OXIDATIVE STRESS PARAMETERS AND ITS ASSOCIATIONS WITH ARTERIAL STIFFNESS IN COMPETITIVE POWERLIFTING ATHLETES AFTER 12-WEEK SUPERVISED STRENGTH TRAINING

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ABSTRACT

Jürgenson, J, Serg, M, Kampus, P, Kals, Z, Zagura, M, Viru, M, Zilmer, K, Zilmer, M, Eha, J, and Unt, E. Oxidative stress parameters and its associations with arterial stiffness in competitive powerlifting athletes after 12-week supervised strength training. J Strength Cond Res XX(X): 000–000, 2019—Available studies have not revealed a clear understanding of the impact of intensive strength training on arterial stiffness and oxidative stress (OxS) parameters, which may have a significant impact on further cardiovascular health of an athlete. The purpose of this study was to evaluate the effect of a 12-week supervised strength training program (SSTP) on oxidative stress indices and its relationship with arterial stiffness in powerlifting athletes. A total of 19 men (28 ± 6 years) exercised for 12 weeks (4 days per week with intensity 60–90% assessed from 1 repetition maximum, 90–120 minutes per session). Oxidative stress parameters and arterial stiffness (Sphygmocor 7.1) were measured before and after SSTP. The study results showed that total peroxide concentration increased and total antioxidant capacity decreased significantly after SSTP. There were no significant changes in carotid-femoral pulse wave velocity (cfPWV) or in the augmentation index. Correlation analysis revealed that the magnitude of the increase of cfPWV was significantly related to the increase of OxS. The current study demonstrated that a 12-week SSTP in powerlifting athletes produced significant changes in OxS indices, which were positively related to increased aortic stiffness. This novel finding may have significant implications about the effect of OxS on cardiovascular health after high-intensity strength training. Furthermore, strength and conditioning coaches may have to consider the long-term exercise-induced changes in OxS on an individual level, where increased OxS leads to impaired arterial stiffness and cardiovascular health.

KEY WORDS resistance training, pulse wave velocity, oxidative stress index, central blood pressure, cardiovascular health

INTRODUCTION

Strenuous exercise increases the production of reactive oxygen species (ROS), which leads to cellular damage as a result of high-grade oxidative stress (OxS) (11,15,33). Damaged mitochondria of muscle cells are the major intracellular source of ROS (11), whereas ROS are also produced by erythrocytes, neutrophils, and lymphocytes (15,33). In a single bout of aerobic exercise, where skeletal muscles produce significant amounts of superoxide anion, the harmful effect of ROS is well known (26). In anaerobic training, other different pathways of reactive oxygen and nitrogen species generations, including xanthine and oxidase production, have shown disruption of iron-containing proteins, prostanoid metabolism, ischemia reperfusion, etc (4,18). Very intensive and long-term physical
**TABLE 1.** Mean anthropometric, hemodynamic, and arterial stiffness parameters of the subjects before and after the 12-week strength training program (n = 19), (x ± SD).*

