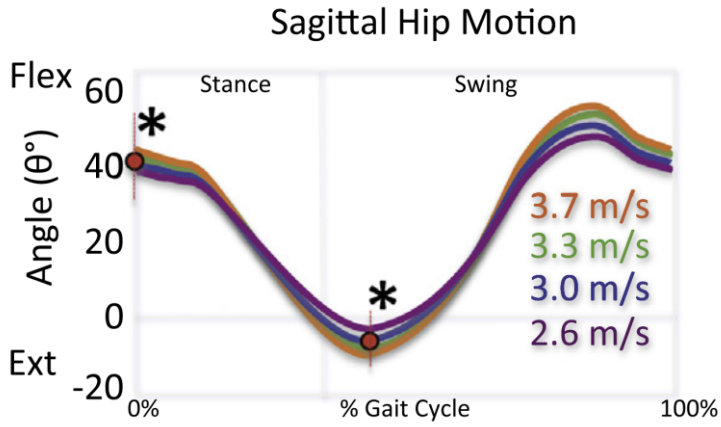

FIGURE 10. Hip angle over one gait cycle at four different running velocities for recreational runners. Horizontal line represents the point of toe-off. (Orendurff et al. 2018).

4.6 Acute responses of strength exercise on running kinematics

Strength exercise induced fatigue has been shown to alter functions of neuromuscular system. These alterations cause variation to running economy through several different mechanisms that are discussed earlier in this text. Many studies have reported changes in running kinematics within 0-48 hours after exercise. Commonly used exercises to induce fatigue include eccentric exercises for the lower extremities (Braun & Dutto 2003; Paschalis et al. 2005) and downhill running (Dutto & Braun 2004; Chen et al. 2007; Chen et al. 2009). Doma & Deakin (2013) utilized "typical" strength exercises that could be performed by endurance athletes with dynamic movements. All these types of exercises also cause exercise-induced muscle damage and muscle soreness, which are suggested to be linked to altered running kinematics, in addition to neuromuscular fatigue.

It appears that the mechanics of the hip (Chen et al. 2009; Doma & Deakin 2013), knee (Dutto & Braun 2004; Chen et al. 2007; Paschalis et al. 2007; Chen et al. 2009), and ankle (Hamill et al. 1991; Chen et al. 2007; Chen et al. 2009) during submaximal running velocities are the most commonly affected kinematic variables by strength exercise. Some studies have also demonstrated alterations in stride length and frequency (Chen et al. 2007; Chen et al. 2009), while others did not find changes in these variables (Hamill et al. 1991; Palmer & Sleivert 2001). It is evident that strength exercise-induced fatigue, exercise-induced muscle damage (EIMD), and delayed onset muscle soreness (DOMS) can cause changes in running kinematics. The magnitude of these changes may vary greatly depending possibly on the type of strength exercise and the background of the test subjects.

It has been suggested that strength exercise induced changes in the range of motion of the hip, knee, and ankle joints is a self-protection mechanism to prevent further damage (Paschalis et al. 2005). According to Doma et al. (2017), there are several mechanisms that can reduce range of motion, including the perception of muscle pain, changes in motor unit activation patterns, reduced stretch reflex sensitivity, and a reduced ability to utilize the stretch-shortening cycle during running. All of these changes can impair movement efficiency and increase energy expenditure, potentially leading to suboptimal endurance adaptations in endurance exercise performed post strength exercise. (Doma et al. 2017)

5 MUSCLE ACTIVATION

When a signal is sent from a nerve to a muscle, it creates a reaction in the muscle that can be detected with electrodes. This measurement is called an electromyogram (EMG). Doctors use electromyogram readings to diagnose issues with the connection between nerves and muscles, while ergonomists use them to figure out what tasks people can do at work. Physiologists study electromyograms to better understand how the neuromuscular system adapts, while biomechanists use them to estimate how much force a muscle can produce. (Enoka 2008, 197)

EMG is used in biomechanics to explore three key areas: understanding when muscles become active, identifying when muscle fatigue occurs, and examining the correlation between EMG signals and the force generated by muscles. By analyzing the timing of muscle activity, we can identify the steps involved in a task that requires multiple muscles, like walking and running. Additionally, by examining the link between EMG signals and muscle force, we can determine how much each muscle contributes to force production. (De Luca 1997)

5.1 Measuring surface EMG

Measuring muscle activation by surface EMG signals is a non-invasive way to measure muscle activity. Needle electrodes are another way but that is an invasive method as with needle electrodes intramuscular EMG is measured within a muscle. (Chowdhury et al. 2013) Surface EMG electrodes serve as a detector of the electrical activity in muscles and their job is to convert the ion flow in the muscle tissue into an electrical current through the metal conductors of the electrode. (Merletti et al. 2009) In EMG measurements, the use of practical surface electrodes has been favored over invasive needle and wire electrodes, which are mainly used to study deeper muscles or the characteristics of individual motor units (Raez et al. 2006).

The surface EMG recording are usually recorded with bipolar setting, where two electrodes (usually silver or silver-chloride) are placed on the muscle and resulting EMG signal represents the difference between the two electrodes (Enoka 2008, 198). The electromyogram represents the summation of all active motor unit action potentials of all muscle fibers that pass through the detection area of the EMG electrodes. When using surface EMG, usually the goal is to observe the combined activity of as many motor units as possible. Surface EMG shows the summation of action potentials of multiple motor units due to the larger surface area of the

electrodes. This provides a more general picture of the overall muscle activity compared to intramuscular electrodes. (Raez et al. 2006; Merletti et al. 2009)

5.2 Factors affecting surface EMG signal

The surface EMG signal can provide information about the amount of force or torque produced by a joint, but accurately determining this relationship is challenging due to the number of factors that can influence the signal. While some of these factors can be controlled, others cannot, and their impact on the signal might be difficult to estimate. (Chowdhury et al. 2013) The frequency and amplitude content of the EMG signal are affected by several factors, which can be categorized into anatomical, physiological, physical, and detection system-based factors (Farina et al. 2002).

Anatomical factors affecting EMG signal are the thickness of skin and subcutaneous tissues, the structure and distribution of motor units, the number and size of muscle fibers, and the location of neuromuscular junctions (DeLuca 1997; Farina et al. 2002). Additionally, anatomical factors also include changes in three-dimensional muscle geometry during muscle contraction, which can cause significant shifts in electrode placement in relation to the muscle, leading to alterations in the recorded EMG signal (Farina et al. 2004).

The EMG signal is influenced by various physiological factors related to active motor units during muscle contractions. Recruitment patterns and the number of active motor units, motor unit firing rate and frequency, and conduction velocity of the action potentials are all important factors affecting the EMG signal (DeLuca 1997; Farina et al. 2002). Synchronization of active motor units is also crucial for EMG signal detection, as it is the summation of all action potentials generated by active motor units. However, if different motor units fire at different times, particularly during surface EMG measurements, it can have a negative effect. If a positive phase of one action potential occurs simultaneously with a negative phase of another action potential, the resulting summed signal will be canceled out. Consequently, the different timing of motor unit action potentials decreases the signal amplitude, which may result in an underestimation of muscle activity. (Yao et al. 2000)

Physical factors that can affect surface EMG measurements include for example the conductivity of the tissues being measured, crosstalk and movement artifacts. Crosstalk refers

to signals from muscles other than the one being studied that are detected by the measurement electrodes. The degree of crosstalk depends on a variety of anatomical factors, such as the thickness of the subcutaneous layer and the length of the muscle fibers, as well as detection system parameters, including the type of spatial filter used, the distance between electrodes, and the size and location of the electrodes. (Merletti & Parker 2004, 249-250) Movement artifacts in the EMG signal can occur due to the movement of the cable connecting the electrode to the amplifier, as well as the interface between the electrode detection surface and the skin (Chowdhury et al. 2013). The quality of interface between electrode and skin is important so that noise and impedance are within recommendations (De Luca 1997). Recommendations related to the electrode placement locations and procedures have been produced by the European cooperation project SENIAM (Surface Electromyography for the Non-Invasive Assessment of Muscles) (Hermens et al. 2000).

5.3 Muscle activity during running

Muscle activity patterns during running have been extensively researched. Typically, muscle activity is highest immediately before (pre-contact activation) and after the initial contact. At these points, muscle contraction appears to be more critical than during the rest of the stance phase or toe-off (Novacheck, 1998). In walking and running gait, muscle activity tends to correspond with changes in speed (Murray et al. 1984; Kyröläinen et al. 2005). As the load and opposing forces increase during movement, the human body must generate more force through muscle contraction, leading to higher levels of muscle activity. With increasing speed, the segments of the limbs move with a greater range of motion in a shorter time (Murray et al. 1984). The greatest increase in muscle activity with increasing running speed is observed during pre-contact activity and the braking and push-off phases of the gait cycle (Kyröläinen et al. 2005).

Figure 11 presents an example of typical interference EMG signals during running over one gait cycle (A) and an averaged EMG signal of eight muscles during running over one gait cycle (B) (Darandeli et al. 2023). Most of the muscles show the highest activation from the late swing phase (to prepare for the ground contact) to the mid-stance phase. For increasing speeds, the activity of rectus femoris and biceps femoris increases significantly, as previously shown in the literature (Kyröläinen et al. 2005). Due to the need for rapid hip flexion and extension during the swing phase, there is an increase in hamstring muscle (hip extensor) and biceps femoris (hip

flexor) activity (Kyröläinen et al. 2005). It should be noted that early cessation of EMG activity in midstance of quadriceps and gastrosoleus does not always indicate lack of force production, as muscles can produce force without noticeable EMG activity (Novacheck 1998).

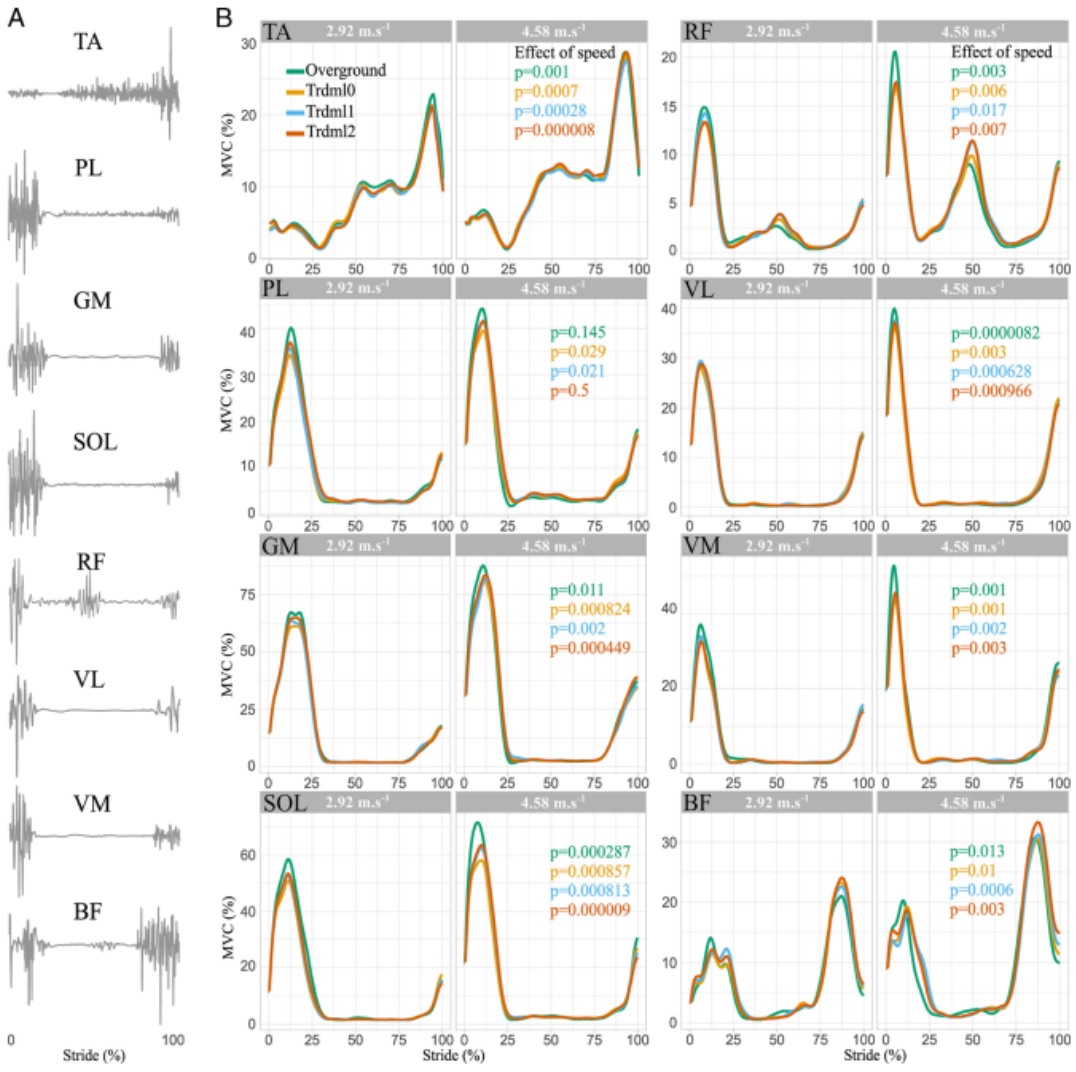

FIGURE 11. (A) Example of typical interference EMG signals during running over one gait cycle. (B) Averaged EMG signal of eight muscle at three different treadmill angle and overground at two different velocities over one gait cycle. (Darendeli et al. 2023)

5.4 Acute responses of strength exercise on muscle activation

There are conflicting results regarding the acute effects of different strength exercises on muscle activation and neural processes. However, there seems to be consensus that hypertrophic strength exercise does not reduce EMG amplitude acutely post-exercise (Izquierdo et al. 2009;

McCaulley et al. 2009; Walker et al. 2012). Although there have been some results where hypertrophic strength exercise induced decrease in EMG amplitude acutely post-exercise (Häkkinen 1994; Ahtiainen & Häkkinen 2009), which might be due to difference in test subjects (trained vs untrained). The lack of reduced EMG amplitude post hypertrophic exercise has been shown mostly on test subjects who doesn't perform strength training regularly (Walker 2019).

