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Institute of Sports Sciences and Physiotherapy, Faculty of Medicine, University of Tartu, Estonia

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TABLE OF CONTENTS

LIST OF ORIGINAL ARTICLES

The thesis is based on the following original publications referred to in the text by Roman numerals (I–III).

PAPER I

Jürgenson, J., Serg, M., Kampus, P., Kals, J., Muda, P., Zagura, M., Viru, M., Zilmer, M., Eha, J., Unt, E. (2019) The effect of pre-seasonal strength training on central hemodynamics and cardiac function in elite powerlifting athletes. *Research Reports in Clinical Cardiology, 10:33–41. doi: 10.2147/RRCC.S198590.*

PAPER II

Jürgenson, J., Serg, M., Kampus, P., Kals, J., Zagura, M., Zilmer, K., Zilmer, M., Eha, J., Unt, E. (2019) Oxidative stress parameters and its associations with arterial stiffness in competitive powerlifting athletes after 12-week supervised strength training. *Journal of Strength and Conditioning Research, 33(7):1816–1822. doi: 10.1519/JSC.0000000000003067.*

PAPER III

Jürgenson, J., Serg, M., Kampus, P., Kals, J., Zagura, M., Zilmer, K., Zilmer, M., Eha, J., Unt, E. (2021) Effect of half-marathon running on arterial stiffness and blood biomarkers in high-level and recreational male athletes. *Journal of Sports Science and Medicine (2021) 20:548–556. doi:* 10.52082/jssm.2021.548.

Author's contribution:

In papers I, II, the author had primary responsibility for study design, recruiting the participants, data collection, data analysis and writing the manuscripts.

In paper III, the author was collaboratively involved in data collection and was responsible for the data analysis and writing the manuscript.

LIST OF ABBREVATIONS

ADMA	asymmetric dimethylarginine
AIx	augmentation index
AS	arterial stiffness
bDBP	brachial diastolic blood pressure
BeP	bench press
BMI	body mass index
BP	blood pressure
bPP	brachial pulse pressure
bSBP	brachial systolic blood pressure
cDBP	central diastolic blood pressure
cfPWV	carotid-femoral pulse wave velocity
CK	creatine kinase
cPP	central pulse pressure
crPWV	carotid-radial pulse wave velocity
cSBP	central systolic blood pressure
CHOL	total cholesterol
CV	cardiovascular
DC	diene conjugates
ECG	electrocardiography
Hct	haematocrit
HDL-C	high-density lipoprotein cholesterol
HGB	haemoglobin
HMRS	half-marathon running study
HR	heart rate
hsCRP	high-sensitive C-reactive protein
JHS	jumping from a half squat
LDH	lactate dehydrogenase
LDL-C	low-density lipoprotein cholesterol
LV	left ventricle
$LVID_d$	left ventricular internal dimension in end-diastole
MAP	mean arterial pressure
NT-proBNP	N-terminal pro B-type natriuretic peptide
OSI	oxidative stress index
OxS	oxidative stress
PA	physical activity
PPA	pulse pressure amplification
PWT_d	posterior wall thickness in end-diastole
PWA	pulse wave analysis
PWV	pulse wave velocity
RBC	red blood cell count
RECOV	post-exercise recovery period
RER	respiratory exchange ratio

RM	repetition maximum
ROS	reactive oxygen species
RT	resistance training
sICAM	soluble intercellular adhesion molecule-1
SSTP	supervised strength training program
SWT_d	septal wall thickness in end-diastole
TAC	total antioxidant capacity
TASV	tricuspid annular systolic velocity
TDI	tissue Doppler imaging
TPX	total peroxide concentrations
WBC	white blood cell count
VO_2max	maximal oxygen consumption
VO_2peak	the highest level of oxygen uptake
2DE	two-dimensional echocardiography

1. INTRODUCTION

It is well known that regular physical activity and exercise training play a significant role in the prevention and treatment of cardiovascular (CV) disease (Gielen *et al.,* 2010; Kodama *et al.,* 2009; Lee *et al.,* 2003; Nystoriak and Bhatnagar, 2018; Thompson *et al.,* 2003). Numerous studies have demonstrated that regular exercise is associated with a better profile of traditional risk factors – overweight, hypertension, dyslipidemia, insulin resistance (Kodama *et al.,* 2007; Mora *et al.,* 2007; Pescatello *et al.,* 2004; Sigal *et al.,* 2007; Stehouver and Ferreira, 2006; Swift *et al.,* 2014; Tambalis *et al.,* 2009) as well as with better profile of novel risk factors – i.e. arterial stiffness, oxidative stress and inflammatory markers (Marchio *et al.,* 2019; Stehouver and Ferreira, 2006).

The pathogenesis of CV disease is multifactorial and therefore the importance of an integrated approach, including the measurement of vascular function, different metabolic markers (atherogenic, oxidative stress, inflammation markers etc.) (Koenig, 2007; Marchio *et al.,* 2019; Stehouver and Ferreira, 2006) is relevant. Repetitive exposure to haemodynamic stimuli during exercise can lead to antiatherogenic adaptations in vascular function and structure (Fiuza-Luces *et al.,* 2018; Gielen *et al.,* 2010; Green *et al.,* 2017; Higashi *et al.,* 1999; Lavie *et al.,* 2015; Nystoriak and Bhatnagar, 2018). Improvements in vascular function are mainly attributable to increased shear stress, which stimulates vasodilatation *via* the release of nitric oxide as well as a decrease of chronic inflammation and oxidative stress (Ashor *et al.,* 2014; Fiuza-Luces *et al.,* 2018).

Physical activity (PA) guidelines (Chaput *et al.,* 2020) recommend a minimum of 75 vigorous-intensity or 150 moderate-intensity minutes of aerobic activity per week for substantial health benefits and doing more than double this amount for achieving additional benefits. Furthermore, PA guidelines stress the importance of strength or resistance training as one component of activity, especially in the elderly people for diminishing the risk for osteoporosis and sarcopenia. However, epidemiological studies behind these recommendations are usually performed in the general population, where the frequency and other qualitative markers of exercise training are relatively moderate. The upper limit of a beneficial or harmful impact with more physical activity is still unclear. In their prospective cohort study, Arem and co-authors (2015) demonstrated additional benefits from increasing the PA level three to five times above the recommended PA minimum and no excess risk at 10 or more times as compared to the minimum.

Both strength training and aerobic training have effects on the CV system. These different exercise modalities result in distinct haemodynamic demands and, possibly, different patterns of adaptation. The popularity of resistance training in fitness centres and long-distance running events has increased during recent years and high training loads with high intensities may reach enormous levels. Endurance athletes often exercise more than 15–20 times the level of the currently recommended minimum of PA. Furthermore, the main knowledge of the beneficial effect of exercise on arterial stiffness and blood pressure mainly comes

from aerobic training studies, where the training loads and intensities are moderate (Laurent *et al.*, 2011; Parry-Williams and Sharma, 2020; Tanaka 2019). Available data show less consistent findings about the effect of vigorous exercise on the cardiovascular risk profile and further cardiac events in well-trained athletes (Burr *et al.,* 2012, 2014; D'Ascenzi *et al.*, 2019; Parry-Williams and Sharma, 2020; Peliccia *et al.*, 2017; Vlachopoulos *et al.,* 2010a). Some studies have revealed that high-level athletes, despite having more favourable overall CV disease risk profiles, may have an increased risk of CV disease (McCullough and Lavie, 2014; Schwartz *et al.,* 2014). The key issue of elevated cardiovascular risk may be related to poor cardiovascular adaptation and increased arterial stiffness of athletes. There are practically no data about the independent effect of strength training on AS in competitive level powerlifting athletes. In summary, the response of vasculature to different exercise modalities (endurance and strength) in well-trained athletes and related biomarkers is the main interest of the present study.

2. LITERATURE REVIEW

2.1. Exercise, blood pressure and cardiac remodelling

It is widely known, that exercise has a blood pressure (BP) lowering effect and this is more demonstrated in hypertensive individuals (Börjesson *et al.,* 2017; Moraes-Silva *et al.,* 2017; Pelliccia *et al*., 2020). The decrease of BP may be significant if there is a decrease of 4–10 mmHg according to available data (Kokkinos, 2014; Pescatello *et al.*, 2004). BP and hypertension prevalence studies in athletes vary considerably due to different measurement methods, cut-off values and confounding factors, but the type and intensity of exercise seem to play also a significant role (Berge *et al.,* 2015).

It has been shown that elevated BP is a common abnormal finding during the periodic health evaluation of athletes (Corrado *et al*., 2008). Some studies demonstrate that strength-trained athletes, i.e. weightlifters have a higher prevalence of hypertension as compared to endurance athletes (Berge *et al.*, 2015; Guo *et al.*, 2013). On the other hand, moderate resistance training has been shown to be effective for reducing blood pressure values in resting state and is thereby recommended in the guidelines for hypertensive individuals (Pelliccia *et al*., 2020; Williams *et al.,* 2018). In their meta-analysis, Fecchio *et al*. (2021) reveal that the main mechanism for a BP lowering effect in dynamic resistance training lies in the vascular function improvement.

Elevated BP is considered a risk factor for left ventricular (LV) hypertrophy with subsequent cardiac events, i.e. myocardial infarction or serious ventricular arrhythmia (Rodriguez *et al*., 2002). Chronic physical training may result in morphological and functional myocardial alterations, which is known as the "athlete's heart" (Lavie *et al.,* 2015; Mihl *et al.,* 2008). Highly trained athletes develop cardiac adaptations, such as enlarged left and right ventricular volumes, increased LV wall thickness and cardiac mass, and increased left atrial size (Lavie *et al.,* 2015; Mihl *et al.,* 2008). The process of this physiological remodelling is influenced by haemodynamic load, neurohumoral activation and additional factors, including endothelin, cytokines, nitric oxide and oxidative stress (Cohn *et al.,* 2000). In endurance training or competition, athletes sustain long intervals of high cardiac output, elevated heart rate (HR), high stroke volume and moderately increased mean arterial blood pressure (MAP) (Schaier *et al.,* 1992). Increased dynamic load leads to left ventricular dilatation and increased left ventricular mass (called eccentric hypertrophy). Strength training is more related with static exercise and in this case the stroke volume is less affected, but a marked elevation in systolic and diastolic pressure is prevalent. At the same time, the increase of cardiac output, HR and oxygen consumption is modest (Fisman *et al.,* 1997). The heart of a strength-trained athlete responds to large pressure overload with concentric left ventricular hypertrophy (Mihl *et al.,* 2008). Taking into account the relative numbers, the extent of LV hypertrophy is greatest in athletes participating in dynamic exercise, averaging 105 g/m^2, compared to

athletes training with high resistance or static exercise, averaging 90 g/m^2, and these data are significantly lower as compared to pathological hypertrophy (Longhurst *et al.,* 1997). However, the degree of cardiac hypertrophy may vary between athletes involved in the same training program and should not be considered an absolute concept. A meta-analysis on cardiac structure (Pluim *et al.,* 2000) showed no significant differences in LV mass between endurance-trained and strength-trained athletes, but strength-oriented athletes showed significantly higher values of mean relative wall thickness. At the same time, all the athletes' groups showed normal systolic and diastolic cardiac functions (Pluim *et al.,* 2000).

Elevated BP and chronic increase in afterload may lead to ventricular hypertrophy in athletes (Berge *et al.*, 2015; Lalande and Baldi, 2007; Volpe *et al*., 2012). However, this topic needs further discussion. Whether LV hypertrophy is a benign physiological adaptation to high BP or the beginning of pathological remodelling is not known (Berge *et al.*, 2015). The concern is related with the effect of vigorous and excessive long-term training on cardiovascular health. It is possible that the harmful effect on CV health is mediated through elevated BP. Data have shown an increased risk of atrial fibrillation in endurance-athletes, which is considered to be associated with repeated bouts of high BP, an acute adverse effect on cardiac right side chambers and subsequent myocardial fibrosis (La Gerche, 2016; O'Keefe *et al.,* 2012; Parry-Williams and Sharma, 2020). During the post-exercise period, the cardiac dimensions are usually restored, but repetitive stretching of these chambers may lead the development of chronic structural changes of the myocardium (O'Keefe *et al.,* 2012).

Thus, the abnormal rise in BP to exercise may carry an elevated CV disease risk. In most studies in athletes, brachial BP is analysed, but central BP is considered a more valid parameter for target organ damage and cardiovascular events than brachial BP (Kampus *et al.,* 2011; Roman *et al*., 2007). However, the clinical impact of exercise-related elevated BP in athletes is not very well-known.

2.2. Arterial stiffness and exercise

2.2.1. Arterial stiffness

Arterial stiffness (AS) is widely used for the evaluation of the severity and progression of cardiovascular disease. At the same time, AS is considered one of the earliest detectable measures of the adverse functional changes of the vasculature (McEniery *et al*., 2005). In addition, AS is associated with a number of traditional and novel cardiovascular risk factors and target organ damage in patients with atherosclerosis and hypertension (Kals *et al*., 2008; Kampus *et al*., 2008). There is also evidence that a reduction in arterial stiffness results in a lower incidence of cardiovascular events (Guerin *et al*., 2001).