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Before SSTP</th>
<th>After SSTP</th>
<th>ρ</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body mass (kg)</td>
<td>99.9 ± 16.5</td>
<td>100.2 ± 17.1</td>
<td>0.614</td>
</tr>
<tr>
<td>BMI (kg·m⁻²)</td>
<td>31.2 ± 5.1</td>
<td>31.3 ± 5.2</td>
<td>0.603</td>
</tr>
<tr>
<td>bSBP (mm Hg)</td>
<td>132.3 ± 8.8</td>
<td>124.3 ± 8.7</td>
<td>0.002</td>
</tr>
<tr>
<td>bDBP (mm Hg)</td>
<td>70.1 ± 5.9</td>
<td>67.7 ± 5.2</td>
<td>0.059</td>
</tr>
<tr>
<td>bPP (mm Hg)</td>
<td>61.2 ± 8.4</td>
<td>58.8 ± 7.3</td>
<td>0.259</td>
</tr>
<tr>
<td>MAP (mm Hg)</td>
<td>88.4 ± 7.4</td>
<td>86.9 ± 5.9</td>
<td>0.359</td>
</tr>
<tr>
<td>cSBP (mm Hg)</td>
<td>110.1 ± 7.7</td>
<td>104.5 ± 8.7</td>
<td>0.008</td>
</tr>
<tr>
<td>cDBP (mm Hg)</td>
<td>71.1 ± 6.3</td>
<td>68.9 ± 5.3</td>
<td>0.078</td>
</tr>
<tr>
<td>cPP (mm Hg)</td>
<td>39.0 ± 3.7</td>
<td>35.6 ± 5.0</td>
<td>0.317</td>
</tr>
<tr>
<td>HR (b·min⁻¹)</td>
<td>62.6 ± 8.6</td>
<td>64.8 ± 9.3</td>
<td>0.093</td>
</tr>
<tr>
<td>AIx (%)</td>
<td>3.3 ± 9.2</td>
<td>5.1 ± 8.9</td>
<td>0.233</td>
</tr>
<tr>
<td>AIx@75 (%)</td>
<td>−2.7 ± 9.3</td>
<td>−2.6 ± 8.3</td>
<td>0.081</td>
</tr>
<tr>
<td>cfPWV (m·s⁻¹)</td>
<td>6.2 ± 0.7</td>
<td>6.5 ± 1.1</td>
<td>0.088</td>
</tr>
<tr>
<td>crPWV (m·s⁻¹)</td>
<td>7.4 ± 0.8</td>
<td>7.5 ± 1.0</td>
<td>0.493</td>
</tr>
</tbody>
</table>

*SSTP = supervised strength training program; BMI = body mass index; bSBP = brachial systolic blood pressure; bDBP = brachial diastolic blood pressure; bPP = brachial pulse pressure; MAP = mean arterial pressure; cSBP = central systolic blood pressure; cPP = central pulse pressure; HR = heart rate; AIx = augmentation index; AIx@75 = augmentation index corrected for a heart rate of 75 b·min⁻¹; cfPWV = pulse wave velocity at carotid-femoral segments.

Load is detrimental on antioxidant mechanisms and may lead to the unfavorable effect by OxS on the health outcomes, all of which are not known (33). To prevent OxS, there is a complex antioxidant defense system consisting of enzymatic and nonenzymatic antioxidants (31). Thus, it is important that the antioxidant defense system is effective and recovers properly after strenuous physical exercise. Regular aerobic training has been shown to improve antioxidant adaptation (15). Kaldur et al. (19) demonstrated that oxidative stress parameters increased after strenuous physical exercise. Regular aerobic training has been shown to improve antioxidant adaptation to exercise (15). Kaldur et al. (19) demonstrated that oxidative stress parameters increased after an acute endurance capacity test in young men in non–heat-acclimated status; however, beneficial adaptive effects in oxidative and inflammatory parameters were found after a 10-day exercise and heat acclimation program. Furthermore, OxS is directly linked to impaired arterial stiffness (20,29), which has emerged as one of the earliest independent determinants of cardiovascular morbidity and mortality (32).

In recent years, the importance of resistance training has been emphasized in general health guidelines (14), including recommendations of moderate resistance training in the prevention programs of sarcopenia and for increasing muscular strength, especially in elderly populations. In previous literature, resistance training also known as strength training (35,8) has been poorly defined and used with a wide variety of modalities and intensities (12). Furthermore, moderate to intensive strength training (60–90% of 1 repetition maximum [1RM]) is used by well-trained weightlifting and powerlifting athletes, whereas most of the OxS and arterial stiffness studies are conducted using low- or moderate-intensity resistance training or in combination with aerobic training (3,5,8). Moreover, data are practically missing regarding the effects of moderate- to high-intensity resistance training on OxS and its relationship with arterial stiffness in well-trained powerlifting athletes.