Most likely due to the high amount of peripheral fatigue caused by hypertrophic strength exercise the neural system does not experience fatigue similarly. Nevertheless, this does not imply that there are no changes within the neural system after hypertrophic strength exercise. Walker et al. (2012) demonstrated a change in the frequency component of the EMG signal, with a reduction of median frequency. The median frequency of the EMG signal represents the average conduction velocity of the firing motor units (Solomonow et al. 1987). According to Walker (2019), this might indicate that the conduction velocity of the action potential decreases within the muscle due to peripheral fatigue. Another explanation is that greater synchronization of the motor units occurs, which reduces the median frequency. When this increase in motor unit synchronization is presented with the expected decrease in motor unit recruitment and firing frequency, it means that the EMG signal amplitude remains almost unchanged. However, as mentioned earlier there have been studies reporting a decrease in EMG amplitude acutely post hypertrophic strength exercise, but these studies have focused on strength athletes as test subjects. It is possible that due to their previous training and already high level of motor unit synchronization, there is not much potential for further improvement compared to untrained individuals. (Walker 2019)

Neural strength exercise causes significant decrease in EMG amplitude and force production capacity post exercise (Linnamo et al. 1998; McCaulley et al. 2009). In previous literature explosive and maximal strength exercise are often considered separately whereas in this study neural strength exercise is used as combination of maximal and explosive strength exercise. Differences between maximal and hypertrophic strength exercise in acute EMG responses post exercise is most likely due to maximal strength exercise inducing stimulus to the nervous system resulting in great amount of central fatigue. This has been shown to induce significant changes in rate of force development immediately after the exercise (Häkkinen 1994) and even 48 hours post exercise (McCaulley et al. 2009). Recovery of rate of force development (RFD) seems to be significantly slower for maximal- compared to hypertrophic strength exercise. Similar results have been shown for explosive strength exercise (Linnamo et al. 1998;

McCaulley et al. 2009) with exception that explosive strength exercise seems to induce more notably changes during first 100 millisecond of muscle contraction (Linnamo et al. 1998). Walker (2019) suggests that when performing explosive exercise, which involves accelerating the load as quickly as possible, fatigue primarily affects the initiation of muscle contraction. These findings may provide insight into the specific adaptations that occur as a result of different strength training programs.

There are only a limited number of studies that have investigated the effects of strength exercise-induced fatigue on muscle activation during running. Kellis and Liassou (2009) examined the impact of ankle and knee fatigue protocols on lower limb kinematics and muscle activity during level running. Running was performed at submaximal intensity immediately after the fatigue protocol. They found that both knee and ankle fatigue protocols increased the activity of vastus medialis, gastrocnemius, and biceps femoris muscles during the swing phase of running, as well as the pre-contact activity of vastus medialis. No changes in muscle activity were observed during the stance phase or at toe-off. These findings are very similar with the changes in EMG amplitude that occur with increasing running speed (Kyröläinen et al. 2005; Darendeli et al. 2023).

5.5 Repeated bout-effect and EMG

Neural adaptations have been shown to occur due to the repeated bout effect (RBE) (Hyldahl et al. 2017). During repeated bouts of strength exercise, an increase in EMG signal amplitude has been observed in relation to force production, suggesting an improved recruitment and synchronization of motor units. Additionally, activation of type I muscle fibers or muscle fibers with less damage may limit stress on damaged fibers (McHugh et al. 2003; Pincheira et al. 2021). Since type II motor units are more susceptible to damage caused by eccentric exercise, it is possible that during repeated exercise, activation of type I motor units increases. This may lead to a decrease in exercise-induced muscle damage and DOMS after the second exercise bout. (Warren et al. 2000)

According to Hyldahl et al. (2017), increasing the synchronization of motor units has been suggested as a mechanism used by the central nervous system to enhance coordination between synergistic muscles. Therefore, it is believed that during repeated submaximal eccentric training, the central nervous system adapts the motoneuron pool to protect the muscle from

further damage by distributing the mechanical load across a greater number of motor units (Hyldahl et al. 2017). However, these adaptations seem to be muscle dependent. For instance, Pincheira et al. (2021) did not observe any changes in medial gastrocnemius muscle activity distribution due to the RBE, but changes in motor unit activation patterns have been observed in the more susceptible muscle (biceps brachii) to eccentric damage. Protective adaptations, therefore, seem to be specific to the muscle or region (upper vs lower extremities). (Pincheira et al. 2021)

6 PURPOSE OF THE STUDY

Previous studies have extensively investigated the acute effects of strength exercise on various endurance-related variables such as running economy, maximal endurance performance, and running kinematics. However, most prior research has focused on either hypertrophic strength exercise or eccentric exercise such as downhill running, as they typically induce EIMD and DOMS, which could have a greater impact on endurance performance. Neural strength exercise is a typical type of training for endurance athletes, so it is important to also investigate the acute effects of neural strength exercise on endurance performance and how they differ from hypertrophic strength exercise. Additionally, there is limited research that has investigated the acute effects of strength exercise on muscle activation during subsequent endurance exercise. The aim of this study is to investigate the acute effects of neural and hypertrophic strength exercise on running economy, running kinematics, and lower extremity muscle activation in a running economy test performed 48 hours after the strength exercise. Furthermore, we aim to determine if any changes occur due to the repeated bout effect when the strength exercise + running economy test is performed three times.

RESEARCH QUESTION 1. Do neural or hypertrophic strength exercises have an effect on endurance performance, running economy, running kinematics, and lower extremity muscle activation during an endurance test performed 48 hours after the strength exercise?

Hypothesis: Hypertrophic strength exercise causes alterations to both running economy and running kinematics due to exercise-induced muscle damage and delayed onset muscle soreness, which are both more prevalent on not strength trained individuals. Through these changes maximal endurance performance is also attenuated due to hypertrophic strength exercise. (Burt et al. 2013; Doma et al. 2015; Doma et al. 2017) Hypertrophic strength exercise does not induce changes in muscle activation as it causes more peripheral than neural fatigue. (McCaulley et al. 2009; Walker et al. 2012)

Neural exercise induces changes in the EMG signal amplitude of lower limb muscles (either a decrease or increase) during running (Kellis & Liassou 2009; Pincheira et al. 2021). It may not significantly affect endurance performance, running economy or kinematics, depending on the amount of EIMD and DOMS. In untrained test subjects, neural strength exercise can also cause significant amount of EIMD and DOMS, but to a lesser extent than hypertrophic strength

exercise. Neural strength exercise causes a noticeable amount of central fatigue that can be observed even 48 hours post-exercise. (McCaulley et al. 2009)

RESEARCH QUESTION 2. Do the acute effects of neural or hypertrophic strength exercise on endurance performance, running economy, running kinematics and lower extremity muscle activation change over the course of three repetitions (one per week) due to repeated bout effect.

Hypothesis: For both types of strength exercises, the repeated bout effect attenuates the acute effect of the exercise on measured variables during a running economy test. Previous research has demonstrated that both neural and peripheral adaptations occur after the first bout of strength exercise, which provides protection for subsequent bouts (Hyldahl et al. 2017). These adaptations can lead to smaller levels of EIMD and DOMS after the second and third bouts, as well as improved motor unit synchronization (McHugh et al. 2003; Hyldahl et al. 2017). Additionally, neural strength exercises have been shown to improve endurance performance and running economy (Paavolainen et al. 1999; Mikkola et al. 2011; Ronnestad & Mujika 2014), which may also contribute to improvements in subsequent bouts of exercise.

7 METHODS

7.1 Participants

Participants in the study were recruited from among healthy men and women between the ages of 18 and 40 years who had a background in endurance training. The background information for each participant is presented in Table 1. To be eligible for the study, participants had to have a minimum of three years of regular endurance training and no prior history of systematic strength training, or they needed to have taken a one-month break from strength training. However, circuit-type strength training was allowed.

14 test subjects started the study of whom 2 discontinued the study due to illness. The final number of participants considered in the results was 6 for each group or 12 in total. All the physiological variables, muscle activity and running kinematic variables are compared between 4 different time points (Control, bout 1, bout 2 and bout 3) with bouts 1-3 preceding either neural or hypertrophic strength exercise 48±3 hours prior to running economy test. For the maximum values of physiological variables one participants' values were marked as missing due to the interruption of the bout 2 running economy test after submaximal loads.

Recruitment was carried out through the Facebook page and email list of the Faculty of Sport and Health Sciences at the University of Jyväskylä. Recruitment letters were also sent via email to endurance sports clubs in the Jyväskylä region. Ethical statement for the study was granted by the Ethical Committee of the University of Jyväskylä.

TABLE 1. Number of test subjects (n) and background information of the subjects of the study. Sex, age, height, weight and $VO2_{max}$ presented as mean ± standard deviation. HYP = hypertrophic strength exercise group, NEU = neural strength exercise group.

Group	*n*	Sex (f/m)	Age (years)	Height (cm)	Body mass (kg)	$VO2_{max}$ (ml/kg/min)
HYP	6	2/4	34.5 ± 8.0	169.4 ± 12.3	65.7 ± 12.3	49.4 ± 5.6
NEU	6	3/3	32.5 ± 5.9	171.9 ± 9.7	71.9 ± 11.2	48.4 ± 6.4
All	12	5/7	33.5 ± 6.8	170.7 ± 10.7	68.8 ± 11.7	48.9 ± 5.8

All the measurements in this study were carried out in the Faculty of Sport and Health Sciences in the University of Jyväskylä during autumn 2019.

7.2 Research design

This study was conducted over a period of 2 weeks of pre-measurements and 2-3 weeks of experimental testing. The research design is shown in Figure 12. All participants underwent 9 visits to the laboratory. The first measurement was a $VO2_{max}$ test, which was the same for all participants. After the test, the participants were randomly assigned to either a neural strength exercise group (NEU) or a hypertrophic strength exercise group (HYP). The randomization was carried out so that there were no significant differences in $VO2_{max}$ between the groups.

The second measurement involved introducing the participants to the strength exercise equipment and protocols and determining the exercise loads. For the hypertrophic strength exercise group, 8RM determination was done for leg press, knee extension, and knee flexion, and 12 RM for calf press. For the neural strength exercise group, 3 RM was determined for leg press and plyometric movements were then trained. Three to five days after this (to ensure adequate recovery), control measurements were taken, including isometric leg press (including RFD=rate of force development measurement), countermovement jump, and running economy test.

After the pre-measurements, there was a recovery period of 5-7 days before the actual experimental weeks. During experimental weeks the test subjects performed either hypertrophic or neural strength exercise, based on their assigned group, and a running economy test 48±3 hours after the strength exercise. This was done three times, once a week, with 4-6 days in between the running economy test and subsequent strength exercise. Both strength exercises and running economy tests were carried out at the same time of day (within 3 hours) to avoid any effects of circadian rhythm on performance.

