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Laura Karavirta

Cardiorespiratory, Neuromuscular and Cardiac Autonomic Adaptations to Combined Endurance and Strength Training in Ageing Men and Women

JYVÄSKYLÄN YLIOPISTO

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ABSTRACT

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Individual differences in the time-course of ageing are largely due to factors related to lifestyle, including regular physical activity. As a result of different adaptations of endurance and strength training, neither training mode alone can substantially improve all the essential aspects of physical performance and health. The competitive nature of endurance and strength training may, however, lead to interference in cardiorespiratory and neuromuscular adaptations when endurance and strength training are performed simultaneously. Therefore, this study examined cardiorespiratory, neuromuscular and cardiac autonomic adaptations to combined endurance and strength training in 40 to 67 year old men and women. 196 healthy subjects completed a 21 week progressive training period consisting one of the following: 1) two endurance, 2) two strength, 3) combination of two endurance and two strength training sessions per week, or 4) no systematic training. The present results indicated that the training mode-specific adaptations in cardiorespiratory endurance and muscular strength were similar after combined endurance and strength training compared with endurance or strength training alone, respectively. However, individual training responses revealed large individual variation in the magnitude of training adaptations in all training groups. Furthermore, the evident goal of combined endurance and strength training, i.e. simultaneous increase in both cardiorespiratory endurance and maximal strength, was only achieved by approximately half of the ageing men and women, while others improved either endurance or strength. Significant muscle fibre hypertrophy was observed in the strength training group, but not in the combined endurance and strength training group. In terms of cardiac autonomic function, adaptations measured by heart rate dynamics seemed to be more notable after endurance training compared to combined training. Strength training alone did not lead to significant improvements in cardiac autonomic function. To summarise, the training mode-specific outcomes of combined endurance and strength training may be individually determined. Furthermore, muscle hypertrophy may be impaired when strength and endurance training are combined over a prolonged period. New means are, therefore, needed to individualise combined endurance and strength training programs for optimal adaptations.

Keywords: endurance training, strength training, training response, heart rate variability, cardiac autonomic function

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"*The whole is different*
from the sum of its parts"

Aristotle

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Jyväskylä, December 2010

Laura Karavirta

ABBREVIATIONS

1RM	one repetition maximum
ATP	adenosine triphosphate
a-v O_2 diff	arterio-venous oxygen difference
BMI	body mass index
C	control (group)
CI	complexity index
CSA	cross-sectional area
DXA	dual-energy x-ray absorptiometry
E	endurance training (group)
ECG	electrocardiography
EMG	electromyography
HFP	high frequency power
HR	heart rate
HR_{max}	maximal heart rate
HRR	heart rate reserve
HRV	heart rate variability
LFP	low frequency power
MVC	maximal voluntary contraction
NNI	normal sinus to normal sinus interval
P_{max}, W_{max}	maximal aerobic power ouput
RRI	R-to-R peak interval
S	strength training (group)
SampEn	sample entropy
SDNN	standard deviation of all NN intervals
SE	combined endurance and strength training (group)
SV	stroke volume
TB	triceps brachii
VCO_2	carbon dioxide output
VI	vastus intermedius
VL	vastus lateralis
VM	vastus medialis
VO_2	oxygen uptake
VO_{2max}	maximal oxygen uptake
VO_{2peak}	peak oxygen uptake

LIST OF ORIGINAL PUBLICATIONS

This thesis is based on the following original articles, which are referred to in the text by their roman numerals.

I Karavirta L, Häkkinen A, Sillanpää E, García-López D, Kauhanen A, Haapasaari A, Alen M, Pakarinen A, Kraemer WJ, Izquierdo M, Gorostiaga E, and Häkkinen K (2009). Effects of combined endurance and strength training on muscle strength, power and hypertrophy in 40–67-year-old men. Scand J Med Sci Sports (Dec 18, 2009, Epub ahead of print).

II Sillanpää E, Häkkinen A, Nyman K, Mattila M, Cheng S, Karavirta L, Laaksonen DE, Huuhka N, Kraemer WJ, Häkkinen K (2008). Body composition and fitness during strength and/or endurance training in older men. Med Sci Sports Exerc 40, 950 – 958.

III Karavirta L, Häkkinen K, Kauhanen A, Arija-Blázquez A, Sillanpää E, Rinkinen N, and Häkkinen A (2010). Individual responses to combined endurance and strength training in older adults. Med Sci Sports Exerc (Aug 2, 2010, Epub ahead of print)

IV Karavirta L, Tulppo MP, Laaksonen DE, Nyman K, Laukkanen RT, Kinnunen H, Häkkinen A, and Häkkinen K (2008). Heart rate dynamics after combined endurance and strength training in older men. Med Sci Sports Exerc 41, 1436–1443.

V Karavirta L, Costa MD, Goldberger AL, Mietus JE, Tulppo MP, Laaksonen DE, Nyman K, Keskitalo M, Häkkinen A and Häkkinen K (2010). Heart rate complexity and variability after endurance, strength and combined endurance and strength training in aging women. (Submitted)

CONTENTS

1 INTRODUCTION

There has been ever growing evidence about the beneficial effects of physical exercise since 1953, when the positive outcomes of occupational physical activity on overall health were discovered (Morris et al. 1953). Since then, numerous studies have confirmed the benefits of regular physical activity on different aspects of physical performance and health. Based on recent findings, however, being physically active may not be enough. One of the main outcomes of regular endurance exercise training, i.e. improved cardiorespiratory endurance, has been shown to be inversely and more strongly associated with cardiovascular and all-cause mortality than physical activity (Lee et al. 2010). Furthermore, endurance training has been shown to enhance cardiac autonomic function, which is an independent determinant of cardiovascular health. Therefore, the focus has been aimed on achieving optimal training outcomes, and guidelines for training prescription have been frequently updated.

Apart from low cardiorespiratory fitness and impaired cardiac autonomic function, low muscular strength has also been recently shown to be an independent predictor of mortality, even after adjusting for cardiorespiratory endurance (Katzmarzyk & Craig 2002; Ruiz et al. 2008). Furthermore, muscular strength contributes to functional capacity (Hunter et al. 2004). These findings emphasise the importance of strength training aside from endurance training as a method to improve functional capacity and overall health. The beneficial effects of both endurance and strength training are, thus, evident. Given the training mode-specific nature of adaptations and the rapid effects of detraining, the aim should be to simultaneously enhance both these aspects of physical performance.

Concurrent endurance and strength training are generally used to seek health benefits beyond endurance or strength training alone. Hickson (1980), however, reported in his renowned study an interference in the development of maximal strength after eight weeks of training when endurance and strength were trained simultaneously several times per week. Since then, in several studies the combination of endurance and strength training regimes has been investigated by modifying both the training protocols and the subject groups.

The complex interplay of several control mechanisms at the muscular and cellular levels that enable the human body to adapt to different external stimuli seem to be on a collision course in combined endurance and strength training, producing smaller training adaptations compared to the adaptations to endurance or strength training alone.

In addition to the health risk and impaired physical performance induced by physical inactivity, ageing of the human body itself leads to inevitable progressive impairment in physiological functions, resulting in a loss of muscular strength, cardiorespiratory fitness and cardiac autonomic function. Furthermore, functional capacity relates to physical performance. The most profound effects of ageing compromise our health and the ability to lead an independent life. The good news is that even if physical training is initiated after the age-related loss of physical performance has already began, considerable benefits in terms of physical performance and health can be achieved. The recent findings on individual training adaptation show, however, that the range in the magnitude of training adaptation is vast and not everyone improves their physical performance even with supervised training. The question remains, what kind of training program is optimal for physical performance and health outcomes. Therefore, this thesis aimed to clarify cardiorespiratory, neuromuscular and cardiac autonomic adaptations to combined endurance and strength training compared to those of endurance or strength training alone in ageing men and women.

2 REVIEW OF THE LITERATURE

2.1 Physiological challenges related to ageing

As a human gets older, physiological changes lead to a gradual decrease in physical performance. Decreases in physical performance can be observed well before any decrements in functional capacity are evident, but the reductions in physical performance imply that a higher percentage of the maximal capacity must be exerted to perform any submaximal exercise task (Fleg et al. 2005; Chodzko-Zajko et al. 2009). Physical performance can be divided into two main components, cardiorespiratory endurance and muscular strength. Cardiorespiratory endurance is characterised by the ability of the body to sustain prolonged exercise including repetitive muscular contractions with low intensity whereas muscular strength refers to the ability of the neuromuscular system to produce high force in few repetitions using near maximal loads.

Functional capacity is not, however, confined to strength and endurance performance but also includes several regulatory mechanisms. The heart is a key organ maintaining all bodily functions, and the cardiovascular system, including the heart, blood vessels and blood, is mainly regulated by the function of the autonomic nervous system. Cardiac autonomic function, determined as heart rate variability (HRV) is also impaired in ageing (Jensen-Urstad et al. 1997). However, the association between physical performance and HRV is not as clear as could be concluded when comparing athletes and sedentary adults (Dixon et al. 1992; Verlinde et al. 2001; Aubert et al. 2003). In an average population, physical performance seems not to explain the interindividual variation in cardiac autonomic function (Melanson & Freedson 2001; Uusitalo et al. 2002; Bosquet et al. 2007). Thus, HRV is an important independent predictor of cardiovascular and all-cause mortality that provides additional information about cardiac health (Tsuji et al. 1994; Tsuji et al. 1996).

Low baseline values of muscular strength and cardiorespiratory endurance in middle-aged women and men forecast future risks of disability (Huang et al. 1998; Rantanen et al. 1999; Blair & Wei 2000). Tasks that require consider-

able effort in unfit individuals are likely to be avoided, leading to inactivity and further reductions in aerobic capacity and muscular strength (Fleg et al. 2005). Therefore, it is crucial to acknowledge the effects of ageing in early life to maintain a good functional capacity in the working population. Age-related reductions in physical performance start already in middle-aged adults, and the rate of reduction accelerates during the sixth decade of life. Even though ageing is inevitable, individuals differ broadly in how they age, which is likely due to both lifestyle and genetic factors (Chodzko-Zajko et al. 2009).

2.1.1 Age-related decrease in cardiorespiratory endurance

The concept of cardiorespiratory endurance

Cardiorespiratory endurance can be defined as the capacity to sustain a given exercise intensity for the longest possible time (Jones & Carter 2000). The single most often used variable for quantifying cardiorespiratory endurance is maximal oxygen uptake (VO_{2max}), i.e. aerobic capacity, first presented by A.V. Hill in the 1920's (Hill & Lupton 1923). VO_{2max} can be determined, based on the Fick equation, as a product of maximal cardiac output and arteriovenous oxygen difference (a-v O_2 difference), where maximal cardiac output is a product of stroke volume (SV) and maximal heart rate (HR_{max}) (Fick 1870). Peak oxygen uptake (VO_{2peak}) is also a commonly used term, which refers to the highest VO_2 attained when VO_{2max}, i.e. a plateau in VO_2, is not reached and the performance is limited by local rather than by central circulatory factors (Rowell 1974). The reason for the popularity of these variables is that they are strongly associated with endurance performance (di Prampero 2003) and inversely related to cardiovascular and all-cause mortality (Sandvik et al. 1993; Blair et al. 1995; Blair et al. 1996).

Due to the prolonged duration of endurance exercise, performance is highly dependent on energy production via the aerobic resynthesis of adenosine triphosphate (ATP), a process that requires oxygen (Jones & Carter 2000). The use of oxygen in the energy production for physical work is a complex process including transferring O_2 from the air to the blood, pumping it to the periphery, and distributing it to the muscle cells for the oxidative production of energy, bound in ATP, to generate muscle contractions (Levine 2008) (Fig. 1). Therefore, VO_{2max} reflects the maximal rate of aerobic energy expenditure. VO_{2max} is limited under most circumstances by the ability of the heart to deliver oxygen to the working muscles (Bassett & Howley 1997; di Prampero 2003). However, VO_{2max} does not equal endurance performance, since there are other factors that contribute to the performance outcome, including exercise-specific economy, anaerobic capacity and neuromuscular factors (Levine 2008). Time to exhaustion in a treadmill test or maximal power output (P_{max}, W_{max}) in a bicycle ergometer test are, therefore, also generally used as indicators of endurance performance in sedentary or physically active, non-competitive individuals (Kuipers et al. 1985; Izquierdo et al. 2005).

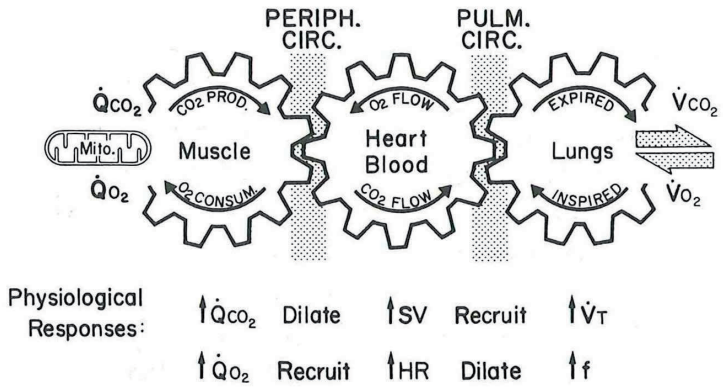

FIGURE 1 Gas transport mechanisms and physiological responses to exercise from the pulmonary to the cellular level. Q_{CO2}, carbon dioxide production; Q_{O2}, oxygen consumption; V_{O2}, oxygen uptake; Vc_{O2}, carbon dioxide output; SV, stroke volume; HR, heart rate; V_T, tidal volume; f, breathing frequency. Adapted from Wasserman et al. (2005).

Effects of ageing

In sedentary adults VO_{2max} is highest at the age of approximately 25 to 30 years (Fitzgerald et al. 1997; Fleg et al. 2005). Both cross-sectional and longitudinal studies have confirmed a gradual decline in VO_{2max} due to ageing (Fleg & Lakatta 1988; Fitzgerald et al. 1997; Proctor & Joyner 1997; Åstrand et al. 1997; Ades & Toth 2005; Fleg et al. 2005; Hakola et al. 2010). The decrease in VO_{2max} has been suggested to start after the age of 45 years (Jackson et al. 2009). A meta-analysis has suggested an average rate of decrease of 10 % per decade (Fitzgerald et al. 1997). Based on longitudinal data it has been observed that the decrease in aerobic capacity is nonlinear as the rate of decline seems to increase with age (Fleg et al. 2005). The average rate of decline in VO_{2peak} relative to body mass was 11 and 11 % during the fifth, 16 and 14 % during the sixth, and 20 and 17 % during the seventh age decade in men and women, respectively (Fig. 2) (Fleg et al. 2005). The increasing rate of the decline in VO_{2peak} was also evident when determined relative to fat free mass.

Longitudinal studies may provide more valid information about the true rate of decline in aerobic capacity, since a cross-sectional study design may produce a selection bias due to the selected survival of the older subjects producing a healthier group of older subjects than younger subjects (Ades & Toth 2005). Furthermore, the predicted decrease per decade may vary depending on the characteristics of the subject group in question, such as lifestyle factors. For example, the level of physical activity can significantly affect the time course of the age-related decrement in cardiorespiratory endurance (Fitzgerald et al. 1997).

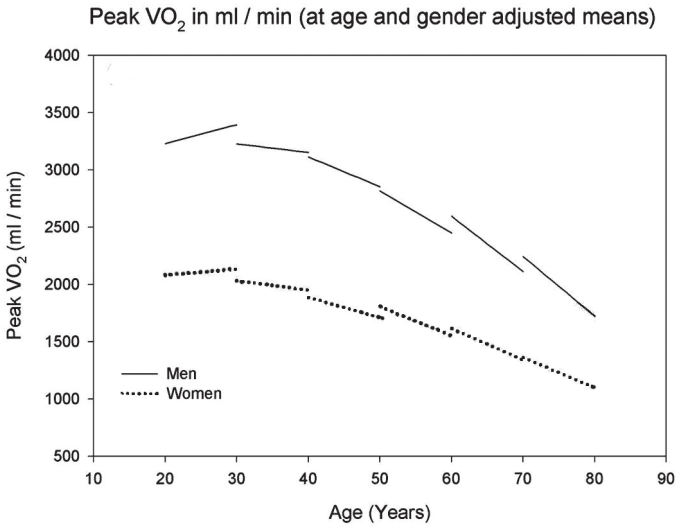

FIGURE 2 Longitudinal changes in VO_{2peak} per decade from 20 to 70 years of age predicted from a mixed-effects linear regression model in men and women (Fleg et al. 2005). Reproduced with permission of Wolters Kluwer Health.

Thus, the decrease in cardiorespiratory endurance in ageing may not be entirely due to ageing itself. Physical activity, which is a major determinant of cardiorespiratory endurance, also decreases in ageing (Bijnen et al. 1998; Westerterp 2000). It is therefore difficult to dissociate the effects of inactivity from the effects of ageing. In fact, it has been shown that three weeks of bed rest may cause a greater decrease in aerobic capacity than 30 years of ageing from the age of 20 to 50 years (McGuire et al. 2001b). Furthermore, the rate of decline in cardiorespiratory endurance in older endurance athletes seems to be highly dependent on the maintenance of training stimulus in ageing (Katzel et al. 2001).

Regular physical activity cannot, however, reverse the effects of ageing. This was demonstrated in a group of untrained subjects who underwent a similar endurance training load as 20 year old adults and 30 years later as middle-aged adults. None of the subjects taking part in the training 30 years later were able to reach as high a level of VO_{2max} as attained after training 30 years before (McGuire et al. 2001a). A meta-analysis has in fact revealed a faster absolute decrease of VO_{2max} in endurance trained versus sedentary women, which possibly is a result of more striking changes in training volume and intensity in trained compared to sedentary subjects (Fitzgerald et al. 1997). Despite the faster rate of decrease, older endurance trained adults still have considerably higher absolute VO_{2max} values compared to their sedentary peers (Fitzgerald et al. 1997).

A major contributor to the lower VO_{2max} observed in older adults seems to be a substantially smaller maximal cardiac output compared to younger counterparts (Stratton et al. 1994). Both SV and HR_{max} may in turn contribute to the age-related decline in cardiac output. A smaller stroke volume may account for

most of the difference in VO_{2max} between young (18 to 31 years old) and older (51 to 72 years old) adults (Ogawa et al. 1992). HR_{max} decreases by approximately 0.7 beats per minute per year due to ageing (Inbar et al. 1994; Tanaka et al. 2001; Balmer et al. 2005), and the decline has been reported to somewhat accelerate with age (Londeree & Moeschberger 1982). However, the decrease in HR_{max} is a relatively small contributor to the age-related decrease in VO_{2max} since it accounts for just 26-30 % of the age-related differences in maximal cardiac output while the remaining proportion is due to the smaller SV (Ogawa et al. 1992).

VO_{2max} is often expressed per kilogram of body mass, especially when cardiorespiratory fitness is evaluated against average values of a population. In fact, VO_{2max} is strongly associated to body mass and especially to fat free mass that may be mainly due to the ability of skeletal muscle to consume oxygen (Fleg & Lakatta 1988; Toth et al. 1994). However, statistically controlling for blood volume and SV seems to eliminate the close association between fat free mass and maximal oxygen consumption ($r = 0.12$, ns.). Thus, the loss of skeletal muscle mass with ageing, i.e. sarcopenia, may contribute to the age-related decline in VO_{2max} mainly due to reductions in the central circulatory factors in healthy sedentary 18 to 75 year old adults (Hunt et al. 1998).

2.1.2 Age-related decrease in neuromuscular performance

Muscular strength

The concept of muscular strength refers to the maximum capacity of skeletal muscle to generate force (Knuttgen & Komi 2003), which is determined by the structure and function of the neuromuscular system, including muscle cross-sectional area (CSA), neural activation, and muscle architecture, as well as by the features of the muscle contraction such as contraction velocity and contraction type. Muscle contraction is initiated by an α-motor neuron, which together with all the muscle fibres it innervates constitutes a component termed a motor unit. Characteristics of motor neurons largely also determine the qualities of individual muscle fibres (Wilmore & Costill 2004).

Muscle fibres are generally grouped into three types according to the myosin heavy chain profile (Monti et al. 2001). Different types of muscle fibres vary in the contraction velocity and fatigue resistance. Type I fibres are most fatigue resistant slow twitch fibres, type IIA fibres are fast twitch fibres with a moderate fatigue resistance, and type IIX fibres, formerly also called IIB, are fast twitch fibres that are sensitive to fatigue. Human muscle also contains so called hybrid types of muscle fibres, e.g. IIAX, that are transforming from one type to another (Klitgaard et al. 1990; Harridge 2007).

Muscle possesses a considerable potential for growth through an increased ratio between protein synthesis and protein degradation (Harridge 2007). Muscular strength, however, is not solely dependent on the muscle CSA. The highest possible force produced, i.e. maximal voluntary contraction (MVC), is also highly dependent on the activation of the central nervous system. Factors

(such as motor unit recruitment, activation of antagonistic muscles, muscle architecture, and tendon stiffness) that contribute to the muscular performance per unit of muscle volume or mass are often referred to as muscle quality (Dutta et al. 1997).

Effects of ageing

In sedentary adults, muscular strength reaches its peak during the third decade of life (Larsson et al. 1979; Häkkinen & Häkkinen 1991; Lindle et al. 1997). The reduction in muscular strength starts in middle-aged adults and accelerates after the onset of the sixth decade (Larsson et al. 1979; Frontera et al. 1991; Häkkinen & Häkkinen 1991; Lindle et al. 1997; Deschenes 2004). The rate of gradual decline in maximal strength after the age of approximately 30 is between eight and ten percent per decade (Lindle et al. 1997), after which the rate of decline is increased to about 12 to 15 % per decade (Fig. 3) (Larsson et al. 1979; Frontera et al. 1991). In addition to maximal strength, the loss of explosive strength or power of the neuromuscular system may occur at an even faster rate than the loss of maximal strength from middle to older age (Häkkinen et al. 1996; Izquierdo et al. 1999). Furthermore, the decrease seems to be more profound in concentric muscle contractions, compared to isometric contractions, whereas actions involving eccentric muscle contractions seem to be impaired at the slowest rate (Vandervoort 2002).

Increased inactivity is a major contributor to the attenuated muscular strength in ageing (Bijnen et al. 1998; Westerterp 2000). For example, due to life-long strength training, master athletes demonstrate a higher muscular strength at the age of 75 (range 69-85) years than sedentary men at the age of 40 (35-45) years (Ojanen et al. 2007). However, regardless of a life-long background of strength training, the age-related decrease is inevitable even in strength athletes.

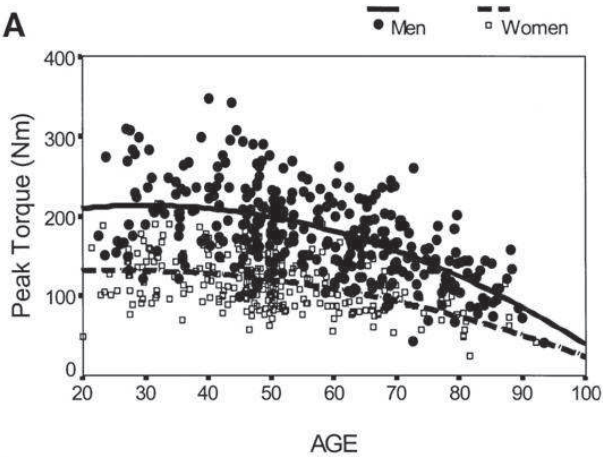

FIGURE 3 Age-related decrease in concentric peak torque of knee extensors in men and women (Lindle et al. 1997). Copyright 1997 by American Physiological Society. Reproduced with permission.

The decrease in maximal strength is mainly due to the age-related decrease in skeletal muscle mass, i.e. sarcopenia (Frontera et al. 1991; Deschenes 2004). The progressive loss of muscle fibres and fibre size has been suggested to begin between the third (Lexell et al. 1988) and the fifth decade (Hepple 2003) and accelerate thereafter. The decrease in muscle mass has been suggested to be fibre type-dependent, since a smaller proportion of type II muscle fibres have been observed in older than young men (Scelsi et al. 1980; Klitgaard et al. 1990). However, similar proportions of fibre types between young and older adults have also been reported (Lexell et al. 1988; Häkkinen et al. 2001). The most prominent difference in the proportion of muscle fibres seems to lay in the higher proportion of hybrid fibres in older compared to younger subjects (Klitgaard et al. 1990). A major contributor to the loss of muscle fibres is motor neuron death coupled with an incomplete reinnervation of abandoned muscle fibres (Enoka 1997). It seems, however, that the rate of decline in muscular strength is faster than that of muscle size suggesting that a decrease also occurs in the muscular strength per unit of muscle mass, i.e. muscle quality (Goodpaster et al. 2006).