The aim of this study was to investigate the effect of a moderate- to high-intensity 12-week supervised strength training program (SSTP) on OxS parameters and its relationship with arterial stiffness in powerlifting athletes. It was hypothesized that 12-week high-intensity strength training increases OxS and inflammation biomarkers, and this may have an adverse effect on arterial stiffness indices.

**METHODS**

**Experimental Approach to the Problem**

A descriptive study was applied for determining the changes of a 12-week SSTP on arterial stiffness and OxS in competitive well-trained powerlifting athletes.
A 12-week SSTP (combination of dynamic and isometric resistance training 4 times per week, 90–120 minutes per session, 60–90% from 1RM) was selected. This training protocol is characteristic for high-level powerlifters in the precompetition phase and may have more practical implications than training protocols for less-conditioned athletes. There was no rationale to involve a control group (12 weeks without strength training) among high-level powerlifting athletes because of training periodization circumstances. Our study subjects were selected as elite-level powerlifters who started the routine preseasonal training program involving a very high training load. This high training load would not be applicable for the untrained or less-trained recreational athletes. Biomarkers of inflammation, muscle damage and oxidative stress data, as well as blood pressure (BP) and arterial stiffness were measured before and after the SSTP.

**Subjects**

A total of 20 voluntary well-trained male powerlifting athletes (28.2 ± 6.1 years, age range: 18 to 44 years, 179.2 ± 5.9 cm, 99.9 ± 16.5 kg) were examined before and after a 12-week SSTP. All subjects were free of acute or chronic illnesses. The study subjects were informed about the details of all training and study procedures. All participants were asked not to take any medication or antioxidant supplements at least 2 months before the study and during the SSTP. One subject was excluded from the data analysis because of an acute injury. Thus, the final study sample was 19.

Informed written consent was obtained from each athlete in accordance with principles of the Declaration of Helsinki. The study protocol was approved by the Research Ethics Committee of the University of Tartu: No. 162/T-12.

**Supervised Strength Training Program.** Before the SSTP, there was an 8-week light training period (without heavy strength training sessions and competitions). During the SSTP, athletes exercised 4 days per week with intensity 60–90% assessed from 1RM. The 12-week SSTP included of combination of dynamic and isometric resistance training. One session lasted 90–120 minutes. A one-week training program consisted of 5–8 working sets (4–12 repetitions) of the bench press (BEP), lateral pull down, standing shoulder press, arm curl and extension, leg press, squat, leg curl, calf press, abdominal crunch, and dead lift. All the major muscle groups were trained twice a week. No aerobic exercise was involved in the training program. All the sessions were supervised by a skilled certified instructor. After the SSTP, all study participants participated in the National Powerlifting Championships. The baseline maximal muscular power, laboratory measurements, and arterial stiffness measurements were performed 1 week before the SSTP and post-training period measurements were collected 1 week after the competition, during which period only recreational activities for the subjects were allowed. There were no training sessions on the day preceding the measurements (maximal muscular power, laboratory, and arterial stiffness measurements).

**Anthropometric Data.** Subjects’ height and body mass were measured using the Martin metal anthropometer (±0.1 cm) and clinical scales (±0.05 kg); body mass index (BMI) was calculated (kg·m$^{-2}$).

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### Table 3. The biochemical parameters of the powerlifters before and after 12-week supervised strength training program. For hsCRP and NT-proBNP medians, 25th percentile and 75th percentile are presented (in brackets), ($n = 19$), (x ± SD).*