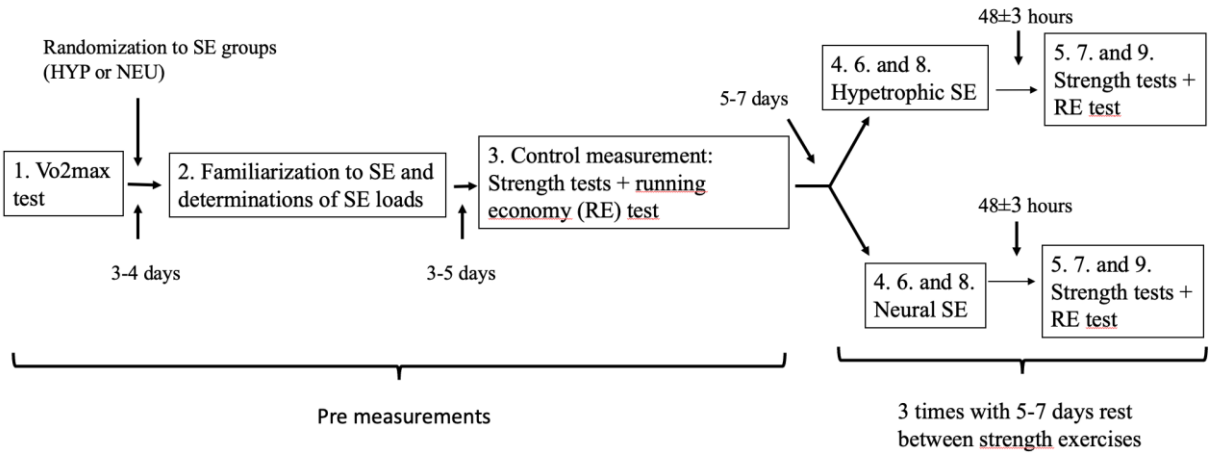

FIGURE 12. Research design. There were altogether nine visits per test subject. First three were premeasurements and on 4th, 6th and 8th visits test subjects performed strength exercise and on 5th, 7th and 9th visit strength and running economy test(s) were performed.

Participants training or nutrition throughout the whole study were not recorded. All participants were asked to come to testing in well rested state and do the preparations for the tests similarly every time. It was also instructed not to perform any heavy training during the study (pre-measurement and experimental weeks). Light training (aerobic) was allowed.

7.3 Data collection

During the first measurement session, participants were provided with comprehensive information about the study and the tests that would be performed. They were required to fill out a consent form, which they had previously reviewed, and complete pre-information forms to assess potential risks and their previous activity levels. In addition, the participants' height and weight were measured.

$VO2_{max}$ test and running economy tests were conducted on a motorized treadmill (OJK -Komi (0402/73, Telineyhtymä Kotka, Finland). Prior to the test a controlled warmup of 10 minutes was performed at a speed that was assessed to be the speed of first load in $VO2_{max}$ test. During warmup test subjects heart rate and appearance was followed and starting speed was adjusted based on those if needed. Throughout the $VO2_{max}$ test, the treadmill angle was maintained at 0.6 degrees, and the speed was increased by 1 km/h every three minutes until voluntary exhaustion. After each speed (3min) treadmill was stopped and lactate sample was collected

from persons fingertip for lactate analysis. Lactate samples were collected also before (rest lactate) and right after test (maximal lactate). Lactate samples were analyzed with Biosen S-line lactate and glucose analyzer (EKF-diagnostic GmbH Eberdorfer Chaussee 3, Germany). Lactate sample collection took approximately 15-25 second so actual running time per load was 2 min 35-45 second.

During $VO2_{max}$ test heart rate was measured using Polar V800 or Polar M430 sport watch with polar H10 heart rate sensor (Polar Electro Oy, Kempele, Finland). The heart rate was measured continuously throughout the test, and the average heart rate over 15 seconds at the start of the last half-minute of the load was recorded as the corresponding load's heart rate. Gas exchange was also measured throughout the test using a Vyntus CPX metabolic cart (Jaeger Vyntus™ CPX, CareFusion Germany 234 GmbH, Hoechberg, Germany). The gas exchange was measured breath by breath and averaged to 30 second intervals. Average of last 30s per load was used for analysis except for $VO2_{max}$ the highest 60 second average was used.

Based on the $VO2_{max}$ test, the aerobic and anaerobic threshold speed, heart rate, oxygen uptake, and lactate were determined for each test subject. The thresholds were calculated using the Klab method, which is widely used and accepted in Finland, with Microsoft Office Excel 2016 software. The aerobic threshold is defined as 0.3 mmol/l above the lowest lactate value during the test. The anaerobic threshold is determined at the intersection of two linear regressions. The regressions were plotted according to the lactate curve, with the first regression drawn between the aerobic threshold and the next lactate value, and the second regression drawn based on all loads where lactate had risen 0.8 mmol/L or more from the previous load. For this study, the anaerobic threshold was the most important because the speeds for running economy tests were determined based on the speed at the anaerobic threshold.

Strength tests were conducted prior to all running economy tests, which included a maximal voluntary isometric contraction (MVIC) in leg press, countermovement jump (CMJ), and assessment of muscle soreness. The isometric leg press was performed on a custom-made machine (Sports and Health laboratory, Jyväskylä, Finland) with a 110-degree knee angle (figure 13). In each measurement, the test subject performed three repetitions with a 2-minute rest period between attempts. Test subjects were strongly encouraged verbally to produce as much force as they could. If the force continued to increase, then a fourth repetition was performed. We also measured the rate of force development (RFD) during the isometric leg

press. Test subjects were instructed to produce as much force as possible as quickly as possible for about 2-4 seconds, until the force curve no longer increased. The maximum force was determined by the highest value achieved during the test, and obvious peaks due to improper technique were not included in the calculations. From the force curve we calculated rate of force development for two different time windows 0-50 milliseconds ($RFD^{0\text{-}50ms}$) and 100-200 ms ($RFD^{100\text{-}200ms}$). RFD has been shown to be useful as indicator for acute muscle damage and exercise-induced neuromuscular fatigue (Oliveria et al. 2013, Rodriquez-Rosell et al. 2017).

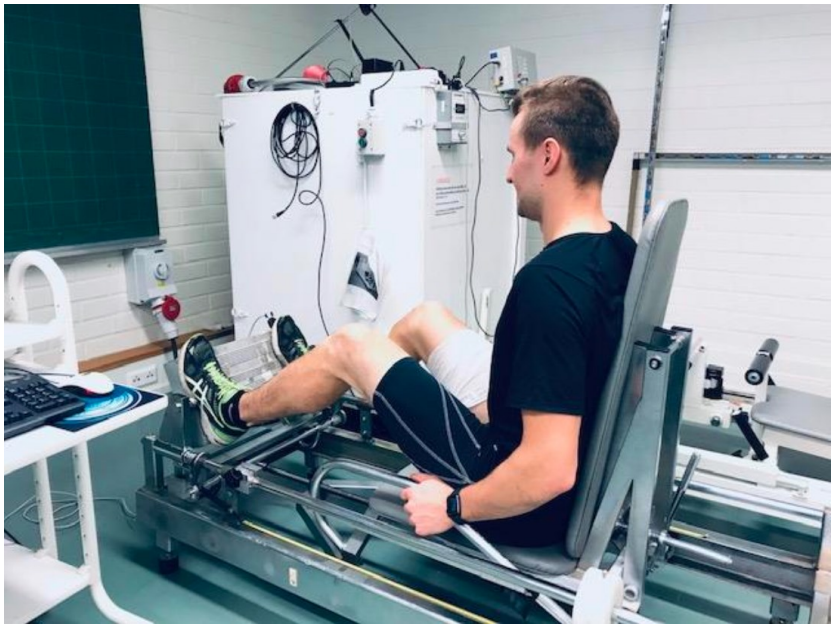

FIGURE 13. Isometric leg press.

Countermovement jumps were conducted using custom made contact mat (Sports and Health Laboratory, Jyväskylä, Finland) as seen in figure 14. Contact mat measured the flight time of the jump from which the jump height was calculated using the formula $h=1/8*g*t^2$, where g represents the acceleration due to gravity (9.81 m/s^2) and t represents the flight time. Three successful repetitions of CMJ were performed with 2 minutes recovery in between attempts. Only the best of the three jumps was used for analysis.

FIGURE 14. Countermovement jump (CMJ) was performed prior to all running economy tests on a custom-made contact mat.

The assessment of muscle soreness was carried out using visual analogue scale (VAS) (appendix 1). To assess muscle soreness, the test subject performed a 90-degree squat with their body weight, and after the squat, they drew a line on a scale to indicate the degree of perceived muscle soreness. We measured the percentage of where the drawn line was on the scale, with 0% indicating no muscle soreness and 100% indicating muscles that were too sore to move. VAS scale has been used in previous studies (Burt et al. 2013) and have been proven to be valid method to measure muscle soreness (Price et al. 1983).

Running economy test was performed four times overall during the study. First on 3rd visit that served as control and then 48 hours after each strength exercise. The warm-up procedure for the test subject comprised 10 minutes of controlled warm-up on a treadmill, starting with a 5-minute run at 60 % speed of anaerobic threshold (vAnT), followed by 2 minutes of 75 % vAnT, 1 minute of 90 % vAnT, and finally 2 minutes at 60 % vAnT. Muscle soreness assessment and strength tests, including maximum voluntary isometric contraction (MVIC) on leg press and countermovement jump, were performed after the warm-up.

The actual running economy test involved running for 10 minutes at 90 % speed of anaerobic threshold, followed by 10 minutes at anaerobic threshold speed, and then gradually increasing

the speed by 1 km/h every minute until voluntary exhaustion. The treadmill was set at a 0.6-degree angle throughout the test. Lactate samples were collected at the end of both 10-minute loads and immediately after the test. Ratings of perceived exertion (RPE) and heart rate were measured at four time points during the test (5, 10, 15, and 20 minutes), with heart rate measured as the average heart rate over 15 seconds at the start of the last half-minute (e.g., at 4 minutes 30 seconds to 4 minutes 45 seconds). Breathing gases were measured during the running economy test, similar to a $VO2_{max}$ test. Additionally, muscle activity from four lower limb muscles and 2D running kinematics were measured during the running economy test.

7.3.1 Muscle activity and running kinematics

Both muscle activity and running kinematics were measured at six time points during the running economy test. Both measurements started at time points 3, 6, 9, 13, 16 and 19 minutes so there were three measurements per one 10 min load.

Muscle activity was measured during running economy test of rectus femoris (RF), vastus lateralis (VL), biceps femoris (BF) and lateral gastrocnemius (GA) from the right-side leg. Bipolar Ag/AgCl EMG electrodes (Ambu bluesensor N) were used to record EMG signal amplitude with 2 cm inter-electrode distance. SENIAM guidelines (Hermes et al. 2000) were followed for electrode placement and skin preparation. Electrode locations were prepared by shaving the skin, using abrasion and cleaning with alcohol. The electrode locations were marked with a permanent marker to ensure consistency between tests. The test subjects were instructed to reinforce the marker between tests', if necessary, as the permanent marker may wear off over time. If marker was worn off in in between RE tests' the location of the marker was palpated again as in the first time. Markers and wires were secured with tape and elastic band to minimize any interference from the movement of wires and/or electrodes. The reference electrode was placed on the lateral malleolus.

The EMG signals were recorded using a portable transmitter (Telemyo 2400T, Noraxon, Scottsdale, AZ, USA), which was attached to the treadmill railings during running. The signal was wirelessly transmitted to a receiver (Telemyo 2400R, Noraxon, Scottsdale, AZ, USA) and sampled at a frequency of 3000 Hz before passing through an analog-to-digital converter (Micro1401, Cambridge Electronic Design, Cambridge, United Kingdom). The Signal software

(Signal 4.11, Cambridge Electronic Design, Cambridge, United Kingdom) was used to capture the EMG data.

The Matlab software, version 9.9.0.1467703 (R2020b) (MathWorks Inc, Massachusetts, United States), was employed to analyze the EMG data. Prior to the analysis, the data was bandpass filtered (15-500 Hz) using custom Matlab scripts. A specific time window (200-400 milliseconds) was determined for each muscle based on the average activation time during the stance phase. Custom Matlab scripts were used to automatically find the highest root mean square (RMS) value for the determined time window, as depicted in Figure 15. For all muscle activity data analysis, the average of 25 consecutive stride cycles was used. Muscle activity values were normalized to the highest single RMS value measured during the second load (anaerobic threshold) of the running economy test.

