

Research article

Effect of Half-Marathon Running on Arterial Stiffness and Blood Biomarkers in High-Level and Recreational Male Athletes

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Abstract

There is no clear understanding about the effect of intensive physical load on arterial stiffness and related biomarkers. The aim of this study was to evaluate the effect of half-marathon running on arterial stiffness and blood biomarkers during post-competitive recovery period in competitive and recreational male athletes. Eleven high-level long-distance runners (27.1 ± 4.8 yrs) and seven recreational athletes (34.3 ± 6.1 yrs), who participated in a half-marathon run were examined. Blood biomarkers and arterial stiffness (SphygmoCor 7.1) were measured at baseline and at 18 to 22 hours after the competition. There were no statistically significant changes between the groups in augmentation index (AIx, AIx@75) or pulse wave velocities at carotid-femoral segment (cfPWV) during recovery period. Between-group comparison did not reveal significant differences in blood pressure and arterial stiffness values at baseline and during recovery period. The change of cfPWV (difference between cfPWV at baseline and cfPWV during post-competitive recovery period) was significantly dependent on race time and sports level of the athlete (high-level or recreational). A significant increase was found in hsCRP, creatine kinase and LDH activity during the post-race period in both groups. No significant changes were found in oxidative stress markers in the groups after the race except for higher diene conjugates level in recreational athletes in comparison with the high-level group during recovery period. Our study results showed that half-marathon competition did not cause any significant changes in arterial stiffness parameters during the recovery period. However, the change in cfPWV was independently associated with half-marathon race time and the athlete's level of training revealing a mild increase of arterial stiffness in high-level athletes and athletes with a faster race time.

Key words: Arterial compliance, elite athletes, physical load, oxidative stress, cardiovascular risk.

Introduction

Regular physical activity plays an independent and significant role in cardiovascular risk reduction. Studies have shown that subjects who exercise regularly have a better profile of biochemical risk markers, including oxidative stress (OS) and other risk factors (overweight, hyperten-

sion, etc.) (Pihl et al., 2003; 2006; Gielen et al., 2010). Furthermore, one of the important protective effects of physical activity on the cardiovascular disease (CVD) mechanism may account for better endothelial function (Gielen et al., 2010; Ashor et al., 2014; Tanaka et al., 2018). The dysfunction of endothelium results in the increase of arterial stiffness, which is hypertension-mediated organ damage (Williams et al., 2018) and independently associated with an elevated CVD risk. Non-invasive pulse wave analysis 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 (Parvathaneni et al., 2002, McEniery et al., 2005).

Published data have shown that regular aerobic exercise is associated with better arterial stiffness indices in comparison with sedentary controls (Laurent et al., 2011; Tanaka, 2019). In general, the beneficial effects are mainly contributed to small and moderate doses of aerobic exercise in healthy subjects (Kobayashi et al., 2020). However, some studies have shown impaired arterial compliance in marathon or ultra-marathon runners, which may be accounted to the chronic response of high-volume training and competitions (Vlachopoulos et al., 2010; Burr et al., 2012, Burr et al., 2014). In contrary, Müller et al. (2017) showed no detrimental effect 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 OS. Several studies have evaluated acute response to arterial stiffness after short-term physical load (Pierce et al., 2018; Saz-Lara et al., 2021). However, most 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). Furthermore, very little data are available in regard to the recovery period over 60 min to 24 hours. A recent meta-analysis has demonstrated

(Saz-Lara et al., 2021), that the acute effect of different types of exercise (aerobic exercise, resistance training, interval training) on PWV varies significantly within 24 hours after exercise.

It has been shown that acute profound exercise-related oxidative stress and inflammation may impair endothelial function and arterial stiffness (Scherr et al., 2011; La Gerche, 2016). Regarding post-exercise late recovery period (24 hours after physical load), our previous study showed that changes in arterial elasticity indices were significantly associated with the exercise-related inflammatory markers and furthermore, changes in inflammatory and arterial stiffness were dependent on the athletes' aerobic fitness level showing better recovery data in those who had higher fitness level (Kampus et al., 2008).