Cross-sectional studies have shown a significant difference between young and older subjects in muscle quality (Welle et al. 1996), although the magnitude of the effect may be dependent on the muscle group and type of muscle action (Lynch et al. 1999). Several factors may explain the age-related decrement in muscle quality. Age-related changes in the duration and amplitude of motor unit action potentials have been demonstrated using electromyography (EMG) techniques (Vandervoort 2002). Furthermore, axonal conduction velocity seems to slow down with ageing (Vandervoort 2002). Older adults may not be able to fully activate the muscles voluntarily, even after strength training (Harridge et al. 1999), which could result from a decrease in the motor unit recruitment or decrease in the average motor unit firing rates (Howard et al. 1988; Kamen & Knight 2004). All studies have not, however, confirmed a lower muscle activation or lower motor unit firing rates in older compared to young adults (Roos et al. 1999). Ageing seems to also induce changes in muscle architecture by, for example, decreasing the fascicle length and pennation angle and thus probably leading to impaired muscle function (Narici et al. 2003).

2.1.3 Effects of ageing on cardiac autonomic function

Heart rate variability as a measure of cardiac autonomic function

The function of the cardiovascular system is mainly regulated by the autonomic nervous system. The function of the autonomic nervous system, on the other hand, is modulated by changes in blood pressure, rate of metabolism and the state of skeletal muscle sensed by baro-, chemo-, or mechanoreceptors, respectively. The autonomic nervous system is divided into the parasympathetic (vagal) and sympathetic branches, which together mediate changes in peripheral vascular tone, blood pressure, and heart rate (HR). Parasympathetic control is mediated through the vagus nerve i.e. the tenth cranial nerve, which enter the

sinoatrial node, atrioventricular conducting pathways, atrial muscle (Loewy 1990) and ventricular muscle (Johnson et al. 2004). Sympathetic neurons, on the other hand, originate from the spinal cord and innervate the entire heart (Loewy 1990). Sympathetic and vagal divisions produce opposite effects on the function of the heart. The activation of the sympathetic division increases heart rate and the force of contraction whereas the activation of the vagal division decreases heart rate and the force of contraction. HR is, therefore, regulated by the interaction of vagal and sympathetic nerves, and the effects can vary independently, coactively or reciprocally (Berntson et al. 1993). Since an increase in HR may arise from vagal withdrawal, sympathetic activation or both, HR alone does not provide information about the autonomic control of the heart.

Heart rate variability (HRV) refers to variation in the time intervals between consecutive heart beats. R peaks of a standard electrocardiographic recording are generally used to detect the interbeat intervals, correspondingly named RR intervals (RRI). HRV at high frequencies is of vagal origin (Akselrod et al. 1981; Akselrod et al. 1985; Martinmäki et al. 2006) and mainly derived from respiration, also named respiratory sinus arrhythmia, which is induced by tachycardia during inspiration and bradycardia during expiration (Yasuma & Hayano 2004). At lower frequencies of around 0.1 Hz HRV is probably of both vagal and sympathetic origin (Akselrod et al. 1981; Martinmäki et al. 2006), although it has also been suggested to be only of sympathetic origin (Malliani et al. 1991). Changes in HR during incremental exercise are mainly modulated by both the parasympathetic and sympathetic nervous systems. During exercise of light intensity, an increase in HR is almost entirely due to decrease in vagal tone. As the exercise intensity increases, further increases in HR are mediated by both increased sympathetic activation and continued withdrawal of vagal tone. During heavy exercise, sympathetic activity is further increased whereas no vagal tone remains.

The function of the parasympathetic nervous system cannot be measured directly in conscious humans and, therefore, the measurement of HRV has been introduced as a valid indirect method for evaluation of vagal tone. Furthermore, a direct assessment of the function of the sympathetic nervous system is not suitable for all situations using, for example, the measurement of muscle sympathetic nerve activity with microneurography (Grassi & Esler 1999). The vast research interest in HRV is due to the observation that abnormalities in HRV are associated with increased mortality after myocardial infarction (Kleiger et al. 1987; Bigger et al. 1992; La Rovere et al. 1998). Increased risk of cardiac events has also been observed in community-based elderly populations with reduced HRV (Tsuji et al. 1996). Therefore, HRV provides additional prognostic information compared with traditional cardiovascular disease risk factors, such as overweight and obesity, high blood pressure, cholesterol, smoking and physical inactivity (Tsuji et al. 1994; Tsuji et al. 1996).

HRV can be quantified using a variety of different methods including time domain, frequency domain (spectral) and nonlinear methods (Malik et al. 1996). Time domain measures are obtained from the RRIs directly or from the differ-

ences between RRIs and are thus relatively simple to calculate. Frequency domain (spectral) analysis provides information about the distribution of power, i.e. variance, as a function of frequency. With short term spectral analysis, three main components can be differentiated in the spectrum: high frequency (HF), low frequency (LF) and very low frequency (VLF) components (Malik et al. 1996). RRI time series also includes nonlinear oscillations that linear time domain and spectral measures alone cannot capture. Nonlinear methods may, therefore, provide additional information concerning the fluctuations of HR (Kuusela et al. 2002).

Several different nonlinear measures have been developed to detect the nonlinear features of HR behaviour. Detrended fluctuation analysis (DFA) quantifies fractal correlation properties of RRI time series (Peng et al. 1995a). Physiologically, the breakdown of the fractal organisation of short-term HR dynamics has been associated with the coactivation of vagal and sympathetic outflow (Tulppo et al. 2005), vagal blockade (Penttilä et al. 2003), vagal tone inhibition due to light-intensity exercise (Tulppo et al. 2001) and heart failure (Peng et al. 1995b; Goldberger et al. 2002). In addition, several studies have concluded that HR complexity, quantified using measures such as approximate entropy (Pincus 1995) and sample entropy derived from a single scale (Richman & Moorman 2000) or multiple scales (Costa et al. 2002) is under control of the autonomic nervous system (Hagerman et al. 1996; Penttilä et al. 2003; Porta et al. 2007). It has been suggested that the loss of complexity in physiological systems contributes to ageing and disease (Lipsitz & Goldberger 1992). Nonlinear measures of HR fluctuations in humans seem to be only moderately influenced by the sympathetic nervous system and mainly by vagal modulation (Beckers et al. 2006).

Effects of ageing

Ageing alters cardiac autonomic function, which may contribute to the higher risk for cardiac events observed in older adults (Lipsitz 2004). Parasympathetic activity is decreased (Schwartz et al. 1991; Ryan et al. 1994; Jensen-Urstad et al. 1997) leading to a domination of sympathetic activity and reduced autonomic responsiveness (Lipsitz et al. 1990). Changes in cardiac autonomic function can be observed, for example, as a decrease in total variation of RRIs or as a decrease in the spectral power of all frequencies of HRV (Gregoire et al. 1996; Umetani et al. 1998; Carter et al. 2003; Vuksanović & Gal 2005).

The age-related decrease pattern of HRV seems to be somewhat dependent on the HRV measure used. A slight gradual decrease reaching approximately 70% of the baseline value by the sixth decade has been observed in the standard deviation of RR intervals (SDNN) which is an estimate of overall HRV. In terms of spectral measures of HRV age-related decreases have been reported to be 30 % in total power between the third and seventh decade (Jensen-Urstad et al. 1997). pNN50 (the percentage of differences between consecutive RR intervals that are > 50 ms), which is mainly a measure of vagal modulation, seems

to decrease more rapidly, reaching 24 % of baseline by the sixth decade (Umetani et al. 1998). It is not clear, however, whether age-related decreases are more prominent for HF power (HFP) (Lipsitz et al. 1990; Vuksanović & Gal 2005) or LF power (LFP) (Bigger et al. 1995; Tasaki et al. 2000). It is possible that changes in spectral measures of HRV occur relatively abruptly, rather than progressively, after 60 years of age (Lipsitz et al. 1990).

Apart from the linear measures of HRV, progressive loss of HR complexity (Pikkujämsä et al. 1999; Vuksanović & Gal 2005) and changes in long-term fractal-like behaviour of HR (Pikkujämsä et al. 1999) have been observed to start at middle age. Negative correlations have been found between age and several measures of HR complexity, including approximate entropy ($r = –0.27$), although correlations tend to be weaker than those between age and linear measures of HRV, for example HFP ($r = –0.62$) (Beckers et al. 2006). Furthermore, Schmitt and Ivanov (Schmitt & Ivanov 2007) did not find any differences in fractal characteristics between healthy young and elderly subjects. Thus, ageing may have more profound effects on linear than nonlinear measures of HRV (Vuksanović & Gal 2005).

Associations between age, cardiorespiratory endurance and HRV

Even though the association between cardiorespiratory endurance and HRV seems evident when comparing athletes and sedentary adults (Dixon et al. 1992; Verlinde et al. 2001; Aubert et al. 2003), ageing rather than low cardiorespiratory endurance seems to have a more evident effect on cardiac autonomic function at supine rest. In fact, an age-related reduction in HRV measured at rest occurs independently from impairments in cardiorespiratory endurance (Byrne et al. 1996; Tulppo et al. 1998). This finding gives further support to the importance of investigating cardiac autonomic function as an independent aspect of physical function in ageing adults. Age-related structural and functional changes reduce the complexity of HR control, impairing the ability to adapt to physiologic stresses such as physical exercise (Lipsitz & Goldberger 1992; Lipsitz 2004). Thus, an age-related decrease in HRV can also be observed during submaximal exercise (Tulppo et al. 1998).

2.2 Endurance and strength training in ageing

Since the first observations about the positive effects of occupational physical activity among bus drivers and conductors (Morris et al. 1953), the effects of regular physical activity have been extensively studied during the past decades. In consequence, regular physical activity has been compellingly shown to be beneficial for physical performance and overall health. However, regular physical activity seems to be most beneficial when it leads to significant adaptations in cardiorespiratory endurance (Lee et al. 2010) and muscular strength (Hunter et al. 2004). These adaptations can be enhanced by a carefully planned exercise

prescription with adjustment of the training design variables. The training design is targeted in order to meet the two main training principles: progressive overload and specificity. Progressive overload is attained with the manipulation of training intensity, frequency and duration. The principle of specificity states, on the other hand, that the adaptations are specific to the mode and intensity of exercise performed. Repeated bouts of exercise inducing a variety of acute physiological responses lead to the chronic adaptations of physical training, e.g. improved physical performance.

The most recent guidelines for exercise prescription by the American College of Sports Medicine suggest three or more aerobic training sessions per week with intensities equal to or above 60 % of HR_{max} for older adults (Chodzko-Zajko et al. 2009) and two to three resistance training sessions per week at fatiguing or near-fatiguing intensity (Ratamess et al. 2009). Long-term participation in aerobic exercise training is recommended, employing large muscle groups in rhythmic and dynamic activities. The recommended volume of endurance exercise depends on the intensity but generally, when exercise intensity is only moderate, two and a half hours of endurance-type activity per week is recommended for adults of all ages (American College of Sports Medicine 2006). The recommended intensity for endurance exercise, in turn, depends on the target population and desired goals. For example, health benefits may be achieved by lower intensity whereas improvements in cardiorespiratory endurance may require higher exercise intensities (American College of Sports Medicine 2006). For strength training a minimum of eight to ten separate exercises are recommended to activate all the major muscle groups. For individuals with no previous strength training experience, loads corresponding to 8–12 repetition maximum are recommended, but progressive increases in the training load are needed for further improvements. Each set should be performed to volitional fatigue (Ratamess et al. 2009).

2.2.1 Cardiorespiratory and cardiac autonomic adaptations to endurance training

Cardiorespiratory adaptations to endurance training

At the onset of aerobic exercise, rapid adjustments in the cardiovascular system allow the human body to meet the requirements of increased energy expenditure, rate of metabolic processes, heat and waste production and acidity. Thus, increases in HR, SV, cardiac output, blood flow, and blood pressure can be observed. However, regular bouts of sustained aerobic exercise are required to produce chronic training adaptations in cardiovascular and respiratory systems and skeletal muscles. A number of training design variables, such as the training intensity, duration and frequency of a single training session, as well as duration of the training program may play a role in the magnitude of the adaptation (Jones & Carter 2000). The adaptive increase in VO_{2max} is fairly rapid, the first adaptations occurring after only 10 days of training in young adults (Mier et al. 1997). In controlled studies with a training program between 16 and 20

weeks of duration, the mean increase in VO_{2max} was reported to be 16 % in older adults (Chodzko-Zajko et al. 2009).

Some studies have suggested that training intensity is the key factor in determining the magnitude of training adaptation (Hickson et al. 1985; Cornelissen et al. 2009). In another study, on the other hand, a comparison of three endurance training programs differing in volume and intensity, a higher volume produced additional benefits in middle-aged (40 to 65 year old) adults compared to the low volume and moderate intensity training with caloric equivalent of walking 19 kilometres per week at 40–55 % of VO_{2peak} (Duscha et al. 2005). Increasing the intensity from 40–55% to 65–80 % of VO_{2peak} did not lead to further improvements in VO_{2peak}, whereas, increasing the volume from 19 to 32 km per week at the intensity of 65–80% of VO_{2peak} led to higher gains in VO_{2peak}. Furthermore, controlling for the total amount of work performed during the training period, exercise at two different intensities seems to result in similar training responses in older men (Gass et al. 2004).

Based on the Fick equation, $VO_{2max} = HR_{max} \cdot$ maximal SV $\cdot$ a-v O_2 difference, there are both central and peripheral mechanisms behind the training-induced increase in aerobic capacity (Fick 1870). The main factor that may explain the considerable difference in the aerobic capacity between endurance athletes and sedentary adults is believed to be the substantially higher cardiac output in the athletes (Levine 2008). HR_{max} has been shown to decrease slightly due to endurance training or not to change at all. Thus, increased SV as a result of increased end-diastolic volume may be the key aspect in the training adaptation (Levine 2008). Increased blood volume (Hagberg et al. 1998) and enhanced cardiac chamber compliance (Levine et al. 1991) seem to contribute to the increased end-diastolic volume through the increased utilisation of the Frank-Starling mechanism. Increases in SV and cardiac output have been observed as rapidly as after ten days of endurance training (Mier et al. 1997). Peripheral adaptations leading to increased a-v O_2 difference have been suggested to include increased capillary density and transformation of fatigue sensitive type IIX muscle fibres into fatigue resistant type IIA fibres (Spina 1999). Furthermore, increased mitochondrial content may result in diminished glycogen depletion and lactate production, and improved oxidation of fat for energy with the same submaximal exercise intensity (Spina 1999).

Cardiorespiratory adaptations in ageing

In an attempt to examine whether the training adaptations observed in young subjects also occur in older adults, some early studies did not show a significant increase in VO_{2max} in older adults, probably as a result of insufficient training stimulus (Benestad 1965; Niinimaa & Shephard 1978). Recent studies have, however, evidenced the significant benefits of endurance training on aerobic capacity in older adults. A direct comparison between previously sedentary younger (20-30 years) and older age groups (60-70 years) has shown similar relative increases in VO_{2max} despite the difference in age (Makrides et al. 1990).

Thus, endurance training seems to lead to adaptations in middle-aged and older adults that are similar in relative magnitude as in young adults (Hartley et al. 1969; Seals et al. 1984; Kohrt et al. 1991; Hepple et al. 1997).

The adaptation mechanisms may also be fairly similar in older and young adults (Stratton et al. 1994). Ageing may, however, change the priority of the mechanisms, highlighting the importance of peripheral adaptations at the expense of increases in cardiac output (Seals et al. 1984; McGuire et al. 2001a). In fact, in young men, the adaptations in VO_{2max} were shown to be coupled with improvements in both central and peripheral factors, whereas in the same men 30 years later VO_{2max} was improved mainly by the increase in a-v O_2 difference without changes in cardiac output (McGuire et al. 2001a). Capillary supply has been observed to increase after endurance training in men aged 65 to 74, and the change in capillary supply has been shown to positively correlate with the changes in VO_{2peak} (Hepple et al. 1997). Furthermore, significant increases in both cardiac output and a-v O_2 difference have also been observed in older men (Makrides et al. 1990; Spina et al. 1993), whereas in older women only changes in a-v O_2 difference were documented (Spina et al. 1993). Relative changes in VO_{2max}, however, seem to be similar in men and women (Spina 1999).

Cardiac autonomic adaptations to endurance training

Training induces a decrease in HR during rest and submaximal exercise. The decrease in the resting (Huang et al. 2005) and submaximal HR (Spina 1999) after endurance training are associated with an increase in stoke volume. Other mechanisms leading to the decreases in HR include increased vagal modulation (Smith et al. 1989; Shi et al. 1995) and decreased sympathetic activation (Smith et al. 1989), although it is likely that changes in sympathetic activation play a significant role only during exercise since sympathetic control is low at supine rest (Pomeranz et al. 1985; Martinmäki et al. 2006).

Training-induced changes in cardiac autonomic function have been studied using both cross-sectional and longitudinal designs. Most but not all (Lazoglu et al. 1996; Uusitalo et al. 1996; Middleton & Vito 2005) studies examining the association between cardiorespiratory fitness and HRV in a cross-sectional design have reported a significantly higher HFP suggesting enhanced vagal modulation in endurance athletes (Goldsmith et al. 1992; Macor et al. 1996; Shin et al. 1997; Verlinde et al. 2001; Mourot et al. 2004; Martinmäki et al. 2006) and in subjects with higher aerobic capacity (Tulppo et al. 1998) than in sedentary subjects or subjects with poor cardiorespiratory endurance. On the other hand, the effect of endurance training status on LFP seems to be negligible (Macor et al. 1996; Middleton & Vito 2005).

Based on several longitudinal studies, convincing evidence also exists about the positive effects of endurance training on selected measures of HRV recorded during supine rest in previously sedentary young (Leicht et al. 2003a; Leicht et al. 2003b) and middle-aged subjects (Carter et al. 2003; Leicht et al. 2003a; Tulppo et al. 2003; Hautala et al. 2004), although not all studies have

documented significant changes in HRV after endurance training (Loimaala et al. 2000; Verheyden et al. 2006). Thus, similarly to other training-induced adaptations, endurance training dosage probably also plays a significant role in the magnitude of training adaptation in terms of HRV. When the training dose has been too low, significant changes in resting measures of HRV have not been observed after 21 weeks of endurance training in young adults (Martinmäki et al. 2008) or even after one year of endurance training in older adults (Verheyden et al. 2006), despite concomitant increases in VO_{2peak}. A long training period may not necessarily lead to larger gains in HRV if the training dose is too low (Uusitalo et al. 2004). Furthermore, HRV has been shown to continue to improve with increased training doses (Okazaki et al. 2005). Training-induced changes have, however, been observed during exercise even if HRV measured at supine rest did not show significant changes with a low training dose (Martinmäki et al. 2008). Other studies have also reported training effects in HRV measured during exercise at the same absolute but not relative exercise intensities (Leicht et al. 2003a; Leicht et al. 2003b).

Similarly to linear measures, fractal scaling measures of HRV have also demonstrated significant changes after endurance training. Fractal scaling exponent decreased towards desirable fractal-like behaviour while resting HR decreased and LFP and HFP increased corresponding to increased vagal activity after endurance training (Tulppo et al. 2003). Information about the effects of endurance training on HR complexity measured as approximate entropy or sample entropy is limited. Cross-sectional data suggests, however, a significant difference between young trained (basketball players) and untrained subjects. The difference in sample entropy was especially evident during standing and a few minutes after exhaustive exercise (Platisa et al. 2008). A longitudinal study did not, however, show any training-induced effect on HR complexity in healthy obese middle-aged men and women (Kanaley et al. 2009).

2.2.2 Neuromuscular adaptations to strength training

Muscle strength varies markedly between individuals, and the most important factor contributing to the individual differences is training status. Through lifelong progressive resistance training, muscle strength can be increased two to three-fold, depending on the muscle group studied, compared with sedentary counterparts (Ojanen et al. 2007). In comparison, age accounts for only 30 % of the variance in strength in adults ranging between 20–93 years of age (Lindle et al. 1997). In middle-aged and older previously sedentary adults, strength training may lead to a 70 % increase in muscular strength during the first year of training when measured as the maximal load that can be lifted once (one repetition maximum, i.e. 1RM) (McCartney et al. 1995; Morganti et al. 1995).

Mechanisms of adaptations

The beneficial effects of chronic strength training are due to the acute neuromuscular and metabolic responses. Overloading a muscle, which occurs in

high-intensity strength training, induces acute physiological responses in the human body leading to a temporary decrease in muscle strength caused by neuromuscular fatigue (Gandevia 2001). Progressive overloading with sufficient rest between training sessions can elicit substantial increases in muscle strength. Careful design of the training program is essential for producing progressive overload and continued improvements in strength (Ratamess et al. 2009). Training intensity seems to be an especially important determinant of the strength training response in older adults, whereas training frequency higher than two times per week may not produce further improvements (Steib et al. 2010). Furthermore, the rate of increase in muscular strength is considerably faster during the first three months of strength training, although further increases also take place thereafter (McCartney et al. 1995; Morganti et al. 1995; Häkkinen et al. 1998a).

The training-induced increase in muscular strength may result from muscular and neural adaptations (Gandevia 2001). It has been suggested that in untrained individuals voluntary activation may be an important limiting factor in force production implicating that maximal voluntary force is smaller than the actual maximal force. The limitation can lie either in the firing of motoneurons or translating the motoneuron firing into force (Gandevia 2001). The increase in strength at the beginning of a strength training program has been shown to be mainly due to changes in neural mechanisms (Moritani & deVries 1979; Häkkinen & Komi 1983; Sale 1988; Staron et al. 1994) (Fig. 4). Changes in neural regulation measured as muscle electromyographic (EMG) activity can be observed during the first few weeks after initiation of training (Sale 1988). The increase in muscle activation may be explained by enhanced neural drive and changes in the pattern of motor unit recruitment (Sale 1988). Increases of 22-143% in mean average voltage of EMG have been reported in young men during an early contraction phase after 14 weeks of resistance training (Aagaard et al. 2002). Training-induced changes in EMG seem to be similar in older and middle-aged compared to young adults (Häkkinen et al. 1998a; Häkkinen et al. 1998b) Neural adaptations generally level out after a few weeks of strength training (Moritani & deVries 1979; Sale 1988).

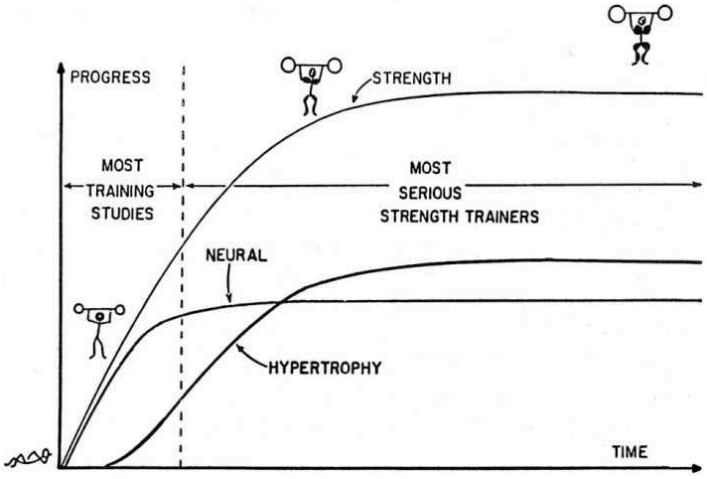

FIGURE 4 A schematic time course of neural and muscular adaptations to strength training. Modified from Sale (1988). Reproduced with permission of Wolters Kluwer Health.

A few weeks after the initiation of strength training, muscle hypertrophy seems to become the primary factor eliciting the further increases in maximal strength (Moritani & deVries 1979; Sale 1988). Muscle hypertrophy assessed with magnetic resonance imaging (MRI) has been generally observed after eight to twelve weeks of strength training (Roman et al. 1993; Häkkinen et al. 1998b; Ivey et al. 2000; Hubal et al. 2005). With high training stimulus three times per week, and with frequent and sensitive assessment of muscle size, quadriceps muscle hypertrophy (3.5–5.2 %) has been observed after only a couple of weeks of training in young adults (Seynnes et al. 2007).