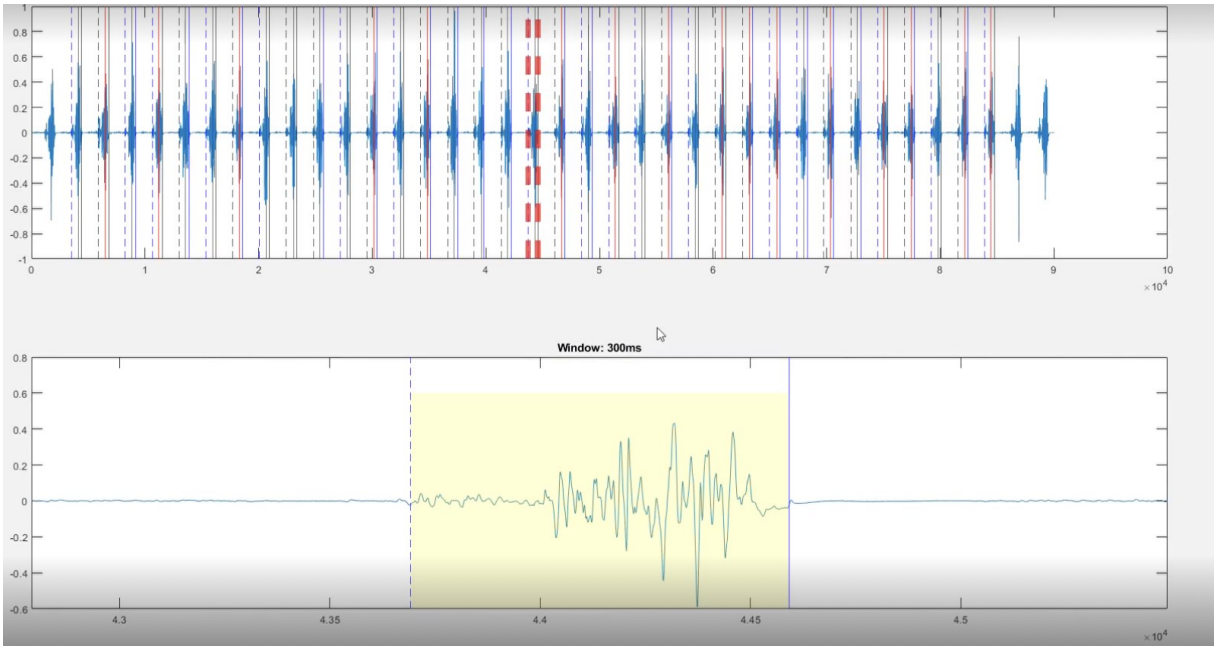

FIGURE 15. Muscle activity analysis for GA-muscle using a custom Matlab script.

Running kinematics was measured using Sony DCS-RX 10 Mark II video camera (Sony Corporation, Tokyo, Japan) with 100 frames per second. Video camera was on a tripod with direct line to runner. Distance from video camera to the nearest rail of treadmill was 3.29 meters (figure 16). Video was calibrated using a metal rectangle frame with dimensions of 200cm x 111 cm. Running kinematics was analyzed using Vicon Motus 10.0.1 software (Vicon motion systems Ltd., Kidlington, United Kingdom). Joint angles were manually digitized at two separate time points within one stride cycle. These time points were initial contact (IC) and toe-

off (TO). Ground contact time was also calculated from data as time between initial contact and toe-off. Joint angles for ankle, knee and hip were measured at those two timepoints from five markers that were placed on the left side of the test subject. Markers were placed on the fifth metatarsal, lateral malleolus, lateral epicondyle, greater trochanter and on anterior superior iliac spine as seen in figure 16. Average of ten consecutive stride cycles was used for all kinematic analysis.

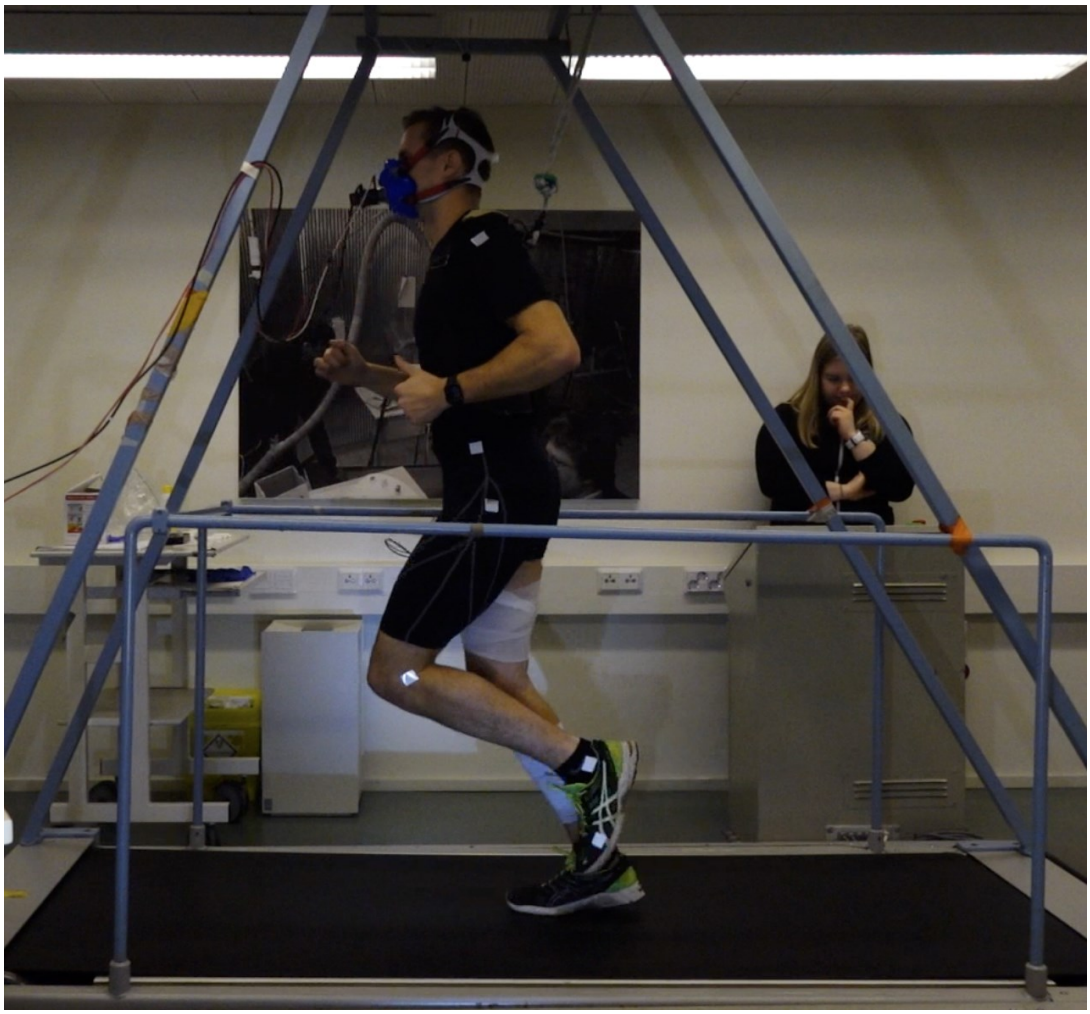

FIGURE 16. Field of view and marker placement in running economy test. Reflective markers were placed on 5 anatomical locations for joint kinematic analysis.

7.3.2 Strength exercises

This study included two different strength exercises. Half of the test subject performed hypertrophic and other half neural strength exercises. Each group had three strength exercises with approximately 1 week in between sessions. During the second visit, the test subjects were

instructed on proper movement techniques and the loads were determined for each strength exercise. The protocol for both strength exercises is presented in table 2. Prior to the strength exercise, the test subjects performed a 10-15-minute non-controlled warm-up, which included cycling on a bicycle ergometer, mobility exercises, and two approach sets on a leg press.

The hypertrophic strength exercise involved four movements with a 2-minute recovery period between sets. Each set was aimed to be performed to failure, and the load was decreased as necessary to enable the subject to complete the required number of repetitions. Additionally, the starting loads for the strength exercises were increased as necessary during the study.
The neural strength exercise consisted of one maximal strength movement and four plyometric movements. The leg press was performed using a 3-repetition maximum (RM) load, with a 4-minute recovery period between sets. For the plyometric movements, the recovery time between sets was 2 minutes. The drop jump was performed from a 30 cm high stand.

TABLE 2. Sets, repetitions and recovery time between sets (RBS) for both strength exercises. CMJ=countermovement jump, DLB = double leg bounce, ALB = alternate leg bouncing.

	Hypertrophic			Neural	
Exercise	Sets x Reps	RBS	Exercise	Sets x Reps	RBS
Leg press	5 x 8	2 min	Leg press	6 x 3	4 min
Leg extension	3 x 8	2 min	CMJ	2 x 6	2 min
Leg curl	3 x 8	2min	Drop jump	3 x 3	2 min
Calf press	5 x 12	2min	DLB	2 x 10	2 min
			ALB	3 x 5	2 min

7.4 Statistical analyses

Data were analyzed using IBM SPSS 28.0 Statistic software (IBM, United States) and Microsoft Office Excel 365 (Microsoft Corporation, United States). All data are expressed as mean ± standard deviation. Normality of data were checked using Shapiro-Wilk test ($n < 50$). For data between repeated measurement (control, bout 1, bout 2 and bout 3) repeated measures analysis of variance and pairwise comparison with Bonferroni's adjustment was used. Mauchly's test of sphericity was used to check the sphericity of data. If sphericity was violated Greenhouse-Geisser correction was used. Paired samples t-test was also used to compare within-group changes between individual measurements sessions. Statistical significance was set as $p < 0.05$.

8 RESULTS

Running speeds for the running economy test were determined based on the maximal oxygen uptake test results for each participant. At 90 % of the anaerobic threshold speed (90 % vAnT), the neural and hypertrophic groups had mean running speeds of 10.7 ± 1.1 km/h and 10.7 ± 1.3 km/h, respectively. At the anaerobic threshold speed (vAnT), the neural and hypertrophic groups had mean running speeds of 11.9 ± 1.2 km/h and 11.9 ± 1.4 km/h, respectively.

8.1 Strength tests and indirect muscle damage markers

The hypertrophic group did not exhibit any statistically significant differences in maximal force production capacity (MVC), rate of force production (RFD), or countermovement jump (CMJ) performance. The neural strength exercise group showed a statistically significant difference in the RFD during the first 50ms of force production when comparing bouts 2 and 3. For the control and bouts 1 and 2, $RFD^{0\text{-}50ms}$ was quite similar in the neural group, but it decreased noticeably for bout 2. In contrast, $RFD^{100\text{-}200ms}$ slightly increased to bout 3 for the neural group, although the change was not significant. Countermovement jump performance did not show any significant changes between different bouts (table 3).

Muscle soreness was significantly higher in the hypertrophic group compared to the neural group at every time point (the morning after the first strength exercise, the morning after the second strength exercise, and immediately before the running economy test). For both groups, muscle soreness was greatest in the morning after the first bout of strength exercise and showed a downward trend after subsequent bouts. In the neural group, the Visual Analog Scale (VAS) score was significantly lower on the morning after the third strength exercise compared to bout 2.

TABLE 3. Strength test results for maximal (MVC) and explosive strength (RFD and CMJ) and muscle soreness questionnaire results (VAS). Strength exercise was not performed prior to control trial so VAS was not measured. Statistical significance: * $p<0.05$, ** $p<0.01$ and *** $p<0.001$, bouts are shown in brackets between which statistical significance occurs.

Variable	Group	Control	Bout 1	Bout 2	Bout 3
MVC (N)	NEU	3467±1422	3693±1786	3638±1692	3540±1768
	HYP	3903±1375	4094±1319	3922±1502	4297±1144
$RFD^{0\text{-}50ms}$(N/s)	NEU	10057±3974	12125±7548	11737±5450	7274±3205 *(2-3)
	HYP	8512±9349	6708±7784	9501±8961	10066±8947
$RFD^{100\text{-}200ms}$ (N/s)	NEU	6645±2643	7578±4683	7246±5125	8285±5474
	HYP	8742±5933	8325±4169	5664±3819	8693±10668
CMJ (cm)	NEU	31.5±7.5	31.4±7.7	31.6±7.9	32.2±6.9
	HYP	30.1±6.6	29.1±6.9	28.0±6.6	29.5±7.1
$VAS_{morning\ 1}$	NEU	-	12.7±9.7	12.0±10.0	11.2±11.9
	HYP	-	43.5±17.3	22.3±10.8	30.5±12.6
$VAS_{morning\ 2}$	NEU	-	20.8±11.9	13.6±11.0	6.8±6.1 *(1-3)
	HYP	-	47.3±17.7	17.2±13.9 *(1-2)	14.1±9.2 ***(1-3)
VAS_{pre}	NEU	-	19.4±16.0	11,4±12,2	6,4±4,0
	HYP	-	36.1±13.3	9.6±10.4 **(1-2)	11.9±9.5 ***(1-3)

Hypertrophic group showed significant decreases in perceived muscle soreness at each time point after first strength exercise. Greatest decrease in muscle soreness was at 2nd morning after the strength exercise when comparing bouts 1 and 3.