The inconsistent results of previous studies regarding the response to exercise may arise from the different timing of measurements after exercise and a heterogeneous fitness level of the subjects. There are limited data on the effect of intensive exercise at competition level on arterial stiffness and blood biomarkers in elite runners who have been at an upper level of sports activity for several years. Moreover, there is no clear understanding and no systematic data on late post-competitive recovery period in case of intensive physical load on arterial stiffness.

The purpose of this study was to evaluate the effect of half-marathon running on arterial stiffness and blood biomarkers during post-competitive recovery period in competitive and recreational male athletes.

Methods

Subjects and study protocol

Eighteen voluntary endurance-trained male subjects who participated in a half-marathon run (21 km) in Estonia were examined. Subjects were divided into two groups: high-level (well-trained long-distance runners, who were members or candidates of the national team), $n = 11$, and recreational athletes (who exercised regularly for recreational purposes), $n = 7$. One week preceding the competition, the subjects passed a physical examination and a maximal cardiopulmonary exercise test on a treadmill. The athletes provided a written consent confirming their willingness to adhere to the study protocol. All the subjects were asked not to drink alcohol during the study as well as advised to maintain their usual diet. Athletes' training habits and training volume as well as sleeping time and sleeping quality during the study were asked for. Participants' food intake for 5 consecutive days was analyzed (3 days before the half-marathon race, on the day of the competition and one day after the race) using the NutriData Estonian food composition database, version 8. The subjects did not use medications and antioxidant supplements at least two months prior to the study.

Venous blood samples and arterial stiffness measurements were taken 24 hours prior to the competition (baseline) and 18-22 hours after the competition (post-exercise recovery period, RECOV). The study protocol was approved by the Medical Ethics Committee, University of Tartu, No 162/T-12. Informed written consent was

obtained from each athlete in accordance with principles of the Declaration of Helsinki.

Anthropometric measurements

The subjects' height and weight were determined by the Martin metal anthropometer (± 0.1 cm) and clinical scales (± 0.05 kg), respectively. The body mass index (BMI) was calculated ($\text{kg}\cdot\text{m}^{-2}$). Body fat percentage was measured by dual-energy X-ray absorptiometry using the DPX-IQ densitometer (Lunar Corporation, Madison, WI, USA).

Cardiopulmonary exercise test

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). For detecting VO_2peak , 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. Gas analysis data were automatically calculated for every 30-second period. 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. HR 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 running, HR was also recorded by the sport-tester and the mean results of the half-marathon competition (time, maximum HR) are presented in Table 1.

Laboratory procedures

Venous blood samples were drawn from the antecubital vein in the morning between following an overnight fast and abstinence from any medication, tobacco, alcohol, tea, and coffee. Venous blood samples were drawn before the arterial stiffness measurement.

QBC Autoread Plus autoanalyzer (QBC Diagnostics, Inc., USA) was used to assess white blood cell (WBC) count, haemoglobin (HGB) and haematocrit (Hct) in whole blood. Lactate dehydrogenase (LDH), creatine kinase (CK), myoglobin, high-sensitivity C-reactive protein (hsCRP), ferritin, glucose, and insulin were measured by standard laboratory methods. All the measurements were performed at the Laboratory Department of the Tartu University Hospital. For other parameters, the blood samples were centrifuged within 15 minutes after collection at 3000 rpm to obtain serum that was frozen at -80°C until further analysis.

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.

Table 1. Mean anthropometric, training, and aerobic capacity characteristics of the subjects. Data presented as means \pm SD and percentage (%).