Muscle hypertrophy, caused by the synthesis of contractile proteins, can be observed both within the whole muscle (which is approximately 5–8% in quantity) and within the myofibres (25–35 %) (Deschenes & Kraemer 2002). Muscle hypertrophy occurs mainly though myofibre hypertrophy with only a small contribution by fibre hyperplasia (Deschenes & Kraemer 2002). To some extent, hypertrophy is fibre type dependent (Roman et al. 1993; Staron et al. 1994) whereby type II fibres have a greater potential for muscle growth and a higher force per unit area than type I fibres (Staron et al. 1990; Kraemer et al. 1995; Harridge 2007). The increase in the size of muscle type II fibres may be over 40 % after 20 weeks of heavy resistance training, whereas type I fibres experience a smaller growth of approximately 15 % (Staron et al. 1990). Furthermore, a cross-sectional study did not observe a significant difference in the CSA of type I fibres between body builders and controls, while the type II fibres were considerably larger in body builders (D'Antona et al. 2006).

Prolonged strength training may also result in transformations in the muscle fibre types. Specifically, an increase in the percentage of type IIA fibres and decrease in the percentage of type IIX fibres has been often reported (Staron et al. 1990; Kraemer et al. 1995). Other studies have also supported the observation

that type IIX fibres are downregulated with resistance training (Staron et al. 1994; Häkkinen et al. 1998b). Training-induced transformation in the fibre types from slow-to-fast or fast-to-slow has not been shown in humans suggesting that the sport-specific variation in fast to slow ratio observed in athletes in different sports (Bergh et al. 1978; Tesch & Karlsson 1985; Aagaard et al. 2007) is regulated by heredity rather than training (Harridge 2007).

Neuromuscular adaptations in ageing

Strength training improves muscle strength with simultaneous increases in muscle size even when training begins in older age (Frontera et al. 1988; Häkkinen et al. 1998a; Häkkinen et al. 2001; Kryger & Andersen 2007). Middle-aged and older adults at the age of 55 to 75 (Jozsi et al. 1999; Tracy et al. 1999; Häkkinen et al. 2001) have shown substantial increases in muscle strength (Tracy et al. 1999; Häkkinen et al. 2001), power (Jozsi et al. 1999; Fielding et al. 2002), muscle size (Häkkinen et al. 2001) and muscle activation (Häkkinen et al. 1998b) after strength training. Training-induced changes in muscular strength may in fact be similar in relative magnitude in middle-aged and older adults compared to young subjects (Ivey et al. 2000). Some studies have, however, found smaller adaptations in older compared to young adults (Welle et al. 1996; Lemmer et al. 2000; Kosek et al. 2006), possibly due to a decreased rate of muscle recovery (Kosek et al. 2006) or lower levels of anabolic hormones (Izquierdo et al. 2001), especially in older women (Häkkinen et al. 2000). Muscle hypertrophy may also be attenuated in older compared to young adults (Welle et al. 1996), and in older women training-induced muscle hypertrophy may be lower especially among those individuals who have attenuated concentrations of serum testosterone (Häkkinen et al. 2001). Yet, with strength training, it is possible to minimise or delay the age-related loss of skeletal muscle mass and decreases in muscle strength and power in middle-aged and older individuals (Hunter et al. 2004; Martel et al. 2006).

2.2.3 Training mode specificity and secondary effects of endurance and strength training

The principle of training specificity states that training adaptations are linked with the mode, intensity and duration of exercise performed (Hawley 2002). This means that the training outcome is determined by the selection of the training design variables (Hawley 2008). Hence, in terms of training mode, endurance training is expected to lead to greater improvements in cardiorespiratory endurance than strength training and vice versa. In general, strength training does not produce significant cardiorespiratory adaptations. Similarly, endurance training is usually not a sufficient stimulus for significant muscle growth or increase in maximal strength (Sipilä & Suominen 1995; Grund et al. 2001). In addition to the specificity between endurance and strength training, specificity is also evident within the training modes. With regard to strength training, for example, gains in maximal power are dependent on the contraction velocity

utilised in strength training, since a training program with low-resistance and high-velocity contractions produces larger increases in power compared to low-velocity contractions in older women (Fielding et al. 2002).

The absolute specificity of training adaptations has, however, been questioned. It seems that endurance training may produce some stimulus for maximal strength development through muscle hypertrophy (Coggan et al. 1992; Harber et al. 2009) and through remodelling of contractile properties of the muscle fibres (Harber et al. 2009). Furthermore, strength training may lead to peripheral changes that improve the capacity of muscle to use oxygen through increased capillarisation (Frontera et al. 1990; Hepple et al. 1997) and conversion of IIX muscle fibres to more fatigue-resistant IIA and IIAX types (Hikida et al. 2000). In addition, the increase in lower extremity strength may increase the time to exhaustion during submaximal cycling exercise, often without concomitant increases in VO_{2peak} (Marcinik et al. 1991). Strength training may even provide an alternative means to improve aerobic capacity. Similar increases in VO_{2peak} have been observed with nine weeks of strength training followed by nine weeks of endurance training compared to 18 weeks of endurance training in older adults (Hepple et al. 1997). Furthermore, the effects of a short-term endurance or strength training program on VO_{2peak} showed that a subgroup of subjects who did not respond to endurance training increased their VO_{2peak} with strength training (Hautala et al. 2006).

Resting HR may be decreased after both endurance and strength training but the change is much more prominent after endurance training (Fleck 1988; Stone et al. 1991). Along with the increased SV, a mechanism behind the training-induced decrease may be increased parasympathetic and decreased sympathetic activity suggested by changes in various measures of HRV (Tulppo et al. 1998; Carter et al. 2003; Leicht et al. 2003a; Tulppo et al. 2003; Hautala et al. 2004). Similarly to the minimal effects of strength training on resting HR, the strength training-induced changes in HRV seem to be minimal compared to the effects of endurance training. Only a couple of studies have shown changes in linear measures of HRV after strength training in healthy adults (Jurca et al. 2004; Hu et al. 2009), while others have shown no effect (Forte et al. 2003; Cooke & Carter 2005; Madden et al. 2006; Heffernan et al. 2007; Collier et al. 2009). HFP expressed in normalised units has been shown to slightly decrease after high-intensity eccentric strength training, suggesting a decrease in HFP in relation to LFP. However, the absolute value of HFP remained unchanged (Melo et al. 2008). Studies have also shown beneficial effects of strength training on arterial blood pressure without marked changes in HRV (Cooke & Carter 2005; Melo et al. 2008).

Aside from the evident benefits of strength training, strength training may also increase arterial stiffness (Miyachi et al. 2004; Cortez-Cooper et al. 2005; Collier et al. 2008) even if arterial blood pressure is decreased (Collier et al. 2008). Arterial stiffness, on the other hand, has been associated with lower HRV (Jensen-Urstad et al. 1999). It has been shown recently, however, that muscular strength is inversely associated with aortic stiffness in young men (Fahs et al.

2010). Nonetheless, nonlinear measures assessing heart rate complexity and fractal behaviour of HR have shown significant positive strength training-induced changes (Heffernan et al. 2007; Heffernan et al. 2008) even without concomitant improvements in the linear measures of HRV (Heffernan et al. 2007). Actually, it has been suggested that nonlinear methods of beat-to-beat HR assessment provide additional information compared to linear measures of HRV (Kuusela et al. 2002). Alterations in fractal scaling properties, for example, have been shown to occur independent of changes in general measures of cardiovascular health such as cardiorespiratory fitness and percentage of body fat (Heffernan et al. 2008).

2.2.4 Individuality and the determinants of trainability

As described above, a considerable number of studies have demonstrated the beneficial effects of physical training in older adults. The mean change within a training group, however, may conceal a wide range of individual differences in trainability ranging from high responders to low and no responders, and even negative responders (Bouchard & Rankinen 2001). The individual differences in the adaptations to physical training have caught only minor research attention considering the large number of studies investigating the effects of training. Since training can be used, for example, for prevention of various diseases and age-related decreases in physical performance, attention should be aimed at individual differences in training adaptations (Kainulainen 2009). Therefore, information that is valuable for optimizing training programs may be lost when drawing conclusions based on average training responses alone (Bouchard & Rankinen 2001). However, as much as half of the interindividual variation in the training response after both endurance (Bouchard et al. 1999) and strength training (Thibault et al. 1986) may be explained by genotype.

After 20 weeks of endurance training, the individual response in VO_{2peak} has been shown to vary from about 0 to 1 $l \cdot min^{-1}$ (Bouchard et al. 1999). Age and gender seem only to have minor effects on the endurance training response (Kohrt et al. 1991; Bouchard & Rankinen 2001; Hautala et al. 2006). Age has been shown to account for 4 % of the individual variation in the VO_{2peak} response after 20 weeks of three times a week endurance training with progressively increasing intensity and duration in a large group of men and women between 17 to 65 years of age (Bouchard & Rankinen 2001). On the other hand, coefficient of determination has been shown to be as high as 16 % after eight weeks of moderate intensity (70-80 % of HR_{max}) endurance training six times a week in 23 to 52 year old men (Hautala et al. 2003). It is possible, therefore, that a shorter training period or a higher training frequency may emphasise the importance of age in the training response (Hautala et al. 2003). The age range of the subject group may also contribute to the association between age and the training response (McGuire 1986). In a study with a prolonged moderate intensity training of nine or twelve months and an average frequency of four times a week, age accounted for only 2 % of the variation in the endurance training response in a small age range (from 60 to 71 years) (Kohrt et al. 1991). A similar

coefficient of determination for age (1 %) has also been shown in middle-aged subjects after only two weeks (ten sessions) of moderate intensity endurance training (Hautala et al. 2006).

Similarly to endurance training adaptations, strength training-induced changes in muscle strength have shown large variations, ranging between a negative change to as much as a 250 % increase in young adults (Hubal et al. 2005). It has also been suggested that the variation in strength adaptations may be even larger in older compared to young adults (Newton et al. 2002). Individual adaptations to a 12 week unilateral strength training program have been studied with a large subject group (Hubal et al. 2005). The non-dominant arm was trained with a progressive increase of the intensity up to loads corresponding to 6 RM. For MVC of the elbow flexor muscles, 119 subjects showed an increase in strength of between 15 and 25 %, whereas 60 subjects gained over 40 %, and 102 subjects gained less than 5 %. Furthermore, 232 subjects of the total number of 585 subjects showed an increase in the CSA of biceps brachii muscle of between 15 and 25 %. In ten subjects, however, CSA increased over 40 %, whereas in 36 subjects less than 5% (Hubal et al. 2005).

The effect of baseline values of VO_{2peak} or muscular strength on the endurance or strength training responses, respectively, varies in different studies. When the whole range of physical performance is investigated, it is evident that sedentary subjects hold a capacity for larger relative improvements in physical performance than highly trained athletes. Furthermore, a large enough sample size of subjects is needed to fully quantify the range and determinants of responses to a given training program (Hubal et al. 2005). In a group of previously sedentary subjects VO_{2peak} at baseline may be an insignificant (Kohrt et al. 1991) or a small contributor (7 %) (Hautala et al. 2006) to the endurance training response. On the other hand, a significant negative correlation between initial VO_{2max} and ΔVO_{2max} (–0.52, $P < 0.05$) has been shown in ten pairs of young monozygotic twins after a 20 week moderate intensity endurance training program with four to five sessions per week (Prud'homme et al. 1984). With regard to the strength training responses, the type of muscle action used in the measurement of maximal strength may contribute to the correlation between muscle strength at baseline and the training responses. A higher negative correlation has been reported in a unilateral dynamic bicep curl (–0.55) compared to a corresponding isometric exercise (–0.27) in the same study (Hubal et al. 2005). Acute responses to a heavy strength exercise bout may also predict chronic training responses. Larger acute reduction in myostatin, which is a negative regulator of muscle mass, has been shown to be associated to larger strength training-induced increase in total body muscle mass (Hulmi et al. 2007).

In absolute terms, strength gains have generally been shown to be greater in men than in women (Cureton et al. 1988; Tracy et al. 1999). Several studies have shown, however, that both men and women may increase their maximal strength similarly in relative terms (Cureton et al. 1988; Abe et al. 2000; Lemmer et al. 2000; Lemmer et al. 2001). This finding was supported, for example, by carrying out nine weeks of high intensity unilateral strength training for the

knee extensors, after which no significant differences in strength gains were found between men and women in either younger (20–30 yr) or older subjects (65–75 yr) (Lemmer et al. 2000). Furthermore, it has been shown with a large sample of subjects (n = 585) that women may actually have larger relative gains in isometric (22 vs. 16 %, $P < 0.001$) and dynamic strength (64 vs. 40 %, $P < 0.001$) than men regardless of larger absolute gains in men, which could be explained by the smaller initial strength levels of women compared to men (Hubal et al. 2005). It seems, however, that the potential for muscle hypertrophy is greater in men (Ivey et al. 2000; Bamman et al. 2003) even though other studies have found no difference between genders in relative terms (Cureton et al. 1988; Tracy et al. 1999), possibly due to the lower training volume compared to the studies that reported a significant difference between genders (Ivey et al. 2000).

2.3 Adaptations to combined endurance and strength training

Combined endurance and strength training refers to the performance of endurance and strength training programs simultaneously within the same training session, during the same day on different occasions or on alternating days. In athletes, a combination of endurance and strength training modes is generally used for maximising sport-specific performance. In sedentary or physically active individuals, however, it is usually applied to seek further health benefits or enhanced physical performance compared to either of the training modes alone (Ferketich et al. 1998; Wood et al. 2001; Izquierdo et al. 2004; Häkkinen et al. 2005). The most recent guidelines for exercise prescription by American College of Sports Medicine suggest three or more aerobic training sessions per week for older adults (Chodzko-Zajko et al. 2009) combined with two to three resistance training sessions per week (Ratamess et al. 2009). The ability to improve both characteristics may, however, be limited. A number of studies have reported interference in the development of maximal strength (Hickson 1980; Bell et al. 1997; Bell et al. 2000) and muscle hypertrophy (Kraemer et al. 1995; Putman et al. 2004) by simultaneous endurance and strength training compared to those by strength training alone. Although rarely reported, the development of aerobic capacity may also be impaired by combined endurance and strength training (Nelson et al. 1990).

Endurance and strength training induce different kinds of requirements to the cardiovascular system and skeletal muscles. Endurance exercise generates a volume challenge to the cardiovascular system due to the prolonged duration of exercise. In contrast, strength training represents a pressure challenge due to the use of very high loads in only a few repetitions (Roy et al. 1988; D'Andrea et al. 2002). From the muscular perspective, endurance exercise is characterised by repetitive muscle contractions with relatively low loads. According to the size principle (Henneman et al. 1965), motor units are recruited in an order that is based on their recruitment threshold. The slower and lower force-producing motor units are activated before the faster and higher force-producing units.

Thus, endurance exercise activates mostly the slower motor units, including mainly fatigue resistant type I muscle fibres while strength training activates also faster units and high force-producing, but fatigue-sensitive fibres. The physiological effects of endurance training on skeletal muscle have been suggested to be antagonistic to increases in strength and muscle CSA, particularly in the muscle fibres that are recruited to perform both types of exercise (Hickson 1980; Kraemer et al. 1995; Bell et al. 2000).

2.3.1 Interference effects of concurrent endurance and strength training

In the context of combined endurance and strength training, interference refers to impaired training adaptations compared to those after similar endurance or strength training alone. The possible occurrence of interference has been suggested to largely depend on acute training design variables, such as training volume, mode of exercise, order of exercise, intensity, rest between sets, repetition velocity and frequency of training (Kraemer & Ratamess 2004), or in the long term, the duration of the training period (Häkkinen et al. 2003). When the volume, frequency and duration of combined endurance and strength training are moderate, the development of muscular strength or cardiorespiratory endurance do not seem to be compromised in young adults (McCarthy et al. 1995; McCarthy et al. 2002; Häkkinen et al. 2003; Leveritt et al. 2003; Izquierdo et al. 2005; Hendrickson et al. 2010). On the other hand, a prolonged combined training period and high training volume have been shown to attenuate strength gains (Hickson 1980; Dudley & Djamil 1985; Bell et al. 1997), muscle hypertrophy (Kraemer et al. 1995; Bell et al. 2000) and occasionally aerobic capacity (Nelson et al. 1990), compared with similar strength or endurance training only.

Inhibition of cardiorespiratory endurance

Many studies comparing the effects of combined endurance and strength training to those of endurance training alone have shown similar or even improved development of aerobic capacity (Haykowsky et al. 2005; Shaw & Shaw 2009; Hendrickson et al. 2010). Submaximal endurance capacity may be especially enhanced with added strength training in both trained young men (Hickson et al. 1988) and older untrained women (Ferketich et al. 1998).

Two studies have, however, documented impaired development of VO_{2max} with combined endurance and strength training compared with endurance training alone (Nelson et al. 1990; Dolezal & Potteiger 1998). In both studies that reported impaired gains in aerobic capacity, strength training was performed before endurance training within the same training session. The other aspects of the training programs were, however, quite disparate. One utilised unilateral strength training for knee extensors and flexors and cycling as the mode of endurance training four times a week (Nelson et al. 1990), whereas strength training in the other study included bilateral exercises for the whole body and endurance training comprised of running three times a week (Dolezal & Potteiger 1998). Interference occurred by the tenth week of training in trained

subjects (Dolezal & Potteiger 1998) and between the eleventh and twentieth weeks of training in untrained subjects (Nelson et al. 1990). The order in which endurance and strength training are performed may be important. In one study, performing endurance training before strength training within the same session led to larger increases in VO_{2max} compared to performing strength and endurance training in reverse order (Chtara et al. 2005).

Muscle hypertrophy may play some role in the interference. As could be expected, hypertrophy of muscle fibres seemed to be larger (25 %), although non-significant, in the combined training group compared to the endurance training group (11 %) in a study reporting attenuated development of VO_{2max} (Nelson et al. 1990). Thus, it could be postulated that significant increases in muscle mass may impair cardiorespiratory endurance, especially in exercise modes where body mass is entirely transported by muscular work, such as in walking and running. In fact, in both studies that reported interference in the development of aerobic capacity, VO_{2max} was measured in a treadmill running test (Nelson et al. 1990; Dolezal & Potteiger 1998). Furthermore, in young distance runners strength training combined with endurance training has been shown to improve anaerobic and neuromuscular characteristics with simultaneous increases in body mass but without a beneficial effect on endurance performance (Mikkola et al. 2007).

Impairment in strength development

It is impossible to simplify the interference effect of combined endurance and strength training into one specific cause leading to inhibition in strength development due to the various training programs utilised in different studies. For example, similar development of maximal strength has been observed with 20 weeks of combined strength and endurance training compared to strength training alone with four training sessions per week (Nelson et al. 1990), whereas attenuated gains in maximal strength have been observed after nine weeks of combined training with two weekly training sessions (Sale et al. 1990a; Gergley 2009). Some general resemblances regarding the combined training program leading to impaired strength gains may, however, be observed in the literature. Almost all studies combining four or more endurance and strength training sessions per week have shown interference in strength development (Hickson 1980; Hennessy & Watson 1994; Kraemer et al. 1995) with one exception (Nelson et al. 1990). In all three above-mentioned studies that documented interference in strength gains, subjects were athletes with a resistance training background (Hennessy & Watson 1994), or already trained (Kraemer et al. 1995) or physically active (Hickson 1980) before the training period. Furthermore, along with the high intensity strength training, endurance training consisted of running (Hennessy & Watson 1994; Kraemer et al. 1995) or both cycling and running (Hickson 1980) including high intensity intervals.

Low training frequency, on the other hand, may prevent the interference effects. A training program consisting of two strength and two endurance train-

ing sessions per week with high intensity on alternating days seems to produce similar strength gains as strength training alone after 21 weeks of training (Häkkinen et al. 2003). Combining three weekly endurance and three strength training sessions show, however, inconsistent results. Several studies show no interference in strength development (McCarthy et al. 1995; McCarthy et al. 2002; Glowacki et al. 2004; Haykowsky et al. 2005; Shaw et al. 2009; Hendrickson et al. 2010), whereas other studies have reported impaired strength gains (Dudley & Djamil 1985; Dolezal & Potteiger 1998; Bell et al. 2000).

A few studies allow a direct assessment of the effects of individual aspects of a training program. A study design comparing the effects between combined training performed on the same day and alternate days suggests that smaller increases in maximal strength may be observed when training for endurance and strength on the same day compared to alternate days (Sale et al. 1990a). Another study evaluated the effect of running compared to cycling as the endurance training mode (Gergley 2009). It was concluded that both cycling and running impaired strength gains compared to strength training alone, but the impairment was larger with combined training involving running. The subjects trained only two times a week, and therefore, overtraining is an improbable reason behind the interference. Other studies without a possibility for a direct comparison between endurance training modes also suggest that simultaneous endurance training by running may impair strength training adaptations (Hickson 1980; Craig et al. 1991; Hennessy & Watson 1994; Kraemer et al. 1995). A comparison between upper body versus whole body strength training combined with endurance training suggested that simultaneous strength and endurance training may compromise strength gains only if both modes of training utilise the same muscle groups (Kraemer et al. 1995).

The time course in the study by Hickson et al. (1980) suggests that strength performance can be improved with combined training similarly to strength only training if the training period is not too long. Indeed, with six weekly endurance and five weekly strength training sessions strength gains were similar in combined and strength only training groups during the first six weeks of the training period. Increase in maximal strength levelled off, however, between training weeks seven and eight. It has also been suggested that different measures of strength may evaluate different mechanistic events and, therefore, some measures may be more sensitive to the interference effects. Comparison of isoinertial, isometric and isokinetic strength measures before and after 6 weeks of combined training implies that susceptibility of the different measures to interference effects may vary (Leveritt et al. 2003). Furthermore, explosive strength seems to be more susceptible to interference than maximal strength (Häkkinen et al. 2003). Studies have shown an attenuated development of performance in exercises requiring rapid force production, such as maximal knee extension with fast angle velocities (Dudley & Djamil 1985) or in the maximal rate of force development in bilateral isometric leg extension without impairment in maximal strength (Häkkinen et al. 2003).

A summary of studies examining the effects of combined endurance and strength training on cardiorespiratory endurance, muscular strength and muscle hypertrophy is provided in table 1. Studies comparing the effects of combined training on similar strength or endurance training alone have been included, while studies with a disparate design have been excluded from the summary (Sale et al. 1990a; Sale et al. 1990b). Furthermore, studies with lower endurance or strength training volume in the combined training group compared to the endurance only group or strength only group, respectively, have also been excluded (Wood et al. 2001; Izquierdo et al. 2004; Izquierdo et al. 2005). Studies that have been summarised in the table have investigated previously untrained or physically active subjects. Studies investigating athletes have, however, been excluded (Hennessy & Watson 1994; Bell et al. 1997).

Inhibition of muscle hypertrophy

It seems to be feasible to train for strength and endurance simultaneously for as long as 21 weeks without significant attenuation in the development of maximal strength or muscle hypertrophy (Häkkinen et al. 2003). However, concurrent endurance training may inhibit muscle growth, at least in the mitochondria-rich type I fibres (Kraemer et al. 1995; Putman et al. 2004). Muscle growth may be inhibited even without simultaneous attenuation in maximal strength development (Bell et al. 1997; Bell et al. 2000; Putman et al. 2004). In the study by Kraemer et al. (1995), high intensity strength training alone increased the CSA of type I fibres, and endurance training alone seemed to decrease CSA, whereas performing both training programs simultaneously induced no change, thus demonstrating an intermediate response (Kraemer et al. 1995). Two other studies have also observed a significant change in CSA of type I fibres with strength training, no change with endurance training, and no change with combined training (Bell et al. 2000; McCarthy et al. 2002).

Proposed mechanisms of interference

Different mechanisms have been proposed to lead to the impaired training adaptations during concurrent endurance and strength training. The mechanisms are related to either fatigue in the short or long term, or divergent physiological adaptations at muscular level. Overreaching and overtraining are phenomena resulting from accumulation of fatigue due to an imbalance between training and recovery (Leveritt et al. 1999). Thus, overreaching may lead to impaired development of cardiorespiratory endurance or especially muscular strength and mass, since training volume is typically considerably larger in the combined training group compared to the groups that perform only endurance or strength training. In addition, the so called acute hypothesis suggests that in the short term, fatigue caused by the preceding endurance training session may decrease the loading and thus the quality of the following strength training session (Leveritt et al. 1999). The chronic hypothesis states, on the other hand, that

at the muscular level the adaptations to endurance and strength training are different thus leading to interference (Leveritt et al. 1999).