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Before SSTP</th>
<th>After SSTP</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>WBC (10 × 9/L)</td>
<td>5.6 ± 1.4</td>
<td>5.6 ± 1.0</td>
<td>0.770</td>
</tr>
<tr>
<td>RBC (10 × 12/L)</td>
<td>5.0 ± 0.3</td>
<td>5.2 ± 0.2</td>
<td>0.106</td>
</tr>
<tr>
<td>Hematocrit (%)</td>
<td>44.3 ± 3.2</td>
<td>45.1 ± 2.1</td>
<td>0.222</td>
</tr>
<tr>
<td>Hemoglobin (g/L)</td>
<td>152.5 ± 10.8</td>
<td>155.5 ± 8.5</td>
<td>0.119</td>
</tr>
<tr>
<td>Creatine kinase (U/L)</td>
<td>329.8 ± 246.7</td>
<td>466.7 ± 294.8</td>
<td>0.026</td>
</tr>
<tr>
<td>Creatinine (μmol/L)</td>
<td>96.5 ± 8.7</td>
<td>94.1 ± 10.4</td>
<td>0.304</td>
</tr>
<tr>
<td>hsCRP (mg/L)</td>
<td>0.73 (0.38; 1.91)</td>
<td>0.64 (0.33; 1.22)</td>
<td>0.717</td>
</tr>
<tr>
<td>Glucose (mmol/L)</td>
<td>5.24 ± 0.32</td>
<td>5.12 ± 0.53</td>
<td>0.345</td>
</tr>
<tr>
<td>CHOL (mmol/L)</td>
<td>4.89 ± 1.21</td>
<td>5.00 ± 1.13</td>
<td>0.524</td>
</tr>
<tr>
<td>HDL-C (mmol/L)</td>
<td>0.96 ± 0.23</td>
<td>1.13 ± 0.27</td>
<td>0.008</td>
</tr>
<tr>
<td>LDL-C (mmol/L)</td>
<td>3.25 ± 1.09</td>
<td>3.41 ± 1.09</td>
<td>0.278</td>
</tr>
<tr>
<td>TG (mmol/L)</td>
<td>1.33 ± 0.62</td>
<td>1.18 ± 0.55</td>
<td>0.187</td>
</tr>
<tr>
<td>NT-proBNP (pg/ml)</td>
<td>20.5 (6.0; 33.3)</td>
<td>21.0 (10.0; 31.0)</td>
<td>0.767</td>
</tr>
</tbody>
</table>

*SSTP = supervised strength training program; WBC = white blood cell; RBC = red blood cell; hsCRP = high-sensitivity 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.
where the external load used was equal to a subject’s individual half, full, or one and a half body mass, and the load was same in both exercises. Muscle power produced during the 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 is described by Bosco et al. (6).

Biochemical Analysis and Oxidative Stress Data. Blood samples were obtained in the morning between 8:00 and 10:00 after an overnight fast. White blood cell count, red blood cell count, hematocrit, hemoglobin, serum creatine kinase, creatinine, glucose, total cholesterol, high-density lipoprotein (HDL)-cholesterol, low-density lipoprotein (LDL)-cholesterol, triglycerides were measured by standard laboratory methods at the local laboratory department of university hospital. 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 immunnoassay (Cobas; Roche Diagnostics GmbH).

For other parameters, the blood samples were centrifuged within 15 minutes after collection at 3,000 rpm to obtain serum that was frozen at −80°C until further analysis. Total peroxide concentrations (TPX) of samples were determined using OXYSTAT 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 (10). Percent ratio of the total peroxide concentration of plasma to the TAC of plasma was accepted as oxidative stress index (OSI), which is an indicator of the degree of Oxs.

Blood Pressure and Arterial Stiffness. Brachial BP was measured in a sitting position from the nondominant arm as a mean of 3 consecutive measurements at 5-minute intervals using a validated oscillometric technique (OMRON M4-I; Omron Healthcare Europe BV, Hoofddorp, the Netherlands). The mean of the 2 closest BP readings was used in further analysis (25). Brachial pulse pressure (bPP) was calculated as the difference between brachial systolic and diastolic BP. No significant changes were found in central systolic blood pressure (cSBP) and arterial stiffness parameters were in normal range in all studied subjects at baseline and after STTP. Brachial systolic blood pressure and central systolic blood pressure (cSBP) decreased significantly after the training period. There were nonsignificant differences after the training period in brachial diastolic blood pressure and central diastolic blood pressure, crPWV, cfPWV, AIx, and Alx@75. After the SSTP, a slight but nonsignificant decrease was detected in central pulse pressure and bPP. No significant changes were found in mean arterial pressure and heart rate after the SSTP.