Several techniques are available for assessing AS, including invasive and non-invasive methods. Non-invasive pulse wave analysis (PWA) and pulse wave velocity (PWV) are reliable methods for the measurement of arterial stiffness and

useful indicators of future CVD risk, particularly in younger individuals and before the manifestation of clinical signs (McEniery *et al.,* 2005; Parvathaneni *et al.,* 2002).

PWA uses pressure waves from the different segments, where carotid, brachial or radial artery and central pressure waveforms are calculated using the transfer function (Townsend *et al.,* 2015). The PWV is calculated as the distance travelled by the pulse wave divided by the time taken to travel the distance (Laurent *et al.,* 2006) (Figure 1).

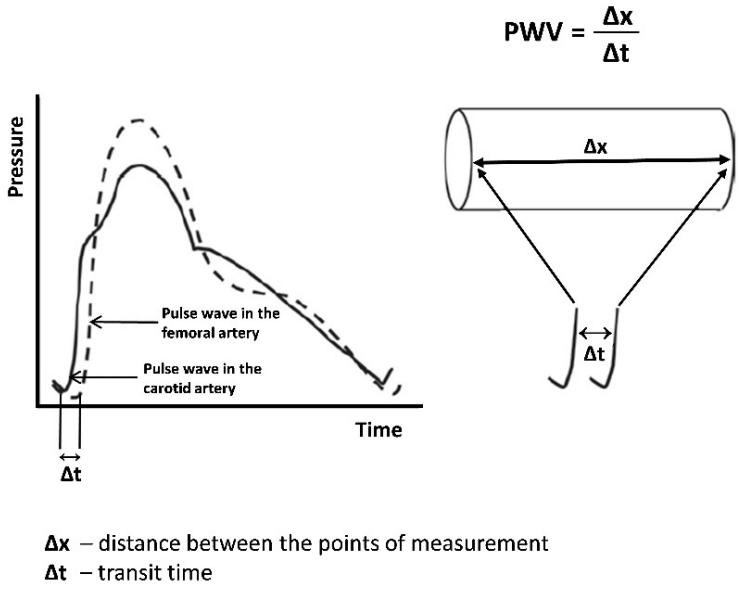

Figure 1. Measurement of arterial PWV with the foot-to-foot method (Adji *et al.,* 2011, modified).

Carotid to femoral pulse wave velocity (PWV) is considered the valid "golden standard" criterion index representing central arterial stiffness (Townsend *et al.,* 2015; Van Bortel *et al.,* 2012). Higher PWV represents stiffer arteries. According to the guidelines, the cut-off value of increased PWV is considered >10 m/s (Van Bortel *et al.,* 2012). In stiffened arteries, waves arrive earlier in the end-systole and augment systolic pressure and this phenomenon can be quantified by means of an augmentation index (Aix) (Townsend *et al.,* 2015). In healthy subjects, AS progressively increases by 40 to 50 % between the ages of 25 to 75 years (Tanaka *et al.,* 2000).

A number of cross-sectional and longitudinal studies have shown the beneficial effect of exercise on arterial stiffness (Ferreira *et al.,* 2006; Miura 2012). The mechanism by which exercise affects AS have not been exhaustively examined. It is known that aerobic exercise improves classical risk factors (body fatness, insulin resistance, blood pressure) and thereby improves AS (Stehouver and Ferreira, 2006). Enhanced endothelium-dependent vasodilatation through the

increased production of nitric oxide has also beneficial effect (Goto *et al.,* 2003; Hambrecht *et al.,* 2000). In addition, exercise-induced improvements in AS could be mediated by concomitant improvements in inflammation and decreasing sympathetic activity (Ferreira *et al.,* 2006). Structural adaptations to exercise, such as increased elastin content and inhibition of collagen activity within the arterial wall may also play significant role, but these beneficial changes are considered more time-dependent (Tanaka and Safar, 2005).

2.2.2. Strength training and arterial stiffness

In recent years, the importance of strength training, also known as resistance training (RT), has been emphasized in general health guidelines (Chaput *et al.,* 2020; Niebauer *et al.,* 2018; Peliccia *et al*., 2020), including recommendations of moderate resistance training in the prevention programs of sarcopenia and for increasing muscular strength. Resistance exercise has been shown to have a beneficial effect on muscular strength, bone mineral density, and body composition (Gómez-Cabello *et al*., 2012). Nevertheless, compared to aerobic exercise there is less evidence to demonstrate a protective effect of resistance exercise on cardiovascular health, particularly on BP and oxidative stress (Cornelissen and Fagard, 2005; Fecchio *et al*. 2021; Yoon *et al*., 2010).

The haemodynamic effect of resistance training differs significantly from aerobic training. In general, resistance training is divided into two subgroups having different effects on the cardiovascular system (Cornelissen and Smart, 2013; Mancia *et al*., 2013):

1) dynamic resistance training (where the force development is associated with movement with intensity between 30% and 100% of 1 repetition maximum – RM) has been shown to reduce BP and is recommended for cardiovascular prevention in the hypertension guidelines;
2) isometric resistance training (where the force development is associated without movement with intensity between 10% and 40% of 1 RM) and this is not recommended for lowering BP because there are not enough studies available.

High-intensity strength training involves slow-speed contractions of the muscles, which are related to transient mechanical compression of resistant vessels increasing peripheral vascular resistance and left ventricular pressure overload during exercise (MacDougall *et al*., 1995). A recent meta-analysis (Pierce *et al.,* 2018) revealed that acute resistance training has an adverse effect on arterial stiffness due to cardiovascular and non-cardiovascular mechanisms. In regard to systematic strength training, some studies have shown stiffer arteries in strength-trained athletes (Bertovic *et al.,* 1999; Miyachi, 2013), while other data have shown a beneficial effect of resistance training on arterial stiffness (Morra *et al*., 2014; Okamoto *et al*., 2008).

In well-trained weightlifting and powerlifting athletes, moderate to intensive strength training (60–90% of 1 RM) is usually used, whereas most of the arterial stiffness studies are conducted with low or moderate intensity RM or in combination with aerobic training (Azizbeigi *et al*., 2013; Bloomer *et al.,* 2005; Cakir-Atabek *et al*., 2015).

In summary, the inconclusive data about the effect of strength training on AS in available studies may depend on the very wide variety of modalities and intensities of training protocols. Furthermore, previous inconsistent study results may depend on the athletes' athletic level and preconditioning status.

2.2.3. Aerobic training and arterial stiffness

Published data have shown that regular aerobic exercise is associated with better arterial stiffness in comparison with sedentary controls (Laurent *et al.,* 2011; Tanaka, 2019). In general, the beneficial effect of aerobic exercise is mainly contributed to small and moderate doses of exercise in healthy subjects (Kobayashi *et al.,* 2020).

On the contrary, some studies have shown impaired arterial stiffness in marathon or ultra-marathon runners, which may be attributed to the chronic response of high-volume training and competitions (Burr *et al.,* 2012, 2014; Vlachopoulos *et al.,* 2010a). However, Müller *et al*. (2017) showed no adverse effects of regular marathon running on vasculature measured by carotid intima-media thickness in their longitudinal study. It must be taken into consideration that high-level endurance runners may experience chronic increases in central blood pressure and altered arterial stiffness due to repeated acute exercise loads and exercise-induced inflammation as well as oxidative stress. Several studies have evaluated an acute response to arterial stiffness after short-term physical load (Pierce *et al.,* 2018; Saz-Lara *et al*., 2021) and some studies have evaluated the post-exercise effect during the early recovery phase (i.e. 5 to 60 min after the exercise), whereas the acute effect of prolonged and intensive physical load on arterial stiffness in aerobically well-trained athletes is less studied (Pierce *et al.,* 2018; Saz-Lara *et al.,* 2021). Very little data are available in regard to the recovery period over 60 min to 24 hours. A recent meta-analysis (Saz-Lara *et al.,* 2021) revealed that the acute effect of different types of exercise (aerobic exercise, resistance training, interval training) on PWV varies significantly within 24 hours after exercise.

2.3. Exercise and blood biomarkers

Intensive training can induce changes in the serum concentrations of numerous blood biomarkers. These changes, especially increases, can often result over the normal range and may be a reason for diminishing physical loads and competitions (Banfi *et al.,* 2012). However, the behaviour of some blood biomarkers in high-level athletes in regard to high physical loads has not been extensively studied. Numerous blood biomarkers may assess different aspects of health, sports performance as well as recovery (Lee *et al.,* 2017). Creatine kinase (CK) and lactate dehydrogenase (LDH) activity and myoblobin are widely used for describing skeletal muscle damage (Banfi *et al.,* 2012; Lee *et al.,* 2017; Nie *et al.,* 2011), and showing a remarkable increase after strenuous exercise (Banfi *et al.,* 2012). There is data available about the alterations of renal as well cardiac markers, where exercise-related ischemia is prevalent (Banfi *et al.,* 2012; Legaz-Arrese *et al.,* 2011). Post-exercise increase of serum troponins and N-terminal pro B-type natriuretic peptide (NT-proBNP) is related to cardiac damage and dysfunction and may reflect a heavy cardiovascular demand during exercise (Banfi *et al.,* 2012; Legaz-Arrese *et al.,* 2011; Rubio-Arias *et al.,* 2021; Scherr *et al*., 2017). Moreover, the increase of NT-proBNP has been shown to be related to increased arterial stiffness (Sung *et al.,* 2009). Nevertheless, the short- or long-term clinical impact of an increase in cardiac-specific markers following intensive exercise or competitions in healthy athletes is not clear.

2.3.1. Exercise and inflammatory markers

Chronic low-grade inflammatory level is considered a strong predictor for atherosclerosis and thereby for CV disease (Ridker *et al.,* 1997; Ross, 1999). Recognition of the importance of the inflammation in CV disease pathogenesis gives an opportunity to evaluate different inflammatory markers as potential predictors of CV risk.

Regular physical activity has been shown to have a beneficial effect on the inflammatory status and therefore may protect against the development of many chronic diseases, including cardiometabolic diseases (Gleeson *et al.,* 2011). The anti-inflammatory effects of regular exercise are mediated by the reduction of visceral fat mass (with a subsequent decreased release of adipokines) and the increase of an anti-inflammatory markers (Gleeson *et al.,* 2011). Cross-sectional studies support the finding that physically active persons have a lower baseline inflammatory level in comparison with sedentary ones (Fernandez *et al.,* 2018; Pihl *et al.,* 2003). On the other hand, there is arising data that frequent strenuous exercise may lead to a proinflammatory condition and subsequent health problems, i.e. overload of the cardiovascular system and increase of the CV risk (O'Keefe *et al.,* 2012; Petersen and Pedersen, 2005). It is not known how much these changes are attributed to physical load *per se* or insufficient recovery (Petersen and Pedersen, 2005). Repeated damage of muscle cells, connective

tissue and bones may add an adverse impact to the inflammatory status. All these adverse effects may lead to the impairment of arterial stiffness and may increase the future risk of CV disease.

During acute exercise, interleukin-6 (IL-6) stimulates the synthesis of other anti-inflammatory cytokines (IL-1a, IL-10), thus providing an inhibitory effect on pro-inflammatory cytokines (IL-1β, TNF-α) (Pedersen, 2013). A widely used marker of low level inflammatory condition is a high-sensitive C-reactive protein (hsCRP). A significant elevation of hsCRP was observed after circuit resistance exercise in previously untrained subjects (Bizheh and Jaafari, 2011). Similarly, a significant increase in white blood cells (WBC) content and CRP level was observed in marathon runners immediately after the race (Weight *et al.*, 1991). It has been suggested that intense muscular contraction and exercise-induced muscle injury increase IL-6 secretion from the skeletal muscle, which in turn stimulates the synthesis of CRP in the liver (Kasapis and Thompson, 2005). In contrast, no significant changes in hsCRP or IL-6 levels were detected after thirty minutes of walking on a treadmill at the intensity of 50% maximal oxygen consumption (VO_2max) in healthy sedentary male subjects (Markovitch *et al.,* 2008). It has been proposed that the CRP-lowering effect of chronic exercise is more related to the fat mass reduction of the subjects (Walsh *et al.,* 2011). Therefore, attention should be paid to the interpretation of the anti-inflammatory effect in overweight subjects in comparison with normal body fatness.

The soluble intercellular adhesion molecule-1 (sICAM) plays an important role in facilitating of leukocytes migration into the subendothelial space and impairing the endothelial function (Kals *et al.,* 2008; Koh and Park, 2018). Studies have shown, that an elevated sICAM level is associated with hypertension and other cardiometabolic diseases (Rohde *et al.,* 1999). There is supporting data that low-to-moderate aerobic exercise reduces the sICAM level in healthy individuals as well as in case of cardiometabolic disease, while high-intensity aerobic exercise increases the sICAM level immediately after the exercise (Koh and Park, 2018). However, there is less information about strength training in relation to adhesion molecules.