A few possible mechanisms for the interference of muscle hypertrophy with combined endurance and strength training have been suggested that are related to the different signalling pathways of these two training modes (Baar 2006; Nader 2006). For example, the mammalian target of Rapamycin (mTOR) signalling is considered as one of the key factors in strength training induced protein synthesis (Bodine 2006). On the other hand, the activity of adenosine monophosphate-activated protein kinase (AMPK), modulated by the ratio of adenosine diphosphate (ADP) to ATP, is increased during endurance type exercise. Using the combination of strength and endurance training, the activation of AMPK may substantially inhibit the mTOR signalling thus inhibiting protein synthesis (Baar 2006; Nader 2006). Another possible factor leading to interference could be the greater potential of aerobic exercise to induce oxidative stress than strength exercise, due to the duration-dependent nature of free radical production. Many biologically important substances involved in muscle hypertrophy, such as nucleic acid and protein, are at a theoretical risk of cellular damage (Pattwell & Jackson 2004; Fisher-Wellman & Bloomer 2009). Combined training has also been suggested to lead to a catabolic state, as evidenced by increased cortisol levels in younger men (Kraemer et al. 1995; Bell et al. 1997).

A model for the interference phenomenon by Docherty & Sporer (2000) is based on the divergent physiological responses to endurance and strength training at the muscular level. The model highlights the role of endurance and strength training intensity on the interference phenomenon. Endurance training including high intensity intervals is suggested to induce mainly peripheral adaptations due to hypoxia experienced by the muscles whereas continuous training at lower intensity may lead to larger cardiorespiratory adaptations (Cunningham et al. 1979). Strength training using intensities between 8 RM and 12 RM, on the other hand, has been shown to induce considerable hypertrophy, whereas higher loads may lead to larger neural adaptations. Thus, performing hypertrophic strength training combined with endurance training including high intensity intervals may in theory be most likely to lead to interference of endurance or strength training adaptations at muscular level (Docherty & Sporer 2000).

TABLE 1 A summary of studies examining the effects of combined endurance and strength training on cardiorespiratory endurance, muscular strength and muscle hypertrophy

Study	Groups (n)	Age	Training duration (wk), frequency and overall program			Main findings
Hendrickson et al. 2010	S (18 W) E (13 W) SE (15 W) C (10 W)	21 (0.5) 21 (0.4) 20 (0.4) 20 (0.5)	8	3 $\cdot wk^{-1}$; SE: same session S first	S: Non-linear periodised model, progressive 3 x 12/8-10/6-8/3-5 RM; E: running, continuous 20-30min, 70-85% of HR_{max} and 400-1600 m intervals	Squat 1RM: S +48%, SE +37% > E +15%; Treadmill VO_{2peak}: E +6%, SE +8 % > S +2%
Gergley 2009	S (8 M, 2 W) SE_C (7 M, 3 W) SE_T (7 M, 3 W)	21 (1) 20 (2) 20 (2)	9	2 $\cdot wk^{-1}$; SE: same day changing order	S: lower body, 3 x 8-12 RM, progressive; E: either cycling or treadmill walking, 65% of HR_{max}, progressive 20-40 min	Leg press 1RM: S +44% > $S+E_C$ +28% > $S+E_T$ +24%
Chtara et al. 2005	S (9 M) E (10 M) S+E (10 M) E+S (10 M) C (9 M)	21 (1)	12	2 $\cdot wk^{-1}$; S+E: same session S first; E+S: E first	S: whole body circuit training, 4 x 6 exercises until fatigue, muscle endurance (wk 1-6) and explosive strength (wk 7-12); E: running, 5 intervals at vVO_{2max}, recovery at 60% of vVO_{2max} (1:1)	Cycling VO_{2max} (ml $\cdot kg^{-0.75} \cdot min^{-1}$): E+S +14% > S+E +11%, E +10% > S +6%
Haykowsky et al. 2005	S E SE C (31 W)	70 (4) 66 (3) 68 (6) 67 (4)	12	3 $\cdot wk^{-1}$; SE: same day	S: upper and lower body, 2 x 10 reps, progressive 50-75 % of 1RM; E: cycling 60-80 % HRR, progressive 15 - 42.5 min	Upper and lower extremity 1RM: S,E+S > E; Cycling VO_{2peak}: E+S = E = S
Putman et al. 2004	S E SE C (24 M, 16 W)	23 (1)^{SEM}	12	S: 3 $\cdot wk^{-1}$ E: 3 $\cdot wk^{-1}$ SE: 3+3 $\cdot wk^{-1}$	S: upper and lower body, 2-6 x 4-10 reps at 70-85% of 1RM; E: cycling, progressive 30-42 min, continuous at VT and intervals at 90 % of VO_{2max}	CSA of type I fibres: only in S +17%; type IIA: S +13%, SE +18%.
Glowacki et al. 2004	S (13 M) E (12 M) SE (16 M)	23 (3) 25 (5) 22 (2)	12	S: 2.5 $\cdot wk^{-1}$ E: 2.5 $\cdot wk^{-1}$ SE: 5 $\cdot wk^{-1}$	S: total body, 3 x 6-10 reps, 8 exercises; E: running, progressive 65-80% of HRR, 20-40min	Leg press 1RM: S +41%, SE +39% > E +20%; Treadmill VO_{2max}: E 8%, SE 3% (ns.), S 4% (ns.)

Study	Groups (n)	Age	Training duration (wk), frequency and overall program			Main findings
Häkkinen et al. 2003	S (16 M) SE (11 M)	38 (5) 37 (5)	21	S: 2 ·wk^{-1}; SE: 2+2 ·wk^{-1}	S: whole body, 3-5 x 10-15/8-12/5-6 reps with progressive 50-80% of 1RM; E: cycling, continuous and intervals, progressive 30-90 min	Leg extension 1RM: S +21% = SE +22%; Explosive strength: S > SE; Quadriceps CSA: S +6% = SE +9%
Leveritt et al. 2003	S (5 M, 3 W) E (3 M, 6 W) SE (3 M, 6 W)	19 (1) 19 (2) 19 (1)	6	3 ·wk^{-1}; SE: same session E first	S: whole body, 3 x 4-8RM for squats, 3 x 6-10RM for other exercises; E: cycling, 5 x 5 min intervals with 5 min passive recovery, 40-70% of VO_{2peak}	Squat 1RM: S = SE; Cycling VO_{2peak}: E,SE↑, S↓
McCarthy et al. 2002	S (10 M) E (10 M) SE (10 M)	28 (1) 27 (2) 27 (2)	10	3 ·wk^{-1}; SE: same session order rotated	S: whole body, 8 exercises, 3 x 5-7RM; E: cycling, 50 min, 70% of HRR	CSA of thigh extensors: S +12%, SE +14% > E +3%; CSA of type I fibres: S +19%, SE +13% (ns.); type II: S +24%, SE +28%
Bell et al. 2000	S (7 M, 4 W) E (7 M, 4 W) SE (8 M, 5 W) C (5 M, 5 W)	22 (3)	12	E: 3 ·wk^{-1}; S: 3 ·wk^{-1}; SE: 3+3 ·wk^{-1}	S: lower and upper body, 2-6 x 4-10 reps with 70-85% of 1RM; E: cycling, progressive 30-42 min, continuous at VT and intervals at 90% of VO_{2max}	Leg press 1RM: S,SE > E; knee extension S > SE > E; Cycling VO_{2max}: E,SE > S; CSA of type I: S ↑, SE (ns.), type II: S, SE ↑
Dolezal & Potteiger 1998	S (10 M) E (10 M) SE (10 M)	20 (2)	10	3 ·wk^{-1}; SE: same session S first	S: whole body, progressive 3 x 10-15/10-12/8-10/4-8 RM; E: running, progressive 65-85% HR_{max} and 25-40 min	Squat 1RM: S +23%, SE +19% > E -1%; Treadmill VO_{2max}: E +13% > SE +7%
Ferketich et al. 1998	E (8 W) SE (7 W) C (6 W)	69 (2) 67 (2) 70 (2)	12	3 ·wk^{-1}; SE: same session E first	S: lower and upper body, 2 x 12-15 reps with 80% of 10RM; E: cycling, 70-80% of VO_{2peak}, 30 min	Leg extension 10RM: SE +112% > E +43%; Cycling VO_{2peak}: E +25% = SE +30%; Submax time to fatigue: SE > E

Study	Groups (n)	Age	Training duration (wk), frequency and overall program			Main findings
McCarthy et al. 1995	S (10 M) E (10 M) SE (10 M)	28 (1) 27 (2) 27 (2)	10	3 $\cdot wk^{-1}$; SE: same session order rotated	S: whole body, 8 exercises, 3 x 5-7RM; E: cycling, 50 min, 70% of HRR	Squat 1RM: S +23%, SE +22% > E -1%; Cycling VO_{2max}: E +18%, SE +16% > S +9%
Kraemer et al. 1995	S (9 M) E (8 M) SE (9 M) S_UE (9 M) C (5 M)	24 (5) 21 (4) 23 (4) 23 (5) 22 (4)	12	4 $\cdot wk^{-1}$; SE: same day E first with 5-6h recovery	S: whole body, 2-5 x 10-5RM; E: running, continuous 40 min and 200 – 800 m intervals; SE: E + S for whole body; S_UE: E + S for upper body	Leg press 1RM: S 30% > SE 20% > S_UE 10% > E 2%; CSA of type I fibres: S > SE, S_UE > E; Treadmill VO_{2max}: E +12%, SE +8%, S_UE +10% > S -1%
Nelson et al. 1990	S (5 M) E (4 M) SE (5 M)	27 (1)^{SEM} 30 (2)^{SEM} 26 (1)^{SEM}	20	4 $\cdot wk^{-1}$; SE: same session S first	S: unilateral knee extension and flexion at 30° $\cdot s^{-1}$, 3 x 6RM; E: cycling, progressive 30-60 min, 75-85% HR_{max}.	Knee extension torque: S, SE > E; Treadmill VO_{2max}: E > SE
Dudley & Djamil 1985	S (6 MW) E (10 MW) SE (6 MW)	26 (2)^{SEM} 21 (1)^{SEM} 22 (2)^{SEM}	7	3 $\cdot wk^{-1}$; SE: alternate days	S: knee extension, 2 x 30s, 4.19 rad $\cdot s^{-1}$; E: cycling, 5 x 5min intervals, near VO_{2peak}	Knee extension torque: S↑ up to the training velocity, SE↑ only up to 1.68 rad $\cdot$s-1; Cycling VO_{2peak}: E = SE
Hickson 1980	S (7 M, 1 W) E (5 M, 3 W) SE (5 M, 2 W)	22 (18-27) 25 (19-36) 26 (18-37)	10	S: 5 $\cdot wk^{-1}$; E: 6 $\cdot wk^{-1}$; SE: >2h recovery	S: lower body, 3/5 x 5RM; E: cycling (continuous) 30-40 min and running (intervals) 6x5 min near VO_{2max}	Squat 1RM: S +44% > SE +34%; Cycling VO_{2max}: E +27% = +24% SE; and Treadmill; E +19% = +18% SE

S, strength training; S_U, strength training for upper body; E, endurance training; E_C, E using cycling; E_T; E using treadmill; SE, combined S and E training; C, control group; M, men; W, women; age reported as mean and SD, unless otherwise indicated; SEM, standard error of mean; (18-27), age range; RM, repetition maximum; VO_{2max}/VO_{2peak}, maximal / peak oxygen uptake; vVO_{2max}, velocity at VO_{2max}; HR_{max}, maximal heart rate; HRR, heart rate reserve; CSA, cross-sectional area; >, significantly larger than the corresponding change in the other group(s); = similar change compared to the other group(s); ↑significant increase.

2.3.2 Potential benefits of combined endurance and strength training in ageing

The individual differences in the time-course of ageing are largely due to factors related to lifestyle, including regular physical activity (Chodzko-Zajko et al. 2009). Neither endurance training nor strength training alone can substantially improve all the essential aspects of physical performance and health in light of the principal of training specificity. Ideally, performing both endurance and strength training in parallel leads to significant increases in both cardiorespiratory endurance and maximal strength that are similar to endurance training or strength training only, respectively (McCarthy et al. 1995; Häkkinen et al. 2003). Thus, a combination of endurance and strength training modes may also combine the beneficial effects of both endurance and strength training leading to improved cardiorespiratory endurance and cardiac autonomic function as well as considerable gains in muscular strength and muscle mass. In ageing adults these adaptations may contribute to maintaining health and functional capacity at work and during leisure time. Problems with the compatibility of endurance and strength training may, however, reciprocally impair the training-specific gains. Furthermore, the mean change within a training group may conceal a wide range of individual differences in the responses (Bouchard et al. 1999; Hubal et al. 2005). Training prescription of a combination of endurance and strength training is, therefore, a challenging task to optimise physical performance and health outcomes.

3 AIMS OF THE STUDY

Due to the specificity and competitive nature of the adaptations of endurance and strength training, performing these two training modes simultaneously may produce different outcomes compared to endurance or strength training alone. The ability to improve both characteristics may thus be limited. Furthermore, even if cardiorespiratory endurance is associated with the function of the autonomic nervous system, training-induced changes in cardiac autonomic function may also occur independently of the changes in physical performance. Therefore, this study examined the effects of combined endurance and strength training on cardiorespiratory endurance, neuromuscular performance and cardiac autonomic function compared with endurance or strength training alone in ageing men and women. The specific aims of the present investigations were:

1) To examine the possible interference in adaptations to combined endurance and strength training in terms of cardiorespiratory endurance, neuromuscular performance and skeletal muscle hypertrophy in previously untrained 40–67 year old adults after a prolonged training program (original papers I and II).

2) To examine individual differences in the responses to endurance, strength and combined endurance and strength training when endurance and strength training programs are performed separately and concurrently (original paper III).

3) To investigate the effects of combining endurance and strength training compared with endurance training or strength training alone on cardiac autonomic function at rest and during exercise (original papers IV and V).

4 METHODS

4.1 Subjects

Healthy untrained 40 to 67 year old men and 40 to 65 year old women were recruited for the intervention by advertising in newspapers and through e-mail lists. The participants were informed about the design of the study and possible risks and discomforts related to the measurements, after which all participants signed a written informed consent. Subjects underwent an examination of general health and a resting electrocardiogram (ECG) administered by a physician. The subjects who passed the medical examination performed a maximal exercise test to voluntary exhaustion with ECG monitoring under the supervision of a physician. Exclusion criteria included cardiovascular diseases, musculoskeletal problems, malfunctions of the thyroid gland, diabetes, or any other disease that may have hindered performing the requested testing or training. Furthermore, exclusion criteria included medications known to influence cardiovascular or neuromuscular performance or cardiac autonomic function. Subjects with a background of systematic endurance or strength training during the year preceding the study were also excluded.

206 subjects entered the actual study, and finally 196 subjects, 100 men and 96 women [mean age 53 (SD 8) years)] completed the intervention (Fig.5). The mean age of men [55 (7) years] was somewhat higher ($P < 0.001$) than that of women [51 (7) years]. The characteristics of the final subject groups at baseline in different original papers are presented in table 2. The study plan was approved by the Ethics Committees of the University of Jyväskylä and the Central Finland Health Care District.

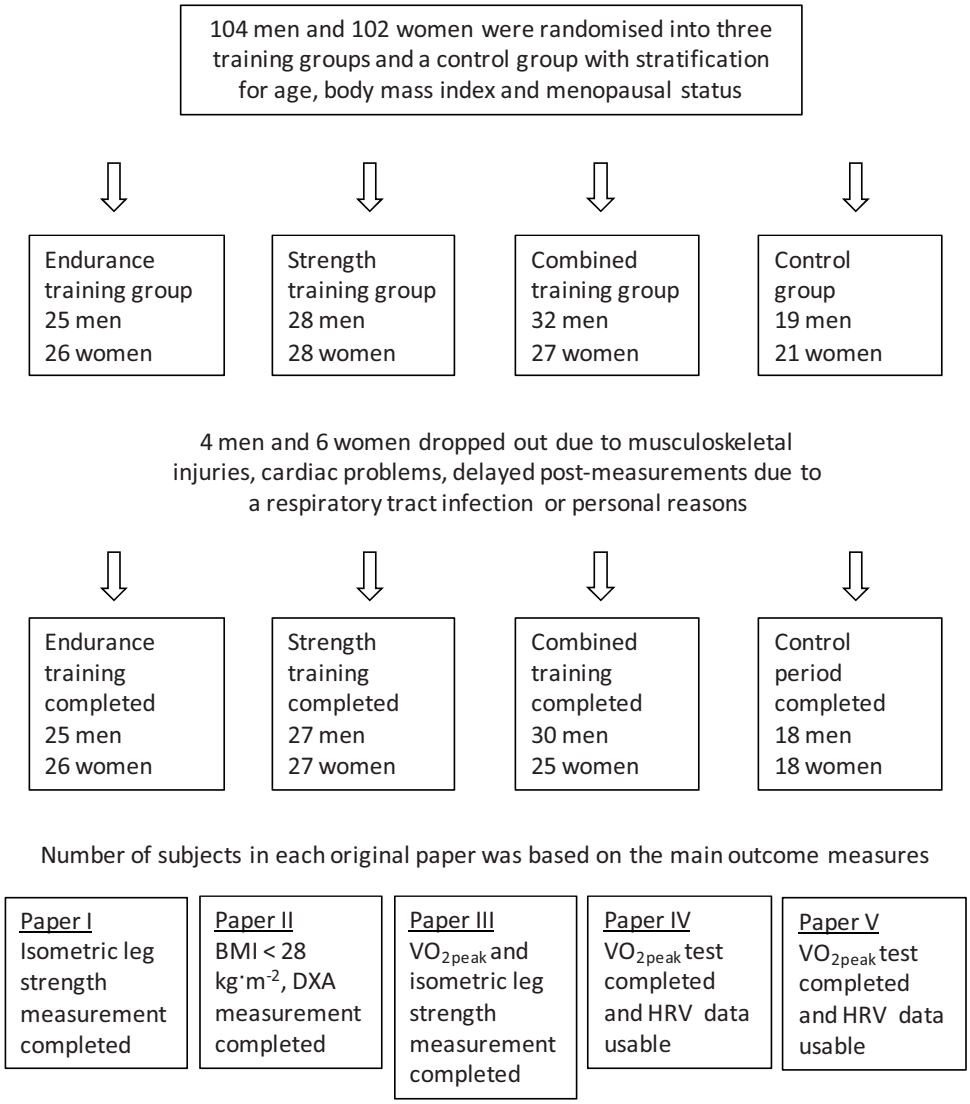

FIGURE 5 Participant flow. BMI, body mass index; DXA, body composition analysis with dual-energy x-ray absorptiometry; VO_{2peak}, peak oxygen uptake; HRV, heart rate variability.

TABLE 2 Characteristics of the subjects in different original publications (papers I – V).

	Paper I	Paper II	Paper III		Paper IV	Paper V
n	96 M	52 M	89 M	86 W	93 M	90 W
Age, yr	56 (7)	55 (7)	55 (7)	51 (7)	56 (7)	51 (7)
Height, cm	177 (7)	177 (7)	177 (7)	165 (6)	177 (6)	164 (6)
Weight, kg	81 (10)	77 (9)	81 (10)	67 (9)	82 (10)	66 (9)
BMI, $kg \cdot m^{-2}$	25.9 (2.9)	24.5 (2.2)	25.7 (2.8)	24.8 (2.8)	26.0 (2.9)	24.7 (2.8)

Values are mean (SD). BMI, body mass index; M, men; W, women.

4.2 Experimental design

The subjects were randomised into endurance training (E), strength training (S), combined endurance and strength training (SE) or control (C) groups separately for each gender. Men and women were investigated separately in consecutive years. The measurements were performed once (cardiorespiratory endurance test and muscle biopsies) or twice (maximal strength, power and HRV at rest) before the training, representing a control period of two weeks (-2), and after 10 and 21 weeks of training. The control group was measured similarly once or twice before the study period and after that once at week 21. Endurance and strength training were performed twice a week so that the E and the S groups trained two times a week and the SE group trained a total of four times a week on alternate days. Both the control group and the training groups were instructed to maintain habitual physical activity throughout the study period at the same level as before.

4.3 Data collection

4.3.1 Cardiorespiratory endurance test

A graded maximal exercise test to volitional exhaustion was performed on a mechanically braked bicycle ergometer (Ergomedic 839E, Monark Exercise AB, Sweden) with simultaneous ECG and blood pressure monitoring. The test was supervised by a physician. The exercise intensity was increased by 20 W every second minute starting with 50 W, and pedalling frequency was sustained at 60 rpm throughout the test. The subjects were verbally encouraged to maintain the required pedalling frequency as long as possible. Oxygen uptake (VO_2), carbon dioxide production (VCO_2), ventilation, breathing frequency and other standard respiratory parameters were measured continuously breath by breath (SensorMedics® Vmax229, SensorMedics Corporation, Yorba Linda, CA, USA). VO_{2peak} was determined as the highest minute average of VO_2 during the test. Maximal aerobic power output (P_{max}) was calculated with the following formula: $P_{max} = P_{com} + t/120 \cdot \Delta P$, where P_{com} is the last cycling power completed, t is the time in seconds the non-completed power was maintained and ΔP is the increment in watts (Kuipers et al. 1985).

4.3.2 Measurements of neuromuscular performance

Maximal strength and power

Isometric bilateral leg extension force was measured as maximal voluntary contraction (MVC) on a dynamometer (Häkkinen et al. 1998a; Häkkinen et al. 1998c) in a seated position with a knee angle of 107° and a hip angle of 110°. Subjects

were instructed to generate maximum force as rapidly as possible against the force plate for a duration of two to four seconds.

Isometric bench press was performed in a seated position with hip and knee angles of 90° (Häkkinen et al. 1998c). Maximal force was exerted against horizontal bars with the upper arm in a horizontal position and an elbow angle of 90°. For both isometric actions, subjects performed a minimum of three trials, and the trial with the highest peak force was selected for further analysis. The force signal was low pass filtered (20 Hz) and analyzed (Signal software Version 2.15, Cambridge Electronic Design Ltd., Cambridge, UK).

Concentric bilateral leg extension strength (hip, knee and ankle extensor strength) based on 1RM was measured on a horizontal leg press dynamometer (David 210, David Fitness and Medical Ltd., Outokumpu, Finland) (Häkkinen et al. 1998a). The test was performed in a seated position with a hip angle of 110°. Subjects performed leg extension from 70° of knee flexion to full extension (180°). The load was increased for each trial until the subject failed to fully extend the knees. The load of the last successful performance was determined as 1RM, which was usually achieved within 3–5 trials.

Maximal concentric power was measured with a load of 50 % of 1RM using a dynamic explosive leg press exercise on a David 210 dynamometer. After the baseline measurements, the load of the explosive leg press exercise was modified according to the changed 1RM value after 10 and 21 weeks of training, in order to maintain the same relative load compared to baseline.

Recordings of electromyographic (EMG) activity

Electromyographic (EMG) activity was recorded from the vastus lateralis (VL) and vastus medialis (VM) muscles of the right leg during the isometric bilateral leg extension, and from the triceps brachii (TB) muscle during the isometric bench press. The skin was carefully prepared, and the bipolar surface electrodes were placed according to SENIAM guidelines (Hermens et al. 1999) (inter-electrode distance 20 mm and resistance < 10 kΩ). The signal was recorded telemetrically (Telemyo 2400R, Noraxon, Scottsdale, Arizona USA), amplified with a factor of 500, band pass filtered (20–350 Hz), digitised at a sampling frequency of 2,000 Hz (Micro1401, CED, Cambridge, UK) and analyzed (Signal software Version 2.15, Cambridge Electronic Design Ltd., Cambridge, UK). Maximum iEMG was calculated for the maximal force phase of the isometric contractions (500–1500 ms) (Häkkinen et al. 1998a). To ensure repeatable measurements throughout the study period, small ink tattoos were used to mark the positions of the electrodes on the skin (Häkkinen et al. 1998a). In carefully controlled conditions, EMG will reflect training-induced changes in the neural drive to the muscle (Gandevia 2001).

