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ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

The effect of physical activity on serum levels of selected biomarkers of atherosclerosis

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Abstract

Background: Physical activity is associated with a lower risk of cardiovascular disease but the mechanism underlying this association is unclear. These benefits of physical activity might result from its effects on inflammation and endothelial function.

Aim: We investigated whether cardiorespiratory fitness and the level of physical activity are associated with biomarkers of atherosclerosis in athletes and nonathletes.

Methods: Forty six athletes and 46 age- and sex-matched subjects who did not exercise regularly were studied. All subjects underwent anthropometric measurements and maximal treadmill cardiopulmonary exercise tests. Physical activity level was assessed using the International Physical Activity Questionnaire-Short Form (IPAQ-SF). Blood samples were taken before and immediately after exercise. Serum interleukin (IL)-6, soluble CD40 ligand (sCD40L) and soluble intercellular adhesion molecule-1 (sICAM-1) levels were determined using the ELISA method.

Results: In all participants, IL-6 level was significantly increased after exercise as compared to baseline (1.35 ± 2.6 vs. 1.46 ± ± 2.1 pg/mL, p = 0.01). Resting IL-6 and sCD40L levels were lower in athletes as compared to nonathletes (0.7 ± 0.92 vs. 1.8 ± 3.52 pg/mL, p = 0.003, and 888.8 ± 892.9 vs. 2367.7 ± 8743.4 pg/mL, p = 0.005, respectively), while sICAM-1 levels did not differ between the two groups. IL-6 level correlated negatively with peak oxygen consumption (r = –0.25, p = 0.035) and the IPAQ-SF score (r = –0.26, p = 0.02), and sCD40L level correlated negatively with the IPAQ-SF score (r = –0.4, p = 0.004).

Conclusions: Intensive exercise training and high exercise capacity are associated with lower serum IL-6 and sCD40L levels. This may constitute an important factor limiting progression of atherosclerosis.

Key words: physical activity, biomarkers, inflammation, endothelium

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INTRODUCTION

Research undertaken in recent years contributed significantly to our understanding of mechanisms underlying development of atherosclerosis [1]. Effects of adverse mechanical, biological, and chemical factors lead to endothelial cell activation and increased expression of adhesive molecules, including soluble intercellular adhesion molecule-1 (sICAM-1). These processes result in leukocyte adhesion to vessel walls and increased penetration of macroparticles and inflammatory cells into the intima. Monocytes accumulating in the intima transform into macrophages and then into foam cells by internalisation of modified lipoproteins. Further stages of atherosclerotic plaque development are characterised by the migration of smooth muscle cells from the media into the intima, the accumulation of extracellular matrix, and the fibrosis leading to formation of a fibrous cap. These phenomena are mediated by various cytokines, including interleukin-6 (IL-6) and soluble CD40 ligand (sCD40L) [2].

Some cytokines and soluble adhesion molecules, receptors, and receptor ligands are released from the vessel wall into bloodstream. Their levels reflect the activity of the atherosclerotic process, have prognostic value, and allow evaluation of the effectiveness of therapeutic interventions [3]. These substances are known as biomarkers. Multiple studies showed independent association of increased sICAM-1, IL-6, and sCD40L levels with coronary artery disease risk and the risk of cardiovascular events [4–6].

An association between physical activity and coronary artery disease risk was shown for the first time in the 1950s [7]. Studies