Adiponectin is a protein that is mostly produced in the adipose tissue and has anti-inflammatory and anti-atherogenic effects (Choi *et al.,* 2020). Plasma adiponectin levels have been shown to be related with arterial stiffness and with the progression of arterial hypertension (Youn *et al.,* 2013). The results of studies assessing exercise and adiponectin level provide inconsistent results. Moderate to high intensity exercise appears to modify the adiponectin level positively, but most of these studies do not consider simultaneous changes in body composition, dietary intake and other confounding factors (Simpson and Singh, 2008).

No sufficient data are available about the effect of different physical loads and recovery period response on inflammatory markers in high-level athletes. Furthermore, very little is known about the inflammatory response to high-intensity resistance training in high-level strength athletes.

2.3.2. Exercise and oxidative stress markers

It is well accepted that strenuous exercise increases the production of reactive oxygen species (ROS), which may lead to cellular damage as a result of high-grade oxidative stress (OxS) (Finaud *et al*., 2006; Guolin, 2013; Withee *et al*., 2017). Damaged mitochondria of muscle cells are the major intracellular source of ROS (Finaud *et al*., 2006), whereas ROS are also produced by erythrocytes, neutrophils, and lymphocytes (Guolin, 2013; Withee *et al*., 2017).

In a single bout of aerobic exercise, where skeletal muscles produce significant amounts of superoxide anion, the harmful effect of ROS is well known (Mastaloudis *et al*., 2006). In anaerobic training, other different pathways of reactive oxygen and nitrogen species generations (including xanthine and oxidase production) have shown a disruption of iron-containing proteins, prostanoid metabolism, ischemia reperfusion, etc. (Bloomer *et al.,* 2006; Jackson, 2000). Very intensive and long-term physical load is detrimental on antioxidant mechanisms and may lead to the unfavourable effects by OxS on health, all of which are not yet known (La Gerche, 2016; Withee *et al*., 2017). For preventing OxS, there is a complex antioxidant defence system in the human organism consisting of enzymatic and nonenzymatic antioxidants (Urso and Clarkson, 2003). Thus, it is important that the antioxidant defence system is effective and recovers properly after strenuous physical exercise. Furthermore, it is demonstrated that an increased level of OxS is directly linked to impaired arterial stiffness (Kals *et al*., 2006; Patel *et al*., 2011).

Regular aerobic training has been shown to improve antioxidant adaptation to exercise (Guolin, 2013; Mozos and Luca, 2017). However, very little data are available regarding the effects of high-intensity exercise on OxS and its relationship with arterial stiffness in well-trained athletes. In the study conducted by Kampus *et al*. (2008), the changes in arterial elasticity indices were significantly related to the exercise-related inflammatory markers and these changes were dependent on the athletes' aerobic fitness level showing better recovery data in those who had a higher fitness level. There are several OxS markers in use: isoprostanes, blood gluthatione, protein carbonyls, diene conjugates and others (Frijhoff *et al.,* 2015). The oxidative stress index (OSI), which is the ratio of total peroxide to the total antioxidant level, is considered one of the precise indices of oxidative stress in the body (Harma and Erel, 2003).

In summary, there are limited data on the effect of intensive exercise at competition level on arterial stiffness and related blood biomarkers in elite-level athletes, who have been at an upper level of sports activity for several years. Moreover, there is no clear understanding and no systematic data on the late post-competitive recovery period in case of intensive physical load on arterial stiffness. Furthermore, the effect of elite-level strength training for lowering BP and cardiac function is still unclear.

3. RESEARCH HYPOTHESIS AND AIMS

The main purpose of this study was to test the hypothesis that a high-level strength training program as well as acute long-distance running may evoke adverse effects on arterial stiffness and related blood markers in high-level athletes.

Specific aims of the study were:

1. To evaluate the effect of a 12-week supervised strength training program on arterial stiffness, blood pressure and cardiac function in male elite powerlifting athletes. (Paper I).
2. To assess the effect of a 12-week supervised strength training program on oxidative stress indices and blood lipids as well as their relationship with arterial stiffness in elite powerlifting athletes. (Paper II).
3. To compare the effect of acute long-distance running on arterial stiffness and related blood biomarkers during the post-competition recovery period in high-level male long-distance runners and recreational level athletes. (Paper III).

4. METHODS

4.1. Participants

4.1.1. Supervised strength training program (Paper I, II)

The main descriptive data (age, anthropometric data) are presented in Table 1. In total, 20 voluntary well-trained male powerlifting athletes were examined before and after a 12-week supervised strength training program (SSTP). Study subjects have exercised in powerlifting sports at a competitive level from 4 to 15 years. Before the study, subjects completed a questionnaire concerning their personal and medical history and lifestyle parameters. All subjects were non-smokers and were free of acute or known chronic illnesses.

One subject was excluded from the data analysis because of an acute injury. Thus, the final study sample was 19. The study subjects were informed about the details of all training and study procedures two months prior to the study.

Table 1. Age, anthropometric, VO_{2peak}, and exercise tolerance data of the subjects at baseline (n=19), x±SD.

Parameter	x ± SD
Age (years)	28.2 ± 6.1
Height (cm)	179.2 ± 5.9
Weight (kg)	99.9 ± 16.5
BMI (kg/m^2)	31.2 ± 5.1
Training experience (yrs)	8.0 ± 4.1
Body fat (%)	16.1 ± 7.9
VO_{2peak} (L/min)	3.8 ± 0.7
VO_{2peak} (ml/kg/min)	38.7 ± 8.9
W_{max} (W/kg)	4.1 ± 0.9

Abbreviations. BMI − body mass index; VO_{2peak} – the highest level of oxygen uptake; W_{max} − exercise tolerance.

4.1.2. Half-marathon running study (Paper III)

In half-marathon running study (HMRS), 18 endurance-trained male subjects who participated in 21 km competition were examined. According to the sports participation level, subjects were divided into two groups: high-level (well-trained long-distance runners, who were members or candidates of the Estonian national team), n=11, and recreational athletes (who have exercised regularly for recreational purposes), n=7. The study subjects were informed about the details of study procedures two months prior to the study. Subjects' anthropometric, aerobic capacity and training characteristics are presented in Table 2.

Table 2. Anthropometric, training, and aerobic capacity characteristics of the subjects, x±SD.

Parameter	Total n = 18	High-level n = 11	Recreational n = 7
Age (years)	29.9 ± 6.3	27.1 ± 4.8	34.3 ± 6.1*
Height (m)	1.80 ± 0.06	1.81 ± 0.04	1.80 ± 0.09
Weight (kg)	74.2 ± 11.1	69.9 ± 5.2	80.0 ± 14.6*
BMI	22.9 ± 2.7	21.4 ± 1.2	25.2 ± 2.9**
Fat percentage (%)	11.7 ± 4.5	10.0 ± 3.2	14.5 ± 5.2*
Training experience (years)	12.4 ± 8.6	10.0 ± 6.2	16.2 ± 10.8
Training volume (km/month)	252 ± 114	296 ± 117	172 ± 48*
VO_2peak (L/min)	4.67 ± 0.48	4.91 ± 0.31	4.28 ± 0.44**
VO_2peak/kg (ml/kg/min)	63.8 ± 10.5	70.0 ± 5.3	54.0 ± 9.3***
Mean competition time (minutes)	84.9 ± 12.7	76.4 ± 5.6	95.9 ± 11.6***
Maximal HR (beats/min)	188 ± 7	189 ± 8	187 ± 5
Mean HR during the half-marathon (beats/min)	175 ± 7	177 ± 9	172 ± 4
% of the maximal HR during the half-marathon	93.1 ± 2.9	93.9 ± 2.5	92.1 ± 3.2

Abbreviations. BMI – body mass index; VO_{2peak} – the highest level of oxygen uptake; HR – heart rate.
* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$ in comparison with high-level group.

In both studies (SSTP, HMRS), informed written consent was obtained from each athlete in accordance with principles of the Declaration of Helsinki. The study protocol was approved by the Medical Ethics Committee, University of Tartu, No 162/T-12.

4.2. Study design

4.2.1. Supervised strength training program (Paper I, II)

Before the SSTP, there was an 8-week off-season period, where systematic strength training sessions and competitions were not allowed.

The 12-week SSTP consisted of a combination of dynamic (muscles contract concentrically during the lifting phase and eccentrically during the lengthening phase) and isometric (muscular force development without movement) resistance training. During the SSTP, one exercise session consisted of 5–8 working sets on one muscle group, and athletes exercised with an intensity of 60–90% assessed from 1 RM. The first set was done with an intensity of 60% of 1 RM and the last one 90% of 1 RM. Variation of repetitions was decreased according to the rise of intensity in one set (4 to 12). One exercise session lasted 90–120 min.

Athletes exercised four days per week and every one-week training program consisted of the following exercises covering all major muscle groups: bench press, lateral pulldown, standing shoulder press, arm curl and extension, leg press, squat, leg curl, calf press, abdominal crunch, and deadlift. All the major muscle groups were exercised twice a week.

The structure of each exercise session was divided into three parts:

1) the first part consisted of a 15-min warm-up, which included stretching and active exercises using body weight or very light weight;
2) the second part was the main training phase, where the structure of the training session was organized according to the ascending pyramid principle (Fleck and Kraemer, 2014). The first series were started with 60% of 1 RM with 12 repetitions, the second series were 70% of 1 RM with 10 repetitions, and the third were 80% of 1 RM with 8 repetitions. The fourth to sixth series were performed with loads ranging from 80% to 90% of 1 RM and 4–6 repetitions were made. In one session, half of the muscle groups were exercised: on Monday and Thursday exercises were performed on chest muscles, *m. latissimus dorsi* muscles, hamstring muscles, abdominal muscles, lower back muscles, and shoulder muscles; on Tuesday and Friday exercises were performed on arm muscles (*m. biceps brachii* and *m. triceps brachii*), *quadriceps* muscles and calf muscles;
3) the third part –15-min cool-down period, which included stretching and foam rolling for all muscles that experienced load in the training session.

No aerobic exercise was involved in the training program and all the sessions were supervised by a skilled certified instructor. After the SSTP, all athletes in the study participated in the Estonian Powerlifting Championships. The training protocol used in this study is characteristic for high-level powerlifters in the pre-seasonal phase.

4.2.2. Half-marathon running study (Paper III)

Before the half-marathon running competition, all the participants performed their usual preparation phase with respective training loads. The goal of running the half marathon competition (21 km) was as fast a time as possible for each athlete. During the competition, HR was recorded by the heart rate monitor Polar RS800CX and analysed with Polar Protrainer 5 software (Polar Electro Oy, Kempele, Finland). The mean results (time) and intensity (maximal HR, % of maximal HR) of the half-marathon competition are presented in Table 2.

4.3. Measurements

4.3.1. Supervised strength training program (Paper I, II)

Anthropometric, laboratory measurements, haemodynamic, echocardiographic and maximal muscular power measurements were performed one week before the SSTP, while post-training period measurements were performed one week following the competitions. During this measurement period, only light recreational activities for the subjects were allowed. All the measurements were performed in the morning between 8 and 10 AM. Blood analyses were collected after haemodynamic measurements. All haemodynamic and echocardiographic examinations were performed by a single experienced investigator.

At baseline (one week before SSTP) a maximal cardiopulmonary exercise test was performed.

4.3.2. Half-marathon running study (Paper III)

One week before the competitions, anthropometric measurements and the cardio-respiratory exercise test were performed. Venous blood samples and arterial stiffness measurements were taken 24 hours prior to the competition (baseline) and during 18–22 hours after the competition (post-exercise recovery period, RECOV) in the morning between 8 and 10 AM.

4.3.3. Anthropometric measurements (Paper I, II, III)

Subjects' height and weight were determined by the Martin metal anthropometer (±0.1 cm) and clinical scales (±0.05 kg), and body mass index (BMI) was calculated (kg/m^2). Body fat percentage was assessed by measuring skinfold thickness at six different anatomical sites (Tipton and Oppliger, 1984) in SSTP. In HMRS, body fat percentage was measured by dual-energy X-ray absorptiometry using the DPX-IQ densitometer (Lunar Corporation, Madison, WI, USA).

4.3.4. Haemodynamic measurements (Paper I, II, III)

4.3.4.1. Brachial blood pressure

Brachial blood pressure was measured in a sitting position from the non-dominant arm as a mean of three consecutive measurements at 5-min intervals using a validated oscillometric technique (OMRON M4-I; Omron Healthcare Europe BV, Hoofddorp, the Netherlands). Three readings were taken at 1 min intervals and the averaged two closest readings were used in further analysis (Mancia *et al.*, 2007). Brachial pulse pressure (bPP) was calculated as the difference between brachial systolic (bSBP) and diastolic BP (bDBP).