4.3.3 Measurements of muscle size

Lower extremity lean mass was estimated by dual-energy x-ray absorptiometry (DXA). DXA systems (LUNAR, GE Healthcare) provide a measure of lean soft

tissue including muscle and other components such as skin, tendons, and connective tissues and excluding fat and bone mineral components. Legs were isolated from the trunk by using regional computer-generated default lines with manual adjustments using the system software (enCORE 2005, version 9.30) and anatomical landmarks. The line between the trunk and legs was positioned at the soft tissue extending from a line drawn through and perpendicular to the axis of the femoral neck and angled with the pelvic brim to the phalange tips (Kim et al. 2002). Metal items were removed from the subject to ensure the accuracy of the measurement. Subjects were positioned in the centre of the table for each scan. The same investigator performed all the measurements and analyses.

Ultrasonography (B-mode, model SSD-2000, Aloka, Tokyo) was used to assess the thickness of the vastus lateralis (VL) and vastus intermedius (VI) muscles (Sallinen et al. 2008). The measurement was performed with a probe frequency of 5 MHz. The thickness of the VL and VI muscles of the right leg were measured from the midpoint of the line formed by anterior spina iliaca superior and lateral side of patella. Ultrasonography measurements were performed three times per muscle using the average of the two closest measurements as the final value of muscle thickness. The positions of the electrodes were marked on the skin by small ink tattoos. Muscle thickness of the knee extensors has been shown to predict muscle volume (Miyatani et al. 2000; Miyatani et al. 2002). The coefficient of variation for the measurement of muscle thickness in older subjects has been reported to be 2.5 % for elbow flexors and 3.6 % for knee flexors (Candow & Chilibeck 2005).

4.3.4 Determination of muscle fibre composition and size

Human skeletal muscle fibre types can be identified based on the histochemical staining properties of the myosin adenosine triphosphatase enzyme (Staron 1997). Due to the large total number of subjects, muscle biopsies were obtained from a subsample of subjects; 11 male and 15 female subjects in S, 7 and 16 in E, 12 and 14 in SE, and 3 and 8 in C, before and after the 21 week training period. Biopsies were taken from the vastus lateralis (VL) muscle, midway between the patella and greater trochanter using the percutaneus needle biopsy technique (Bergström & Hultman 1966). After the training period, muscle biopsies were carefully obtained from the same depth, 0.5 cm lateral to the preceding biopsy. The samples were mounted on a cork and frozen immediately in isopentane (–160 °C) and stored at –80 °C. For histochemical analysis, serial cross-sections (8 μm thick) were cut on a cryomicrotome (Leica CM 3000) at –24 °C, and stained with a myofibrillar ATPase method after preincubation at a pH of 4.37, 4.60, and 10.30 to define four different fibre types (Types I, IIA, IIAX, and IIX) (Ennion et al. 1995). The stained cross-sections were analyzed using a microscope (Olympus BX50, Olympus Optical Co., Tokyo, Japan) and a colour video camera (Sanyo High Resolution CCD, Sanyo Electronic Co., Osaka, Japan) combined with image-analysis software (Tema, Scan Beam, Hadsund, Denmark).

Fibre type distribution and CSA were determined from a mean fibre number of 147 (SD 43) and 137 (46) in S, 145 (54) and 157 (30) in E, 142 (65) and 165 (52) in SE, and 149 (41) and 132 (30) in C, at the pre and post training measurements, respectively.

4.3.5 Measurement and analysis of heart rate dynamics

RR interval recording

The resting measurement was carried out after an overnight fast in the morning between 7:00 and 9:00 a.m. The measurement took place in a quiet room at a thermo neutral temperature. The RR intervals were recorded in a supine position for ten minutes, and the last five minutes of the data was used in the analysis allowing an additional five minute rest period before the actual measurement in men. In women, recording of ten minutes was used for the analysis. The breathing frequency was spontaneous and remained at the normal respiratory frequency of supine rest throughout the recording. RR intervals were also recorded during a five minute steady state exercise on a bicycle ergometer with a power output of 50 W. Furthermore, in men RR intervals were recorded during a maximal graded cycling test with 20 W increments starting from 50 W. Each two minute test stage was analyzed separately.

RR intervals were collected for further analysis with Polar S810i HR monitors by using online recording with infrared interface and Polar Precision Performance software (Polar Electro Ltd., Kempele, Finland). RR intervals were recorded with 1 ms time resolution. The Polar S810i HR monitor has been validated for RRI recording both at rest and during exercise with accuracy better than 2 ms when compared with an ECG method (Kingsley et al. 2005; Gamelin et al. 2006). The subjects whose data contained more than 15% noise or ectopic beats were discarded from further HRV analysis. For the other subjects, the measurement errors and the ectopic heart beats were eliminated by both an automatic process in the software and visual inspection followed by manual filtering for possible artefacts. In the automatic process, the errors were corrected according to the length of the error sequence, taking the previous and the next normal RR interval into account and maintaining the local trend in the RR interval time series (Jurca et al. 2004). The NN interval, used in the abbreviations of many HRV measures, e.g. SDNN, refers to the corrected time series including only normal sinus to normal sinus interbeat intervals.

Time domain and spectral analysis

The average of all NN intervals and the corresponding average HR were computed. Standard deviation of all NN intervals (SDNN) was computed as the square root of variance. Analysis of power spectral density provides information about how the variance (power) is distributed as a function of frequency (Malik et al. 1996). Frequency domain variables HFP (0.15–0.40 Hz) and LFP (0.04–0.15 Hz) were analyzed using an autoregressive (AR) model of order 18 in

men (paper IV) and a Lomb periodogram (Moody 1993) in women (paper V). As distinct from the AR method, the Lomb periodogram allows power spectral density estimation without resampling the time series at uniform intervals, which may produce a more accurate estimation of the high frequency components. AR and Lomb spectra closely resemble each other, but in the presence of noise and ectopic beats, the Lomb periodogram may produce a more robust power spectral density estimation (Moody 1993). Due to the short duration of the recordings total power was not included in the analysis. The assessment of HFP and LFP components was made in absolute values of power (milliseconds squared, ms^2).

Detrended fluctuation analysis (DFA)

Fractal HR dynamics describe correlative properties of HR. A short-term scaling exponent that represents the slope of the plotted root mean square fluctuations of the integrated and detrended data was analyzed. The short-term (from 4 to 11 beats) scaling exponent (α_1) was calculated by integrating the RR interval time series and fitting least-squares line to the data. Integrated time series was then detrended by subtracting a local trend. A relationship between the average fluctuation and the size of the window of observation was then drawn to a double log graph. The scaling exponent represents the slope of this line (Peng et al. 1995b). DFA analysis was performed using the Hearts software (Heart Signal Co, Oulu, Finland).

Multiscale entropy analysis

The multiscale entropy method was developed to quantify a multiscale variability of complex signals (Costa et al. 2002), and sample entropy (SampEn) is a measure of irregularity. The general complexity of a signal is calculated by integrating the values obtained for a pre-defined range of scales (Costa et al. 2008). The algorithm comprises two steps, a coarse-graining procedure and the quantification of the degree of irregularity of each coarse-grained time series, which can be accomplished using sample entropy (Richman & Moorman 2000).

The analysis of multiscale entropy is illustrated in figure 6. A simulated time series u[1],…,u[N] illustrates the procedure for calculating sample entropy. Dotted horizontal lines around data points u[1], u[2] and u[3] represent u[1]±r, u[2]±r and u[3]±r, respectively. Two data points match each other if the absolute difference between them is ≤ r. In the present study an r value of 8 ms was used. The symbols ○, x and Δ are used to represent data points that match the data points u[1], u[2] and u[3], respectively. Consider the two-component ○-x template sequence (u[1], u[2]) and the three-component ○-x-Δ template sequence (u[1], u[2], u[3]). For the segment shown, there are two ○-x sequences, (u[13], u[14]) and (u[43], u[44]), that match the template sequence (u[1], u[2]), but only one ○-x-Δ sequence that matches the template sequence (u[1], u[2], u[3]).

These calculations are repeated for the next two-component and three-component template sequence, which are (u[2], u[3]) and (u[2], u[3], u[4]), respectively. The number of sequences that match each of the two- and three-component template sequences are summed and added to the previous values. This procedure is then repeated for all other possible template sequences to determine the ratio between the total number of two-component template matches and the total number of three-component template matches. SampEn is computed as the natural logarithm of this ratio and it corresponds to the probability that sequences that match each other for the first two data points will also match for the following point (Costa et al. 2005).

Subsequently, the sample entropy for different coarse-grained time series (different scales) was calculated. A complexity index (CI) (Kang et al. 2009) was computed by plotting the sample entropy of each coarse-grained time series as a function of scale and integrating the curve over a scale of 1 to 5 for the RR interval recordings at rest (CI_{1-5}) and over a scale of 1 to 2 for the recordings during exercise (CI_{1-2}) due to the shorter duration of the exercise recording (5 vs. 10 minutes). For regular signals, SampEn is low. For uncorrelated random signals, SampEn is the highest (Costa et al. 2005).

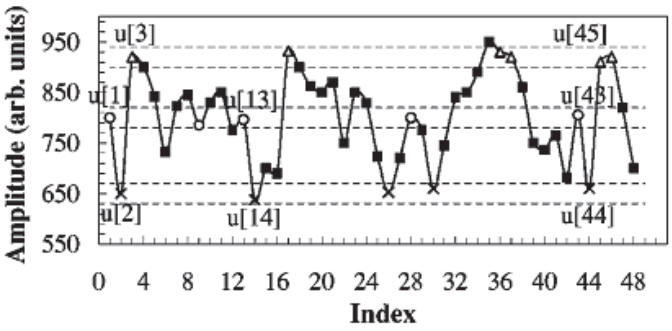

FIGURE 6 Simulated time series u[1],…,u[N] for illustrating the procedure for calculating sample entropy (Costa et al. 2005). Copyright (2005) by The American Physical Society.

4.4 Training programs

Training programs were selected according to the previous training interventions performed in our laboratory to ensure continuum of the series of investigations regarding the effects of endurance training (Martinmäki et al. 2008), strength training (Häkkinen et al. 1998a; Häkkinen et al. 2000; Häkkinen et al. 2001) and combined endurance and strength training (Häkkinen et al. 2003).

4.4.1 Endurance training

Endurance training was carried out twice a week. The heart rate levels for endurance training were determined based on respiratory parameters and blood lactate concentrations, as described in detail previously (Aunola & Rusko 1984). All training sessions were supervised, and heart rate monitoring was used. During the first 7 week period, the subjects trained on a bicycle ergometer for 30 min below the level of the aerobic threshold. Weeks 5–7 during the first period also included three training sessions during which the subjects were accustomed to the intensity above the aerobic threshold by a 10 minute interval in the middle of the sessions. During weeks 8–14, one weekly session of 45 min included a 10-min interval between the aerobic-anaerobic thresholds and a 5-min interval above the anaerobic threshold, in addition to a 15-min warm-up and a 15-min cool down below the aerobic threshold. The other weekly training session involved 60 min of cycling below the aerobic threshold. The focus of training during weeks 15–21 was to improve maximal endurance. One of the weekly sessions lasted for 60 min, which included two 10-min intervals between the aerobic-anaerobic thresholds, two 5-min intervals above the anaerobic threshold, and 30 min below the aerobic threshold. The other weekly session included 90 min cycling at a steady pace below the aerobic threshold.

4.4.2 Strength training

Strength training was carried out twice a week. All strength training sessions were supervised. The strength training program included 7–10 exercises that activated all of the main muscle groups. Every training session included two exercises for the leg extensors (leg press and knee extension), one exercise for the knee flexors (leg curl), and one to two other exercises for the lower extremities (seated calf raise, hip abduction or adduction). For the upper body, each session included three to four exercises (bench press, biceps curl, triceps pull-down, lateral pull-down), and one to two exercises for the trunk (abdominal crunch, back extension). The overall training intensity increased progressively throughout the 21-week training period (Häkkinen et al. 1998a).

The training period was divided into three 7 week cycles to optimise strength gains and muscle hypertrophy. The focus of the first cycle was to accustom the subjects to high intensity training and to improve muscle endurance and strength using light loads (40–60 % of 1RM) and a high number (12–20) of repetitions, and by performing 3 sets. The second cycle (weeks 8–14) was designed to produce muscle hypertrophy to further increase the total muscle mass by increasing the loads progressively up to 60–80 % of 1RM, with 5–12 repetitions and 2–4 sets. To optimise maximal strength development and to further produce hypertrophy during weeks 15 – 21, higher loads of 70–85 % of 1RM together with 5–8 repetitions and 2–4 sets were used. In addition, due to the significance of maximal power in daily life, low-resistance high-velocity contractions are recommended to be included into strength training programs (Hunter et al. 2004). Therefore, approximately 20 % of the leg press, knee exten-

sion and bench press exercises were performed with light loads of 40 to 50 % of 1RM and 5–8 repetitions to meet the requirements of a typical explosive strength training protocol. With the light loads, each repetition was executed as rapidly as possible (Häkkinen et al. 1998a).

4.4.3 Combined endurance and strength training

The subjects in the combined endurance and strength training group performed endurance training twice a week and strength training twice a week, performing a total of four training sessions per week on alternate days as described in the preceding paragraphs (Häkkinen et al. 2003).

4.5 Statistical analyses

Standard statistical methods were used to compute means and standard deviations (SD). Men and women were examined separately in consecutive years and therefore, papers I, II, IV and V only involved either male or female subjects. Thus, analysis of variance (ANOVA) with repeated measures was used for investigation of training group and time interactions. One-way ANOVA for relative changes at post-training measurements was additionally used. The assumptions for repeated measures ANOVA, homogeneity of variance, sphericity, and normal distribution, were tested with the Levene's test, Mauchly's test and Shapiro-Wilk test, respectively. If the assumptions were not met, even after a natural logarithm transformation, an analogous nonparametric test was used. ANOVA for repeated measures was also used to detect within-group changes followed by pair wise comparisons between time points using Bonferroni correction.

Paper III differed in a design investigating individual training responses in both genders separately and together. Thus, individual values and 95 % confidence intervals were reported, and differences between genders in addition to differences between the training groups were assessed using multifactor ANOVA (for training group, gender and time interactions). Variability in the training response was calculated as the coefficient of variation, and Levene's test was used to assess the equality of variances between the training groups. Spearman's rank-order correlation coefficient was used to study the relation between the training responses in aerobic capacity and maximal strength within the training groups.

Associations between variables were studied using the Pearson's correlation coefficient. The critical level of significance was set at $P = 0.05$. The statistical analyses were carried out using SPSS software for Windows (SPSS Inc., Chicago, IL).

5 RESULTS

5.1 Training adherence and values at baseline

The average training adherence was 99 (3) % in the endurance and 99 (2) % in the strength training sessions. All subjects who completed the training period performed at least 90 % of the total training volume. Furthermore, there were five or less subjects in each group who performed < 95 % of the total training volume when strength and endurance training programs were analyzed separately in the SE group. There were no differences between groups in training adherence. The randomised groups were similar (P = 0.24-0.83) within genders at baseline in terms of age, height, weight, BMI, VO_{2peak}, P_{max}, and leg press 1RM and MVC. Resting HR (P = 0.050) and submaximal HR (P = 0.007) were lower in C compared to the training groups in men. In women, HR was similar between groups both at rest (P = 0.65) and during exercise (P = 0.95).

5.2 Training-mode specific and individual adaptations in physical performance in men and women (I, III)

5.2.1 Average training-induced changes in cardiorespiratory endurance and muscular strength

Body mass only slightly decreased in the male and female E groups [-1 (2) kg, P < 0.05 for both genders] and the male SE group [-1 (2) kg, P = 0.010] and thus, the changes in the absolute values of VO_{2peak} were similar to VO_{2peak} values normalised to body mass. VO_{2peak} ($ml \cdot kg^{-1} \cdot min^{-1}$) increased only in SE and E and not in S or C (time and group interaction P < 0.001) in both men and women after 21 weeks of training (table 3). Endurance performance, measured as maximal power output (P_{max}) in the maximal incremental cycling test, also increased significantly in S aside from the E and SE groups. The average in-

creases in P_{max}, however, were significantly higher in the E and SE groups than in the S group. Both VO_{2peak} and P_{max} increased similarly with combined endurance and strength training compared to endurance training alone (P = 1.00 for both variables). The average changes in P_{max} were 14 (9) and 17 (8) % in E, 13 (7) and 18 (8) % in SE, 6 (6) and 7 (9) % in S, and –2 (5) and 2 (7) % in C, in men and women, respectively. A significant difference between genders in the training response was observed in the relative changes of VO_{2peak} (Fig. 7) and P_{max} in SE, whereby women showed a larger mean response than men.

No significant changes were observed in isometric or dynamic strength, concentric power or muscle activation in any of the groups during the control period (between weeks –2 and 0) in men. In women leg press 1RM was 4 (7) and 3 (5) kg higher at week 0 compared to week –2 in S (P = 0.017) and SE (P = 0.010), respectively.

Training-induced changes in muscular strength were similar between the SE and S groups in both men and women. SE and S showed larger increases in 1RM than C or E in both men and women. In addition, the mean increase in leg press MVC was larger in SE and S compared to C and E. Men showed larger relative increases in leg press 1RM than women in all the training groups ($P \leq$ 0.001). The relative changes were, however, similar between genders in leg press MVC.

The findings related to bench press and maximal concentric power are only reported in men. (Data for women will be reported later outside this thesis). All training groups showed significant increases in bench press MVC but in SE and S the changes were significantly different from the control group (table 3). Maximal concentric power only increased in the S and SE groups.

5.2.2 Individual differences in the training responses

For the analysis of individual training adaptations both genders were analysed together. Large individual differences in trainability were observed in all training groups (Fig. 7). Coefficient of variation (CV) of ΔVO_{2peak} was similar in E (0.92) and SE (0.90), and CV of ΔMVC was similar in S (0.85) and SE (0.82). Large interindividual variation was also observed in ΔVO_{2peak} after strength training, and in ΔMVC after endurance training. In S, 27 % of the subjects increased their VO_{2peak} more than the upper 95 % confidence interval of the control group (4.5 %). In E, 33 % increased their MVC more than the upper 95 % confidence interval of the control group (10.1 %).

There were no significant correlations between the training responses in VO_{2peak} and MVC in E (r = 0.097, P = 0.54), S (r = 0.059, P = 0.69), or SE (r = 0.078, P = 0.58). Figure 8 shows that even though a few subjects in the SE group showed a negative training response in VO_{2peak} or MVC, none of the subjects showed a negative change in both. In addition, none of the subjects reached the highest quintile in both ΔVO_{2peak} and ΔMVC (Fig. 8), and only two subjects reached the highest quartile and seven subjects the highest tertile in both ΔVO_{2peak} and ΔMVC. Furthermore, only 55 % of the subjects increased both

their VO_{2peak} and MVC more than the upper 95 % confidence interval of the control group.

The training-induced change in VO_{2peak} correlated significantly with the baseline only in women (r = -0.72 to -0.43, P < 0.05). Significant correlations were not found between ΔMVC and MVC at baseline. Age was not a significant contributor to the training response in terms of VO_{2peak} or MVC.

TABLE 3 Measures of physical performance before (0) and after 10 and 21 weeks of training and in the control group in men and women.

Men	Endurance (n = 25)	Strength (n = 25)	Combined (n = 30)	Control (n = 16)	Time x Group
VO_{2peak}, $ml \cdot kg^{-1} \cdot min^{-1}$					
0	32.9 (7.2)	33.2 (6.4)	32.5 (4.2)	34.8 (5.5)	
10	34.9 (6.5) **	34.0 (5.6)	34.3 (4.7) **		
21	36.6 (7.9) ***	33.1 (5.9)	35.7 (5.1) ***	34.8 (6.0)	*P* < 0.001
VO_{2peak}, $l \cdot min^{-1}$					
0	2.58 (0.57)	2.80 (0.51)	2.70 (0.45)	2.77 (0.45)	
10	2.74 (0.55) **	2.86 (0.49)	2.85 (0.54) **		
21	2.84 (0.63) ***	2.76 (0.49)	2.93 (0.54) ***	2.77 (0.55)	*P* < 0.001
P_{max}, W					
0	202 (43)	212 (34)	210 (33)	216 (42)	
10	216 (43) ***	218 (31) **	226 (39) ***		
21	227 (41) ***	225 (30) ***	237 (41) ***	211 (40)	*P* < 0.001
1RM leg press, kg					
0	154 (34)	166 (32)	158 (29)	153 (20)	
10	160 (33) *	181 (34) ***	176 (33) ***		
21	166 (37) ***	200 (36) ***	193 (38) ***	161 (21) ***	*P* < 0.001
MVC leg press, N					
0	2735 (670)	2827 (692)	2776 (668)	2642 (389)	
10	2874 (716) *	3098 (644) ***	3111 (772) ***		
21	2963 (731) **	3177 (666) **	3314 (815) ***	2793 (473) ***	*P* < 0.001
MVC bench press, N					
0	728 (134)	757 (123)	692 (110)	726 (130)	
10	752 (127) *	803 (135) *	743 (123) ***		
21	769 (156) **	838 (127) ***	788 (127) ***	725 (101)	*P* < 0.001
Power, W					
0	1836 (374)	2093 (351)	2000 (477)	1843 (440)	
10	1799 (480)	2314 (468)	2216 (500) *		
21	1724 (589)	2397 (502) *	2250 (555) *	1843 (411)	*P* = 0.020

(continues)

TABLE 3 (continues)

Women	Endurance (n = 21)	Strength (n = 25)	Combined (n = 23)	Control (n = 17)	Time x Group
VO_{2peak}, ml·kg^{-1}·min^{-1}					
0	25.3 (5.2)	26.0 (5.3)	26.8 (4.9)	26.6 (6.1)	
10	28.2 (5.7) ***	28.5 (5.2) ***	30.5 (4.8) ***		
21	29.8 (4.4) ***	27.3 (4.9)	31.1 (5.0) ***	26.8 (5.8)	$P < 0.001$
VO_{2peak}, l·min^{-1}					
0	1.79 (0.40)	1.75 (0.38)	1.79 (0.32)	1.80 (0.43)	
10	1.95 (0.41) ***	1.93 (0.36) ***	2.02 (0.32) ***		
21	2.07 (0.35) ***	1.83 (0.34)	2.07 (0.31) ***	1.81 (0.42)	$P < 0.001$
P_{max}, W					
0	157 (26)	153 (30)	155 (23)	152 (33)	
10	173 (25) ***	162 (30) ***	174 (23) ***		
21	181 (24) ***	164 (30) ***	181 (24) ***	155 (35)	$P < 0.001$
1RM leg press, kg					
0	103 (13)	98 (14)	101 (15)	93 (13)	
10	105 (14)	104 (16)	109 (15)		
21	105 (14)	107 (16)	112 (15)	94 (13)	$P < 0.001$
MVC leg press, N					
0	1992 (431)	1965 (431)	1977 (498)	1882 (278)	
10	2082 (489)	2193 (536) ***	2278 (613) ***		
21	2105 (455) *	2293 (587) ***	2446 (793) ***	1970 (239)	$P = 0.001$

Values are mean (SD). VO_{2peak}, peak oxygen uptake; P_{max}, maximal aerobic power output; 1RM leg press, one repetition maximum in a leg press exercise; MVC, maximal voluntary contraction in isometric leg press and bench press exercises; Power, maximal concentric power of leg extension. Significant difference compared to baseline at * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$.