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performed in the next decades clearly indicated that both low physical activity and poor fitness are strong and independent predictors of cardiovascular morbidity and mortality [8]. Numerous data suggest a positive effect of exercise on inflammation and endothelial function, factors playing an important role in the pathogenesis of atherosclerosis [9]. For example, Pedersen and Febbraio [10] evaluated the effect of physical training on the level of IL-6, a multifunctional cytokine involved in the formation, progression, and destabilisation of atherosclerotic plaques. He showed that IL-6 is produced and released into the bloodstream by skeletal muscle during exercise. IL-6 gene expression in contracting myocytes is induced by such factors as reduced glucose availability, changes in Ca^{2+} level, and increased synthesis of reactive oxygen species. Paradoxically, skeletal muscle-derived IL-6 has a number of beneficial effects. Large amounts of IL-6 released during exercise act on the adipose tissue by inducing lipolysis and regulating gene transcription in subcutaneous abdominal fat. In addition, exercise-related IL-6 has an anti-inflammatory effect mediated by IL-1RA and IL-10 release, which may reduce tumour necrosis factor alpha (TNF-α) synthesis and thus result in reduced insulin resistance. Regular physical training reduces resting IL-6 levels by improving insulin sensitivity, reducing oxidative stress, and stimulating release of anti-inflammatory cytokines such as IL-1RA and IL-10, thus constituting one plausible mechanism of beneficial effects of exercise in the prevention of cardiovascular disease.

Previous studies evaluating the relationship between physical activity and other biomarkers of atherosclerosis gave some inconsistent results. Only few such studies were performed in young subjects free from cardiovascular disease. The aim of the present study was to assess the influence of physical activity, evaluated using a questionnaire, and physical fitness, assessed during cardiopulmonary exercise test, on serum IL-6, sCD40L, and sICAM-1 levels.

METHODS

Study group

We studied 46 athletes (including 29 male soccer players and 17 female basketball players) aged 22.89 ± 5.1 years. The control group consisted of 46 healthy volunteers matched for gender, age, and body mass index (BMI) who did not participate in competitive sports. The exclusion criteria included past or current smoking; kidney or liver failure; history of cancer; infection, inflammatory disorder or major trauma within the past 3 months; and history of allergic disease.

The study protocol was approved by a local ethics committee, and all subjects gave informed consent for study participation.

Evaluation of physical activity and cardiorespiratory fitness

Physical activity was evaluated using the International Physical Activity Questionnaire-Short Form (IPAQ-SF) [11]. Based on questionnaire data, daily physical activity score was calculated using the following formula:

$$\text{Daily physical activity score} = (3.3 \times W + 4 \times M + 8 \times I) / 7$$

where W is total weekly walking time, M is total weekly moderate activity time (e.g. carrying smaller weights, bicycle riding at an average speed, volleyball playing), and I is total intensive activity time (e.g. lifting heavy weights, digging, aerobic, bicycle riding at a high speed).

Treadmill cardiopulmonary exercise test was performed to evaluate cardiorespiratory fitness of the studied subjects. Athletes underwent testing using a specifically designed protocol (Table 1), and the Bruce protocol was used in the control group [12]. We estimated peak oxygen consumption (VO_{2peak}, mL/kg/min), oxygen consumption at the anaerobic threshold (VO_{2AT}, mL/kg/min), exercise time to the anaerobic threshold (T_{AT}, min), total exercise time (T, min), and maximal heart rate (HR, bpm).

Biomarkers

Serum IL-6, sCD40L and sICAM-1 levels were determined in blood samples taken from the antecubital vein before exercise (baseline) and at peak exercise (IL-6 baseline, IL-6 peak; sCD40L baseline, sCD40L peak; sICAM-1 baseline, sICAM-1 peak). All subjects were fasting at the time of blood sample collection. Biomarker levels were determined by the ELISA method with the use of R&D SYSTEMS kits (sensitivity 0.039 pg/mL, 2.1 pg/mL and 0.049 ng/mL, respectively; intra-series variability 7.68%, 5.91% and 7.09%, respectively; inter-series variability 9.03%, 7.75% and 8.41%, respectively).

Statistical analysis

Continuous variables were presented as mean values ± standard deviation (SD). We used nonparametric Wilcoxon test to compare biomarker levels before and after exercise, and the Mann-Whitney U test to compare values between the two groups. Relationships between study variables were tested in the total population of athletes and controls using the Pearson linear correlation coefficient. For all study participants, a multivariable regression analysis was performed with baseline sCD40L level as a dependent variable, and baseline IL-6 level, oxygen consumption at the anaerobic threshold, peak oxygen consumption, and daily physical activity score as independent variables. $P < 0.05$ was considered statistically significant. All analyses were performed using the Statistica 9.0 package.