4.3.4.2. Central blood pressure and arterial stiffness

Radial artery waveforms were recorded with a high-fidelity micromanometer (applanation tonometry SPT-301B; Millar Instruments, Houston, TX, USA) from the wrist of the dominant arm. Pulse wave analysis was performed on the systolic portion (SphygmoCor, version 7.1, AtCor Medical, Sydney, Australia) of the pulse curve in accordance with guidelines (Mackenzie *et al.*, 2001). The difference between the carotid and the femoral path lengths was estimated from the distance of the sternal notch to the femoral pulse measured in a direct line (Laurent *et al.,* 2006). The corresponding ascending aortic waveforms were then generated using a validated transfer function (Laurent *et al.,* 2006; Mackenzie *et al.*, 2001; Pauca *et al.,* 2001), from which central haemodynamics (pulse wave velocities at carotid-femoral [cfPWV] and carotid-radial segments [crPWV]), augmentation index (AIx), central systolic (cSBP) and diastolic BP (cDBP) and central pulse pressure (cPP) were calculated. The AIx was defined as the difference between the second and the first systolic peaks of the central arterial waveform expressed as the percentage of central pulse pressure (Mackenzie *et al.*, 2001). The AIx was corrected for heart rate of 75 beats per minute (AIx@75). Mean arterial pressure (MAP) was calculated from the integration of the radial artery waveform. The degree of pulse pressure amplification (PPA) was calculated as brachial pulse pressure divided by central pulse pressure (bPP/cPP). Resting heat rate (HR) was recorded during arterial stiffness measurements. Haemodynamic measurements were obtained by trained investigators according to current guidelines (Mattace-Raso *et al.,* 2010; Townsend, 2017).

4.3.4.3. Carotid-femoral pulse wave velocity

The carotid-femoral pulse wave velocity (cfPWV), a direct measure of arterial stiffness, was determined by sequential acquisition of pressure waveforms from the carotid and femoral arteries using the same tonometer (SphygmoCor, version 7.1, AtCor Medical). The timing of these waveforms was compared with that of the R wave on a simultaneously recorded electrocardiography (ECG). The cfPWV was determined by a calculation of the difference between the carotid and the femoral path length divided by the difference in the R wave to waveform foot times. The within-observer coefficient of variation for cfPWV was 2.3%. The pulse wave analysis and cfPWV measurements were made in duplicate and their mean values were used in the data analysis.

For haemodynamic measurements, all the subjects fasted and refrained from tobacco, alcohol, tea, and coffee consumption overnight.

4.3.5. Echocardiography (Paper I)

In SSTP study, an echocardiographic measurement was performed using a commercially available device (Sonos7500, Philips Medical Systems, Inc., Highland Heights, OH, USA) with a 3.5-MHz transducer and digital recording by an experienced cardiologist. The images were stored digitally, coded with a random number and read by a blinded observer. Left atrial dimension was measured using the two-dimensional echocardiography (2DE) anteroposterior linear method. For left ventricular mass calculations, the 2DE and the M-mode were used. Long-axis measurements were obtained at the level distal to the mitral valve leaflets. Left ventricular internal dimension and septal and posterior wall thickness were measured at the end of the diastole (subscript "d") according to the recommendations of the current guidelines (Lang *et al.*, 2015). Left ventricular mass was calculated using the formula 0.8{1.04[(LVIDd+PWTd+SWTd)3-(LVIDd)3]}+0.6g (LVIDd – left ventricular internal dimension in end-diastole, PWTd – posterior wall thickness in end-diastole, SWTd – septal wall thickness in end-diastole) (Lang *et al.*, 2015). For 2DE volume calculations, the modified Simpson's rule was used according to the current guidelines (Lang *et al.*, 2015). For left ventricular diastolic function assessment, medial E/e' ratio and mitral valve E/A ratio was measured in accordance with the recent guidelines (Nagueh *et al.*, 2016). Left ventricular outflow tract velocity-time interval was measured with the pulsed-wave Doppler sample volume at the level of the left ventricular outflow tract in 3- or 5-chamber view. Tissue Doppler imaging (TDI) was performed in the apical four-chamber view. Peak systolic velocity of tricuspid annulus by TDI was measured as an indicator for right ventricular systolic function.

4.3.6. Blood sampling and blood biomarkers (Paper II, III)

Venous blood samples were drawn from the antecubital vein in the morning between 8:00 and 10:00. The subjects were fasted and refrained from tobacco, alcohol, tea, and coffee consumption overnight.

Biochemical and hormonal parameters

QBC Autoread Plus autoanalyzer (QBC Diagnostics, Inc., USA) was used to assess white blood cell (WBC) count, red blood cell count (RBC), haemoglobin (HGB) and haematocrit (Hct) in whole blood. Lactate dehydrogenase (LDH), serum creatine kinase (CK), creatinine, myoglobin, high-sensitivity C-reactive protein (hsCRP), ferritin, glucose, total cholesterol (CHOL), high-density lipoprotein-cholesterol (HDL-C), low-density lipoprotein-cholesterol (LDL-C), and triglycerides (TG) and insulin were measured by standard laboratory methods.

High-sensitivity C-reactive protein (hsCRP) was determined by a latex particle-enhanced immunoturbidimetric assay (Roche Diagnostics GmbH, Mannheim, Germany). Serum levels of N-terminal pro B-type natriuretic peptide

(NT-proBNP) were measured using a commercially available chemiluminescent sandwich immunoassay (Cobas; Roche Diagnostics GmbH).

In the supervised strength training study – WBC, RBC, Hct, HGB, CK, creatinine, hsCRP, glucose, CHOL, HDL-C, LDL-C, TG and NT-proBNP were analysed.

In the half-marathon running study – WBC, HGB, CK, hsCRP, ferritin, myoglobin, LDH, glucose and insulin were analysed.

All the biochemical analyses were performed at the Laboratory Department of the Tartu University Hospital.

Oxidative stress and inflammatory markers

Total peroxide concentrations (TPX) of samples were determined using OXY-STAT Assay Kit Cat. No BI-5007 (Biomedica Gruppe; Biomedica Medizinprodukte GmbH & Co, Kg, Vienna, Austria). Total antioxidant capacity (TAC) was measured by using an automated measurement method by Erel (2004). Percent ratio of the total peroxide concentration of plasma to the TAC of plasma was used as oxidative stress index (OSI).

For the measurement of diene conjugates (DC), blood samples were incubated at 37 °C for 25 min, 0.25% BHT and lipids were extracted by heptane/ isopropanol (1:1). Blood samples were then acidified by 5 M hydrochloric acid and extracted by heptane. After centrifugation and absorbance of heptane, the fraction was measured spectrophotometrically at an absorbance maximum between 220 and 250 nm.

Protein carbonyls were determined as described previously (Bogdanovic *et al*., 2001). After homogenization (1:10 in 10 mM Hepes buffer containing 0.5 μg/ml of aprotinin) and standing for 15 min at 4 °C, the samples were centrifuged at 1,500 g for 10 min (4 °C). The supernatant was mixed with streptomycin sulphate (final concentration 1%), which stayed at room temperature for 15 min and centrifuged again as described above. 0.1 ml of supernatant was mixed either with 0.4 ml of 10 mM 2,4-dinitrophenylhydrazine, a carbonyl-specific reagent, in 2 M HCl or 0.4 ml 2 M HCl alone. The different spectrum of the 2,4-dinitrophenylhydrazine derivatives versus HCl blanks was followed spectrophotometrically at 350–380 nm.

The plasma level of asymmetric dimethylarginine (ADMA) was determined by an ELISA using a commercially available kit (DLD Diagnostika ®, Hamburg, Germany).

The plasma level of soluble intercellular adhesion molecule-1 (sICAM) was measured by an enzyme-linked immunosorbent assay using a commercially available kit (Human soluble intercellular adhesion molecule-1 Immunoassay; R&D Systems; Minneapolis, USA).

The plasma concentration of adiponectin was analysed by a quantitative sandwich enzyme immunoassay technique using commercially available kits (R&D Systems, Minneapolis, MN, U.S.A.).

The blood samples were centrifuged within 15 minutes after collection at 3,000 rpm to obtain serum that was frozen at –80 ºC until further analysis.

Measurements were analysed at the Institute of Biomedicine and Translational Medicine (University of Tartu).

In the supervised strength training study – TPX, TAC were used and OSI was calculated.

In the half-marathon running study – DC, sICAM, protein carbonyls, adiponectin and ADMA were used.

4.3.7. Assessment of maximal muscular power (Paper I, II)

Maximal muscular power was tested before and after the SSTP with bench press (BeP) and jumping from a half squat (JHS) with a barbell.

Procedure

After the warm-up, each athlete performed maximal effort in bench press and jumping from a half squat by the bar with his body weight following the SSTP study protocol. Bench press was performed in supine position by barbell, which included pressing a weight upward as fast as the athlete was able from starting position to chest and back to starting position, but no bouncing effect was allowed. Jumping from a half-squat exercise was performed in standing position, where the main task was to perform a half squat with barbell and after that jump off the ground as high as an athlete was able and then land back in the starting position on the subject's feet. The muscle power produced during squat jump was measured with a linear encoder attached to the barbell and fitted to the Musclelab system (Ergotest Innovation a.s., Porsgrunn, Norway). The method was previously described by Bosco *et al.,* (1995).

4.3.8. Cardiopulmonary exercise test and heart rate measurement (Paper I, II, III)

At baseline, all subjects underwent a maximal exercise test to determine the highest level of oxygen uptake (VO_{2peak}) using a breath-by-breath metabolic system (MasterScreen CPX, Viasys Healthcare GmbH, Hoechberg, Germany) and a motorized treadmill (Viasys/Jaeger LE300 C, Viasys Healthcare GmbH, Hoechberg, Germany). Gas analysis data were automatically calculated for every 30-second period.

In the supervised strength training study, a modified Balke test on a treadmill was used. The treadmill was kept at a constant speed of 6.0 km/h with grade starting at 1.5% and increasing by 3.5% every 2 min until self-determination of exhaustion.

In the half-marathon running study, an incremental running test was used with the initial speed of the treadmill at 8.0 km/h with an incline starting at 1.5% and increasing by 2 km/h every 3 min until self-determination of exhaustion. VO_2peak was considered as the highest VO_2 rate achieved within 30 seconds at the end of the exercise test. Secondary criteria for achieving VO_2peak included respiratory exchange ratio (RER) > 1.15 and heart rate (HR) > 95% of the subject's age-predicted maximum. Heart rate was recorded continuously at 5-second intervals during the cardiopulmonary exercise test by a sport-tester (Polar Electro, Finland). The exercise tests were carried out within two to four hours after breakfast. During the half marathon competition, HR was recorded by a sport-tester as well.

4.3.9. Food Intake (Paper I, III)

Study subjects were asked not to take alcohol or antioxidant supplements and not to change their usual diet during the supervised strength training study or half-marathon running study.

Food intake of the subjects was analysed using the NutriData Estonian food composition database, version 8. In the the supervised strength training study, all subjects assessed their food intake three times during the study period for five consecutive days during the 12-week SSTP.

In the half-marathon running study, all participants' food intake was analysed for 5 consecutive days (3 days before the half-marathon competition, on the day of the competition and one day after the competition).

The main purpose of the food intake data collection and analysis in the SSTP study was to control and avoid significant changes in subjects' nutrition during the study. In HMRS, the main purpose was to compare the study groups in their caloric and nutrient intake.

4.3.10. Statistical analysis (Paper I, II, III)

Statistical analysis was performed with SPSS (SPSS Inc., Chicago, Illinois, USA), version 22 (Paper I, II) and version 25.0 (Paper III). All data were checked for normal distribution using the Kolmogorov-Smirnov test. Data were expressed as means (x) and standard deviations (SD). The data were analysed using the paired-samples t-test. For skewed data distribution, the Mann-Whitney U-test and for nonparametric analysis for related samples, the Wilcox on Rank test was used. The Pearson or Spearman correlation analysis was used to determine the relationships between the variables. Stepwise multiple regression analysis was performed to determine independent associations between variables. The variables were selected according to univariate analysis. For the observed power estimation, the general linear model, repeated measurements and power calculations were performed. Statistical significance was defined as two-sided $p<0.05$.

5. RESULTS

5.1. Supervised strength training study (Paper I, II)

5.1.1. Anthropometric and food intake data

Subjects' mean body weight, body fat percentage and BMI values after the 12-week strength training period did not differ significantly from baseline values (99.9±16.5 *vs* 100.2±17.1 kg, p=0.614; 16.1±7.9 *vs* 15.8±7.7, p=0.906; 31.2±5.1 *vs* 31.3±5.2, p=0.603, respectively).

Table 3 gives descriptive information about the powerlifters' mean dietary intake during the study [total caloric intake, protein, fat, and carbohydrate intake in grams and total energy intake (%) for proteins, fats and carbohydrates].

No significant differences in caloric and nutrient intake between three measurements were found (data not shown).

Table 3. Nutritional daily intake of the subjects (x±SD), percentages from total energy intake.