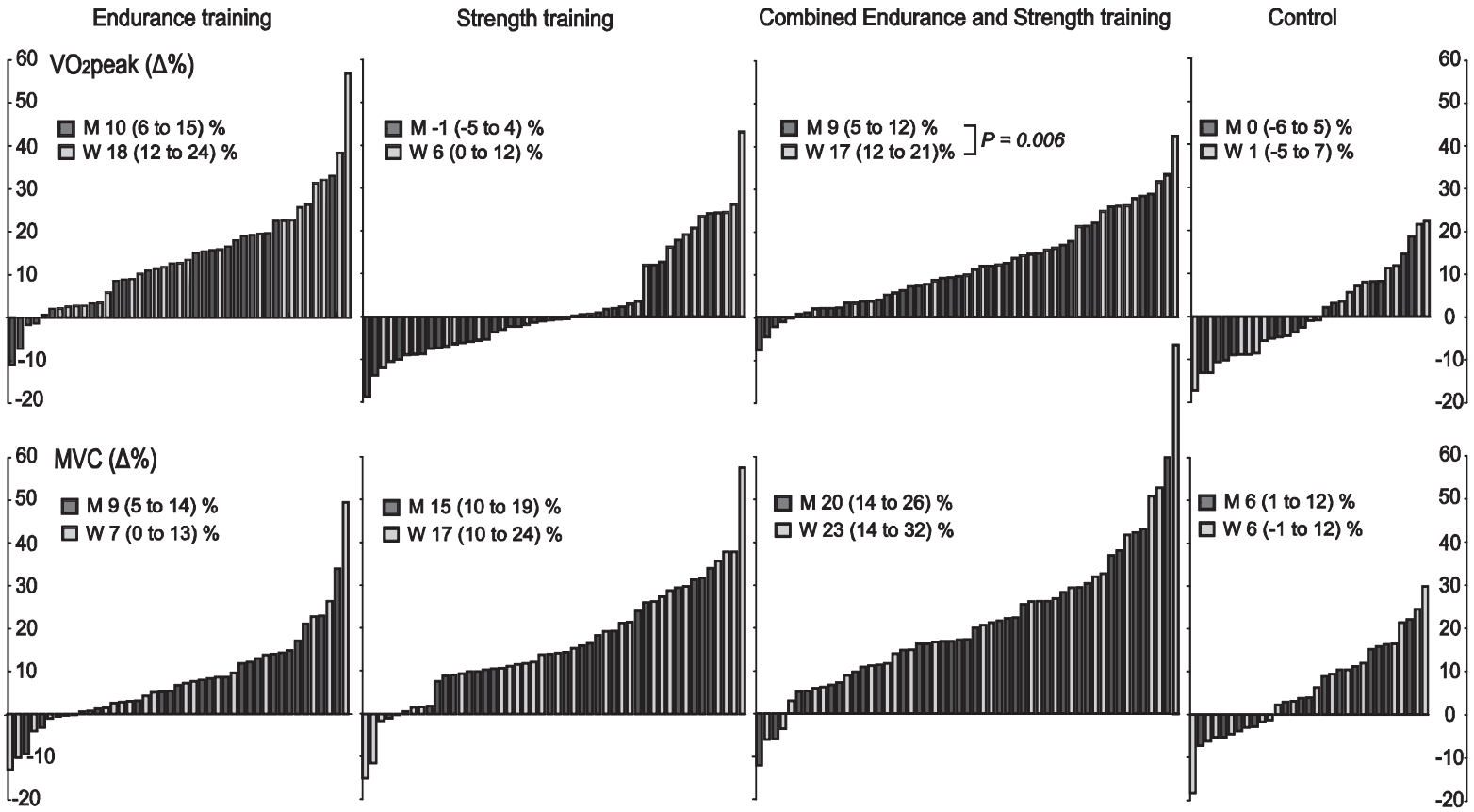

FIGURE 7 Individual (bars) and mean (95 % confidence interval) responses to endurance, strength, and combined endurance and strength training in men (M) and women (W). The control group is also shown for comparison.

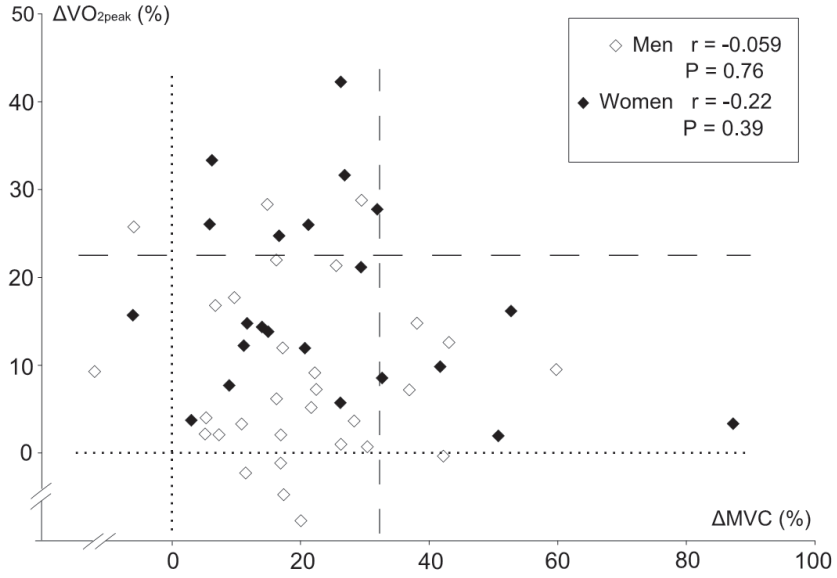

FIGURE 8 Correlations between changes in maximal isometric force (MVC) and peak oxygen uptake (VO_{2peak}) in the combined endurance and strength training group in men and women. Dashed lines represent the highest quintile, and dotted lines represent a negative response in both VO_{2peak} and MVC.

5.3 Muscle hypertrophy and muscle activation (I, II)

5.3.1 Muscle hypertrophy

In men the lean mass of the legs measured by DXA only increased significantly in the S group (Fig. 9). In women, lean mass of the legs increased in all training groups by 2.2 – 2.9 % (P = 0.004–0.010, data not shown).

There was high reproducibility between control and baseline measurements of muscle thickness using ultrasound (intraclass correlation coefficient 0.92 and 0.95 for combined thickness of VL+VI and thickness of TB, respectively). During the first 10 weeks of training, the combined thickness of VL and VI muscles increased (P < 0.001) in all three training groups [in E 8 (8) %, in S 9 (5) %, and in SE 9 (6) %, Fig. 9] in men, but during the last 10 weeks of training, only S showed a minor non-significant increase up to 11 (7) %. In women the thickness of VL+VI also increased in all training groups during the first 10 weeks, but only the S group showed a further significant increase during the latter half of the training period. The thickness of VL+VI increased by 6 (6), 8 (5) and 12 (7) % in E, S and SE, respectively (P < 0.001), after 21 weeks of training compared to baseline.

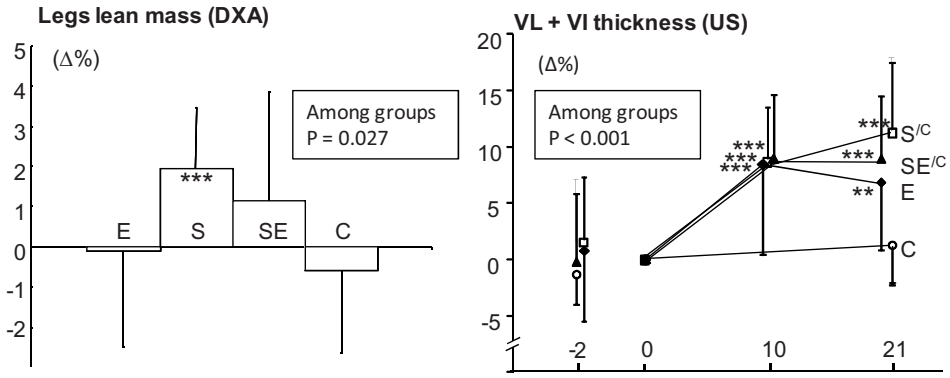

FIGURE 9 Changes in the lean mass of the legs measured by dual-energy x-ray absorptiometry (DXA, left) and in combined thickness of vastus lateralis (VL) and the vastus intermedius (VI) muscles measured by ultrasonography (US, right) after 21 weeks of training in the endurance (E), strength (S), combined endurance and strength training (SE) and the control group (C) in men. Significant differences between the groups are indicated with superscripts, /C = significantly different from C. Significant difference from baseline at ** $P < 0.01$, *** $P < 0.001$.

5.3.2 Fibre type composition and cross-sectional area of muscle fibres

In S, the proportion of type IIA fibres increased ($P < 0.05$) and the proportion of type IIAX fibres decreased ($P < 0.05$) in both men and women (table 4). In women, the percentage of type IIAX and IIX fibres also decreased in SE. The proportion of type I fibres did not change in any of the groups.

When all three subtypes of type II muscle fibres were combined in the analysis, the cross-sectional area (CSA) of type II fibres increased in S without significant changes in any other group in men (Fig. 10). The individual changes in the CSA of type II fibres revealed a rather consistent increase in the S group especially in men, whereas the SE group showed an inconsistent, non-significant change in both men and women (Fig. 11). In women, CSA of type II fibres also increased in E ($P < 0.001$) due to the significant increase in the CSA of type IIA fibres. The increase in the CSA of type IIA fibres also approached significance ($P = 0.050$) in the male S group with a 16 (24) % increase, and did not change in the other groups. No changes were observed in the CSA of type I fibres in any of the groups.

TABLE 4 Muscle fibre type distribution before (pre) and after (post) 21 wks of training.

Men	E (n = 7)		S (n=11)		SE (n = 12)		C (n = 3)	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post
Type I (%)	44 (15)	48 (17)	40 (18)	45 (13)	45 (6)	42 (14)	46 (10)	55 (6)
Type IIA (%)	35 (13)	41 (14)	36 (20)	50 (12) *	41 (8)	49 (14)	23 (3)	22 (7)
Type IIAX (%)	14 (4)	9 (7)	18 (12)	5 (6) **	13 (8)	6 (10)	31 (7)	20 (7)
Type IIX (%)	7 (5)	2 (4)	6 (9)	0.2 (0.4)	1 (1)	3 (7)	0.5 (0.5)	3 (4)

(continues)

TABLE 4 (continues)

Women	E (n = 16)		S (n = 15)		SE (n = 14)		C (n = 8)	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post
Type I (%)	51 (17)	49 (15)	47 (14)	46 (13)	42 (9)	46 (13)	51 (8)	49 (11)
Type IIA (%)	38 (14)	42 (13)	36 (12)	46 (14)*	42 (10)	49 (13)	34 (8)	37 (12)
Type IIAX (%)	9 (7)	8 (9)	9 (5)	5 (5)*	12 (8)	5 (4)**	11 (5)	11 (5)
Type IIX (%)	2 (2)	1 (2)	8 (9)	3 (6)	3 (3)	1 (1)*	3 (4)	2 (3)

Values are mean (SD). S, strength training group; E, endurance training group, SE combined strength and endurance training group; C, control group. Significant difference from baseline at * $P < 0.05$, ** $P < 0.01$.

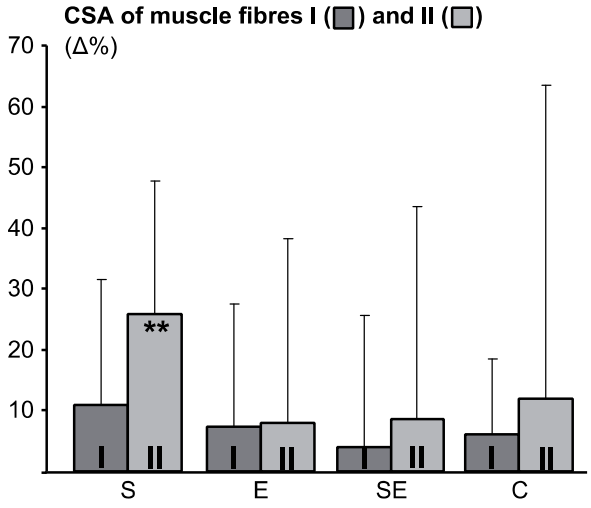

FIGURE 10 Changes (Δ%) in the mean cross-sectional area (CSA) of type I and II muscle fibres in vastus lateralis muscle after 21 weeks of strength (S), endurance (E), combined strength and endurance (SE) training and in the control group (C). **significantly ($P < 0.01$) different from the baseline measurement.

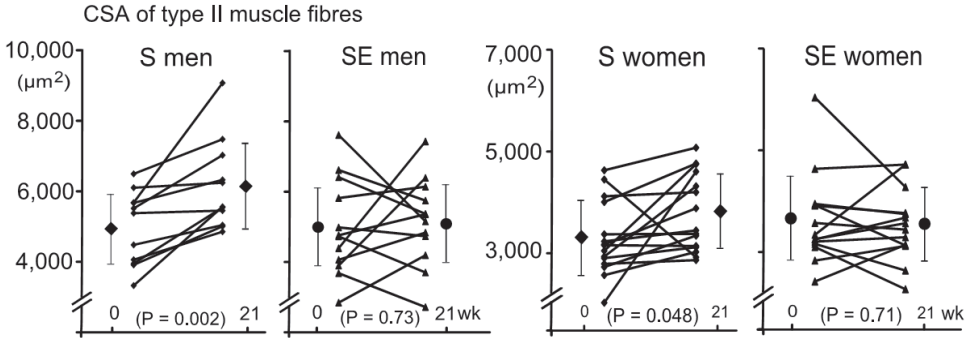

FIGURE 11 Individual changes in the cross-sectional area (CSA, μm²) of type II muscle fibres in the strength (S) and combined strength and endurance (SE) training groups in men and women. *P* value indicates the significance of the difference between the baseline measurement (0 week) and post measurement (21 weeks).

5.3.3 Muscle activation in men

In maximal isometric leg press, EMG activity of vastus medialis (VM) increased significantly more in SE and S than E or C (P = 0.042). In S and SE, EMG activity increased in VM by 41 (47) % and 18 (28) % (Fig. 12A), and in VL by 35 (49) % and 11 (18) %, respectively, between training weeks 0 and 10, whereas between weeks 10 and 21 there was no further increase. EMG activity of VL did not show significant time x group interaction. At week 10, a significant difference between groups was observed in the relative changes (P < 0.01), whereby the increase in EMG activity of VM and VL in S was larger than in SE or E. EMG activity of TB seemed to increase throughout the training period in SE and S (P < 0.05), and the change was not significant in any other groups (Fig. 12B).

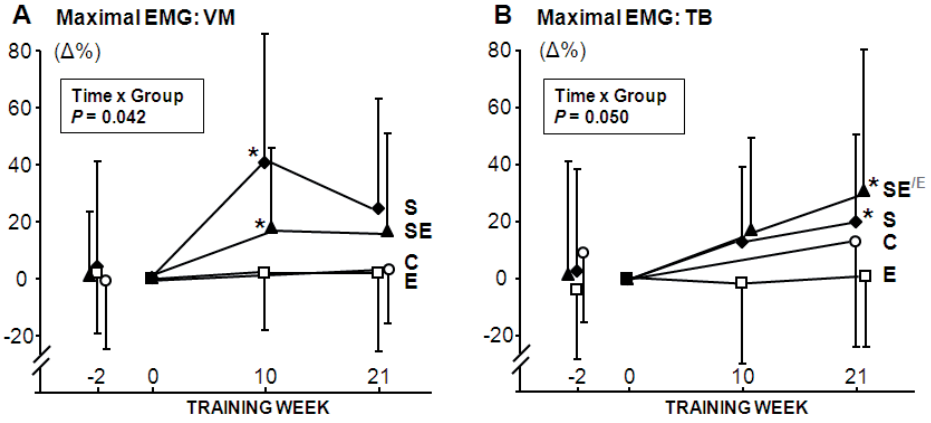

FIGURE 12 Maximal electromyographic (EMG) activity in vastus medialis (VM, A) and triceps brachii (TB, B) muscles during maximal isometric leg press and bench press, respectively, during the control period (-2 and 0 weeks), and after 10 and 21 weeks of strength (S), endurance (E) or combined strength and endurance (SE) training, and in the control group (C). Significantly different from the baseline measurement at *P < 0.05.

5.4 Heart rate dynamics after training in men and women (IV, V)

5.4.1 Heart rate dynamics at rest

During the control period (from -2 weeks to 0 weeks) HR or HRV at supine rest did not change significantly in men except the LFP/HFP ratio which decreased in E (P = 0.023) due to the tendency of HFP to increase (P = 0.053). In the female E group, however, several HRV measures at rest including SDNN, HFP and complexity index were higher at week 0 compared to week -2 (P = 0.012-0.038). In addition, the LFP/HFP ratio was lower at week 0 compared to week -2 (P = 0.045). According to the intraclass correlation coefficient (ICC) between the two baseline recordings the reproducibility of the HRV measures at supine rest was

moderate. ICC tended to be higher in men (between 0.80 and 0.91) than in women (between 0.63 and 0.84, and as low as 0.40 in LFP). In general, the lowest reproducibility was observed in the spectral measures.

Overall, the effects of the present training program on HR dynamics recorded during supine rest were minor in both men and women. In men, resting HR slightly decreased in E and SE, but not in S or C (table 5). In women, resting HR remained unchanged (table 5). SDNN or the spectral variables of HRV measured during supine rest did not, however, show any training-induced changes in men or women. Fractal scaling exponent α_1 decreased significantly in the male SE group towards fractal like dynamics (i.e. α_1 = 1.0) and also showed a decreasing trend in the male E group, although differences between the groups were not significant. No changes were observed in the HR complexity measure $CI_{1\text{-}5}$ in men or women at rest.

TABLE 5 Selected HRV indices at rest before (0), and after 21 weeks of training in the endurance, strength, combined endurance and strength training, and control group in men.

Men	Endurance (n = 23)	Strength (n = 25)	Combined (n = 29)	Control (n = 16)	Time x Group
HR, min^{-1}					
0	61 (10)	59 (9)	58 (8)	54 (5)	
21	57 (8) *	59 (8)	57 (7) *	53 (6)	*P* = 0.026
SDNN, ms					
0	46.5 (19.7)	45.0 (16.3)	43.0 (24.6)	57.8 (29.6)	
21	45.1 (13.7)	40.0 (18.1)	45.0 (25.2)	44.4 (20.3)	*P* = 0.063
HFP (ms^2)					
0	369 (389)	383 (430)	241 (292)	570 (595)	
21	375 (372)	224 (185)	341 (406)	472 (498)	
HFP, ln (ms^2)					
0	5.3 (1.3)	5.3 (1.2)	5.0 (1.3)	5.7 (1.2)	
21	5.4 (1.2)	5.0 (1.0)	5.3 (1.3)	5.6 (1.1)	*P* = 0.067
LFP, (ms^2)					
0	748 (697)	627 (542)	476 (445)	631 (449)	
21	644 (548)	630 (913)	507 (519)	582 (548)	
LFP, ln (ms^2)					
0	6.1 (1.1)	6.1 (0.9)	5.9 (1.0)	6.2 (0.6)	
21	6.1 (1.0)	5.8 (1.1)	5.8 (1.0)	6.0 (0.9)	*P* = 0.56
LFP/HFP					
0	3.21 (2.83)	2.39 (1.41)	3.32 (3.18)	2.33 (2.04)	
21	3.44 (4.89)	2.62 (1.88)	2.25 (1.82)	1.94 (1.47)	*P* = 0.24
α_1					
0	1.21 (0.19)	1.17 (0.18)	1.18 (0.20)	1.06 (0.27)	
21	1.11 (0.22)	1.17 (0.18)	1.11 (0.21)*	1.08 (0.25)	*P* = 0.28
$CI_{1\text{-}5}$					
0	8.55 (2.88)	8.23 (1.99)	8.20 (2.91)	9.45 (3.14)	
21	8.28 (2.25)	7.97 (3.00)	7.86 (1.96)	8.49 (2.14)	*P* = 0.72

(continues)

TABLE 5 (continues)

Women	Endurance n = 26	Strength n = 26	Combined n = 22	Control n = 17	Time x Group
HR, min^{-1}					
0	63 (6)	62 (7)	62 (7)	65 (6)	
21	60 (7)	61 (7)	62 (6)	62 (6)	$P = 0.36$
SDNN, ms					
0	50.2 (15.5)	53.4 (22.9)	52.3 (15.2)	45.1 (16.5)	
21	49.3 (20.7)	57.0 (23.3)	49.2 (15.2)	47.0 (19.5)	$P = 0.68$
HFP (ms^2)					
0	564 (528)	844 (1308)	635 (521)	431 (618)	
21	616 (678)	1115 (1631)	659 (910)	563 (684)	$P = 0.55$
HFP, ln (ms^2)					
0	6.06 (0.71)	6.12 (1.06)	6.10 (0.92)	5.54 (1.01)	
21	5.99 (0.93)	6.24 (1.27)	5.97 (0.98)	5.83 (1.03)	$P = 0.31$
LFP (ms^2)					
0	601 (410)	709 (675)	625 (483)	544 (530)	
21	637 (604)	716 (724)	510 (304)	654 (899)	$P = 0.71$
LFP, ln (ms^2)					
0	6.16 (0.72)	6.11 (1.05)	6.24 (0.62)	5.95 (0.86)	
21	6.01 (1.01)	6.16 (0.96)	6.03 (0.68)	5.97 (1.01)	$P = 0.70$
LFP/HFP					
0	1.35 (0.90)	1.58 (2.54)	1.49 (1.16)	1.70 (0.80)	
21	1.29 (1.06)	1.27 (1.24)	1.48 (1.24)	1.67 (1.72)	$P = 0.89$
CI_{1-5}					
0	8.34 (1.60)	8.52 (2.35)	8.82 (1.70)	7.86 (1.96)	
21	8.40 (2.89)	9.01 (3.21)	8.17 (1.88)	8.41 (2.58)	$P = 0.30$

HR, heart rate; SDNN, standard deviation of NN intervals; HFP, high frequency power; LFP, low frequency power; LFP/HFP, ratio between LFP and HFP; α_1, short-term fractal scaling exponent; CI_{1-5}, complexity index of multiscale entropy analysis using the scales from 1 to 5. Significant difference compared to baseline at * $P < 0.05$.

5.4.2 Heart rate dynamics during exercise

In men, submaximal HR decreased significantly after endurance and combined training at moderate and higher exercise intensities from 90 W onwards, but not at 50 and 70 W (Fig. 13). The time and group interaction was also significant at 90 – 150 W but not at 50 or 70 W. The results in women were alike in that the time and group interaction was only significant from 90 W onwards (Fig. 14). The decrease in submaximal HR was, however, significant at all exercise intensities from 50 to 130 W in E and SE. The training-induced changes in submaximal HR seemed to mainly occur during the first 10 weeks of training without further decreases during the latter half of the training period. In both men and women the S group also showed significant within-group changes at moderate to high intensities which were not, however, different from those of the C group.

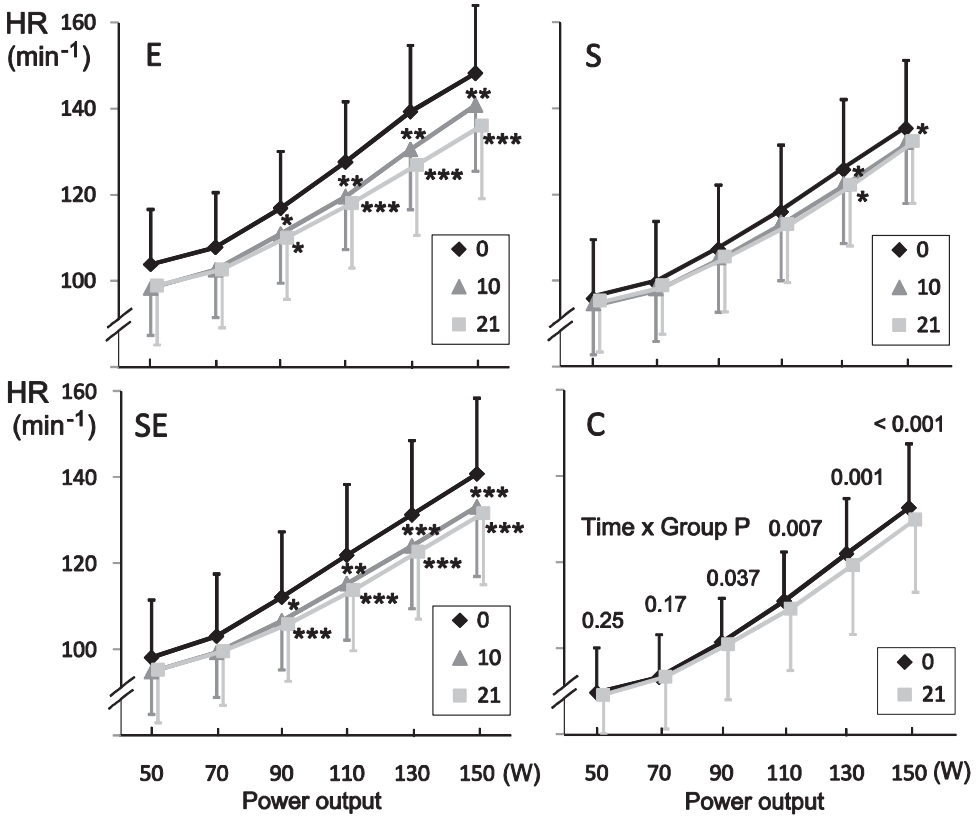

FIGURE 13 Heart rate (HR) at exercise intensities of 50, 70,…, 150 W before (0), and after 10 and 21 weeks of training in the endurance (E), strength (S), combined endurance and strength training (SE), and control group (C) in men. Significant difference compared to baseline at * ($P < 0.05$), ** $P < 0.01$, *** $P < 0.001$.