FIGURE 1. Total peroxides of the powerlifters before and after 12-week supervised strength training program. Data are presented as mean ± SD (n = 19). TPX = Total peroxide concentrations; SSTP = supervised strength training program. *p < 0.01, in comparison with before SSTP.
Maximal Muscular Power Data

Maximal muscular power indices improved in all subjects, where mean values of BEP and jumping from half squat increased significantly after the SSTP in comparison with baseline values (Table 2).

Biochemical and Oxidative Stress Data

Subjects’ biochemical data are presented in Table 3. The mean value of CK and HDL-C was significantly higher after SSTP as compared to respective baseline values. Total peroxide concentrations as well OSI increased (\( p = 0.008 \) and \( p = 0.001 \), respectively) and TAC decreased (\( p = 0.033 \)) significantly after the SSTP as compared to baseline data (Figures 1–3). No significant changes were found in other biochemical parameters.

Correlation Analysis

Correlation analysis revealed that the magnitude of the increase of cfPWV (cfPWV before SSTP − cfPWV after SSTP) was significantly positively related to the increase in TPX (TPX before SSTP − TPX after SSTP) and OSI (OSI before SSTP − OSI after SSTP), \( (r = 0.596, p = 0.007; r = 0.568, p = 0.011, \) 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).

DISCUSSION

In recent years, research has emerged about the beneficial effect of resistance exercise on metabolic health in addition to the well-known benefits on muscular strength, bone health, and body composition (9). However, there are still scarce data available regarding the impact of powerlifting, which is used by competitive athletes, on cardiovascular health and, in particular, on oxidative stress and arterial stiffness.

In this study, we focused on the changes in OxS and its relationship with arterial stiffness in well-trained powerlifting athletes after a 12-week SSTP. Our data revealed that the 12-week SSTP significantly increased OxS status, and the magnitude of OxS was related to the increase in cfPWV—the gold standard measure of arterial stiffness with a valuable cardiovascular prognostic impact (32). At the same time, mean arterial stiffness parameters of the subjects did not change significantly during the SSTP. In addition, we found that the strength training protocol resulted in a significant decrease in brachial and in central systolic BP. Beneficial effects of the 12-week SSTP on peripheral and cSBP are demonstrated in Table 1.

Available evidence clearly demonstrates that exercise is associated with an increase of ROS generation, and this may have dual effects. It is known that elevation of ROS have high reactivity to most biological macromolecules, but growing evidence shows that exercise-induced OxS may act as signaling molecules to mediate useful cellular adaptation to exercise (15). 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 overproduction of reactive species and high-grade OxS is relevant for the development of cardiovascular diseases (24).

In our study, an increase in OxS indices after the intense resistance training program was recorded—significantly higher levels of TPX and OSI were found after SSTP as compared with the baseline data (Figures 1 and 3). In addition, there was a concurrent decrease in the TAC (Figure 2). Previously published studies investigating the effect of resistance training on OxS parameters have reported controversial findings. Azizbeigi et al. (2,3) reported decreased oxidative stress and an increase of antioxidant capacity after an 8-week resistance training program. Panza et al. (28) and Bloomer et al. (4) 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. (2,3), untrained men with no previous experience of resistance training or regular physical activity were recruited as participants, whereas Panza et al. (28) recruited men engaged in recreational-level weight training, and Bloomer et al. (4) 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. (28) and Bloomer et al. (4) were designed to test the effect of a single bout of resistance exercise on OxS indices, either a BEP or a squat, with blood samples collected up to 15 minutes or 24 hours after exercise, respectively. Azizbeigi et al. drew blood samples 72 hours after the completion of an 8-week resistance training program (2,3). In our study, post-training period measurements were performed 1 week after the competitions excluding the impact of extreme acute physical exertion.