RESULTS

Table 2 shows characteristics of the study group. The results of physical activity questionnaire analysis are shown in Table 3. In all participants, IL-6 level was significantly increased after exercise as compared to baseline (1.35 vs. 1.46 pg/mL, $p = 0.01$). In contrast, sCD40L and sICAM-1 levels did not change significantly after exercise (1813.16 ± 2266.46 vs. 2316.25 ± ± 2615.57 pg/mL, $p = 0.069$; and 171.86 ± 39.71 vs. 172.31 ± ± 41.46 ng/mL, $p = 0.49$, respectively). Athletes were characterised by higher daily physical activity scores, increased

Table 1. Exercise test protocol in athletes

Stage	Speed [km/h]	Slope [%]	Duration [min]	Workload [MET]
1	9.0	0	3	9.6
2	10.0	0	2	10.5
3	11.0	0	2	11.5
4	12.0	0	2	12.4
5	13.0	0	2	13.4
6	14.0	0	2	14.3
7	15.0	0	2	15.3
8	16.0	0	2	16.2
9	17.0	0	2	17.2
10	18.0	0	2	18.1
Recovery 1	5.0	0	2	3.4
Recovery 2	0	0	2	1.0

Table 2. Selected evaluated parameters (mean ± SD) in athletes and controls

Parameter	Athletes (n = 46)	Controls (n = 46)	P
Age [years]	22.8 ± 5.1	24.3 ± 5.6	NS
Body mass index [kg/m^2]	22.3 ± 1.4	22.9 ± 2.6	NS
DPAS [MET-min/day]	1083.2 ± 227.9	285.3 ± 152.8	< 0.001
VO_{2AT} [mL/kg/min]	34.7 ± 10.3	25.6 ± 8.4	< 0.001
Exercise time [min]	17.4 ± 3.1	14.3 ± 2.9	< 0.001
Maximum heart rate [bpm]	185.2 ± 12.6	177.1 ± 8.4	NS
VO_{2peak} [mL/kg/min]	53.3 ± 5.1	40.9 ± 10.2	< 0.001
IL-6 baseline [pg/mL]	0.7±0.9	1.8 ± 3.4	0.03
IL-6 peak [pg/mL]	1.2±1.4	1.9 ± 2.5	NS
Δ IL-6 [pg/mL]	0.4±0.9	0.3±3.2	NS
sCD40L baseline [pg/mL]	888.8 ± 892.9	2367.7 ± 8743.4	0.005
sCD40L peak [pg/mL]	1270.7 ± 1205.4	2943.5 ± 3021.8	NS
Δ sCD40L [pg/mL]	381.8 ± 876.1	558.2 ± 2590.6	NS
sICAM-1 baseline [ng/mL]	169.7 ± 39.4	173.5 ± 40.3	NS
sICAM-1 peak [ng/mL]	173.9 ± 44.1	175.3± 39.7	NS
Δ sICAM-1 [ng/mL]	4.2 ± 31.4	3.3 ± 39.3	NS

DPAS — daily physical activity score; VO_{2AT} — oxygen consumption at the anaerobic threshold; VO_{2peak} — oxygen consumption at peak exercise; IL-6 baseline — fasting serum interleukin-6 level at rest; IL-6 peak — serum interleukin-6 level at peak exercise; Δ IL-6 — change in serum interleukin-6 level at peak exercise compared to baseline; sCD40L baseline — fasting serum soluble CD40 ligand level at rest; sCD40L peak — serum soluble CD40 ligand level at peak exercise; Δ sCD40L — change in serum soluble CD40 ligand level at peak exercise compared to baseline; sICAM-1 baseline — fasting serum intercellular adhesion molecule-1 level at rest; sICAM-1 peak — serum intercellular adhesion molecule-1 level at peak exercise; Δ sICAM-1 — change in serum intercellular adhesion molecule-1 level at peak exercise compared to baseline

total weekly moderate and intensive activity times, higher oxygen consumption at the anaerobic threshold, and higher peak oxygen consumption. Compared to controls, athletes had significantly lower baseline IL-6 (Fig. 1) and sCD40L levels (Fig. 2), while sICAM-1 level did not differ significantly between the two groups.