In agreement with the submaximal HR data, HR measured during steady state exercise at 50 W did not significantly decrease in any of the groups in men (Table 6), whereas the female E group showed a significant decrease (Fig. 15). Accordingly, in the E group SDNN and HFP significantly increased in women (Table 6). In men, SDNN and HFP did not change during the training period in any of the groups. The α_1 value, however, increased in the male SE group between training weeks 10 and 21, but did not show any changes in the other groups. Complexity index $CI_{1\text{-}2}$ measured during steady state exercise increased, however, in both men and women. Even though the absolute baseline value of $CI_{1\text{-}2}$ was higher ($P = 0.004$) in men than in women, there were no difference between genders in the absolute change of $CI_{1\text{-}2}$, and thus, the genders were also analyzed together. The analysis showed a significant increase in HR complexity only in the E group with a significant time and group interaction (Fig. 16).

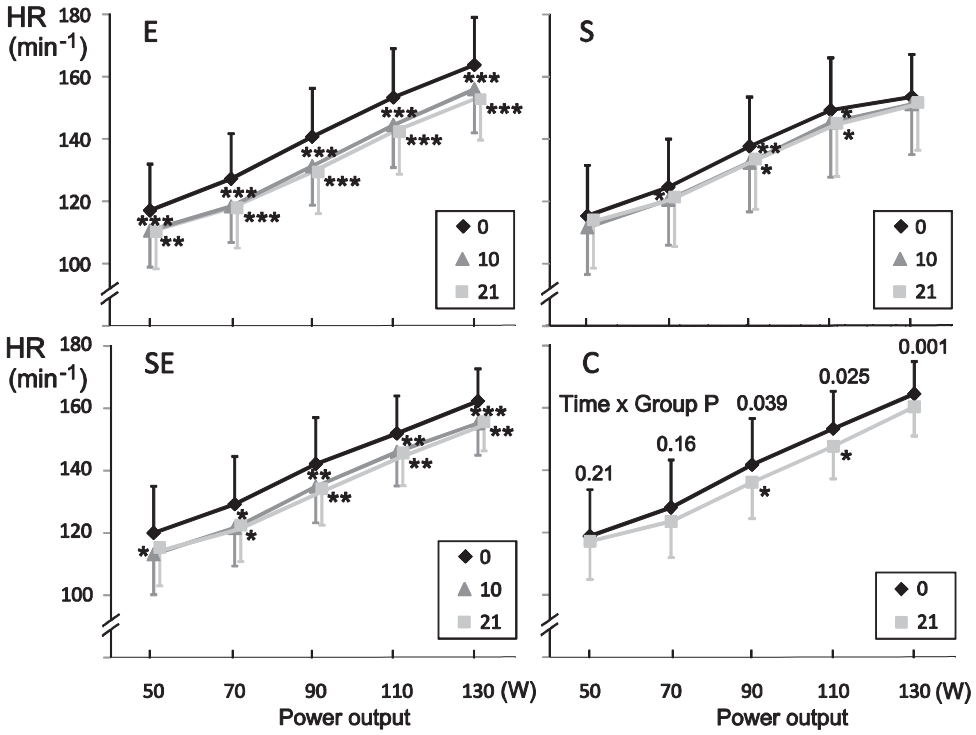

FIGURE 14 Heart rate (HR) at exercise intensities of 50, 70,…, 130 W before (0), and after 10 and 21 weeks of training in the endurance (E), strength (S), combined endurance and strength training (SE), and control group (C) in women. Significant difference compared to baseline at * ($P < 0.05$), ** $P < 0.01$, *** $P < 0.001$.

5.4.3 Association between heart rate dynamics and cardiorespiratory endurance

Resting HR correlated significantly with VO_{2peak} at baseline in men (r = –0.34, P = 0.001) but not in women (r = –0.20, P = 0.066). In addition, the baseline values of SDNN (r = 0.23, P = 0.028) and α_1 (r = –0.28, P = 0.008) measured during supine rest correlated significantly with baseline VO_{2peak} in men. Complexity index $CI_{1\text{-}5}$ correlated significantly with baseline VO_{2peak} in both men (r = 0.31, P = 0.003) and women (r = 0.27, P = 0.013). Correlations during steady state exercise are not reported due to the same absolute thus different relative exercise intensity used in all subjects with varying levels of VO_{2peak}, therefore resulting in an inevitable relationship.

Overall, correlations between training-induced changes in HR dynamics and in VO_{2peak} were weak. The only significant correlation was found between the change in α_1 and ΔVO_{2peak} in the male SE group (r = –0.40, P = 0.036).

TABLE 6 Selected HRV indices recorded during submaximal exercise at baseline (0) and after 10 and 21 weeks of endurance, strength or combined endurance and strength training and 21 weeks after a control period.

Men	Endurance (n = 22)	Strength (n = 25)	Combined (n = 28)	Control (n = 15)	Time x Group	Time
HR, min^{-1}						
0	101 (12)	93 (12)	95 (11)	87 (10)		
21	96 (12)	93 (10)	92 (9)	88 (12)	$P = 0.22$	$P = 0.065$
SDNN, ms						
0	27.1 (13.0)	27.1 (8.1)	28.8 (9.6)	34.9 (12.1)		
21	26.9 (9.3)	27.4 (10.7)	28.3 (8.4)	32.7 (8.2)	$P = 0.90$	$P = 0.55$
HFP, ms^2						
0	37.1 (65.2)	29.3 (30.3)	54.6 (82.2)	89.9 (110.2)		
21	37.7 (33.5)	31.5 (35.6)	44.8 (55.4)	72.7 (111.8)		
HFP, ln (ms^2)						
0	2.60 (1.46)	2.86 (1.16)	3.24 (1.23)	3.88 (1.27)		
21	3.03 (1.32)	2.92 (1.09)	3.27 (1.06)	3.73 (1.00)	$P = 0.31$	$P = 0.39$
α_1						
0	1.49 (0.20)	1.45 (0.21)	1.39 (0.19)	1.41 (0.25)		
21	1.42 (0.23)	1.47 (0.21)	1.50 (0.16) *	1.37 (0.20)	$P = 0.010$	$P = 0.82$
CI_{1-2}						
0	1.17 (0.75)	1.41 (0.67)	1.54 (0.88)	2.06 (0.68)		
21	1.47 (0.67) *	1.35 (0.70)	1.53 (0.82)	1.92 (0.74)	$P = 0.070$	$P = 0.709$
Women	**Endurance (n = 26)**	**Strength (n = 26)**	**Combined (n = 21)**	**Control (n = 17)**	**Time x Group**	**Time**
HR, min^{-1}						
0	116 (14)	114 (15)	117 (15)	117 (12)		
21	109 (11) ***	112 (13)	113 (11)	115 (12)	$P = 0.28$	$P = 0.001$
SDNN, ms						
0	14.4 (5.4)	16.3 (8.7)	17.9 (7.2)	16.0 (6.9)		
21	19.0 (8.4) **	17.7 (7.8)	18.6 (6.8)	15.4 (6.3)	$P = 0.070$	$P = 0.037$
HFP, ms^2						
0	31.4 (56.3)	42.9 (82.4)	29.9 (35.7)	31.6 (33.1)		
21	46.4 (56.7)	41.7 (72.8)	27.5 (27.1)	38.1 (82.2)		
HFP, ln (ms^2)						
0	2.56 (1.37)	2.61 (1.57)	2.57 (1.47)	2.69 (1.46)		
21	3.21 (1.20) ***	2.69 (1.50)	2.80 (1.11)	2.58 (1.49)	$P = 0.18$	$P = 0.10$
CI_{1-2}						
0	0.86 (0.62)	0.94 (0.75)	0.87 (0.61)	0.89 (0.69)		
21	1.18 (0.62) **	0.96 (0.71)	0.98 (0.59)	0.86 (0.64)	$P = 0.16$	$P = 0.091$

HR, heart rate; SDNN, standard deviation of NN intervals; HFP, high frequency power; α_1, short-term fractal scaling exponent; CI_{1-2}, complexity index of multiscale entropy analysis using the scales from 1 to 2. Significant difference compared to baseline at * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$.

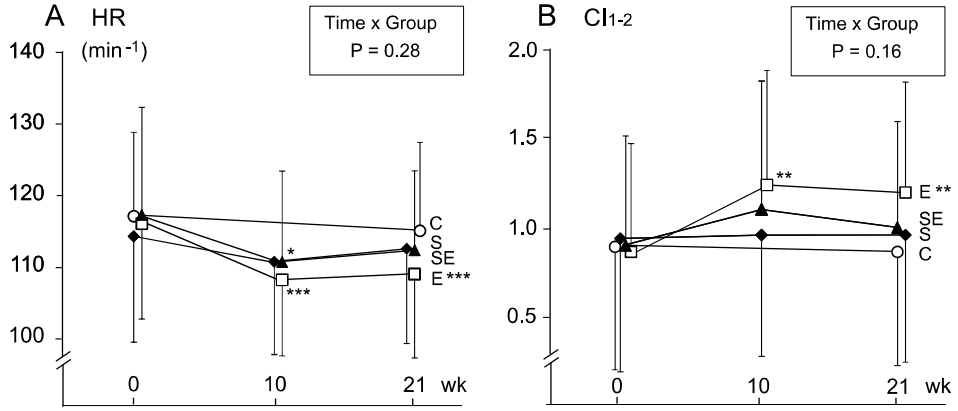

FIGURE 15 Heart rate (HR, A) and complexity index of multiscale entropy analysis ($CI_{1\text{-}2}$, B) measured during steady state exercise at baseline (0) and after 10 and 21 weeks of endurance (E), strength (S) or combined endurance and strength training (SE) and in the control group (C) in women. Significant difference compared to baseline at * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$.

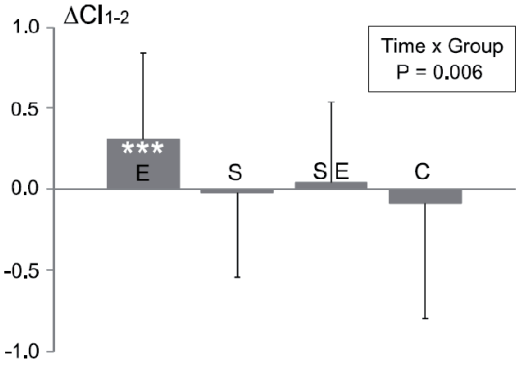

FIGURE 16 The absolute change (from the baseline to 21 weeks) in the complexity index of multiscale entropy analysis ($CI_{1\text{-}2}$) measured during steady state exercise in the endurance (E), strength (S), combined endurance and strength training (SE), and the control group (C) The data are from the compound groups of men and women. ***significant difference ($P < 0.001$) compared to baseline.

6 DISCUSSION

This thesis evaluated the effects of combined endurance and strength training compared with endurance or strength training alone in previously untrained 40–67 year old men and women. The progressive training program was performed twice a week in the endurance training group and twice a week in the strength training group. The combined endurance and strength training group performed identical endurance and strength training programs on alternate days with a total of four weekly training sessions. The control group were advised to continue their physical activities as before.

The specific aims of the present investigation were, firstly, to examine the possible interference effects of combining endurance and strength training on muscular strength, skeletal muscle hypertrophy and cardiorespiratory endurance during a prolonged 21-week training program. Secondly, individual differences in the training response to endurance and strength training were examined when endurance and strength training programs were performed separately or concurrently. Thirdly, the effects of the present training programs on heart rate dynamics were investigated at rest and during exercise as indices of cardiac autonomic adaptations to provide additional information about the effects of combined endurance and strength training on cardiac health.

6.1 Compatibility and training mode-specificity of strength and endurance training adaptations

6.1.1 Improvements in cardiorespiratory endurance

Endurance training with only two training sessions per week led to specific improvements in cardiorespiratory endurance in both the endurance training group and the combined endurance and strength training group. Changes in VO_{2peak} and endurance performance measured as maximal aerobic power output (P_{max}) during incremental cycling were similar in the E and SE groups, suggesting that strength training combined with endurance training for a total of

four training sessions per week did not compromise the endurance-training induced improvements in cardiorespiratory endurance. While the S group did not significantly improve VO_{2peak}, P_{max} increased significantly with strength training in both men and women, although the increase was smaller than with endurance or combined training. Combining endurance and strength training, however, did not further improve endurance performance compared to endurance training only.

Although VO_{2peak} did not show a significant mean increase in the strength training group, approximately one fourth of the subjects in S were able to improve their VO_{2peak}. Previous studies have also shown some improvements in endurance performance measured as time to exhaustion during submaximal cycling after strength training (Marcinik et al. 1991). The increase in endurance performance with strength training seems to occur independently of changes in VO_{2peak} (Marcinik et al. 1991). On the other hand, some subjects may also respond to strength training by increasing VO_{2peak} (Hautala et al. 2006). It could be postulated that improvements in both endurance performance and VO_{2peak} result from increased maximal leg strength, especially when cycling is used as the exercise mode (Marcinik et al. 1991; Hautala et al. 2006). This notion was not, however, supported by the present correlation between Δ1RM of leg press and ΔP_{max} in the strength training group ($r = 0.090$, $P = 0.54$). The mechanisms through which cardiorespiratory endurance may be improved with strength training are thus unclear. Strength training has been shown to lead to peripheral changes that improve the capacity of muscle to use oxygen, such as increased capillarization (Frontera et al. 1990; Hepple et al. 1997) and conversion of muscle fibres to more fatigue-resistant types (Hikida et al. 2000). A shift from muscle fibre type IIAX and IIX (ns.) towards type IIA was also observed after the present strength training program.

Extensive comparisons between endurance and strength trained athletes show that endurance and strength training induce different structural adaptations to the heart. High left ventricular end diastolic diameter in endurance athletes seems to explain at least part of the greater maximal exercise workload of endurance athletes compared to strength athletes. Strength training-induced structural changes to the heart such as increased wall thickness seem to favour higher peak systolic blood pressure during exercise (D'Andrea et al. 2002).

6.1.2 Development of muscular strength

The present results suggest that combined endurance and strength training with a total of four training sessions per week leads to significant mean gains in maximal strength that are as large as with strength training alone. The diversity of possible training programs comprised of training design variables such as volume, frequency, duration and intensity raises some controversy in the literature about the universal nature of interference effects (Leveritt et al. 1999). The similar development of muscular strength in the S and SE groups in the present study supports previous observations in younger adults (Sale et al. 1990b; McCarthy et al. 2002; Häkkinen et al. 2003), suggesting that with a moderate

volume or frequency, concurrent endurance training may not counteract strength gains. Therefore, the present results, together with a previous investigation with three weekly strength and three endurance training sessions for 12 weeks in somewhat older subjects (Haykowsky et al. 2005) suggest that combined training can also lead to similar adaptations in muscular strength than strength training alone in older adults. The present study additionally showed that combined training on alternate days can be continued even for 21 weeks without compromising strength gains in the lower or upper body.

Two sessions per week of strength training alone led to predictable specific improvements in strength performance. Men showed significantly larger training-induced relative changes in leg press 1RM than women. The relative changes were, however, similar between genders in leg press MVC. In absolute terms, strength gains have generally been shown to be greater in men than in women (Cureton et al. 1988; Tracy et al. 1999). Several studies show, however, that both men and women may increase their maximal strength similarly in relative terms (Cureton et al. 1988; Abe et al. 2000; Lemmer et al. 2000; Lemmer et al. 2001). Women may actually have larger relative gains than men, which could be explained by the smaller initial strength levels of women (Hubal et al. 2005). The potential for muscle hypertrophy may, however, be greater in men (Ivey et al. 2000; Bamman et al. 2003).

In the present training program, approximately 20 % of the volume of strength training for the lower extremities was performed explosively. Accordingly, both S and SE training led to improvements in explosive strength performance measured as maximal concentric power during the leg press. A non-significant decrease in maximal concentric power was observed in E between training weeks 10 and 21, although it is possible that a longer endurance training period would have induced a significant decrease.

Endurance training led to a statistically significant but rather small increase in maximal strength of the lower extremities in both men and women, which could be due to the potential of high intensity cycling to also produce some stimulus for maximal strength development in subjects without a previous history of systematic training. Increases in maximal strength were demonstrated in approximately one third of the present subjects in the E group. Endurance training adaptations in muscular strength in older adults may occur through muscle hypertrophy (Coggan et al. 1992; Harber et al. 2009) and remodelling of contractile properties of the muscle fibres (Harber et al. 2009). In the present male subjects significant muscle hypertrophy was not, however, observed after endurance training in either the CSA of muscle fibres or lean mass of legs measured with DXA. The compound thickness of VL and VI muscles increased significantly, however, during the first 10 weeks of training. Muscle activation was not significantly increased in the VL or VM muscle. In women, both lean mass of the legs, CSA of type IIA muscle fibres and VL+VI thickness increased. Thus, decisive conclusions about the mechanisms leading to increased muscle strength cannot be made based on the present study. It can be speculated that sensitivity or statistical power may not have been sufficient

to detect small changes in fibre hypertrophy in men or that muscle activation may have been altered in muscles not examined in the present study, such as antagonists.

In both the strength and the combined training groups in men, the improvements in strength were associated with enhanced muscle activation. It has been suggested that when interference is observed in muscle hypertrophy, strength gains may be maintained by neural mechanisms (Leveritt et al. 1999). This seems not to be the case in the present study, however, because at training week 10, the S group showed a larger increase in muscle activation in VL and VM than the SE group. However, significant differences were not found between groups after 21 weeks of training. As we have no data on muscle hypertrophy at training week 10, it is not appropriate to make comparisons between muscular and neural adaptations. EMG was recorded from the VL and VM muscles during isometric leg extension. It should be noted therefore that the recording of EMG in only two leg extensor muscles may not be sensitive enough to detect changes in neural activity of both agonistic, synergistic and especially antagonistic muscles in a multi-joint exercise.

6.1.3 Muscle hypertrophy

Interestingly, no increases in the CSA of any of the fibre types were observed with the present prolonged strength training program combined with two weekly sessions of endurance training, including cycling with high intensity. Despite the similar development of strength in the S and SE groups, the CSA of type II muscle fibres only increased with strength training in men and women and with endurance training in women, but not with combined training in either gender. This observation implies that muscle hypertrophy might be compromised when combining the two different training modes, despite the similar strength gains in the S and SE groups. Even though the difference between the SE and S groups was not significant, the large interindividual variation in SE suggests diverse adaptations in muscle hypertrophy, compared with a systematic increase in S (Fig. 11). It should be noted that interference in hypertrophy was observed in approximately one half of the subjects in SE, whereas the other half were able to increase their fibre size. CSA of type II fibres increased in S by 26 % and 20 % in men and women, respectively, after 21 weeks of progressive strength training two times a week. Similar or somewhat smaller gains have been reported previously in older men with two to three strength training sessions per week (Häkkinen et al. 2001; Martel et al. 2006).

The interference in muscle hypertrophy was also partly supported by the present data on lean mass of the legs measured by DXA and the pooled thickness of VL and VI muscles measured by ultrasound. In men, the lean mass of the legs only significantly increased in S, whereas in women lean leg mass increased in all training groups. On the other hand, in both men and women the thickness of VL+VI increased significantly in all training groups during the first 10 weeks of training but during the latter 11 weeks of training, only S showed a further minor increase in both men and women. The magnitude of the increase

in muscle thickness was, however, not different between the S and SE groups. One factor that may also go against the interference in muscle hypertrophy with combined training is the significant increase in the CSA of type IIA muscle fibres observed in the female E group. Thus it seems that endurance training had similar rather than divergent effects on muscle growth. It is possible that in the present previously untrained middle-aged women the high intensity cycling also produced a stimulus for muscle growth in the fast twitch fatigue-resistant IIA fibres. Interestingly, in women a significant increase in the CSA of IIA fibres was observed in S and E, but not in the SE group.

Combined training has previously been reported to interfere with muscle hypertrophy in young adults, although mainly with regard to type I fibres (Kraemer et al. 1995; Bell et al. 2000; McCarthy et al. 2002; Putman et al. 2004). In contrast, hypertrophy of muscle fibres has also been shown to be similar after combined training compared to strength training alone (Häkkinen et al. 2003). In the study by Kraemer et al. (1995), strength training alone increased the CSA of type I fibres and endurance training alone decreased CSA, whereas combined training induced no change, thus demonstrating an intermediate response. Three other studies have observed a significant change in CSA of type I fibres with strength training, no change with endurance training and no change with combined training (Bell et al. 2000; McCarthy et al. 2002; Putman et al. 2004). In older adults, however, type I fibre CSA does not seem to increase in response to strength training as much as in young adults (Charette et al. 1991; Martel et al. 2006; Kryger & Andersen 2007).

Besides the older age of the subjects, the current strength training program could explain why the interference effect was found in the type II fibres, without significant increases in the type I fibres. In the present study, approximately 20% of the volume of strength training for the leg extensors was performed using a typical explosive strength training protocol with lower loads and a short duration of each repetition. In addition, during the first seven weeks, strength training was performed with relatively low loads that are not likely to produce major hypertrophy. The incompatibility of endurance and strength training in type II fibres observed in the present study may be especially important in older adults, because type II fibres have a greater potential for muscle growth and a higher force per unit area than type I fibres (Harridge 2007).

6.1.4 Theoretical mechanisms of interference in muscle hypertrophy

In contrast to several earlier studies, endurance and strength training were performed on alternate days in the present study. Thus, the acute hypothesis suggesting that fatigue caused by the preceding endurance training session may decrease the quality of the following strength training session is unlikely to apply in this case. Sale et al. (1990a), for example, reported larger strength gains when strength and endurance training were performed on alternate days compared to performing both on the same day. Studies applying the design of performing endurance training first have also not supported the acute hypothesis (Collins & Snow 1993; Leveritt et al. 2003).

The chronic interference in muscle hypertrophy with combined endurance and strength training may be related to the different signalling pathways of these two training modes (Baar 2006; Nader 2006). Furthermore, aerobic exercise may induce oxidative stress due to the duration-dependent nature of free radical production leading to a theoretical risk of cellular damage (Pattwell & Jackson 2004; Fisher-Wellman & Bloomer 2009). Combined training may also lead to a catabolic state (Kraemer et al. 1995; Bell et al. 1997), which may interfere with muscle hypertrophy.

Protein synthesis induced by hypertrophic strength training may also be impaired due to hypoxia in the muscle caused by endurance training which includes high intensity intervals (Docherty & Sporer 2000). Thus, the high intensity intervals applied in the present endurance training program together with the hypertrophic strength training performed especially during the second strength training cycle (weeks 8–15) may have accounted for the interference in muscle hypertrophy. Not all studies have, however, supported the interference model based on intensity of endurance and strength training. Continuous endurance training combined with strength training has also been shown to impair muscle hypertrophy (McCarthy et al. 2002). In addition, determination of training intensity in relation to whole body VO_{2peak} may not closely reflect the stimulus experienced by the muscles (McPhee et al. 2009). Furthermore, older subjects may rely more on peripheral mechanisms in terms of endurance training adaptations (Seals et al. 1984; McGuire et al. 2001a). Thus, the model highlighting the importance of training intensity on the interference phenomenon requires further investigation.

Similar to many studies examining the interference phenomenon in combined endurance and strength training, the training volume was considerably larger in the combined training group compared to the groups that performed only endurance or strength training. Thus, the possibility of overreaching as a mechanism behind impaired muscle hypertrophy cannot be precluded with the present study design. Interference has, however, been also observed with very small training volumes (Dudley & Djamil 1985). In the present study, the training volume was also rather low including only two endurance and two strength training sessions per week.