As expected, all our study subjects improved their maximal muscular power (Table 2) showing significant improvement in all measured variables from 3.4 to 12.3% as compared to baseline values. After the SSTP, subjects’ mean CK (Table 3) was significantly elevated reflecting muscle injury response to strength training and competition. However, these data are within the normal physiological range in athletes with high muscle mass. There were no subjects with extremely elevated CK and inflammation indices, which may confound other variables.

Studies have shown that, for improving lipid profile, greater exercise stimulus and energy expenditure is needed (7). In our study, we used a moderate- to high-intensity training protocol, and there was a significant increase in HDL-cholesterol but no effect on other blood lipids (total cholesterol, LDL-cholesterol, and triglycerides), or fasting glucose was found. Our data are in accordance with longitudinal studies on weight training, which have shown an increase in HDL-C values (17). However, Kelley and Kelley (22) showed in their meta-analysis that resistance training reduces total cholesterol, LDL-cholesterol, and triglycerides in adults, whereas no significant increase in HDL-cholesterol was detected.

Our study results showed that both peripheral systolic BP and central SBP decreased significantly after 12 weeks, but the changes in arterial stiffness parameters remained nonsignificant. These data are in accordance with previous studies (1), where resistance training significantly reduces BP in normotensive and hypertensive adults. Furthermore, the impact of resistance exercise on arterial stiffness is still under discussion. It is widely accepted that arterial stiffness is associated with a number of traditional and novel cardiovascular risk factors, and cfPWV is a marker of subclinical organ damage (23). In regard to resistance training, available studies show controversial findings (9). However, our study revealed no significant changes in mean values of aortic pulse wave velocity or augmentation index (Table 1). These results might be explained by the fact that our study group consisted of previously well-trained young men, who were conditioned for such strenuous training. In addition, their arterial stiffness indices and lipoprotein profile were in normal range at baseline.

On the other hand, before the study, we hypothesized that a 12-week SSTP may increase inflammation and OxS and, through this mechanism, alter arterial stiffness. The status of inflammation and OxS has been demonstrated to be associated with arterial stiffness (21). 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 is demonstrated by correlation analysis. These data suggest that OxS has an impact on aortic stiffness, which is an independent predictor of increased cardiovascular risk in the general population (32). 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 (13). 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 (16). This novel finding may have significant implications about the effect of OxS on cardiovascular health in powerlifting athletes.

In conclusion, a 12-week SSTP in powerlifting athletes significantly increased oxidative stress and HDL-C concentration. There was no significant effect of SSTP on mean aortic pulse wave velocity, but the increase in oxidative stress indices was significantly positively related to increased aortic pulse wave velocity.

**Practical Applications**

The values of biomarkers after intensive and long-term physical load vary significantly depending on the type, intensity of exercise performed, and training status of the athlete. It is important that the antioxidant defense system is effective and recovers properly after strenuous physical exercise. Excessive oxidative stress impairs arterial stiffness that may lead to unfavorable cardiovascular conditioning of the athlete. Thus, strength and conditioning coaches may have to consider the long-term exercise-induced changes in OxS and arterial stiffness on an individual level, where increased OxS leads to impaired arterial stiffness and cardiovascular health. Therefore, the use of arterial stiffness assessment method in combination of specific biomarkers before preseason and during the season would be applicable for elite-level strength training athletes because it gives additional information about the athlete’s physiological adaptation to exercise and to recovery. Furthermore, monitoring of arterial stiffness, which is a simple, noninvasive, and valid method, would be necessary for the evaluation cardiovascular risk of the elite-level strength training athletes.
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REFERENCES