8.2 Running performance

Table 4 shows the results for running performance in running economy tests. Running performance increased for each subsequent test for both groups as indicated by improvements in time-to-exhaustion (TTE). For neural group time to exhaustion increased significantly from control to bout 3 and from bout 2 to bout 3. Time-to-exhaustion in bout 3 was noticeably highest

for both groups compared to other bouts. For hypertrophic group TTE improvement from bout 1 to bout 3 showed statistical significance (table 4).

TABLE 4. Time-to-exhaustion (TTE), peak oxygen uptake ($VO2_{peak}$), peak heart rate (HR_{peak}) and -lactate (La_{peak}) in running economy tests. Statistical significance: * $p<0.05$, ** $p<0.01$ and *** $p<0.001$, bouts are shown in brackets between which statistical significance occurs.

Variable	Group	Control	Bout 1	Bout 2	Bout 3
TTE (s)	NEU	1408±52	1409±117	1427±56	1463±50 *(c-3) *(2-3)
	HYP	1386±92	1422±27	1435±38	1452±33 ***(1-3)
$VO2_{peak}$ (ml/kg/min)	NEU	47.1±5.9	47.4±5.3	48.1±6.0 *(c-2)	49.0±5.3 *(c-3)
	HYP	49.9±8.9	48.9±7.1	47.5±7.3 *(c-2)	48.3±6.2
Hr_{peak}(1/min)	NEU	191±4	191±7	191±6	192±5
	HYP	190±8	189±12	188±11	190±10
La_{peak} (mmol/l)	NEU	7.3±0.5	8.3±1.5	7.8±1.3	8.9±1.4 *(c-3)
	HYP	8.5±1.2	8.9±1.3	9.3±2.3	10.5±2.5

Peak oxygen uptake showed variation between the groups. In the hypertrophic group, $VO2_{peak}$ decreased from the control to bouts 1 and 2 and then showed slight increase in bout 3. The decrease was statistically significant from control to bout 2. In contrast, in the neural group $VO2_{peak}$ was higher for each subsequent test with significant increases from the control to bouts 2 and 3.

Peak heart rate showed no changes between tests. In the hypertrophic group, maximal lactate levels consistently increased for each subsequent test, albeit without reaching statistical significance. Conversely, in the neural group, maximal lactate levels displayed some variability, with a significant increase observed from the control test to bout 3.

8.3 Running economy – physiological variables

In the running economy (RE) tests, running economy was assessed at the end of both 10-minute loads, at 90% of the speed of anaerobic threshold and at the anaerobic threshold speed. The running speeds for these loads were 10.7±1.1 km/h and 11.9±1.2 km/h for the neural group and

10.7±1.3 km/h and 11.9±1.4 km/h for the hypertrophic group. Figure 17 displays the physiological variables measured during the RE tests.

The heart rate of the hypertrophic group showed a decreasing trend from control to bouts 1 and 2. From bout 2 to bout 3, the heart rate increased slightly and significantly at 90% of the speed of anaerobic threshold (160±11 beats/min vs 163±11 beats/min, $p < 0.01$). The neural group did not display any significant changes in heart rate. The rate of perceived exertion only showed slight alterations in both groups, with a statistically significant decrease from bout 2 to bout 3 (12.83±1.94 vs 11.83±2.48, $p < 0.05$) for the neural group at 90% of the speed of anaerobic threshold.

The hypertrophic group showed statistical significance in oxygen uptake at the anaerobic threshold between bouts 1 and 2 (45.6±6.9 vs. 44.3±6.5 ml/kg/min, $p < 0.05$). Oxygen uptake decreased further for bout 3, but the change was not statistically significant. Similarly, the respiratory exchange ratio (RER) at the anaerobic threshold decreased gradually from bout 1 to bout 3 with significant changes between bouts 1 and 3 (1.01±0.03 vs. 0.98±0.03, $p < 0.05$). For the neural group, neither oxygen uptake nor RER showed significant changes, although the RER at 90% of the speed of anaerobic threshold increased with each subsequent bout, but the changes were not statistically significant.

The neural group showed statistical significance in lactate values at the anaerobic threshold between control and bout 3 (5.36±0.82 mmol/l vs. 4.82±0.81 mmol/l, $p < 0.05$). In contrast, the hypertrophic group did not show any significant changes in lactate values measured during the running economy tests.

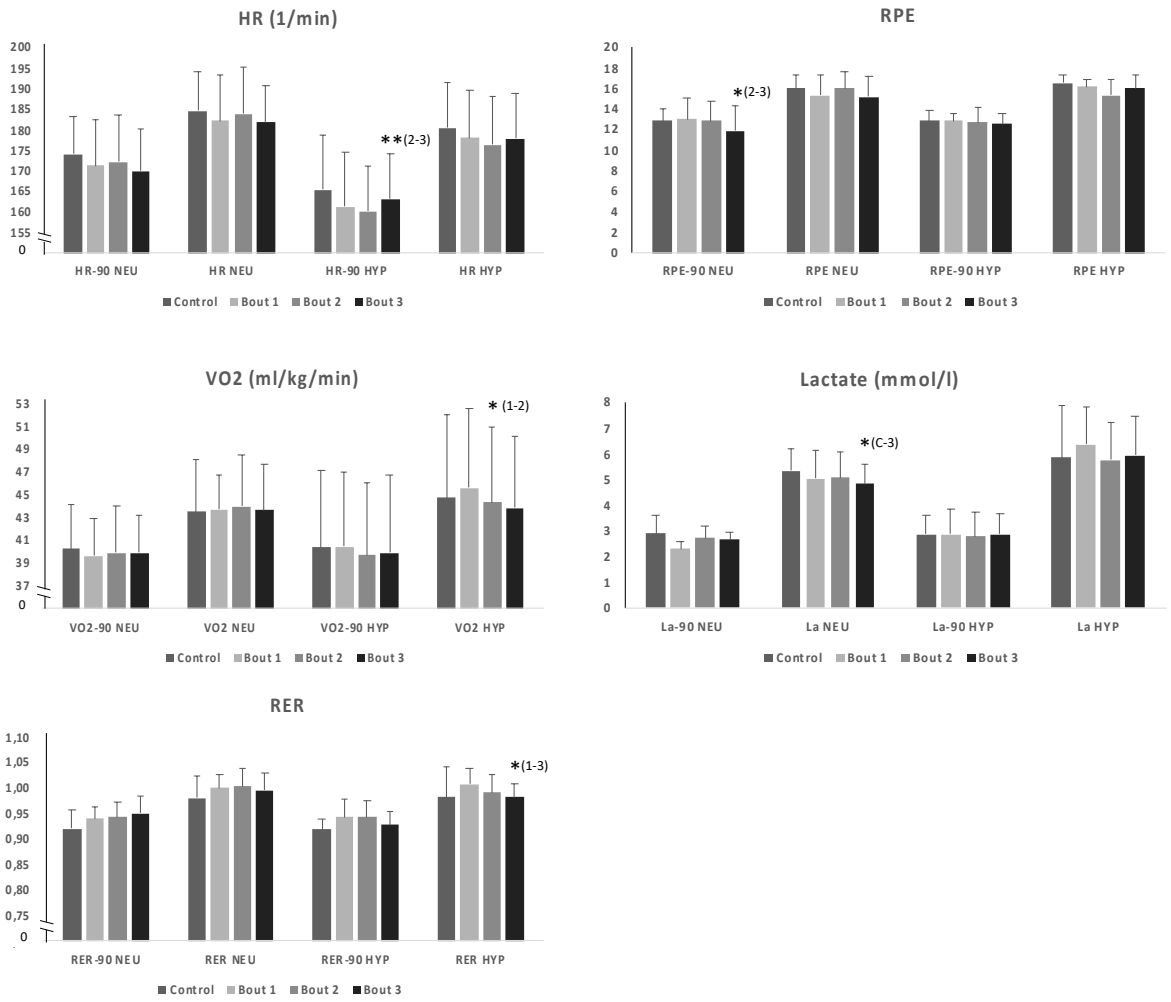

FIGURE 17. Mean (±SD) heart rate (HR), rate of perceived exertion (RPE), oxygen uptake (VO2), lactate and respiratory exchange ratio (RER) in running economy tests at 90 % of anaerobic threshold speed (90) and anaerobic threshold speed for neural (NEU) and hypertrophic (HYP) group. Statistical significance: * $p<0.05$, ** $p<0.01$, bouts are shown in brackets between which statistical significance occurs.

8.4 Kinematic variables

Kinematic variables were measured in running economy tests at three time points (3min, 6min, 9min) from each running speed. All kinematic data are an average of ten consecutive stride cycles. Figure 18 shows the ground contact time, step length and stride frequency for both neural and hypertrophic group.

8.4.1 Ground contact time, step length and stride frequency

Neither the neural nor hypertrophic strength exercise resulted in any significant changes in ground contact time, step length, or stride frequency during running economy tests performed 48±3 hours after the strength exercise. However, the hypertrophic group showed a decrease in step length from control to bout 1, followed by a slight increase in each consecutive bout at every time point. Similarly, the stride frequency for the hypertrophic group increased from control to bout 1 and then decreased (with some alterations) in the subsequent bouts.

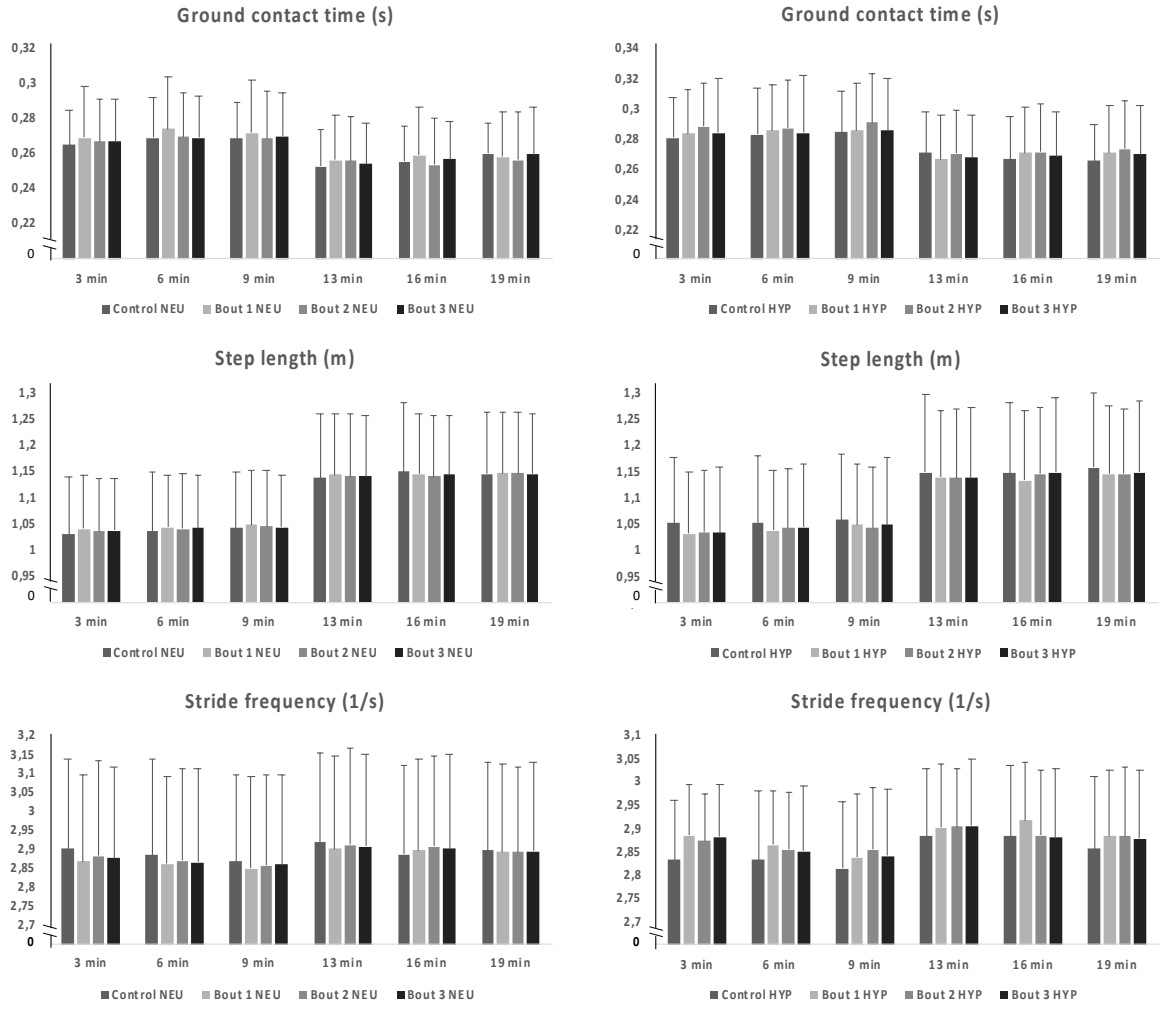

FIGURE 18. Mean (±SD) ground contact time (GCT), step length and stride frequency at six time points in running economy tests for both neural (right side) and hypertrophic group (left side). Statistical significance: * $p<0.05$, bouts are shown in brackets between which statistical significance occurs.