Parameter	Total n = 18	High-level n = 11	Recreational n = 7
Age (years)	29.9 \pm 6.3	27.1 \pm 4.8	34.3 \pm 6.1*
Height (m)	1.80 \pm 0.06	1.81 \pm 0.04	1.80 \pm 0.09
Weight (kg)	74.2 \pm 11.1	69.9 \pm 5.2	80.0 \pm 14.6 *
BMI (kg·m ⁻²)	22.9 \pm 2.7	21.4 \pm 1.2	25.2 \pm 2.9 **
Fat percentage (%)	11.7 \pm 4.5	10.0 \pm 3.2	14.5 \pm 5.2 *
Training experience (years)	12.4 \pm 8.6	10.0 \pm 6.2	16.2 \pm 10.8
Training volume (km·month ⁻¹)	252 \pm 114	296 \pm 117	172 \pm 48 *
VO ₂ peak (l·min ⁻¹)	4.67 \pm 0.48	4.91 \pm 0.31	4.28 \pm 0.44 **
VO ₂ peak/kg (ml·kg ⁻¹ ·min ⁻¹)	63.8 \pm 10.5	70.0 \pm 5.3	54.0 \pm 9.3 ***
Mean competition time (minutes)	84.9 \pm 12.7	76.4 \pm 5.6	95.9 \pm 11.6 ***
Maximal HR (beats·min ⁻¹)	188 \pm 7	189 \pm 8	187 \pm 5
Mean HR during the half-marathon (beats·min ⁻¹)	175 \pm 7	177 \pm 9	172 \pm 4
% of the maximal HR during the half-marathon	93.1 \pm 2.9	93.9 \pm 2.5	92.1 \pm 3.2

* p < 0.05; ** p < 0.01; *** p < 0.001 in comparison with high-level group. BMI – body mass index; VO₂peak – the highest level of oxygen uptake; HR – heart rate.

After centrifugation and absorbance of heptane, the fraction was measured spectrophotometrically at an absorbance maximum between 220 and 250 nm. 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).

Protein carbonyls were determined as described previously (Bogdanovic et al., 2001). After homogenization (1:10 in 10 mM Hepes buffer containing 0.5 μ g/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%), allowed to stand 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 concentration of adiponectin was analyzed by a quantitative sandwich enzyme immunoassay technique, using commercially available kits (R&D Systems, Minneapolis, MN, U.S.A.). The plasma level of asymmetric dimethylarginine (ADMA) was determined by a competitive ELISA using a commercially available kit (DLD Diagnostika®, Hamburg, Germany). Measurements were performed at the Institute of Biomedicine and Translational Medicine (University of Tartu).

Blood pressure and arterial stiffness measurements

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

Sphygmocor apparatus (SphygmoCor, version 7.1, AtCor Medical, Australia) was used to assess arterial stiffness. A high fidelity micromanometer (SPT-301B;

Millar Instruments®, Texas, USA) was employed to record the peripheral pressure waveforms from the radial/femoral arteries. The corresponding ascending aortic waveforms were then generated by using a transfer function in accordance with established guidelines (Laurent 2006). Central hemodynamics (cSBP, cDBP), augmentation index (AIx), pulse wave velocities at carotid-femoral segment (i.e. aortic stiffness) (cfPWV) were calculated. 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 2006). 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.

All measurements were made in triplicate by one trained investigator, and the mean values were used in subsequent analysis. Brachial and central blood pressure as well as arterial stiffness measurement method is described in more detail previously (Jürgenson et al., 2019).

Statistical analysis

Statistical analysis was performed with SPSS for Windows software, version 25.0 (SPSS Inc., Chicago, Illinois, USA). All data were checked for normal distribution using the Kolmogorov-Smirnov test. Data are expressed as means and standard deviation (SD) for the normally distributed data and as medians and 25th and 75th percentage for skewed data. The data were analyzed using the paired-samples t-test. For skewed data distribution, the Mann-Whitney U-test and for nonparametric analysis for related samples, the Wilcoxon on Rank test was used. The Pearson or Spearman correlations 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. Statistical significance was defined as p < 0.05.