Significant correlations were found between total weekly moderate and intensive activity times and questionnaire-based daily physical activity scores and oxygen consumption at the anaerobic threshold and peak oxygen consumption (Table 4).

Negative correlations were found between baseline IL-6 level and peak oxygen consumption, and between baseline IL-6 and sCD40L levels and total weekly moderate and intensive activity times and daily physical activity score (Table 5). No correlations were found between resting and postexercise sICAM-1 levels and spiroergometric exercise test parameters and physical activity. Multivariate regression analysis showed that baseline sCD40L level was related to daily physical activity score ($p = 0.007$). No associations were found between biomarker levels and age, body mass, and BMI.

Table 3. Analysis of physical activity questionnaire data (mean ± SD)

Parameter	Athletes	Controls	P
DPAS [MET-min/day]	1083.2 ± 277.9	285.3 ± 157.8	< 0.001
Intensive exercise [min/week]	660.0 ± 177.8	59.2 ± 76.9	< 0.001
Moderate exercise [min/week]	420.0 ± 177.8	97.5 ± 70.4	< 0.001
Walking [min/week]	344.6 ± 232.4	343.4 ± 226.4	NS

DPAS — daily physical activity score

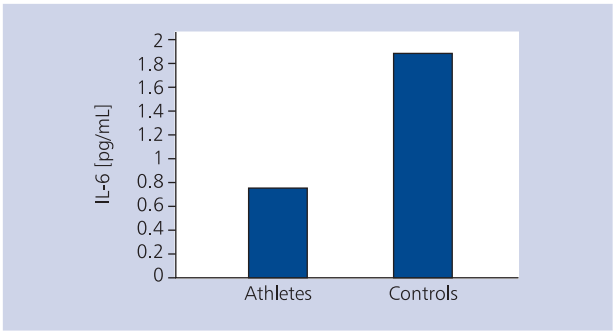

Figure 1. Baseline interleukin-6 (IL-6) level in athletes and controls

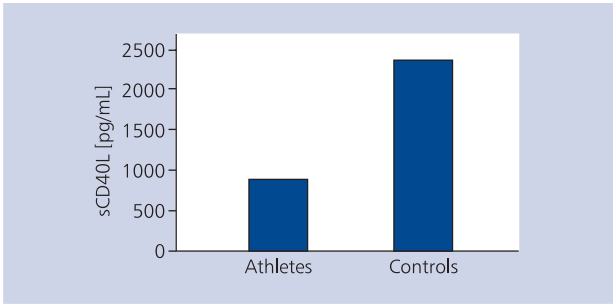

Figure 2. Baseline soluble CD40 ligand (sCD40L) level in athletes and controls

Table 4. Correlations between physical activity questionnaire data and oxygen consumption at peak exercise and the anaerobic threshold

	DPAS [MET-min/day]	Intensive exercise [min/week]	Moderate exercise [min/week]	Walking [min/week]
VO_{2AT} [mL/kg/min]	r = 0.2; p = 0.01	r = 0.5; p < 0.001	r = 0.6; p < 0.001	NS
VO_{2peak} [mL/kg/min]	r = 0.5; p < 0.001	r = 0.5; p < 0.001	r = 0.5; p < 0.001	NS

Abbreviations as in Table 2.