8.4.2 Ankle Angle

The ankle angle exhibited considerable variation in both groups. Statistically significant differences were observed at the 19-minute time point for the hypertrophic group at initial contact between bouts 2 and 3 ($p < 0.05$) and for the neural group at toe-off between control and bout 1 ($p < 0.05$). As seen in figure 19, the hypertrophic group showed an increase in ankle angle from control to bouts 1 and 2 at both initial contact and toe-off, followed by a decrease to nearly the initial level (control) in bout 3, although these changes were not statistically significant.

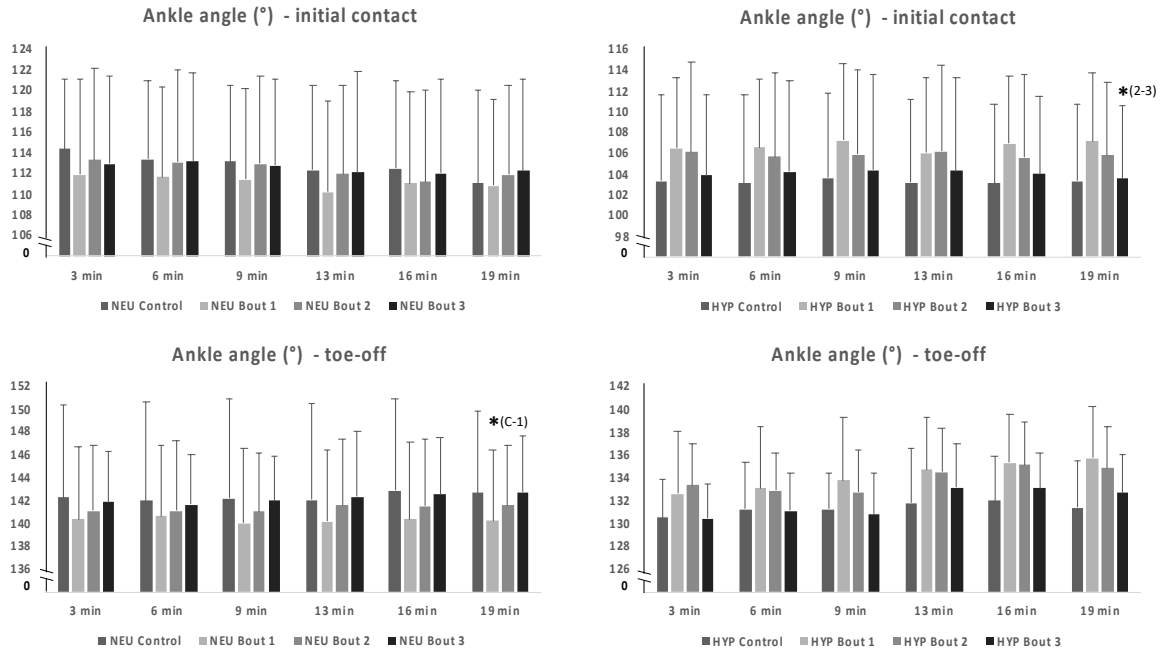

FIGURE 19. Mean (±SD) ankle angle at initial contact and toe-off in running economy tests for both neural (left side) and hypertrophic (right side) groups. Statistical significance: * $p<0.05$, bouts are shown in brackets between which statistical significance occurs.

8.4.3 Knee angle

Figure 20 displays the knee angle at two points of the stride cycle, initial contact, and toe-off. The neural group showed statistical significance at the 9-minute time point between control and bouts 1, 2 and 3 ($p < 0.05$). At the 16-minute time point, significant differences occurred between control and bouts 2 and 3 ($p < 0.05$). Hypertrophic group showed no significant

changes in knee angle at initial contact, although bout 3 shows the lowest values at each time point.

At toe-off, the knee angle appears to increase from the control to the other bouts for the neural group, but significant differences occur only at the 19-minute time point between control and bout 1 ($p < 0.05$). For the hypertrophic group, knee angle at toe-off shows some variation. At the 13-minute time point, there are significant decreases between control and bouts 1-3($p < 0.05$). There is also a statistically significant difference between bout 2 and 3 ($p < 0.05$) at the 13-minute time point and between control and bout 2 ($p < 0.05$) at the 16-minute time point.

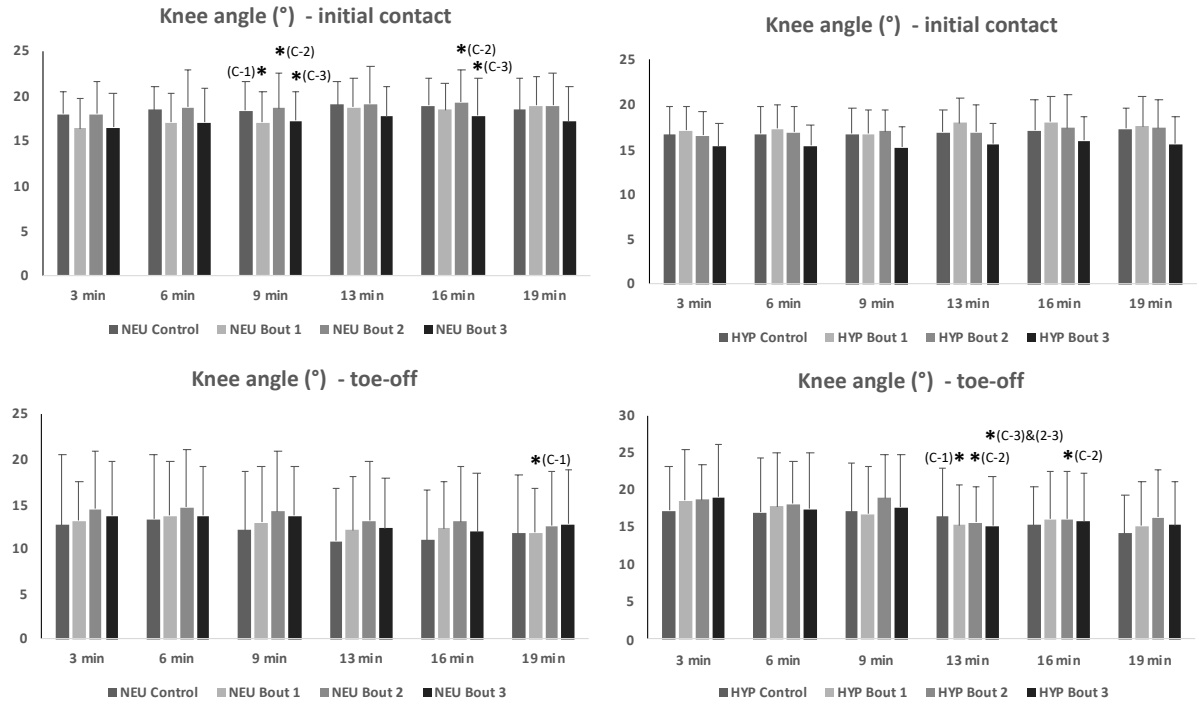

FIGURE 20. Mean (±SD) knee angle at initial contact (upper graphs) and toe-off (lower graphs) in running economy tests for both neural (left side) and hypertrophic (right side) groups. Statistical significance: * $p<0.05$, bouts are shown in brackets between which statistical significance occurs.

8.4.4 Hip angle

Figure 21 indicates that hypertrophic strength exercise did not result in any significant changes in hip angle at the points of initial contact or toe-off. However, for the neural group, hip angle at toe-off exhibited two statistically significant changes: first at the 3-minute timepoint between

the control and bout 3 ($p < 0.05$), and second at the 6-minute timepoint between bout 1 and 2 ($p < 0.05$).

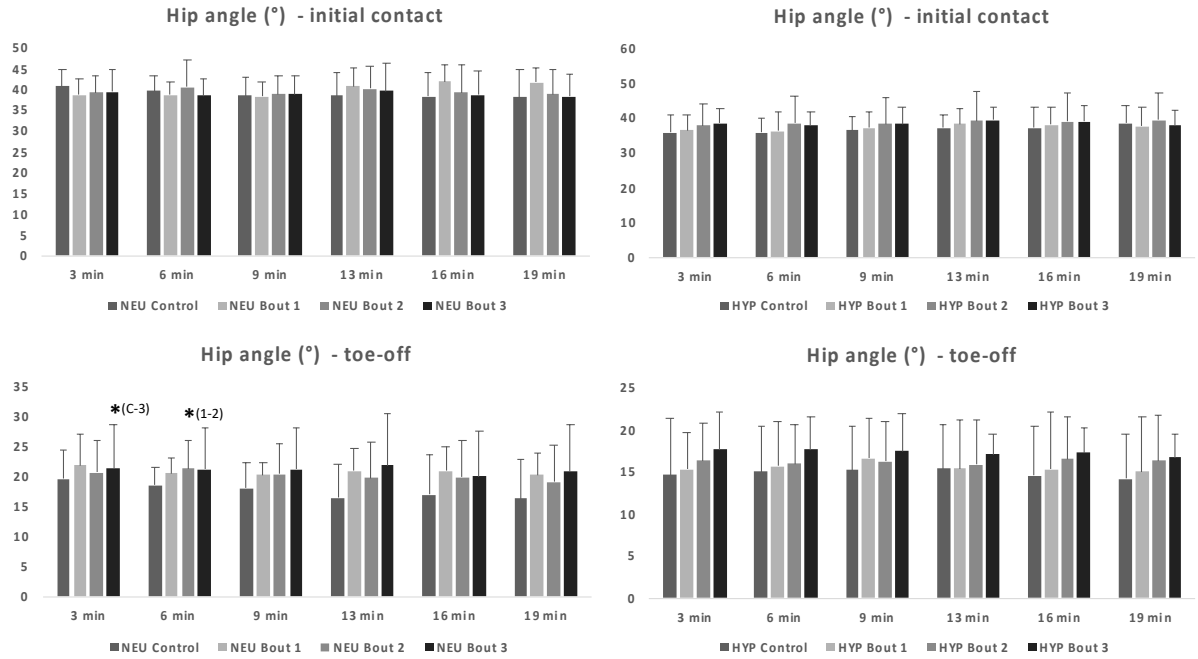

FIGURE 21. Mean (±SD) hip angle at initial contact and toe-off in running economy tests for both neural (left side) and hypertrophic (right side) groups. Statistical significance: * $p<0.05$, bouts are shown in brackets between which statistical significance occurs.

8.5 EMG

One test subject of the neural group was not included in the EMG results due to poor quality of data. There were no statistically significant changes observed in gastrocnemius activation in either of the groups (figure 22). In the hypertrophic group, gastrocnemius activation showed a decreasing trend from the control condition to bout 1 at 90% of the speed of AnT. However, for bouts 2 and 3, activation increased nearly to the level of the control bout. Biceps femoris activation showed a statistically significant change from the control to bout 2 at the 19-minute time point for the hypertrophic group. At time points 3-9, biceps femoris activation showed an increasing trend from the control condition to bouts 1 and 2, but these changes were not statistically significant.

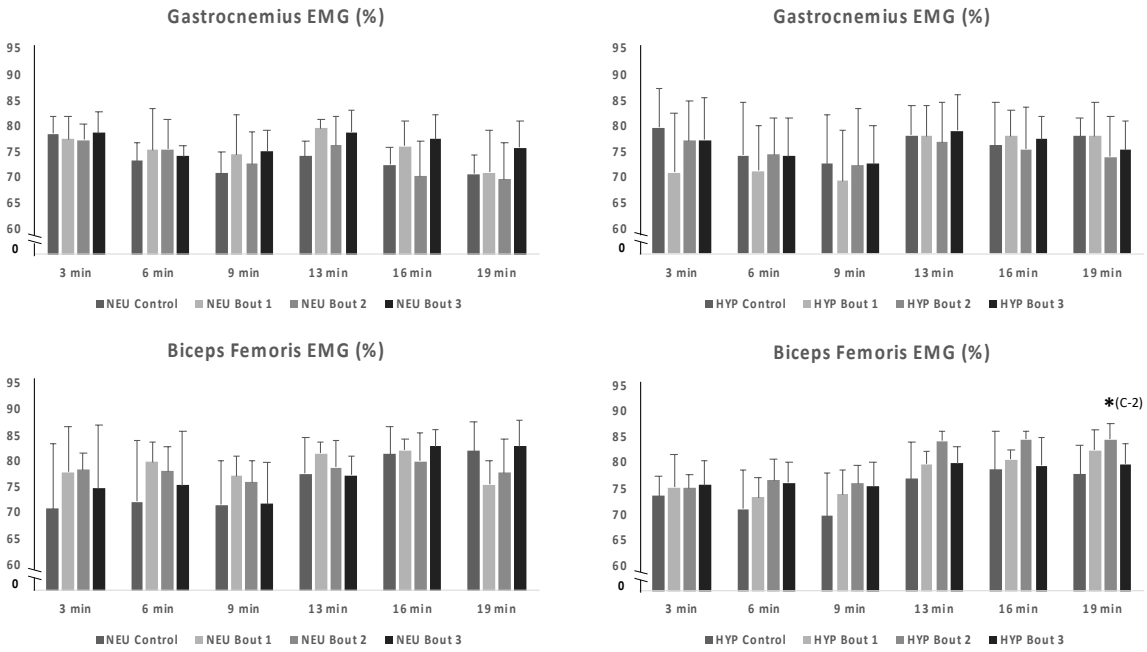

FIGURE 22. Mean (±SD) muscle activation of gastrocnemius and biceps femoris at six time points during running economy test. Data is represented as percentage of highest RMS value measured during second speed (AnT) of RE test. Statistical significance: * $p<0.05$, bouts are shown in brackets between which statistical significance occurs.