Results

Anthropometric, VO₂peak and food intake data

The baseline characteristics of mean anthropometric data, training, the highest level of oxygen uptake and half-

marathon competition (race time and heart rate data) of the subjects are presented in Table 1. Recreational athletes were significantly older and their body mass, BMI and fat percentage were higher as compared to high-level athletes ($p < 0.05$). High-level athletes showed a higher training volume as well as better VO_{2peak} and faster half-marathon race time as compared to the recreational athletes' group ($p < 0.05$). The mean heart rate % of the maximal HR during the race exceeded 90% in both groups.

Table 2 presents descriptive information about the subjects' mean dietary intake. There were no statistically significant differences in relative energy intake (%) for proteins, fats and carbohydrates between the study groups. Subjects' average sleeping time was 8.1 hours before the baseline measurements and 7.2 hours before recovery period measurements and none of the subjects reported disturbed sleeping.

Table 2. Daily nutritional intake of the subjects. Data presented as means \pm SD and percentages (%).

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

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

Blood pressure and arterial stiffness data

The hemodynamic indices at baseline and during the post-competitive recovery period (RECOV) are presented in Table 3. Resting blood pressure and arterial stiffness parameters were in normal range in all study subjects. During RECOV, brachial blood pressure (bSBP, bDBP) showed lower values as compared to baseline data in both study groups, but the decrease remained nonsignificant ($p > 0.05$). No 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 group and in cDBP among the recreational group. MAP

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 post-exercise recovery period (>0.05).

There were no statistically significant changes in cfPWV, AIx and AIx@75 during the RECOV among the groups (Table 3).

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.

Biochemical and oxidative stress data

Subjects' baseline biochemical data are presented in Table 4. In the high-level group, more significant changes were detected in the baseline as compared to post-competitive recovery period data. The WBC, hsCRP and LDH increased significantly during the RECOV as compared to baseline data. In the recreational athletes' group, significant changes were established in hsCRP, myoglobin, LDH and CK during RECOV as compared to baseline data. Between-group comparison showed that recreational athletes had higher WBC at baseline, lower LDH activity and higher insulin level during post-exercise recovery period measurements in comparison with the high-level group.

Oxidative stress indices of the two groups are presented in Table 5. Diene conjugates level showed higher values among the recreational group as compared to the respective data of the high-level group, but no significant differences were found in respective RECOV period data. No significant post-race changes or between-group differences were found in other oxidative stress markers.

Correlation analysis did not show any significant associations between the cfPWV at baseline, during RECOV period and the change of cfPWV (difference between cfPWV at baseline and cfPWV at post-exercise 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 ($p > 0.05$).

Table 3. Blood pressure and arterial stiffness data of the subjects before the race (Baseline) and during post-competition recovery period (RECOV). Data presented as means \pm SD.

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

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

Regression analysis revealed that the change in pulse wave velocity was significantly dependent on half-marathon competition race time and the athlete's level (competitive vs recreational). The athlete's level ($\beta = -$

1.010; $t = -2.741$) and race time ($\beta = 0.780$; $t = 2.116$) explain 59% of the variance in cfPWV change ($p = 0.049$). This association remained significant after adjustment for potential confounders.

Table 4. Biochemical and hormonal parameters before the race (Baseline) and during post-competition recovery period (RECOV). Data presented as means \pm SD, for hsCRP medians, 25th percentile and 75th percentile are presented.