Parameter	x ± SD
Total energy intake (kcal)	4200±845
Protein (g)	190±60
Fat (g)	168±56
Carbohydrate (g)	495±147
Protein (% energy)	18.4±4.8
Fat (% energy)	35.5±4.8
Carbohydrate (% energy)	46.1±10.5

5.1.2. Blood pressure and arterial stiffness data

Blood pressure and arterial stiffness data at baseline and after the 12-week SSTP are presented in Table 4. Study results showed that subjects' mean brachial and central systolic BP were significantly lower after the training period as compared to the baseline (before the SSTP) data. There were no significant differences after the training period in cfPWV (both adjusted and unadjusted) and in AIx. After the SSTP, a slight decrease was detected in central pulse pressure and brachial pulse pressure, but the decrease remained nonsignificant. No significant changes were found in MAP, PPA (bPP/cPP), and heart rate after the SSTP.

Table 4. Blood pressure and arterial stiffness parameters of the subjects before and after the 12-week strength training period (SSTP), x± SD.

Parameter	Before SSTP	After SSTP	*p*-value
bSBP (mmHg)	132.3± 8.8	124.3± 8.7	**0.002**
bDBP (mmHg)	70.1± 5.9	67.7± 5.2	0.059
bPP (mmHg)	61.2± 8.4	58.8± 7.3	0.259
MAP	88.4± 7.4	86.9± 5.9	0.359
cSBP (mmHg)	110.1± 7.7	104.5± 8.7	**0.008**
cDBP (mmHg)	71.1± 6.3	68.9± 5.3	0.078
cPP (mmHg)	39.0± 3.7	35.6± 5.0	0.317
PPA	1.6± 0.1	1.6± 0.1	0.650
HR (beats/min)	62.6± 8.6	64.8± 9.3	0.093
AIx (%)	3.3± 9.2	5.1± 8.9	0.233
AIx@75 (%)	–2.7± 9.3	–2.6± 8.3	0.081
cfPWV (m/s)	6.2± 0.7	6.5± 1.1	0.088
Adjusted cfPWV (m/s) $^{\beta}$	6.2± 1.4	6.4± 1.9	0.309
crPWV (m/s)	7.4± 0.8	7.5± 1.0	0.493

Abbreviations. bSBP – brachial systolic blood pressure; bDBP – brachial diastolic blood pressure; bPP – brachial pulse pressure; MAP – mean arterial pressure; cSBP – central systolic blood pressure; cDBP – central diastolic blood pressure; cPP – central pulse pressure; PPA – pulse pressure amplification; HR – heart rate; Aix – augmentation index; AIx@75 – augmentation index corrected for a heart rate of 75 beats per minute; cfPWV – carotid-femoral pulse wave velocity; crPWV – carotid-radial pulse wave velocity; $^{\beta}$ – adjusted to MAP.

5.1.3. Echocardiography

Echocardiographic parameters at baseline and after the 12-week SSTP are presented in Table 5. At baseline, left ventricular systolic function (ejection fraction 52 to 72%) and left ventricular mass index (49 to 115 g/m^2) were in normal range in all study subjects. However, increased left ventricular end-diastolic volume (>64 mL/m^2) was found in six subjects. Left atrial dimension and E/A ratio remained unchanged after SSTP. E/e' ratio decreased significantly with SSTP ($p<0.05$). There was a statistically significant increase in systolic velocity of tissue Doppler Imaging (TDI) of tricuspid annulus ($p<0.01$).

Table 5. Echocardiographic parameters of the subjects before and after the 12-week strength training program (SSTP), x ± SD.

Parameter	Before SSTP	After SSTP	*p*-value
Left ventricular ejection fraction (%)	63.5±4.5	62.1±4.3	0.056
Left ventricular end-diastolic volume/BSA (ml/m^2)	58.4±9.5	57.7±10.5	0.437
Cardiac output (L/min)	5.6±0.9	5.5±0.9	0.703
Mitral valve E/A ratio	1.6±0.4	1.5±0.4	0.319
IVS thickness (cm)	1.0±0.2	1.0±0.2	0.447
Left atrial dimension (cm)	3.63±0.40	3.58±0.35	0.295
E/e' ratio	7.8±1.3	7.1±1.2	**0.026**
LVMI (g/m^2)	84.4±14.2	84.5±17.3	0.971
Relative wall thickness	0.39±0.05	0.40±0.07	0.380
Tricuspid systolic annular velocity, TDI (cm/s)	13.0±2.3	14.2±2.2	**0.006**

Abbreviations. BSA – body surface area; E/A – ratio of peak velocity blood flow from left ventricular relaxation in early diastole (E wave) to peak velocity flow in late diastole caused by atrial contraction (A wave); IVS – interventricular septal; E/e' – the ratio of mitral peak velocity of early filling (E) to early diastolic mitral annular velocity (e'); LVMI – left ventricular mass index; TDI – tissue Doppler Imaging.

5.1.4. Blood biomarkers

Subjects' data are presented in Table 6. The mean value of CK and HDL-C was significantly higher after SSTP in comparison with baseline values. Total peroxide concentration as well as OSI increased, whereas TAC decreased significantly after the SSTP as compared to baseline data. No significant changes were found in other blood biomarkers.

Table 6. The blood biomarkers of the powerlifters before and after the 12-week supervised strength training program, x ± SD, for hsCRP and NT-proBNP medians, 25th percentile and 75th percentile are presented (in brackets).

Parameter	Before SSTP	After SSTP	*p*-value
WBC (10 × 9/L)	5.6± 1.4	5.± 1.0	0.770
RBC (10 × 12/L)	5.0± 0.3	5.2± 0.2	0.106
Haematocrit (%)	44.3± 3.2	45.1± 2.1	0.222
Haemoglobin (g/L)	152.5± 10.8	155.5± 8.5	0.119
Creatine kinase (U/L)	329.8± 246.7	466.7± 294.8	**0.026**
Creatinine (μmol/l)	96.5± 8.7	94.1± 10.4	0.304
hsCRP (mg/L)	0.73 (0.38; 1.91)	0.64 (0.33; 1.22)	0.717
Glucose (mmol/L)	5.24± 0.32	5.12± 0.53	0.345
CHOL (mmol/L)	4.89± 1.21	5.00± 1.13	0.524
HDL-C (mmol/L)	0.96± 0.23	1.13± 0.27	**0.008**
LDL-C (mmol/L)	3.25± 1.09	3.41± 1.09	0.278
TG (mmol/L)	1.33± 0.62	1.18± 0.55	0.187
NT-proBNP (pg/mL)	20.5 (6.0; 33.3)	21.0 (10.0; 31.0)	0.767
TPX (nmol/ml)	252.8± 128.2	380.2± 191.5	**0.008**
TAC (mmolTroloxeqv/L)	1.44± 0.35	1.22± 0.42	**0.033**
OSI (%)	18.6± 10.0	36.6± 23.9	**0.001**

Abbreviations. SSTP – supervised strength training program; WBC – white blood cells; RBC – red blood cells; hsCRP – high-sensitive C-reactive protein; CHOL – serum total cholesterol; HDL-C – high-density lipoprotein-cholesterol; LDL-C – low-density lipoprotein-cholesterol; TG – triglycerides; NT-proBNP – N-terminal pro B-type natriuretic peptide¸ TPX – total peroxide concentration; TAC –total antioxidant capacity; OSI – oxidative stress index.

5.1.5. Maximal muscular power

Subjects' data are presented in Table 7. Study results showed that all mean values of jumping from half squat (JHS) as well as of bench press (BeP) were significantly higher after the SSTP in comparison with baseline values. These results improved in all subjects. The observed power of effect size for BeP and JHS was between 0.41 and 0.46 for within-subjects effect and 0.96 and 1.0 for between-subjects effect, respectively.

Table 7. The power indices of jumping from half squat (JHS) and bench press (BEP) of the powerlifters before and after the 12-week strength training program, x ± SD.

Parameter	Before SSTP	After SSTP	Change (%)	*p*-value
JHS 0.5 body weight (w)	786.2± 123.0	822.8± 134.8	4.7%	**0.009**
JHS 1.0 body weight (w)	1222.5± 209.4	1264.1± 212.7	3.4%	**0.001**
JHS 1.5 body weight (w)	1397.3± 268.7	1447.5± 258.4	3.7%	**0.002**
BEP 0.5 body weight (w)	734.3± 199.0	797.7± 183.3	8.7%	**0.000**
BEP 1.0 body weight (w)	641.2± 148.3	719.6± 145.6	12.3%	**0.001**
BEP 1.5 body weight (w)	502.0± 159.4	534.0± 165.1	6.4%	**0.008**

Abbreviations. SSTP – 12-week supervised strength training program; JHS – jumping from half squat; BEP – bench press; 0.5 body weight – weight equal to the half of the body weight; 1.0 body weight – weight equal to the subjects' body weight; 1.5 body weight – weight equal to the subjects' 1.5 of body weight.

5.1.6. Associations between blood pressure, arterial stiffness, echocardiographic parameters, maximal muscular power and blood biomarkers

An inverse correlation was found between the change in tricuspid annular systolic velocity TDI and the change in mean arterial pressure (MAP before SSTP – MAP after SSTP ($r=-0.503$; $p<0.05$). The magnitude of the increase of cfPWV (cfPWV before SSTP – cfPWV after SSTP) was significantly positively related to the increase in total peroxide concentrations (TPX) (TPX before SSTP – TPX after SSTP) and oxidative stress index (OSI) (OSI before SSTP – OSI after SSTP), ($r = 0.596$, $p <0.01$; $r = 0.568$, $p < 0.01$, respectively). There were no significant relationships between the changes in arterial stiffness indices (cfPWV, crPWV, AIx, and AIx@75) or changes in maximal muscular power and other biomarkers (CK, hsCRP, lipoproteins, and NT-proBNP).

5.2. Half-marathon running study (Paper III)

5.2.1. Anthropometric, VO_2peak and food intake data

Recreational athletes were significantly older and their body mass, fat % and BMI were higher as compared to high-level athletes ($p < 0.05$) (Table 2). As expected, high-level athletes showed a higher training volume as well as better VO_2peak and faster half-marathon competition time as compared to the recreational athletes' group ($p < 0.05$). The mean heart rate percentage of the maximal HR during the race exceeded 90% in both groups. Table 8 presents descriptive information about the subjects' mean dietary intake. There were no statistically significant differences in energy intake for proteins, fats and carbohydrates between the two study groups.

Table 8. Daily nutritional intake of the subjects, x ± SD, percentages from total energy intake.

Parameter	High-level n = 11	Recreational n = 7
Total energy intake (kcal)	3229± 617	2571± 636*
Protein (g)	120.5± 23.1	97.2± 6.5*
Fat (g)	127.3± 13.5	103.6± 12.3
Carbohydrate (g)	393.8± 104.8	298.3± 33.4
Protein (% energy)	15.3± 2.0	15.5± 3.4
Fat (% energy)	34.7± 10.4	35.5± 5.7
Carbohydrate (% energy)	50.0± 3.1	49.0± 1.8

* $p < 0.05$ in comparison with high-level group.

5.2.2. Blood pressure and arterial stiffness data

Blood pressure and arterial stiffness data at baseline and during the post-competition recovery period (RECOV) are presented in Table 9. In all study subjects, resting blood pressure and arterial stiffness parameters were in normal range. During RECOV, mean brachial systolic and diastolic blood pressure showed lower values as compared to baseline data in both study groups, but the difference remained nonsignificant ($p > 0.05$). No statistically significant changes were determined in central blood pressure data. However, a slight decrease was detected in cSBP as well as in cDBP among the high-level athletes' group and in cDBP among the recreational group. Mean arterial pressure showed a slight decrease in high-level athletes but not in recreational athletes. Mean resting heart rate showed a lower value in comparison with baseline data among the recreational-level athletes' group measured in the RECOV period.

There were no statistically significant changes in arterial stiffness parameters (cfPWV, AIx and AIx@75) during the RECOV among the groups.

The between-group comparison did not reveal significant differences in blood pressure or arterial stiffness values, but high-level athletes' mean resting heart rate was significantly lower as compared to recreational athletes' mean value.

Table 9. Blood pressure and arterial stiffness data of the subjects before the competition (baseline) and during the post-competition recovery period (RECOV), x ± SD.