There were no statistically significant changes observed in rectus femoris activation in either group (figure 23). However, in the neural group, rectus femoris activation decreased quite noticeably from the control condition to bout 1 at all time points. The activation of the vastus lateralis showed a statistically significant change for the hypertrophic group from the control to bout 1 at the 9-minute time point. Although vastus lateralis activity for the hypertrophic group decreased at time points 3 and 6 minutes, these changes were not statistically significant.

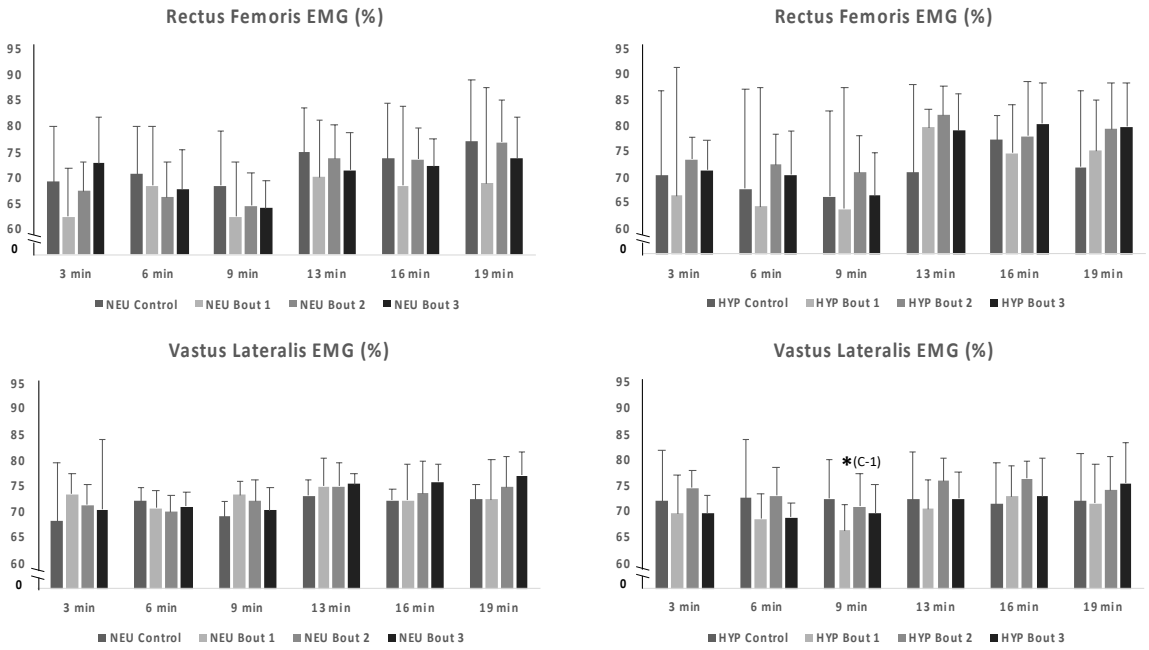

FIGURE 23. Mean (±SD) muscle activation of rectus femoris and vastus lateralis at six time points during running economy test. Data is represented as percentage of highest RMS value measured during second speed (AnT) of RE test. Statistical significance: * $p<0.05$, bouts are shown in brackets between which statistical significance occurs.

9 DISCUSSION

In this study it seemed that hypertrophic strength exercise induced great amount of muscle soreness which led to small changes in running economy, running kinematics and muscle activation in running. These changes attenuated over the course of second and third bouts which indicated the influence of repeated bout effect. Neural strength exercise did not show similar effects as its influence on measured variables during running economy test was minor. This suggest that hypertrophic strength exercise can impair the quality of endurance exercise session performed 48 hours later but neural strength exercise might not.

The running economy test did not reveal any consistent, statistically significant changes in physiological variables. However, for the hypertrophic group, there was a slight trend of decreased running economy from the control test to bout 1, followed by a return to near-control levels in bouts 2 and 3. This trend was observed in oxygen uptake, respiratory exchange ratio, and lactate values at anaerobic threshold, which increased from the control test to bout 1 and then gradually decreased to bouts 2 and 3. Notably, the decrease in respiratory exchange ratio at anaerobic threshold was statistically significant from bout 1 to bout 3. These findings are consistent with the results of the VAS, which showed that subjective muscle soreness was highest after the first strength exercise and then significantly decreased in bouts 2 and 3. These observations are in line with previous studies (Chen et al. 2007; Burt et al. 2013; Doma & Deakin 2013), which have shown that repeating strength exercises reduces their acute effects on running economy. It should be noted that the effects of hypertrophic strength exercise on running economy were smaller in our study than in many previous studies, as we only observed a few statistically significant changes.

Neural group showed contradictory results as for example lactate showed a decreasing trend from control to subsequent bouts (suggesting improved RE) while RER showed an increasing trend (suggesting decreased RE). These changes might be only due to diurnal variations of performance (Knaier et al. 2022) and the results seems to suggest that neural strength exercise does not affect the running economy of subsequent endurance running session 48 hours post exercise, even though presence of some amount (19.4±16 % after first strength exercise) of muscle soreness.

The results for indirect muscle damage markers (i.e., MVC, RFD, and CMJ) showed high variation, and therefore, limited interpretation can be made. However, an interesting observation was made for the countermovement jump performance of the hypertrophic group. Although the changes were not statistically significant, the CMJ performance was found to be lowest after the second strength exercise, whereas the running economy variables showed that running economy was worst after the first strength exercise. Previous literature has shown that there is not always a direct correlation between indirect muscle damage markers and running economy. Several studies have indicated that following the initial bout of strength exercise, there is a clear decrease in both neuromuscular performances, as indicated by muscle damage markers, and running economy. However, subsequent recovery of neuromuscular performance has been found to occur more rapidly than that of running economy (Chen et al. 2009; Doma & Deakin, 2013; Doma et al. 2015).

Kinematic variables, similar to physiological variables, did not show consistent, statistically significant changes in running kinematics in either group. Spatiotemporal variables were almost unchanged for the neural group throughout the study, as ground contact time, step length, and stride frequency showed only very minor changes. For the hypertrophic group, step length showed a small trend to decrease, and consequently, stride frequency increase in bouts 1-3. The decrease in step length and increase in stride frequency seems to be highest at bout 1. The increase in oxygen uptake was also highest at bout 1 for the hypertrophic group. This correlates well with the findings by Chen et al. (2007), which showed that an increase in stride frequency suggests that more energy is required for running at the same intensity.

The changes in spatiotemporal variables for the HYP group are well in line with previous literature, as many studies (Braun & Dutto 2003; Dutto & Braun 2004; Chen et al. 2007; Chen et al. 2009) have shown a decrease in running stride length after strength exercise. This has been suggested to be mainly due to EIMD and DOMS (Dutto & Braun 2004), which may explain why there were only very minor changes in the neural group. Although some studies have not observed alterations in stride length even in the presence of DOMS (Hamill et al. 1991; Palmer & Sleivert 2001). It should be noted that previous studies have found significant differences in spatiotemporal variables, whereas our study did not. The amount of DOMS might be one reason for this, as most previous studies have used eccentric exercises, which generally induce a high amount of EIMD and DOMS, while this study used dynamic exercise with mainly concentric movements.

The changes in joint angles showed some contradictory results for both groups. The ankle angle for the neural group showed a small decrease, to a less plantarflexed position, from control to bout 1 and then gradually returned near the initial level. The ankle angle behaved similarly at both initial contact and toe-off. Previous studies have demonstrated that strength exercise-induced fatigue causes the ankle to be more plantarflexed at initial contact (Christina et al. 2001; Kellis & Liassou 2009). There is a limited number of studies exploring the effect of fatigue on ankle angle at toe-off. Kellis & Lissou (2009) showed no changes, and a meta-analysis and systematic review by Zandbergen et al. (2023) showed that running-induced fatigue showed no changes in ankle angles at toe-off. However, there is clear evidence that ankle range of motion is decreased post strength exercise (Dutto & Braun 2004; Cheng et al. 2007; Cheng et al. 2009). At toe-off, the neural group showed decreased plantarflexion, which might indicate that ankle ROM is compromised. But at initial contact, plantarflexion was also decreased, indicating the opposite. Although some changes in ankle kinematics were observed after the neural strength exercise, most of these changes were not statistically significant. Additionally, previous studies have shown that fatigue effects are more pronounced at initial contact. Based on these findings, it could be concluded that the neural strength exercise did not have a significant impact on ankle kinematics in this study.

The hypertrophic strength exercise group showed a slight increase in ankle plantar flexion at initial contact from the control to bout 1, although this change was not statistically significant. This is consistent with previous studies that have shown that strength exercise-induced muscle damage and DOMS can compromise ankle kinematics (Dutto & Braun, 2004; Cheng et al. 2007; Cheng et al. 2009). However, at toe-off, the ankle angle also showed an increased plantar flexion, which may indicate that the ankle's range of motion was not compromised. Kellis and Lissou (2009) reported similar results after ankle-fatiguing exercise, with a statistically significant 5-degree increase in ankle plantar flexion at initial contact and a 2-degree, statistically nonsignificant, increase in plantar flexion at toe-off. Similar 2-degree statistically nonsignificant increase in plantar flexion at toe-off was reported by Dutto and Braun (2004) in running performed 48 hours after downhill running exercise. It should be noted that the increase in ankle plantar flexion at initial contact was greatest in bout 1 and gradually returned to the initial level, perhaps due to the RBE. Overall, these findings suggest that hypertrophic strength exercise may compromise ankle kinematics at initial contact which is well in line with changes observed in physiological variables of running economy.

Previous research has shown that strength exercise or running-induced fatigue can increase knee flexion angle at both initial contact (IC) and toe-off (TO), while also decreasing range of motion (ROM) (Dutto & Braun, 2004; Chen et al. 2007; Paschalis et al. 2007; Kellis & Lissou, 2009; Landberg et al. 2022). Increasing knee flexion during the impact phase can be a protective mechanism that reduces the risk of injury by decreasing the amount of force applied to the ground and improving shock absorption (Kellis & Lissou 2009). In our study, there were no consistent changes in knee angles for either group. However, the hypertrophic group showed a small increase in knee flexion at most time points from control to bout 1. At the 13-minute time point at TO, there was actually an increase in knee extension from control to bout 1, indicating inconsistent results. It is possible that our strength exercises did not induce sufficient EIMD and DOMS to affect the knee mechanics. Overall, changes in knee kinematics in our study were small, and neither neural nor hypertrophic exercise negatively affected knee angles at IC or TO during the running economy test.

Similar to knee angle, hip angle showed inconsistent results in this study. Previous studies have shown that strength exercise induced fatigue can decrease hip flexion angle at TO (Kellis & Lissou 2009) and hip range of motion (Chen et al. 2009; Doma & Deakin 2013). There are also studies where no changes in hip range of motion were observed (Chen et al. 2007; Paschalis et al. 2007; Satkunskiene et al. 2015). In this study, no changes in hip angle were observed at initial contact for both groups, which is consistent with previous research, as to my knowledge no other studies have found that strength exercise induces changes to hip angle at IC. However, there was a small increase in hip flexion angle at toe-off for both groups, which contradicts some other studies that showed a decrease in hip flexion due to exercise-induced fatigue. Although, Kellis & Lissou (2009) showed a significant decrease in hip flexion after knee fatiguing protocol but increase after ankle fatiguing protocol. So, the type of previous strength exercise as well as the intensity of running might affect the magnitude of changes in hip mechanics, as Chen et al. (2009) noticed changes in hip ROM at only higher running intensities.