Parameter	Baseline	RECOV	Baseline	RECOV
	High-level n = 11		Recreational n = 7	
WBC ($\times 10^9/L$)	4.78 \pm 0.97	5.76 \pm 1.38 #	6.17 \pm 1.33 *	5.81 \pm 1.69
Hgb (g/L)	141.5 \pm 8.7	136.9 \pm 8.3 ##	147.3 \pm 8.2	144.3 \pm 12.1
Hct (%)	40.7 \pm 2.9	39.3 \pm 2.4 ##	42.6 \pm 1.5	41.3 \pm 2.5
hsCRV (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 #
Ferritine (ng/mL)	60.84 \pm 38.8	68.25 \pm 45.00	90.30 \pm 46.97	99.39 \pm 59.47
Myoglobin (ng/mL)	39.0 \pm 9.7	86.8 \pm 59.6 #	42.2 \pm 10.9	85.6 \pm 39.3 #
LDH (U/l)	389.1 \pm 80.3	458.8 \pm 72.4 ###	344.3 \pm 24.5	385.9 \pm 40.8 # *
CK (U/l)	254.7 \pm 185.2	771.1 \pm 380.6 ###	221.9 \pm 77.5	797.3 \pm 543.4 #
Glucose (mmol/L)	4.43 \pm 0.45	4.70 \pm 0.23	4.51 \pm 0.20	4.77 \pm 0.5
Insulin (ng/mL)	3.98 \pm 2.14	3.84 \pm 1.21	6.12 \pm 3.09	6.60 \pm 4.00 *

$p < 0.05$; ## $p < 0.01$; ### $p < 0.001$ in comparison with respective baseline value; * $p < 0.05$; in comparison with high-level group. WBC – white blood cells; Hgb – haemoglobin; Hct – haematocrit; hsCRV – high-sensitive C-reactive protein; LDH – lactate dehydrogenase; CK – creatine kinase.

Table 5. Oxidative stress parameters before the race (Baseline) and during post-competition recovery period (RECOV). Data presented as means \pm SD.

Parameter	Baseline	RECOV	Baseline	RECOV
	High-level (n = 11)		Recreational (n = 7)	
DC (microM)	37.56 \pm 8.84	36.50 \pm 3.62	45.63 \pm 12.76	46.90 \pm 10.92 *
sICAM (ng/ml)	198.9 \pm 60.99	183.1 \pm 42.9	221.1 \pm 17.3	210.9 \pm 19.0
Carbonyls (nmol/mg prot)	0.13 \pm 0.06	0.15 \pm 0.08	0.13 \pm 0.05	0.15 \pm 0.08
Adiponectin (ng/ml)	5130 \pm 1819	4681 \pm 2030	5377 \pm 2904	4569 \pm 1907
ADMA (μ mol/L)	0.51 \pm 0.07	0.55 \pm 0.06	0.53 \pm 0.04	0.50 \pm 0.13

* $p < 0.05$; in comparison with high-level group; DC – diene conjugates; sICAM – soluble intercellular adhesion molecule-1; ADMA – asymmetric dimethylarginine

Discussion

Our study results showed that there were no significant changes in hemodynamic and arterial stiffness parameters during post-competition recovery period (18-22 hours after the half-marathon race) in high-level and recreational athlete groups. However, the change of cfPWV was significantly dependent on race time and sports level of the athlete (high-level or recreational). 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 recovery period, except for the higher diene conjugates level in recreational athletes in comparison with the high-level group.

Previous studies have shown that relatively short duration aerobic training may improve arterial function (Tanaka et al., 2000; Maeda et al., 2001). 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 may play a role in acute effect of exercise on arterial stiffness. It has been shown that exercise intensity and volume may also have an impact on acute response to arterial stiffness (Ferreira et al., 2006; Sugawara et al., 2006; Saz-Lara et al., 2021).

Recent meta-analysis 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 activity, which exceeds anaerobic threshold and has 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, and which were expected to perform at a higher intensity (% HR or % VO_2) than 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 was more than 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 aortic and brachial blood pressure was observed immediately after the race, whereas aortic stiffness did not change significantly (Vlachopoulos et al., 2010). Previous data suggest that both the time of PWV measurement and the type of exercise should be considered when analyzing the effect of physical activity on arterial stiffness. However, there is no available data about the late post-competition recovery period (i.e. more than 90 min), which may provide a more useful understanding for the recovery process and returning to the habitual training process, especially in highly trained athletes.