Parameter	Baseline	RECOV	Baseline	RECOV
	High-level n = 11		Recreational n = 7	
bSBP (mmHg)	117.2± 8.4	116.0± 9.3	122.2± 6.3	117.2± 7.4
bDBP (mmHg)	63.3 ± 9.6	60.4± 5.3	64.5± 3.5	62.7± 7.0
bPP (mmHg)	53.9± 8.3	55.6± 7.9	57.7± 5.4	54.5± 5.6
MAP	79.1± 9.4	76.3± 7.3	83.2± 6.7	82.6± 6.6
cSBP (mmHg)	96.8± 9.7	95.2± 7.4	101.0± 3.9	103.1± 9.2
cDBP (mmHg)	64.4± 10.9	60.7± 5.2	65.1± 4.1	63.4± 6.7
cPP (mmHg)	32.4± 5.3	37.9± 14.3	36.6± 4.0	39.8± 6.7
HR (beats/min)	49± 10	51± 11	57± 7*	53± 6
AIx (%)	0.18± 9.5	0.36± 11.7	3.9± 8.1	9.8± 15.1
AIx@75 (%)	–12.0± 9.4	–9.6± 11.3	–4.8± 8.8	–0.7± 15.5
cfPWV (m/s)	5.14± 0.50	5.38± 0.88	5.52± 0.58	5.41± 0.28

Abbreviations. bSBP– brachial systolic blood pressure; bDBP – brachial diastolic blood pressure; bPP – brachial pulse pressure; MAP –mean arterial pressure; cSBP – central systolic blood pressure; cDBP – central diastolic blood pressure; cPP – central pulse pressure; HR – heart rate; AIx – augmentation index; AIx@75 – augmentation index corrected for a heart rate of 75 beats per minute; cfPWV – carotid-femoral pulse wave velocity.
* $p < 0.05$ in comparison with high-level group.

5.2.3. Blood biomarkers

Subjects' baseline blood biomarkers data are presented in Table 10. In the high-level group, the WBC, hsCRP and LDH were significantly higher during the recovery period as compared to baseline data. In the recreational athletes' group, significant changes were shown in hsCRP, myoglobin, LDH and CK during the recovery period as compared to baseline data. The between-group comparison showed that recreational athletes had higher WBC at baseline, lower LDH activity and a higher insulin level during post-exercise recovery period measurements in comparison with the high-level group.

The recreational group showed a higher diene conjugates level as compared to the respective data of the high-level group in the recovery period, but no significant differences were found in respective baseline data between the two groups. No significant post-competition recovery period changes or between-group differences were found in other oxidative stress markers.

Table 10. Blood biomarkers before the competition (Baseline) and during the post-competition recovery period (RECOV), x ± SD. For hsCRP – medians, 25th percentile and 75th percentile are presented (in brackets).

Parameter	Baseline	RECOV	Baseline	RECOV
	High-level n = 11		Recreational n = 7	
WBC (10^9/L)	4.78± 0.97	5.76± 1.38$^{\#}$	6.17± 1.33*	5.81± 1.69
Hgb (g/L)	141.5± 8.7	136.9± 8.3$^{\#\#}$	147.3± 8.2	144.3± 12.1
Hct (%)	40.7± 2.9	39.3± 2.4$^{\#\#}$	42.6± 1.5	41.3± 2.5
hsCRP (mg/l)	0.29 (0.10; 0.54)	2.63 (2.03; 4.15)$^{\#}$	0.67 (0.45; 1.71)	6.58 (3.44; 10.12)$^{\#}$
Ferritin (ng/mL)	60.84± 38.8	68.25± 45.00	90.30± 46.97	99.39± 59.47
Myoglobin (ng/mL)	39.0± 9.7	86.8± 59.6$^{\#}$	42.2± 10.9	85.6± 39.3$^{\#}$
LDH (U/l)	389.1± 80.3	458.8± 72.4$^{\#\#\#}$	344.3± 24.5	385.9± 40.8*
CK (U/l)	254.7± 185.2	771.1± 380.6$^{\#\#\#}$	221.9± 77.5	797.3± 543.4$^{\#}$
Glucose (mmol/L)	4.43± 0.45	4.70± 0.23	4.51± 0.20	4.77± 0.5
Insulin (ng/mL)	3.98± 2.14	3.84± 1.21	6.12± 3.09	6.60± 4.00*
DC (microM)	37.56± 8.84	36.50± 3.62	45.63± 12.76	46.90± 10.92*
sICAM (ng/ml)	198.9± 60.99	183.1± 42.9	221.1± 17.3	210.9± 19.0
Carbonyls (nmol/mg prot)	0.13± 0.06	0.15± 0.08	0.13± 0.05	0.15± 0.08
Adiponectin (ng/ml)	5130± 1819	4681± 2030	5377± 2904	4569± 1907
ADMA (μmol/L)	0.51± 0.07	0.55± 0.06	0.53± 0.04	0.50± 0.13

Abbreviations. WBC – white blood cells; Hgb – haemoglobin; Hct – haematocrit; hsCRP – high-sensitive C-reactive protein; LDH – lactate dehydrogenase; CK – creatine kinase¸ DC – diene conjugates; sICAM – soluble intercellular adhesion molecule-1; ADMA – asymmetric dimethylarginine.
$^{\#}$ $p < 0.05$; $^{\#\#}$ $p < 0.01$; $^{\#\#\#}$ $p < 0.001$ in comparison with respective baseline value;
* $p < 0.05$ in comparison with high-level group.

5.2.4. Associations between athletes' sports level, arterial stiffness and blood biomarkers

Correlation analysis did not reveal any significant associations between the carotid-femoral pulse wave velocity (cfPWV) at baseline, during recovery period and the change of cfPWV (difference between cfPWV at baseline and cfPWV at post-competition recovery period), age, VO_2peak, BMI, baseline levels and respective changes of all blood biomarkers in the total study group as well as in the two subgroups separately ($p > 0.05$).

Regression analysis revealed that the change in pulse wave velocity was significantly dependent on the half-marathon competition race time and the athletes' higher performance level (competitive *vs* recreational). The athlete's level ($\beta = -1.010$; $t = -2.741$) and race time ($\beta = 0.780$; $t = 2.116$) explained 59% of the variance in cfPWV change ($p = 0.049$). This association remained significant after adjustment for potential confounders.

6. DISCUSSION

6.1. Supervised strength training study (Paper I, II)

Our study results revealed that the 12-week pre-seasonal strength training in elite powerlifting athletes did not cause adverse effects on blood pressure and cardiac function. Furthermore, our study findings demonstrate a beneficial effect of strength training on central blood pressure and improvement of the ventricular function of the heart. Study results showed a significant increase in total peroxide level and oxidative stress index after the supervised strength training program, which were associated with increased arterial stiffness.

The novelty of our study is that it was performed in elite-level powerlifters and evaluated the effect of a high training regimen, which is characteristic only for this target group. According to our knowledge, this is the first study in high-level strength-oriented athletes, where the improvement of cardiac function has been demonstrated.

Brachial and central blood pressure

According to previous studies, there is less evidence about the protective effect of resistance training on cardiovascular health, particularly on haemodynamic parameters, as compared to aerobic exercise (Cornelissen and Fagard, 2005; Yoon *et al.*, 2010). Dynamic resistance training has been shown to reduce BP (Cornelissen and Smart, 2013; Mancia *et al.;* 2013; Peliccia *et al.*, 2020), but there are still not enough studies available in regard to the BP-lowering effect of isometric resistance training. It is also relevant to note that previous research has shown that isometric and isotonic exercise may have a different effect on brachial and central blood pressure (Bentley *et al.*, 2018; Tanaka *et al.*, 2014; Vanhees *et al.*, 2012). In the study by Tanaka *et al.* (2014), central systolic blood pressure increased after hand-grip, but not after ergometer exercises. It has been suggested that peripheral arterial dilatation caused by isotonic exercise might reduce the amplitude of reflected pressure waves (Hashimoto *et al.*, 2018). In contrast, isometric exercise could elevate arterial resistance and thereby increase the amplitude of the radial reflected wave (Hashimoto *et al.*, 2018).

In their systematic review, Berge *et al.* (2015) showed that elevated BP and prevalence of hypertension was found in strength-trained athletes as compared to endurance athletes. Furthermore, studies have shown a significant relationship between elevated BP and left ventricular hypertrophy (Berge *et al.*, 2015; Lalande and Baldi, 2007; Rodriguez *et al.,* 2002; Volpe *et al.*, 2012).

In the present study, we found that the 12-week strength training program, which involved a combination of dynamic and isometric resistance training with the intensity of 60–90% of 1 RM, resulted in a significant decrease in brachial and also central systolic BP. In a study conducted by Tanaka *et al.* (2014), central BP was evaluated by another device, which is based on the measurement of late

systolic pressure of peripheral pressure wave. In our study, the SphygmoCor device was used, in which the estimation of central BP is based on the general transfer function (Butlin and Qasem, 2016). Moreover, our study evaluated central BP and AS in elite powerlifting athletes, whereas Tanaka *et al.* (2014) studied clinically healthy subjects from the general population. In our study, the subjects' mean brachial systolic and diastolic BP reduced by 8 and 2.4 mmHg, respectively. These results support the evidence that strength training, where the dynamic and static exercise is combined, has a significant BP-lowering effect in well-trained elite powerlifting men. In addition, in our study, the exposure to heavy training loads was supervised during the whole study period. As expected, all our study powerlifting athletes improved their maximal power, showing a significant improvement in all measured variables from 3.4 to 12.3% as compared to baseline values. The BP-lowering effect of strength training is probably related to the reduction in brachial vascular resistance and HR change, which moves the reflection point of the forward moving pulse wave more distally causing the BP to decrease (Fagard, 2006; Fecchio *et al.,* 2021; Umpierre and Stein, 2007). In our study, the reduction of the brachial and central blood pressure after the strength training program of powerlifting athletes could be explained by the change in the intensity and volume ratio of training. Presumably, the deconditioning period (two months preceding the SSTP, where subjects were instructed to avoid strength training) may be associated with a decrease in left ventricular wall thickness in highly trained athletes (Maron *et al.,* 1993). Consistent with these findings, two months of detraining have shown to reduce stroke volume, left ventricular end-diastolic diameter and cardiac output in highly trained subjects (Martin *et al.,* 1986).

Arterial stiffness

Increased arterial stiffness is a well-known risk factor for cardiovascular disease (Payne *et al*., 2010). Controversy exists in the literature with regard to the impact of resistance exercise on arterial stiffness. Available mid- to long-term studies show an adverse (Bertovic *et al.,* 1999; Collier *et al.,* 2008; Miyachi *et al.,* 2004; Miyachi, 2013), neutral (Rakobowchuk *et al.,* 2005), or beneficial (Morra *et al.,* 2014; Okamoto *et al.,* 2008) effect of resistance training on arterial stiffness. However, many of these studies were performed in relatively small samples in different age and sex groups and by a large variation in training intensity (Ceciliato *et al.,* 2020). Furthermore, most of the intervention studies were performed in untrained, recreational level, obese or pre-hypertensive individuals. In addition, most of the previously published study protocols are not comparable, especially in regard to intensive physical loads. Unfortunately, there are no data available for high-intensity strength training protocols, which is predominantly used by well-trained elite powerlifting athletes.

In our study, we did not find significant changes in cfPWV or AIx after the 12-week SSTP. It is possible that the changes in arterial stiffness were minor because we studied well-trained powerlifters, who were well-conditioned for

such strenuous training. In addition, the 12-week training period might not be enough to evaluate the absolute effect of strength training on arterial stiffness in athletes. It is also important to note, that all our study subjects were young and their arterial stiffness data stayed in normal range.

Cardiac function

Left ventricular hypertrophy is an independent cardiovascular risk factor in the general population (Levy *et al*., 1990) as well as in hypertensive patients (Koren *et al.,* 1991). In our study, there were no subjects with left ventricular hypertrophy, and we did not find a significant correlation between left ventricular mass and change in brachial BP. Our findings are consistent with previous research conducted by Spence and co-authors (2011), where the 6-month resistance training did not show an effect on left ventricular remodelling. Furthermore, it is noted that 12 weeks is a short period of time to observe structural changes in the heart (Kampus *et al.,* 2011; Yoon *et al*., 2010).

The assessment of diastolic function with echocardiography includes a complex of several variables: one of these variables, the – E/e' ratio, is the most frequently used method for left ventricular filling pressure assessment (Lang *et al.,* 2015). Adler *et al.* (2008) showed in their study that resistance training increases mitral valve E/A ratio (marker of the function of the left ventricle) and decreases deceleration time, which are both beneficial changes in terms of diastolic function. However, tissue Doppler was not used in this study. Two major factors influencing left ventricular filling pressure are left ventricular compliance and wall thickness (Mihl *et al.,* 2008). In our study, SSTP improved left ventricular filling pressure, which indicates that the change in central, not only brachial BP, might improve left ventricular diastolic function. Left ventricular diastolic function can be used as an early marker of myocardial function change. As our athletes' mean left ventricular wall thickness did not significantly change during the 12-week SSTP, it is possible that SSTP had an impact only on their left ventricular compliance. While the left ventricle pumps into the systemic circulation, which has a significant reserve to decrease resistance and increase compliance with exercise, the right ventricle empties into the pulmonary circulation, which is characterized by low resistance and high compliance already at rest. Because there is a limited capacity to accommodate the blood flow in the pulmonary circulation with exercise, the right ventricle needs to maintain the necessary output (La Gerche *et al.,* 2016). This explains the increase in the right ventricular systolic function parameter, i.e. tricuspid annular systolic velocity TDI in our study. Interestingly, we also found that an increase in right ventricular systolic function is associated with BP reduction. However, due to the design of our study, we cannot assess the causal relationship between these changes.