Table 5. Correlations between peak oxygen consumption and physical activity questionnaire data and the evaluated biomarkers

	IL-6 baseline [pg/mL]	sCD40L baseline [pg/mL]	sICAM-1 baseline [ng/mL]
VO_{2peak} [mL/kg/min]	r = –0.25; p = 0.035	NS	NS
DPAS [MET-min/day]	r = –0.26; p = 0.02	r = –0.4; p = 0.004	NS
Intensive exercise [min/week]	r = –0.27; p = 0.028	r = –0.37; p = 0.006	NS
Moderate exercise [min/week]	r = –0.25; p = 0.035	r = –0.31; p = 0.022	NS
Walking [min/week]	NS	NS	NS

Abbreviations as in Table 2

DISCUSSION

In our study, we found an effect of intensive exercise and high level of cardiorespiratory fitness on serum IL-6 and sCD40L levels. Many previous studies using various methods to evaluate physical activity both in healthy subjects and coronary artery disease patients showed a negative correlation between physical activity and IL-6 level, but most of these studies were performed in older subjects [13]. The relationship between physical fitness and IL-6 level was evaluated in few studies that gave inconsistent results, which may have partially resulted from small samples and the use of indirect methods to evaluate fitness [13]. Our finding of a significant post-exercise increase in IL-6 level compared to baseline is in accordance with previous results. IL-6 level increases already during exercise, and the size of this increase is related to exercise intensity, duration, and the number of muscle groups involved [10].

Athletes who are characterised by a high level of cardiorespiratory fitness had lower serum sCD40L levels in our study. In addition, we showed an inverse relationship between serum sCD40L level and both daily physical activity score and total weekly intensive and moderate activity time. Until now, few studies evaluated the relationship between physical fitness and se-

rum sCD40L level. Geertsema et al. [14] investigated the effect of prolonged intensive exercise on this biomarker level, but this study was performed only in athletes. These authors found that serum sCD40L level decreased following physical activity, reaching a nadir at 24 hours after exercise, and then returned to baseline values by the next 24 hours. They suggested that regular exercise might result in lowering the baseline level of this biomarker. Hammett et al. [15] studied middle-aged women and evaluated the effect of a 12-week training (45 minutes thrice weekly at an intensity that allows maintaining 60–70% of the maximum heart rate) on sCD40L level. In that study, no difference in sCD40L level between and after the intervention was found. It is thus possible that some level of exercise intensity is required to induce significant changes in this biomarker level, which is consistent with our findings, but mechanisms of this phenomenon are not clearly understood at the present time.

In our study, we did not find differences in baseline sICAM-1 level between athletes and controls, and serum level of this biomarker did not change significantly after a single exercise session. It has been suggested that a significant increase in sICAM-1 level may only be induced by prolonged intense exercise, as observed in previous studies. Silvestro et al. [16] did not find significant changes in this biomarker level following short intense exercise, while Nielsen and Lyberg [17] found sICAM-1 level to increase in marathon runners. In our study, we also did not find any relationship between sICAM-1 level and physical activity level and parameters of the cardiopulmonary exercise test. An inverse relationship between serum sICAM-1 level and physical activity was observed in some previous studies. For example, Mora et al. [18] in a cross-sectional study that included 27,158 women without clinical manifestations of coronary artery disease (mean age 54.7 years) found an inverse relationship between physical activity evaluated using a questionnaire and serum sICAM-1 level. In addition, sICAM-1 level decreased after physical training in some interventional studies. In a study by Adamopoulos et al. [19] in patients with heart failure, a 12-week training that included 30-minute sessions five days a week, resulted in a significant reduction of sICAM-1 level. Physical activity likely reduces sICAM-1 level only in those subjects in whom baseline level of this biomarker is increased, and this phenomenon is mediated by reduced ICAM gene transcription related to an increased shear stress during exercise, along with reduced stimulation by inflammatory cytokines, free oxygen species, and oxidised low-density lipoproteins [20].

Limitations of the study

Limitations of our study included its small sample, different exercise protocol used in each group, and evaluation of physical activity using a questionnaire. Of note, however, the questionnaire we used showed good sensitivity and reproducibility in previous studies [21], and in our study we found a significant relationship between physical activity score based on the questionnaire and cardiorespiratory fitness parameters as determined during the cardiopulmonary exercise test. In addition, levels of the evaluated biomarkers might be affected by numerous factors, including polymorphisms of genes that code for these proteins. For example, serum sICAM-1 level is affected by diet and the timing of blood collection during the day, and sCD40L level is highly sensitive to preanalytical errors.