It seems that the interference effect cannot be directly generalised based on training design variables, such as volume, frequency or duration of the training period alone. The reasons leading to impairment in endurance or strength development during combined training may involve a complex interplay of numerous mechanisms of training adaptation. Comparison of the studies utilizing combined endurance and strength training is difficult due to the range of different strength measurements and training protocols used. In addition to the training design variables, nutritional factors and the background of the participants, including training history and genetic predisposition, may contribute to the interference effect. Methodological problems in the comparison between studies may also be caused by insufficient statistical power (Leveritt et al. 2003).

6.2 Individual adaptations to combined endurance and strength training

Training responses to the present combined endurance and strength training varied from –8 to 42 % in VO_{2peak} and from –12 to 87 % in MVC. This finding in ageing adults further confirms the wide range in individual training adaptations that has been previously reported in younger subjects separately with endurance (Lortie et al. 1984; Bouchard et al. 1999; Bouchard & Rankinen 2001) or strength training (Newton et al. 2002; Hubal et al. 2005). The range in training responses seems to be similar in the present E and SE groups in terms of VO_{2peak} as well as in S and SE in terms of MVC. At the group level, no interference due to the present combined training program was observed in the development of VO_{2peak} or MVC. Individual values revealed, however, that only some of the men and women showed large increases in both VO_{2peak} and MVC, since only a few subjects fell into the upper tertile in both ΔVO_{2peak} and ΔMVC. An individual's ability to improve both characteristics with combined endurance and strength training may thus be limited, which may not have been observed from the comparisons between the group mean values. Thus, the interference effect of combined training seems to be complex including an individual optimum for the combination of training design variables as well as proportions of endurance and strength training to enhance the training outcomes in both VO_{2peak} and MVC.

As the examination of possible determinants of trainability was beyond the scope of the present study, the possibility to discuss the factors contributing to the training response is limited. Previous studies have shown that age only has minor if any effects on the relative endurance training response (Kohrt et al. 1991; Bouchard & Rankinen 2001; Hautala et al. 2006). In addition, VO_{2peak} at baseline may be an insignificant (Kohrt et al. 1991) or a small contributor (Hautala et al. 2006) to the training response. In the present study the gains in VO_{2peak} were correlated with baseline values in all training groups in women but not in men. A similar kind of trend has been found previously in a study investigating both men and women, but the correlations were not significant in either men ($r = 0.04$) or women ($r = –0.27$) (Kohrt et al. 1991). The effect of age on ΔVO_{2peak} seemed to be minor in this study, and was only significant in the SE group. Age, gender and baseline MVC were not correlated with ΔMVC in the present subjects. Based on earlier studies, genetic factors may explain as much as half of the interindividual variation in the training response after both endurance (Bouchard et al. 1999) and strength training (Thibault et al. 1986). However, a question remains regarding how this information can be used to effectively individualise endurance and strength training programs.

It must be noted that even if only a few of the subjects showed large increases in both VO_{2peak} and MVC, it cannot be concluded with the present study design whether these subjects would have larger gains when performing endurance or strength training only. Furthermore, the range in the training re-

sponse in the SE group was similar than in the E and S groups, suggesting no interference in the development of muscular strength or cardiorespiratory endurance. Thus, the observation about the ability to considerably improve either strength or endurance may be due to individuality in the responsiveness to different modes of training rather than the interference effect related to concurrent strength and endurance training.

6.3 Cardiac autonomic adaptations to combined endurance and strength training

The present training programs induced only minor positive changes in the cardiac autonomic function evaluated by indices of heart rate variability (HRV) in the healthy middle-aged men and women. In women only endurance training seemed to increase HRV whereas after strength training HRV was not altered. Endurance training combined with strength training also seemed to be ineffective at producing any significant changes in women. Even though the changes in E were not significantly different from those of SE, the finding suggests that simultaneous training for strength may affect the endurance training adaptations. It cannot be concluded, however, whether strength training as such or the higher training volume in the combined group compared to the endurance training group led to these different adaptations. Therefore, it is possible that some subjects in the SE group became overreached towards the end of the training period due to increased volume of high intensity training. This hypothesis is supported by earlier findings that showed significant improvements in HR complexity (Heffernan et al. 2007) and fractal scaling properties of HR (Heffernan et al. 2008) with strength training, thus supporting the notion of positive effects of strength training on cardiac autonomic function. In the present study, however, the only significant HR related change after strength training was a slight decrease in submaximal HR.

The above-described finding from women was only partly supported by the data from men. HR complexity determined during steady state exercise was only increased in the endurance group also in men, and merging the data from the male and female groups further supported this finding. A significant decrease in the fractal scaling exponent α_1 towards fractal-like behaviour, however, was only observed in the male SE group ($P = 0.039$), although the decrease observed in the E group also tended to be significant ($P = 0.061$). In contrast, during steady state exercise α_1 increased in the SE group referring to a negative change towards Brownian noise, i.e. a smoother signal. Physiologically the fractal scaling exponent α_1 is determined by the interaction of sympathetic and vagal modulation (Tulppo et al. 2005). During exercise α_1 is increased (Tulppo et al. 2001) which was also observed in the present study.

The significant endurance training-induced increase in the HFP of heart rate variability in women during exercise suggests that the decrease in sub-

maximal HR was mediated, at least in part, by enhanced vagal modulation of HR. The decrease in HR may have also resulted from decreased sympathetic activity. However, without direct measurement of the function of the sympathetic nervous system its role cannot be exactly determined, since most of the HRV indices include both vagal and sympathetic components but none of the HRV indices solely reflect sympathetic activity.

Overall, submaximal HR decreased especially in the endurance training group and combined endurance and strength training group. The changes seemed to be more evident at moderate and higher exercise intensities than at low intensities, especially in men. Similarly to the submaximal HR data, HR measured during steady state exercise at 50 W did not significantly decrease in men whereas the female E group showed significant decreases. The absolute exercise intensities were similar in the maximal cycling test for men and women but because of the relative nature of the training intensity prescription, the absolute training intensities were significantly lower in women. The principle of training specificity may, therefore, explain why women showed significant endurance training-induced changes also at lower exercise intensities whereas the men did not. The problem with the selection of an absolute exercise intensity of 50 W was that it was relatively high for some subjects but quite low for others, as a consequence of the individual differences in cardiorespiratory endurance at baseline.

The training-induced changes in HR dynamics with the present short-term recordings were mainly observed during exercise. Martinmäki et al. (2008) also reported significant effects of endurance training on HRV when measured during exercise but not when measured during supine rest. The simultaneous recordings of respiration revealed that the increased HRV during incremental exercise was most likely due to alterations in cardiac autonomic function, and not in tidal volume or respiration frequency (Martinmäki et al. 2008). Some of the previous studies have reported fairly poor reproducibility of short-term indices of HRV at rest during both paced and spontaneous breathing (Pitzalis et al. 1996; Sandercock et al. 2005) but improved reproducibility during physical exercise (Tulppo et al. 1998). In the present study, it was not possible to determine the reproducibility during exercise, since exercise HRV was recorded only once at baseline. At supine rest the lowest reproducibility was observed in the spectral measures, which could be due to the larger effect of outliers for the spectral measures than time domain measures even if the data are appropriately filtered. The alterations in autonomic function due to exercise may reduce the effect of unwanted confounding factors such as mental and nutritional state (Tulppo et al. 1998). Therefore, the measurement of HR dynamics during exercise may be especially applicable for detecting subtle training-induced effects with short-term recordings of RR intervals.

The association between HR dynamics and cardiorespiratory endurance was relatively weak in the present study. Previous studies have reported a significant association between cardiorespiratory endurance and HRV when comparing athletes and sedentary adults (Dixon et al. 1992; Verlinde et al. 2001; Au-

bert et al. 2003) but a rather negligible relationship in an average population (Melanson & Freedson 2001; Uusitalo et al. 2002; Bosquet et al. 2007). Cardiac vagal function measured during exercise, however, may be associated with cardiorespiratory fitness even if cardiac vagal function at rest could not separate subjects of different fitness levels (Tulppo et al. 1998). The present data also showed that overall the correlations between the training-induced changes in HR dynamics and VO_{2peak} were negligible. Thus, the rather weak associations between cardiorespiratory endurance and HRV further emphasise the importance of HR dynamics as independent measures of cardiovascular adaptations to physical training.

6.4 Methodological considerations and limitations

Experimental design and subject group

The present randomised controlled study design primarily aimed to compare the development of muscular strength, cardiorespiratory endurance and cardiac autonomic function in the combined strength and endurance training group compared to strength or endurance training alone. Therefore, the training mode-specific volume was similar in all training groups, resulting in four weekly training sessions in the combined group compared to two sessions in the endurance training and strength training groups. Therefore, it cannot be determined whether the combination of the two divergent training modes as such or the higher training volume in the combined group led to differences in the training adaptations. Even in the case of matched training frequency in all groups, however, it is problematic to make direct comparisons between the volume of endurance and strength training due to the training mode-specific construction of the prescription.

Four weekly training sessions in the combined training group was considered as a reasonable upper limit of the training volume for untrained ageing subjects over the present fully supervised training period of 21 weeks. Therefore, the training volume was rather low in the groups performing either endurance or strength training. The prolonged length of the supervised training intervention together with the older age of the present subjects compared to earlier studies are, however, important strengths of the present study. A limitation related to the prolonged training period is a possible seasonal variation in physiological variables and physical activity. The intervention was initiated in winter and completed in summer. The statistically significant but small increase observed in maximal strength in the control group may be explained, for example, by increased physical activity during spring and summer compared to that during winter time.

This study covers a wide age range of the middle-aged population (40-67 years old) that is still at a working age and starting to show notable age-related decreases in physical performance. Although most studies have shown that

relative training adaptations are similar in older compared to younger subjects, the broad age span might impact the responses to training at least in absolute terms. Significant correlations were not, however, found between age and the improvements in the variables examined suggesting that age was not a significant contributor to the present training adaptations within the subject group.

In the present study, men and women were mainly analyzed separately due to the gender-specific nature of physiological variables. Genders were also investigated in consecutive years and using partly different analysis methods in terms of HRV, which weaken the reliability of directly comparing the training adaptations between genders. Some suggestive differences between men and women were, however, observed that may be of interest in future studies examining optimal training prescriptions for men and women.

The present sample size was rather large compared to similar training studies and most likely large enough for performance outcome measures. It is possible, however, that the sample size was not large enough to show training-induced changes in HR dynamics due to the large interindividual variation in the HRV indices. The number of subjects for the muscle biopsies was considerably smaller than for the other measurements, which decreases the statistical power to detect changes in response to training. The subjects selected for muscle biopsies were randomly selected, however, and showed similar adaptations in physical performance compared to the total groups.

It should also be pointed out that the participants were volunteers from the Jyväskylä city region and thus, did not represent the average middle-aged population of the area. Furthermore, subjects with diabetes, or cardiovascular, musculoskeletal, or other problems preventing high intensity training were excluded. Therefore, the subjects were probably healthier, leaner and physically more fit than Finnish middle-aged people on average. Thus, the present results can be generalised to healthy, previously untrained ageing adults.

Measurements

The present subjects were examined before, in the middle of and after the intervention by diverse physiological measurements, and thus, reproducibility is essential for obtaining reliable results. Some but not all measurements were repeated twice at baseline allowing the evaluation of reproducibility of only part of the present measurements. Reproducibility of short-term recordings of HRV may, for example, be rather poor in some conditions. Overall, the present reproducibility values of HRV were fairly similar to those reported earlier. It was not possible to determine the reproducibility during exercise, however, since HRV during exercise was recorded only once at baseline. The effect of respiratory frequency on spectral measures of HRV should also be acknowledged. The breathing frequency was spontaneous in the present study in order to maintain a high comparability between measurements at rest and during exercise. However, based on earlier studies, the reproducibility of HRV indices may vary independently of the control of breathing.

Special attention was paid to standardising the measurements in terms of the time of day, and subject instructions, preparation and encouragement. There are, however, possible sources of error in the present physiological measurements. Dual-energy x-ray absorptiometry (DXA) is considered as a valid technique for assessment of lean tissue mass (Mazess et al. 1990). There are some methodological considerations in measuring muscle thickness by ultrasonography, even though the measurements were standardised. Ultrasonography may produce a larger error than, for example, magnetic resonance imaging (MRI) (Miyatani et al. 2002). In our laboratory, the ultrasonography method has been recently shown to accurately detect training-induced changes in muscle thickness compared to the MRI method (Ahtiainen et al. 2010).

Furthermore, the electrical activity of the muscle was recorded with surface EMG electrodes with careful skin preparation and electrode positioning. With surface EMG recordings the interpretations should be done with caution, since the central nervous system adaptations resulting in increased motor unit recruitment cannot be separated from the possible changes in the peripheral factors such as changes in the amplitude of a single fibre or the altered electrical conductivity around the muscle fibres and between the electrodes. Furthermore, several factors may affect the action potential of a muscle fibre, such as changes in fibre type (Gandevia 2001).

6.5 Perspectives

The most recent guidelines for exercise prescription by American College of Sports Medicine recommend three or more endurance training sessions per week with intensities equal to or above 60 % of maximal heart rate for older adults (Chodzko-Zajko et al. 2009) and two to three resistance training sessions per week with fatiguing or near fatiguing intensity (Ratamess et al. 2009). Combining endurance and strength training modalities may ideally also combine the training-specific positive effects on ageing muscle, cardiorespiratory endurance and cardiac autonomic function of middle-aged and older adults.

Based on previous studies in young subjects, the combination of these two different training modes may interfere with optimal neuromuscular adaptation, depending on the training design variables, such as intensity, frequency and duration. However, information from younger subjects may not be directly applicable to older subjects. The present results showed that combined strength and endurance training for 21 weeks in previously untrained 40–67 year old men and women may improve muscular strength to the same extent as strength training alone when both strength and endurance training modes are performed twice a week. Furthermore, combined endurance and strength training for a total of four sessions per week did not compromise the improvements in concentric power or aerobic capacity. However, the significant changes in muscle fibre size only found in the strength training group and not in the combined training group may indicate diminished muscle hypertrophy when strength

and endurance training are combined over a prolonged period. Furthermore, individual data showed that only approximately half of the subjects in the combined endurance and strength training group were able to simultaneously improve both endurance and strength. Therefore, when designing combined training programs for middle-aged and older adults to delay the age related loss of skeletal muscle mass, the possible interference of muscular adaptation, particularly in type II fibres, should be taken into account. Training prescription for optimal muscle hypertrophy may require more subtle individualisation or periodization.

Previous studies have shown that endurance training may have positive effects on cardiac autonomic function as determined by HR dynamics if the training intensity and volume are sufficient. Strength training has also been previously shown to produce some positive effects on nonlinear, but not on traditional linear indices of HRV. In the present study the training-induced changes in the healthy ageing adults were minor and mostly evident only when determined during exercise. It may be, therefore, that the present minor changes in HRV resulted from the rather low endurance training volume. Increasing the training volume by added strength training did not, however, produce significant gains at least in women. Even in men, HR complexity was only increased in the endurance training group. Based on the present study it cannot be concluded, however, whether the combination of these two different training modes or the larger training volume is responsible for the disparate cardiac autonomic adaptations.

7 MAIN FINDINGS AND CONCLUSIONS

Combined endurance and strength training with a total of four training sessions per week led to significant gains in maximal strength that were as large as with strength training alone. Furthermore, peak oxygen uptake increased with two weekly endurance training sessions, and the increases were similar when endurance training was performed simultaneously with strength training compared to those of endurance training alone. However, the CSA of type II muscle fibres only increased with strength training, but not with combined training. Large individual variation in VO_{2peak} and MVC responses to combined endurance and strength training was observed, which was similar to endurance or strength training alone, respectively. Moreover, the combined training group did not show a significant correlation between the individual changes in VO_{2peak} and MVC. This finding suggests that the same subjects were not systematically low or high responders to both endurance and strength training when these training modes were performed concurrently for a prolonged period. In terms of HR dynamics, endurance training led to the most prominent training-induced changes, whereas combining endurance training with strength training seemed to produce somewhat insignificant changes. Furthermore, the training-induced changes in HR dynamics with the present short-term recordings were mainly observed during exercise but not at rest.

The conclusions of the present study are as follows:

1) Previously untrained ageing men and women may be able to improve muscular strength by combining two weekly endurance and two weekly strength training sessions for a prolonged period to the same extent as with strength training alone.

2) Combined endurance and strength training seems to induce similar cardiorespiratory adaptations compared to endurance training. Even though strength training also led to small improvements in endurance performance, combined training did not produce any synergistic gains.

3) Muscle hypertrophy may be compromised by combined endurance and strength training compared to that of strength training alone, despite the similar strength gains.

4) A large individual variation in the training response was observed after combined endurance and strength training, which was similar to that of endurance or strength training only.

5) Examination of individual responses revealed that the apparent goal of combined endurance and strength training – increasing both cardiorespiratory endurance and muscular strength simultaneously – was only achieved by approximately half of the ageing subjects. The interference effect of combined endurance and strength training may thus be individually determined.

6) In contrast to the similar changes in cardiorespiratory endurance, combined endurance and strength training seems to produce cardiac autonomic adaptations measured by changes in heart rate dynamics that differ somewhat from the endurance training adaptations. It is not evident, however, whether the lack of significant changes in the combined training group results from strength training as such or the larger training volume of combined training in previously untrained ageing adults.

YHTEENVETO (FINNISH SUMMARY)

Yhdistetyn kestävyys- ja voimaharjoittelun vaikutukset hengitys- ja verenkiertoelimistön sekä hermo-lihasjärjestelmän toimintaan ja sydämen autonomiseen säätelyyn ikääntyvillä miehillä ja naisilla

Ikääntymisen aikaansaamat muutokset fyysisessä suorituskyvyssä ja sydämen toiminnassa etenevät yksilöllisellä nopeudella, johon vaikuttavat huomattavasti monet tekijät, kuten fyysinen aktiivisuus. Säännöllisellä liikuntaharjoittelulla voidaankin parantaa suorituskykyä ja terveyttä ikääntyessä, mutta myös harjoitteluvaikutuksissa on suuria yksilöllisiä eroja. Lisäksi erilaisilla harjoittelumuodoilla on hyvin erilaisia fysiologisia vaikutuksia, joten yhdistämällä kestävyys- ja voimaharjoittelu voitaisiin periaatteessa saavuttaa monipuolisimmat hyödyt. Aiemmat tutkimukset ovat kuitenkin osoittaneet, että kestävyyden ja voiman samanaikainen harjoittaminen saattaa joissakin tapauksissa huomattavasti heikentää näiden ominaisuuksien kehittymistä. Liikuntaharjoittelun optimaalista annos-vastesuhdetta ei siten tunneta riittävän tarkasti. Tämän väitöskirjatutkimuksen tarkoituksena oli selvittää yhdistetyn kestävyys- ja voimaharjoittelun vaikutuksia hengitys- ja verenkiertoelimistön sekä hermo-lihasjärjestelmän toimintaan ja sydämen autonomiseen säätelyyn 40–67-vuotiailla miehillä ja naisilla.

Tutkimukseen osallistui yhteensä 206 vapaaehtoista, joilla ei ollut säännöllistä liikuntaharjoittelutaustaa. Tutkimusjakson suoritti loppuun 100 miestä ja 96 naista, joiden keski-ikä oli 53 (keskihajonta 8) vuotta. Tutkimusmittaukset suoritettiin ennen 21 viikon harjoittelujaksoa, jakson puolivälissä sekä harjoittelujakson jälkeen. Tutkittavat satunnaistettiin neljään ryhmään: kestävyysharjoittelu-, voimaharjoittelu-, yhdistettyyn kestävyys- ja voimaharjoittelu- sekä kontrolliryhmään. Kestävyysryhmä ja voimaryhmä harjoittelivat kahdesti viikossa, ja yhdistelmäharjoitteluryhmä suoritti samat kestävyys- ja voimaharjoitteluohjelmat harjoitellen yhteensä neljästi viikossa. Kontrolliryhmää ohjeistettiin säilyttämään fyysinen aktiivisuutensa tutkimusta edeltävällä tasolla. Kestävyysharjoittelu suoritettiin ohjatusti polkupyöräegometrillä ja se sisälsi sekä matalaintensiteettistä harjoittelua että kovatehoisia intervalleja. Harjoitusten kesto ja intensiteetti nousivat asteittain, ja harjoittelun intensiteettiä valvottiin sykemittareiden avulla. Voimaharjoitteluohjelma sisälsi harjoitteita kaikille kehon päälihasryhmille. Kuormia nostettiin progressiivisesti harjoittelun edetessä ja samalla toistojen määrää vähennettiin.

Hengitys- ja verenkiertoelimistön suorituskyky (maksimaalinen hapenottokyky) parani merkitsevästi kahdesti viikossa toteutetulla kestävyysharjoittelulla ja maksimivoima parani voimaharjoittelulla. Harjoittelun aikaansaama muutos maksimaalisessa hapenottokyvyssä ja voimassa vaihteli kuitenkin huomattavasti yksilöiden välillä. Yhdistetyn voima- ja kestävyysharjoitteluryhmän ja voimaharjoitteluryhmän välillä ei havaittu tilastollisesti merkitsevää eroa maksimivoiman kehittymisessä. Lisäksi yhdistelmäharjoittelun vaikutukset maksimaaliseen hapenottokykyyn ja kestävyyssuorituskykyyn olivat sa-

manlaiset kuin pelkää kestävyyttä harjoiteltaessa. Yksilöllisiä harjoitteluvasteita tutkittaessa havaittiin kuitenkin, että vain noin puolet yhdistelmäharjoitteluryhmän miehistä ja naisista paransi sekä kestävyyttä että voimaa verrattuna kontrolliryhmän vastaaviin muutoksiin. Yhdistelmäharjoittelun aikaansaamien muutosten suuruus vaihteli yksilöllisesti –10:stä 40 prosenttiin maksimaalisessa hapenottokyvyssä ja jopa 80 prosenttiin maksimivoimassa. Lisäksi voimaharjoittelun aikaansaama lihassolujen kasvu näytti heikentyneen osalla miehistä ja naisista samanaikaisen kestävyysharjoittelun takia, koska vain pelkkä voimaharjoittelu johti merkitsevään lihassolujen kasvuun.

Sydämen autonomisessa säätelyssä, jota määritettiin sykevaihtelun avulla, havaittiin positiivisia muutoksia erityisesti kestävyysharjoittelun jälkeen, kun taas voimaharjoittelun tai yhdistelmäharjoittelun vaikutukset eivät pääosin olleet merkitseviä. Voimaharjoittelu sinänsä ei kuitenkaan välttämättä heikennä kestävyysharjoittelun positiivisia vaikutuksia sydämen autonomiseen säätelyyn, vaan yhdistelmäharjoittelun erilaiset harjoitteluvasteet saattavat johtua myös suuremmasta harjoittelumäärästä kestävyysharjoitteluryhmään verrattuna. Harjoittelun aikaansaamat muutokset sykevaihtelussa eivät olleet yhteydessä suorituskyvyssä tapahtuneisiin muutoksiin, mikä korostaa sykevaihtelun itsenäistä merkitystä sydämen ja verenkiertoelimistön harjoitteluvasteiden tarkastelussa.

Väitöskirjatutkimus tukee aiempia havaintoja suurista yksilöllisistä eroista kestävyys- ja voimaharjoitteluvasteissa, vaikka harjoittelu on tarkasti kontrolloitua ja yksilölliset erot lähtötasossa on huomioitu harjoitteluintensiteettiä määritettäessä. Samanlaista suurta yksilöllistä vaihtelua harjoitteluvasteessa havaittiin nyt myös yhdistetyn kestävyys- ja voimaharjoittelun jälkeen. Kestävyyden ja voiman yhtäaikainen harjoittaminen saattaakin yksilöllisesti parantaa vain joko kestävyys- tai hermo-lihasjärjestelmän ominaisuuksia. Lisäksi voimaharjoittelun aikaansaama lihaskasvu voi estyä, kun voimaharjoitteluun yhdistetään kestävyysharjoittelua, mikä on tärkeää huomioida erityisesti ikääntyvillä lihasmassan vähentyessä. Toisaalta kuitenkin erityisesti kestävyysharjoittelun avulla voidaan positiivisesti vaikuttaa sydämen autonomiseen säätelyyn. Harjoitteluohjelmien laatimisessa tulisi siten entistä enemmän painottaa tavoitteiden asettelua ja yksilöllisyyttä optimaalisten harjoitteluvaikutusten aikaansaamiseksi ikääntyvällä väestöllä.

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