Blood biomarkers

In the present study, we also focused on the changes in OxS and its relationship with arterial stiffness after a 12-week SSTP. Our data revealed that the strength training program significantly increased OxS, and the magnitude of OxS was related to the increase in cfPWV.

Available studies have shown that exercise is associated with an increase of ROS generation and this may have dual effects – the elevation of ROS has high reactivity to most biological macromolecules, but growing evidence reveals that exercise-induced OxS may act as signalling molecules to mediate beneficial cellular adaptation to exercise (Guolin, 2013). Although OxS is a common response of cells or tissues to exercise, it does not seem that all subjects respond to the same exercise in a similar way. However, long-lasting or acute over-production of reactive species and high-grade OxS are considered relevant for the development of cardiovascular diseases (Madamanchi *et al.,* 2005).

In our study, an increase in OxS indices was demonstrated after the 12-week training program – significantly higher levels of TPX and OSI were found after SSTP as compared with the baseline data. In addition, a concurrent significant decrease in the TAC was observed. Previously published studies investigating the effect of resistance training on OxS parameters have reported controversial findings. Azizbeigi *et al.* (2013, 2015) reported beneficial changes in OxS after an 8-week resistance training program. Panza *et al.* (2008) and Bloomer *et al.* (2006) did not reveal significant changes in OxS after a single bout of resistance training.

However, the dissociation of OxS in the studies may be explained by the differences in the training status of study subjects and training protocols. In both studies conducted by Azizbeigi *et al.* (2013, 2015), untrained men with no previous experience of resistance training or regular physical activity were recruited, whereas Panza *et al.* (2008) recruited men engaged in recreational-level weight training, and Bloomer *et al.* (2006) studied cross-trained men.

Furthermore, exercise-induced OxS parameters may depend on the time of collecting blood samples after physical exertion. Studies by Panza *et al.* (2008) and Bloomer *et al.* (2006) were designed to test the effect of a single bout of resistance exercise on OxS indices, either a bench press or a squat, with blood samples collected up to 15 minutes or 24 hours after exercise, respectively. Azizbeigi and co-authors (2013, 2015) collected blood samples 72 hours after the completion of an 8-week resistance training program. In our study, post-training period measurements were performed 1 week after the competitions, which minimises the impact of extreme acute physical exertion.

After the SSTP, subjects' mean CK increased significantly and this mainly reflects a muscle injury response to strength training and competition. It is important to note that CK values were within the normal physiological range in athletes with high muscle mass. In our study, there were no subjects with extremely elevated CK and inflammation indices, which may confound AS variables.

There is little evidence, that strength training may improve lipoprotein profile (Braith and Stewart, 2006). Studies have shown that for improving the lipid profile, a greater physical load and beneficial changes in body composition are needed (Braith and Stewart, 2006)). In our study, we used a moderate- to high-intensity training protocol, and there was a significant beneficial effect on HDL-cholesterol but no effect on other blood lipids or fasting glucose was found. Our data are in accordance with a longitudinal study on weight training, which showed an increase in HDL-C (Hurley *et al*., 1988). However, Kelley and Kelley (2009) showed in their meta-analysis that resistance training reduces more total cholesterol, LDL-cholesterol, and triglycerides in adults, whereas the increase in HDL-cholesterol is not characteristic. Our study results reveal supportive evidence about the independent effect of strength training on HDL-C level, where subjects' body weight and body composition did not show a significant change during the strength training study. Epidemiologic evidence clearly demonstrates that HDL-C is a key component for CV risk profiling (Conroy *et al*., 2003; Rader and Hovingh, 2014).

Before the study, we hypothesized that a 12-week SSTP may increase inflammation and OxS and, and through this mechanism, alter arterial stiffness. Previously, the inflammation and OxS have been demonstrated to be associated with increased arterial stiffness (Kawamoto *et al*., 2016). Our data revealed a significant increase in OxS but not in inflammatory markers or mean pulse wave velocity in the carotid-femoral segment. However, the magnitude of the increase in cfPWV was significantly related to the increase in OxS, which was demonstrated by correlation analysis. Our study results are in accordance with the data, where the impact of OxS on arterial stiffness has been shown (Vlachopoulos *et al.,* 2010a).

Resistance training may be linked to arterial stiffening by several mechanisms. High-intensity resistance training increases the activity of the sympathetic nervous system, which stimulates vasoconstrictions and increases arterial stiffness (Fu and Levine, 2013). Alternatively, OxS associated with intensive resistance training may lead to endothelial dysfunction, which in turn could decrease the synthesis of NO and cause arterial stiffening (Higashi *et al.,* 2009).

Limitations of the study

There was no intention to involve a control group in this study due to several reasons. Firstly, it was not possible to randomize elite level powerlifting athletes as a control group for 12 weeks without systematic strength training. Secondly, appropriate matched controls were not available for the present study group's characteristics due to the circumstance that elite level powerlifters have a higher BMI and remarkably higher muscle mass than other age- and weight-matched men.

In summary, the results of our study provide more practical knowledge about the cardiovascular risk level for competitive powerlifters and other strength-oriented athletes and do not support the understanding that strength training has a deleterious impact on resting BP and cardiac function. However, the elevated oxidative

stress level after the training program may have some adverse effects on cardiovascular health in general, and this might be taken into account by the athletes who train at extremely high training loads and do not recover sufficiently.

6.2. Half-marathon running study (Paper III)

Our study results showed that there were no significant changes in haemodynamic and arterial stiffness parameters during the post-competition recovery period (18–22 hours after the half-marathon competition) in high-level and recreational athlete groups as compared to baseline data. However, the change of cfPWV was significantly dependent on race time and a higher performance level of the athlete (high-level *vs* recreational), revealing that more intensive performance may affect vascular function during the recovery period.

A significant increase was found in hsCRP, creatine kinase and LDH activity during the post-competition recovery period in both groups. No significant changes were found in oxidative stress markers in the groups during the recovery period, except for the higher diene conjugates level in recreational athletes in comparison with the high-level group.

Arterial stiffness

Previous cross-sectional and longitudinal studies have shown that aerobic training may improve arterial function (Maeda *et al.,* 2001, Saz-Lara *et al.,* 2021; Tanaka *et al.,* 2000, 2018; Tanaka and Safar, 2005). Endurance training increases the bioavailability of nitric oxide, which leads to a reduction of vascular smooth muscle cell tone and arterial function improvement (Maeda *et al.*, 2001; Miura, 2012). At the same time, inflammatory mediators and antioxidant capacity play a role in the acute effect of exercise on arterial stiffness. It has been shown that exercise intensity and volume may also have an impact on the acute response to arterial stiffness (Ferreira *et al.,* 2006; Saz-Lara *et al.,* 2021; Sugawara *et al.,* 2006).

Recent meta-analysis has revealed that acute aerobic exercise reduces arterial stiffness, mainly between 30 to 60 min after exercise (Saz-Lara *et al.,* 2021), but there is less data available for a longer recovery period, i.e. up to 24 hours after exercise. Furthermore, there is less data about excessive high-intensity physical loads, which exceed the anaerobic threshold and may have a different effect on arterial stiffness and should be differentiated from conventional aerobic activity. In our study, all the subjects were experienced aerobically well-trained runners who participated in a half-marathon competition, in which the running intensity is higher than in a marathon or ultra-marathon race. Competitive athletes showed better half-marathon race time than recreational athletes, while the intensity of the race was very high among both groups, which was reflected by mean heart rate in absolute values as well as in relative indices (% of the maximal HR exceeded 90% of the individual maximal HR) during the competition.

According to the available data, there is a complex relationship between acute intensive exercise and arterial stiffness. In marathon runners, a decrease in wave reflection as well as in aortic and brachial blood pressure was observed immediately after the competition, whereas arterial stiffness did not significantly change (Vlachopoulos *et al.,* 2010b). Previous data suggest that both the timing of PWV measurement and the type of exercise should be considered when analysing the effect of acute physical exercise on arterial stiffness. It is important to note that there is no available data about the late post-competition recovery period (i.e. more than 90 min), which may provide a more comprehensive view and understanding about the recovery process and return to the habitual training process, especially in highly trained athletes.

Our study did not reveal any significant post-competition recovery period changes in brachial and central blood pressure or in AIx and AIx@75 in comparison with baseline values among either of the groups. No differences were found between the groups in any measured parameters but, as expected, high-level athletes had a significantly lower resting HR as compared to recreational athletes. A relatively low mean resting HR of both groups is very characteristic to aerobically-trained subjects and reflects good cardiovascular adaptation to the sports activity. In our study, regression analysis revealed that the change in cfPWV (difference between cfPWV at baseline and cfPWV during post-competition recovery period) was independently associated with half-marathon race time and the athlete's level of training (competitive *vs* recreational). Thus, a mild but statistically non-significant increase in cfPWV was observed in the high-level group and a minimal decrease in cfPWV was noted in the recreational group. It could be hypothesized that a half-marathon run may have a different effect on arterial stiffness depending on the level of sports performance of an athlete. However, larger-scale studies are required to confirm this hypothesis.

Blood biomarkers

Monitoring the fasting blood glucose and insulin level describe the athletes' nutritional adequacy of their diet (Lee *et al.,* 2017). In our study, the fasting insulin level was higher in recreational athletes as compared to high-level, where a significant difference was found during the recovery period. These data reveal that endurance training may have a beneficial effect on insulin sensitivity. It has been demonstrated that physically active persons have lower baseline hsCRP values in comparison with sedentary ones (Fernandez *et al.,* 2018; Pihl *et al.,* 2003), which is considered to be associated with lower cardiovascular risk level. However, the effect of acute physical load and recovery period response on hsCRP has not been sufficiently studied. The present half-marathon run study data showed that hsCRP increased significantly during the post-competition recovery period in recreational athletes and in the high-level group. Similarly, our study results are in accordance with previous studies (Kampus *et al*., 2008; Kasapis and Thompson, 2005; Weight *et al*., 1991), where an increase of CRP level was observed after strenuous physical load. It seems that exercise intensity

also plays an important role, because no significant increase has been observed after light to moderate intensity aerobic exercise (Markovitch *et al.,* 2008). In summary, exercise-related acute inflammatory response may be affected by the duration and intensity of physical activity. However, regular exercise has been found to reduce chronic inflammation (Rose *et al.,* 2021).

High-grade oxidative stress has also been associated with strenuous physical exercise. Extreme aerobic or anaerobic exercise may contribute to excessive pro-oxidant production through the mechanical injury of skeletal muscle, ischemia and reperfusion injury, disruption of the mitochondrial electron transport chain and other mechanisms (Powers *et al.,* 2016). Conversely, long-term moderate intensity exercise increases antioxidant defences of the body and maintains redox homeostasis (Caimi *et al.,* 2009). The present study demonstrates that the diene conjugates (DC) level was lower in the high-level group in comparison with the recreational athletes. Similarly, serum levels of DC were lower in professional road-racing cyclists in comparison with healthy male volunteers (Pittaluga *et al.,* 2006). In our study, no significant changes were found in pre- and post-competition DC levels in elite or recreational athletes, which might suggest an adequate adaptation of the antioxidant defence in the study subjects (Cases *et al.,* 2006).

Limitations of the study

Our half-marathon running study has a number of limitations. Firstly, according to standardization procedures for the measurement of arterial stiffness with applanation tonometry (Laurent *et al.*, 2006), immediate post-competition measurements during the early stage of recovery were not applicable. However, we consider the late recovery data more valid for the recovery response description due to the elimination of other confounding factors – blood glucose fluctuation, increased inflammation and oxidative stress as well as elevated parasympathetic tone. Secondly, post-competition recovery measurements were performed 18 to 22 hours after the marathon competition, which is not a very limited time point for all participants. The main reason for this is a time-consuming measurement procedure (approximately 20 minutes per one subject). Furthermore, our case-control comparison reflects only descriptive data for the post-competitive recovery period, not a causal effect of the intensive physical load and arterial stiffness.

CONCLUSIONS

1. The 12-week pre-seasonal strength training program had a beneficial effect on brachial and central systolic blood pressure in male elite powerlifting athletes. The strength training program improved left and right ventricular cardiac function. There was no effect of strength training on arterial stiffness. (Paper I)

2. The 12-week pre-seasonal strength training program showed a significant increase in the total peroxide level and oxidative stress index, which were associated with increased arterial stiffness. A beneficial effect of the strength training program was shown on high-density lipoprotein cholesterol concentration. (Paper II)

3. Generally, half-marathon running did not affect arterial stiffness parameters in competitive and recreational male athletes during the post-competition period. However, deteriorated arterial stiffness occurred in athletes with the fastest race time and higher performance level, whereas a mild systemic inflammatory response not related to the impairment of arterial stiffness may have occurred. (Paper III).