Our study did not reveal any significant post-race changes in peripheral and central blood pressure or in AIx and AIx@75 in comparison with baseline values among both groups. This might be explained by a shorter race distance as well as the differences in age and other clinical characteristics of the study participants. No differences were found between the groups in all measured parameters but, as expected, high-level athletes had significantly lower resting heart rate as compared to recreational athletes. A relatively low mean resting heart rate of both groups is very characteristic to aerobically-trained subjects and reflects good cardiovascular adaptation to the sports activity. 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 of cfPWV was noted in the recreational group. It could be hypothesized that a half-marathon race may have a different effect on arterial stiffness depending on the level of physical capacity of an athlete. However, larger-scale studies are required to confirm this hypothesis.

It has been demonstrated that physically active persons have lower baseline hsCRP values in comparison with sedentary ones (Pihl et al., 2003; Fernandez et al., 2018), which is related to lower cardiovascular risk level. However, the effect of acute physical load and recovery period response on hsCRP has not been sufficiently studied. A significant elevation of hsCRP was observed after circuit resistance exercise in the general population (Bizheh and Jaafari, 2011). Similarly, a significant increase in WBC and CRP level was observed in 90 marathon runners immediately and 24 hours after the race, which returned to baseline two to six days after exercise (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% VO_2 max in healthy sedentary male subjects (Markovitch et al., 2008). Our study data show that hsCRP increased significantly during the post-competition recovery period in recreational athletes and in the high-level group. However, in our study the exercise intensity was very high, whereas Markovitch and co-authors assessed the serum level of hsCRP after moderate intensity exercise. Thus, exercise-related inflammatory response may be affected by the duration and intensity of physical activity.

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/reperfusion injury, disruption of the mitochondrial electron transport chain and other mechanisms (Powers et al., 2016). Conversely, long-term moderate intensity exercise increases antioxidant defenses of the body

and maintains redox homeostasis (Caimi et al., 2009). Previously, our group has demonstrated that physically active former athletes have a lower oxidized LDL (oxLDL), oxLDL/LDL ratio and DC levels compared to sedentary controls (Pihl et al., 2003). The present study further extends our previous findings by showing that DC levels were lower in the high-level group in comparison with the recreational athletes. Similarly, serum levels of DC were lower in professional road-racing cyclists than in healthy male volunteers (Pittaluga et al., 2006). In our study, no significant changes were found in pre- and post-race DC levels in the elite and recreational athletes, which might suggest an adequate adaptation of the antioxidant defense in the study subjects (Cases et al., 2006).

This study has a number of limitations. Firstly, according to standardization procedures for measurement of arterial stiffness with applanation tonometry (Laurent et al., 2006), the immediate post-race 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, elevated inflammation and oxidative stress as well as elevated parasympathetic tone. Secondly, post-competitive recovery measurements were performed 18 to 22 hours after the marathon competition, which is not very limited time point. 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. On the other hand, the novelty of our study is that it was performed in elite-level highly-trained athletes evaluating the effect of a real competitive physical load on arterial stiffness and blood biomarkers.

Conclusion

In conclusion, our study results revealed that half-marathon running did not have any significant effects on arterial stiffness parameters during the post-race period but the change in cfPWV was independently associated with half-marathon race time and the athlete's level of training. A significant increase was found in hsCRP, creatine kinase and LDH activity during the post-race period. No significant changes were found in oxidative stress markers in groups during the post-competitive period, except a higher diene conjugates level in recreational athletes in comparison with the high-level group. However, more extensive studies evaluating the acute effect of intensive exercise on arterial stiffness and markers of oxidative stress are needed.

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Key points

- Half-marathon race did not cause significant changes in blood pressure and arterial stiffness parameters during post-competition recovery period in high-level and recreational athletes.
- The change of arterial stiffness after intensive half-marathon race depends on the training level of the athlete, revealing a mild increase of arterial stiffness in high-level athletes and athletes with a faster race time
- High-sensitivity CRP increased significantly during the recovery period in recreational and high-level athletes.
- Half-marathon running did not influence serum level of oxidative stress biomarkers except for higher diene conjugate concentration in recreational athletes as compared to high-level runners during recovery period.

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