EMG amplitude have been shown to decrease acutely after neural strength exercise (Linnamo et al. 1998; McCaulley et al. 2009), but not after hypertrophic strength exercise (Izquierdo et al. 2009; McCaulley et al. 2009; Walker et al. 2012). However these changes have been measured immediately after the strength exercise. Similarly, Kellis & Lissou (2009) found out that knee and ankle fatigue protocol increased the activity of vastus medialis, gastrocnemius, and biceps femoris muscles during the swing phase of running, as well as the pre-contact

activity of vastus medialis. In this case also running was performed immediately after the strength exercise. To my knowledge no other research have done where lower limb muscle activity in running was measured after certain recovery period (in our case 48 hours) after the exercise.

Gastrocnemius (GA) EMG amplitude showed high variation in both groups. For hypertrophic group at speed of 90 % AnT, GA muscle activity decreased (nonsignificantly) from control to bout 1, which might indicate that some amount of central fatigue was presence from the first bout of hypertrophic strength exercise. However, it should be noted that muscle damage might increase presynaptic inhibition of group III and IV muscle afferents (Avela 1998) which could explain decreased EMG amplitude. Similar decrease was not observed at higher speed or after bouts 2 and 3. It's also possible that because of EIMD of gastrocnemius the muscle activation shifted to less fatigued muscle, perhaps soleus. However, GA muscle activation showed high variance and in this study it seems that neither strength exercise caused significant changes in EMG amplitude of gastrocnemius muscle.

In contrast to the GA, the biceps femoris (BF) exhibited an increase in EMG amplitude in the hypertrophic group after bouts 1 and 2, with a statistically significant change at the 19-minute time point between the control and bout 2. An increase in EMG amplitude at the same submaximal speed has been shown to be associated with decreased running economy (Doma et al. 2017). This may be due to exercise-induced muscle damage (EIMD) and delayed onset muscle soreness (DOMS), which likely caused some concurrent alterations in running kinematics as discussed previously. Kellis & Lissout (2009) suggested that increased biceps femoris activity in stance phase might be indicative of increased effort to control knee joint motion. Interestingly in this study, BF activity was greatest in the hypertrophic group after the second strength exercise, even though muscle soreness was already greatly reduced compared to bout 1.

In both groups rectus femoris EMG amplitude showed considerable amount of variation and high standard deviation. There were no consistent differences in RF muscle activation between bouts. So, in the scope of this study it seems that neither of the strength exercise caused significant changes in the rectus femoris activation in running economy test. For the neural group vastus lateralis activation showed very minor changes at all time points and it seems that neural strength exercise did not affect VL activity. For hypertrophic group in most of the time

points VL activity decreased to bout 1 then increased to bout 2 and decreased again to bout 3. It should be noted that only decrease at 9 min time point between control and bout 1 was statistically significant. Reductions of EMG amplitude at bout 1 is interesting, since it is clearly shown that running- and resistance training induced fatigue should increase rms EMG at the same submaximal intensity (Plattner et al. 2011). One explanation for this is that because of the EIMD and DOMS the activation of vastus lateralis was shifted to less fatigued muscles (Kellis & Lissou et al. 2009).

It should be noted that in this study we were not able to identify EMG values in different phases of the stride cycle (pre-activation, stance, swing). Therefore, conclusions from EMG results are very limited compared to many previous studies as it has been shown that usually there is changes is muscle activiation only some (and not all) phases of the stride cycle (Kyröläinen et al. 2005; Kellis & Lissou 2009; Darendeli et al. 2023).

Running performance improved significantly as time-to-exhaustion of running economy tests increased in each subsequent test, with statistical significance observed between the control and bout 3, as well as bouts 2 and 3 for the neural group, and between bouts 1 and 3 for the hypertrophic group. These findings contradict previous studies, as several investigations have shown that although there were no changes in running economy at submaximal intensities, maximal running performance was impaired (Chen et al. 2007; Doma & Deakin 2014). Reasoning behind the maximal endurance performance impairment might be that type II muscle fibers are more prone to EIMD and DOMS following strength exercises, and these fibers are used during endurance exercises only at high to maximal intensities (Doma et al. 2017). Interestingly, no decrease in maximal performance was observed in our study, even after the first strength exercise bout, despite significant muscle soreness in both groups, particularly in the hypertrophic group. There were quite clear (eventhough not statisctically significant) increase in time-to-exhaustion from control to bout 1. One test subject, in particular, demonstrated a remarkable increase in TTE, from 1200 seconds in the control test to 1394 seconds in bout 1, an increase of 194 seconds, or 3 minutes and 14 seconds. Notably, 1200 seconds equals to 20 minutes so in control test subject had to stop after the two 10-minute loads. One possible explanation for this increase is that the participant was unaccustomed to the RE test, and thus found it "easier" to perform after having completed the test once before, even though participant had already done one maximal test ($VO2_{max}$ test) before the first RE test.

The neural group exhibited significant increases in maximal oxygen uptake during each subsequent RE test, with significant changes from control to bouts 2 and 3. In contrast, the hypertrophic group demonstrated a decrease in $VO2_{max}$ from control to bout 2, followed by a slight increase to bout 3. Maximal lactate levels also showed an increasing trend, although the only statistically significant change was observed between control and bout 3 for the neural group. The increase in maximal lactate levels indicates that participants were able to reach their current subjective maximum performance. Additionally, the increase in lactate levels suggests that the use of anaerobic metabolism was increased. This would explain the significant increase in time-to-exhaustion for the hypertrophic group, despite a decrease in maximal oxygen uptake. In contrast, the neural group exhibited increases in both $VO2_{max}$ and maximal lactate, which explains the observed increase in time-to-exhaustion.

9.1 Strengths and limitations of the study

Limitations of the study include some modifications to the schedule of the measurements for a few test subjects. Few of the participants had longer than a week between training sessions due to illness or other unexpected reasons. In a few cases, the participant was able to complete the strength exercise but was not able to complete the endurance test within 48 ± 3 hours, so both the strength exercise and subsequent endurance exercise were repeated the following week. This change in timeline might potentially affect the responses to the strength exercises and therefore variables of the running economy test.

In this study nutrition, activity outside exercise and menstrual cycles were not followed. Seven of our test subjects (58%) were women and it has been shown that the phase of menstrual cycle might have significant effect on performance (Carmichael et al. 2021). EMG was not synced with stride cycle, so different phases couldn't be analyzed separately. There were also notably variation in joint angle data, that could have been reduced with better executed measurements. All these changes would have improved the quality of the data and the research as well as helpen in the analysis of the results.

One of the strengths of this study was the exploration of variables that previous research has overlooked, despite the extensive amount of research on the subject. Previous studies have focused only on the acute effects of either hypertrophic or eccentric strength exercise on running economy, while neglecting the common use of neural strength exercise among endurance

athletes. This study also investigated possible changes in muscle activation during running economy tests, which have not been studied in this kind of setting before.

Another strength of the study is its protocol, which was designed to be comparable to previous research on the same topic while adding a new perspective. The study's relatively homogenous group of participants, standardized conditions (exercises and measurements conducted in the same place with the same equipment), and progressive increase in strength training loads to match participant ability level are also noteworthy strengths of the study.

9.2 Conclusion

In this study, it appeared that hypertrophic strength exercise resulted in a significant amount of muscle damage, which led to minor changes in running economy, running kinematics, and muscle activation during running. Nonetheless, these changes decreased during subsequent strength exercise bouts, indicating the occurrence of the repeated bout effect. In contrast, neural strength exercises had a negligible impact on running economy. Therefore, it is possible that performing hypertrophic strength exercises could negatively effect on the quality of an endurance workout conducted 48 hours later, whereas neural strength exercises may not.

The changes caused by hypertrophic strength exercise are consistent with previous research, with the exception that the changes observed in this study appeared to be less pronounced than those reported in prior literature. It is possible that endurance athletes don’t need to be concerned about a certain amount of strength exercise-induced muscle damage, as it appears to have only a slight impact on endurance performance 48 hours later. Moreover, maximal endurance performance increased considerably in both groups during this study, suggesting that hypertrophic strength training can be performed concurrently with endurance training, at least in the short term. Additional research is needed to investigate chronic adaptations. However, neural strength exercise did not elicit any significant changes in running economy, running kinematics, or muscle activation. Therefore, incorporating neural strength training into an endurance training regimen is safe. Additionally, prior research recommends neural strength exercise, particularly for sports that require a low body weight, such as endurance running, as it does not substantially increase muscle mass.

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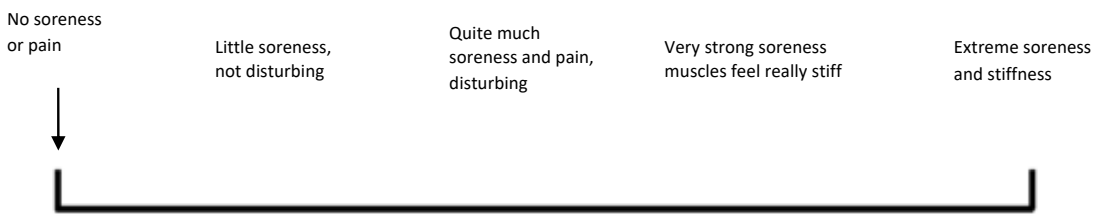
APPENDIX 1. Muscle soreness questionnaire.

MUSCLE SORENESS QUESTIONAIRE

Name: _____

DOMS means Delayed Onset of Muscle Soreness caused by strength exercise. Typically, it is strongest two days after exercise. This soreness can be felt in muscle as soreness when you touch the muscle as soreness and stiffness during any movements.



Mark vertical line ” I ”, to a point of line that describes the muscle soreness you experience at the moment

1. morning

2. morning

Before endurance test

APPENDIX 2. Health and physical activity questionnaire.

JYVÄSKYLÄN YLIOPISTO

On tärkeää, että tiedämme elintavoistasi ja aiemmista liikuntatottumuksista ennen kuin testaamme sinut. Vastaa seuraaviin kysymyksiin huolellisesti

Nimi:_____ Synt.aika:_____paino____pituus_____

Oireet viimeisen 6 kk aikana:	Kyllä	Ei	En osaa sanoa
1. Onko sinulla ollut rintakipuja? Levossa? Rasituksessa?			
2. Onko sinulla ollut rasitukseen liittyvää hengenahdistusta?			
3. Onko sinulla ollut huimausoireita?			
4. Onko sinulla ollut rytmihäiriötuntemuksia?			
5. Onko sinulla ollut harjoittelua estäviä kipuja liikuntaelimissä? Missä?			
6. Oletko tuntenut ylikuormitus- tai stressioireita?			

Todetut sairaudet: Onko sinulla tai onko sinulla ollut jokin/joitakin seuraavista? (ympyröi)

01 sepelvaltimotauti	02 sydäninfarkti	03 kohonnut verenpaine	04 sydänläppävika
05 aivohalvaus	06 aivoverenkierron häiriö	07 sydämen rytmihäiriö	08 sydämentahdistin
09 sydänlihassairaus	10 syvä laskimotukos	11 muu verisuonisairaus	12 krooninen bronkiitti
13 keuhkolaajentuma	14 astma	15 muu keuhkosairaus	16 allergia
17 kilpirauhasen toimintahäiriö	18 diabetes	19 anemia	20 korkea veren koleste
21 nivelreuma	22 nivelrikko, -kuluma	23 krooninen selkäsairaus	24 mahahaava
25 pallea-, nivus- tai napatyrä	26 ruokatorven tulehdus	27 kasvain tai syöpä	28 leikkaus äskettäin
29 mielenterveyden ongelma	30 tapaturma äskettäin	31 matala veren K tai Mg	32 kohonnut silmänpair
33 näön tai kuulon heikkous	34 urheiluvamma äskettäin		

muita sairauksia tai oireita, mitä:_____

Lääkitys: Käytätkö jotain lääkitystä tai lääkeainetta säännöllisesti tai usein? 1 En 2 Kyllä, mitä:_____

Tupakoitko? 1 En 2 Kyllä 3 Olen lopettanut

Koska olet viimeksi nauttinut alkoholia?_____ Kuinka paljon?_____

Raskaus/synnytykset: 1 Olen raskaana, raskausviikko_____ 2 Olen synnyttänyt _____kk / v sitten

Kuumetta, flunssaista oloa tai muuten poikkeavaa väsymystä viimeisen kahden viikon aikana: 1 Ei 2 Kyllä

Onko lähisuvussasi ennenaikaiseen kuolemaan johtaneita sydänsairauksia? 1 Ei 2 Kyllä
Lähisukulainen? _____Minkä ikäisenä?_____
Onko todettu synnynnäinen sydänvika? _____