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SUMMARY IN ESTONIAN

Kolmekuulise jõutreeningu programmi ja poolmaratoni jooksmise mõju arterite jäikusele ning vere biomarkeritele hästitreenitud meessportlastel

Sissejuhatus

Arterite jäikus iseloomustab arterite laienemisvõimet vererõhu mõjul ning nende jäigastumise korral tõuseb südame vasaku vatsakese järelkoormus, mis omakorda põhjustab vasaku vatsakese ülemäärast hüpetroofiat. Pulsilaine leviku kiiruse hindamist aordis peetakse tänapäeval arteriaalse jäikuse hindamise kuldseks standardiks ning selle kiirenemist üheks varajasemaks ebasoodsaks ilminguks kardiovaskulaarse haiguse korral.

Varasemad uuringud on näidanud, et kehalisel aktiivsusel on arterite jäikusele soodne mõju. Paraku enamus neist uuringuist on teostatud treenimata, ülekaalulistel või kõrge vererõhuga isikutel, rakendades kerge kuni mõõduka intensiivsusega aeroobseid koormusi. Tippsportlaste kehaline koormus on väga suur, ületades oluliselt kehalise aktiivsuse üldisi soovitusi. Selle kohta, kuidas mõjub arterite jäikusele jõutreening, on vähe andmeid. Samas on jõualade sportlastel võrreldes vastupidavusalade esindajatega täheldatud sagedamini kõrgvererõhktõbe ja südame ülemäärast hüpertroofiat.

Aeroobsetel koormustel on südame-vereringesüsteemile üldiselt soodne toime. Teisalt on teada, et vastupidavusaladel võib võistluste ajal pingutuse intensiivsus olla väga kõrge, ületades anaeroobse läve. Selline olukord esineb näiteks poolmaratoni jooksuvõistlustel. Seega on õigustatud küsimus, kas korduvad jõutreeningule omased intensiivsed koormused või ühekordne tugev vastupidavuslik pingutus võistlustel ning sellele järgnev puudulik taastumine võivad suurendada sportlaste kardiovaskulaarse riski taset, sealhulgas mõjudes negatiivselt arterite jäikusele. Arterite seisundit võivad mõjutada ka koormusest tingitud põletikulised protsessid ning oksüdatiivse stressi taseme tõus.

Uurimistöö eesmärgid

Antud doktoritöö üldiseks eesmärgiks oli hinnata erineva suunitluse ja kestusega kehalise koormuse mõju arterite jäikusele ning leida selle seoseid vere biomarkeritega hästitreenitud meessportlastel.

Uuringu konkreetsemateks eesmärkideks oli hinnata:

1. kolmekuulise jõutreeningu programmi mõju arterite jäikusele, vererõhule ning südame funktsioonile tipptasemel meessoost jõutõstjatel;
2. kolmekuulise jõutreeningu programmi mõju oksüdatiivse stressi näitajatele ja lipiidide tasemele tipptasemel meessoost jõutõstjatel ning hinnata nende seoseid arterite jäikuse muutustega;

3. poolmaratoni jooksmise mõju arterite jäikusele ning vere biokeemilistele näitajatele taastumisperioodil võrdlevalt kõrge treenitusega sportlastel ning harrastustasemel meesjooksjatel.

Uuritavad ja metoodika

Esimese uuringu raames viidi läbi spetsiaalse programmiga juhendatud jõutreening 19-l meessoost eliittasemel jõutõstjal (vanus 28,2±6,1 aastat, pikkus 179,2±5,9 cm, kehamass 99,9±16,5 kg, KMI 31,2±5,1, keha rasvaprotsent 16,1±7,9). Treeningud viidi läbi 12-nädalasel perioodil, 4 korda nädalas intensiivsusega 60–90% ühest kordusmaksimumist, ühe treeningu kestus oli 90–120 minutit. Treeningprogrammi järgselt osalesid kõik uurimisalused Eesti Meistrivõistlustel jõutõstmises.

Teises uuringus osalesid kokku 18 meessoost jooksjat (vanus 29,9±6,3 aastat, pikkus 180,0±6,0 cm, kehakaal 74,2±11,1 kg, KMI 22,9±2,7, keha rasvaprotsent 11,7±4,5), kes jaotati sportliku taseme järgi kahte gruppi: 1) kõrge treenitusega jooksjad, kes kuulusid Eesti koondisesse või olid koondise kandidaadid, n=11; 2) ja harrastustasemel jooksjad, n=7. Uurimisalused läbisid ühekordselt poolmaratoni võistluse (21 km).

Kõik uuringus osalejad olid mittesuitsetajad, neil ei olnud ägedaid ega kroonilisi haigusi ning nad ei tarbinud ravimeid. Samuti oli uuritavatel palutud eelneva kahe kuu jooksul mitte tarbida toidulisanditena antioksüdante. Kõik uuringus osalejad andsid kirjaliku nõusoleku uuringus osalemiseks ning uuring oli heaks kiidetud Tartu Ülikooli inimuuringute eetikakomitee poolt.

Uuritavate vererõhu ja arterite jäikuse hindamine kui vereproovide kogumine veeniverest viidi läbi hommikul kella 8–10 vahelisel ajal pärast üleööpaastu. Brahiaalset (perifeerset) vererõhku mõõdeti istuvas asendis mittedomineerival käel (Omron M4-I, Omron Healthcare Europe BV) peale 15-minutilist rahulolekut. Arterite jäikuse uuring teostati mitteinvasiivsel meetodil, kasutades aparatuuri SphygmoCor (7.1, AtCor Medical, Austraalia). Aplanatsiooni tonomeetriga registreeriti pulsilaine radiaalarteril ning, kasutades valideeritud ülekandefunktsiooni, arvutati tsentraalse pulsilaine parameetrid: augmentatsiooniindeks (AIx, AIx75), mis iseloomustab pulsilaine tagasipeegeldumist ning tsentraalse vererõhu väärtused. Lisaks arvutati pulsirõhu amplifikatsioon, mis iseloomustab arterite jäikust ja pulsilainete tagasipeegeldumist. Pulsilaine kiiruse leviku hindamiseks registreeriti pulsilained ühisunearterilt ja -reiearterilt samaaegselt elektrokardiogrammi registreerimisega. Pulsilaine tekke ajastuse ja pulsilaine poolt läbitud vahemaa andmete põhjal arvutati pulsilaine leviku kiirus nimetatud segmendis (cfPWV). Veenivere analüüsid (haemogramm, erinevad biokeemilised ja oksüdatiivse stressi näitajad) võeti peale arterite jäikuse uuringut ning analüüsiti Sihtasutus Tartu Ülikooli Kliinikumi Ühendlaboris ning Tartu Ülikooli biokeemia instituudis. Antropomeetrilised mõõtmised, arterite jäikuse ning vere biomarkerite hindamine toimus jõutõstjate uuringus üks nädal enne treeningutega alustamist ja üks nädal peale 12-nädalast treeningperioodi ja selle

lõpus toimunud võistlust. Poolmaratoni uuringus teostati eelpoolninmetatud mõõtmised üks nädal enne võistlust ning 18–22 tunni vältel peale võistluse lõppu (taastumisperioodil).

Uuritavatel viidi läbi koormustest liikurrajal (Viasys/Jaeger LE300 C, Viasys Healthcare GmbH, Hoechberg, Germany) kõrgeima hapnikutarbimise võime hindamiseks, kasutades otsest gaasianalüüsi meetodit (MasterScreen CPX, Viasys Healthcare GmbH, Hoechberg, Germany). Koormustest ning toitumise kvantitatiivne analüüs (NutriData, versioon 8) viidi läbi üks kord enne uuringutega alustamist.

Jõutõstjate uuringus hinnati üks nädal enne ja üks nädal pärast 12-nädalast treeningperioodi jõunäitajate võimsust kahe testiga: poolkükist üleshüppel ja lamades surumisel, kasutades selleks oma keharaskusega kangi (Ergotest Innovation a.s., Porsgrunn seadet) ning viidi läbi ehhokardiograafiline uuring (Sonos7500, Philips Medical Systems, Inc., Highland Heights, OH, USA).

Uuringuandmete statistiliseks analüüsiks kasutati SPSS for Windows 22.0 programmi (IBM Corporation) jõutõstjate puhul ja SPSS for Windows 25.0 programmi (SPSS Inc.) jooksjate puhul.

Tulemused

Jõutõstjate uuringus langes pärast treeningperioodi oluliselt uuritavate keskmine perifeerne ja tsentraalne süstoolne vererõhk (132,3±8,8 *vs* 124,3±8,7 mmHg, p=0,002 ja 110,1±7,7 *vs* 104,5±8,7 mmHg, p=0,008), samas uuritavate kehamass ja keha rasvaprotsent oluliselt ei muutunud. Ootuspäraselt paranesid uuritavatel lihasvõimsust näitavad testid − poolkükist üleshüpe ja lamades surumine (p<0.001), seda 3,4% kuni 12,3% ulatuses. Uuritavate arterite jäikust peegeldavates keskmistes parameetrites (cfPWV, AIx, AIx75) olulist muutust ei täheldatud. Ühelgi uuringus osalenud jõustõstjal ei sedastatud vasaku vatsakese hüpertroofiat ning olulist statistilist seost vasaku vatsakese massi ja vererõhu vähenemise vahel ei olnud. Jõutreening parandas oluliselt uuritavate vasaku vatsakese diastoolset funktsooni (E/e' – varajase mitraalse sissevoolu kiiruse ja mitraalrõnga varajase diastoolse kiiruse suhte paranemise näol), mis näitab, et tsentraalse, mitte ainult perifeerse vererõhu muutus võib parandada vasaku vatsakese diastoolset funktsiooni. Lisaks paranes oluliselt uuritavate parema vatsakese diastoolset funktsiooni peegeldav näitaja TDI. Käesolev uuring on teadaolevalt esimene uuring, kus on seoses jõutreeningutega näidatud nii parema kui vasaku vatsakese funktsiooni paranemist.

Uuritavatel täheldati pärast 12-ne nädalast treeningperioodi oluliselt kõrgemat HDL-C taseme tõusu võrreldes uuringueelse tasemega (p<0.005), mida võib pidada oluliseks soodaks nihkeks kardiovaskulaarse riski langetamisel. Hinnates oksüdatiivse stressi näitajaid, olid jõutreeningute programmi järgselt uuritavate vereseerumi peroksiidide üldkontsentratsioon (TPX) ja oksüdatiivse stressi indeks (OSI) oluliselt kõrgemad ning totaalne antioksüdantne mahtuvus madalam kui enne treeningutega alustamist. Korrelatsioonianalüüs näitas, et cfPWV suurem muutus oli oluliselt seotud TPX ja OSI suurenemisega.

Poolmaratoni uuringus oli võistlusjärgsel taastumisperioodil mõlema uuringugrupi (kõrge treenitusega sportlased *vs* harrastajad) keskmine perifeerne vererõhk küll madalam kui enne võistlust, kuid see langus jäi statistiliselt ebaoluliseks ($p>0,05$). Tsentraalse diastoolse vererõhu osas täheldati mõningast langust mõlema rühma seas, kuid muutus jäi statistiliselt ebaoluliseks. Võrreldes võistluseelse tasemega, ei esinenud võistlusjärgsel taastumisperioodil statistiliselt olulist muutust arterite jäikust iseloomustvates näidajates (cfPWV, AIx, AIx75). Põletikulistes näitajates täheldati võistlusjärgselt mõlemas uuringurühmas mõningast tõusu, viidates tugeva koormuse järgsele põletikulisele foonile. Oksüdatiivse stressi markerites ei leitud olulisi võistlusjärgseid muutusi ega olulisi rühmadevahelisi erinevusi (va taastumisperioodi kõrgem dieenkonjugaatide tase harrastustasemega sportlastel). Regressioonanalüüs näitas, et pulsilaine kiiruse muutus sõltus oluliselt poolmaratoni läbimise ajast ja sportlase tasemest, näidates pulsilaine kiiruse mõningast halvenemist parema võistlustulemuse ja kõrgema sportliku taseme korral.

Järeldused

1. Kolmekuuline jõutreening alandas oluliselt kõrge treenitusega meessoost jõutõstjate perifeerset ja tsentraalset süstoolset vererõhku, samas arterite jäikus ei muutnud. Uuritavatel rakendatud jõutreeninguprogramm mõjutas soodsalt südame vasaku ja parema vatsakese funktsiooni.
2. Kolmekuuline jõutreening suurendas kõrge treenitusega jõutõstjate oksüdatiivse stressi näitajaid ning selle oluliselt kõrgem tase seostus ebasoodsama nihkega arterite jäikuses. Jõutreeningu programmi järgselt ilmnes uuritavatel soodne muutus HDL-kolesterooli tasemes.
3. Poolmaratoni jooksuvõistluse läbimine erineva treenitusega jooksjatel ei avaldanud olulist mõju arterite jäikuse näitajatele võistlusjärgsel taastumisperioodil. Mõningane arterite jäikuse halvenemine seostus kõrgema treenituse taseme ja võistlusdistantsi läbimise lühema ajaga, mis ei sõltunud põletikumarkerite tõusust.

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Peamised uurimisvaldkonnad:

Arterite jäikuse ja hemodünaamika hindamine sportlastel ja füsioteraapia.

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