CONCLUSIONS

Intensive exercise training and high exercise capacity were associated with lower serum IL-6 and sCD40L levels. Physical activity of this sort may play a role in prevention of atherosclerosis.

Conflict of interest: *none declared*

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Wpływ aktywności fizycznej na stężenia w surowicy wybranych biomarkerów miażdżycy

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Streszczenie

Wstęp: Wiele danych wskazuje, że wysiłek fizyczny korzystnie wpływa na proces zapalny i funkcję śródbłonka naczyniowego, które odgrywają istotną rolę w patogenezie miażdżycy. W dotychczasowych pracach analizowano związek wydolności organizmu i różnych form aktywności fizycznej z biomarkerami miażdżycy, jednak wyniki badań są w wielu miejscach niespójne.

Cel: Celem pracy była ocena związku aktywności ruchowej określanej na podstawie kwestionariusza i wydolności fizycznej organizmu ze stężeniem w surowicy krwi interleukiny-6 (IL-6), rozpuszczalnego ligandu dla receptora CD40 (sCD40L) i rozpuszczalnej cząsteczki przylegania międzykomórkowego ICAM-1 u sportowców i osób nieuprawiających wyczynowo sportu.

Metody: Badaniami objęto 46 sportowców (w tym 29 piłkarzy i 17 koszykarek) w wieku 22,89 ± 5,1 roku. Grupę kontrolną stanowiło 46 zdrowych ochotników dobranych pod względem płci, wieku i wskaźnika masy ciała, nieuprawiających wyczynowo sportu. Aktywność fizyczną oceniono przy użyciu krótkiej wersji Międzynarodowego Kwestionariusza Aktywności Fizycznej. W celu oceny wydolności fizycznej u osób badanych wykonano test ergospirometryczny na bieżni ruchomej. Stężenie IL-6, sCD40L i sICAM-1 oznaczono metodą ELISA w próbkach krwi pobranych z żyły łokciowej bezpośrednio przed wysiłkiem i na szczycie wysiłku.

Wyniki: W całej badanej populacji zanotowano istotny statystycznie wzrost stężenia IL-6 na szczycie wysiłku w porównaniu z wartościami wyjściowymi (1,35 *v.* 1,46 pg/ml; p = 0,01), natomiast nie stwierdzono istotnych powysiłkowych zmian stężenia sCD40L i sICAM-1 (odpowiednio 1813,16 ± 2266,46 *v.* 2316,25 ± 2615,57 pg/ml; p = 0,069 i 171,86 ± 39,71 *v.* 172,31 ± 41,46 ng/ml; p = 0,49). Grupa sportowców charakteryzowała się większym dziennym wskaźnikiem aktywności fizycznej (1083,2 ± 227,9 *v.* 285,3 ± 152,8 MET-min/dzień; p < 0,001), większą liczbą minut spędzonych tygodniowo na wysiłku intensywnym (660,0 ± 177,8 *v.* 59,2 ± 76,9 min/tydzień; p < 0,001) i umiarkowanym (420,0 ± 177,8 *v.* 97,5 ± ± 70,4 min/tydzień; p < 0,001) oraz większym zużyciem tlenu na kilogram masy ciała w punkcie beztlenowym (34,7 ± 10,3 *v.* 25,6 ± 8,4 ml/kg/min; p < 0,001) i na szczycie wysiłku (53,3 ± 5,1 *v.* 40,9 ± 10,2 ml/kg/min; p < 0.01). Zaobserwowano, że grupa sportowców w porównaniu z grupą kontrolną charakteryzowała się istotnie statystycznie mniejszym spoczynkowym stężeniem IL-6 (0,7 ± 0,9 *v.* 1,8 ± 3,5 pg/ml; p = 0,003) i sCD40L (888,8 ± 892,9 *v.* 2367,7 ± 8743,4 pg/ml; p = 0,005), natomiast nie wykazano istotnych różnic między stężeniem we krwi sICAM-1. Analiza korelacji przeprowadzona łącznie w obu grupach, sportowców i kontrolnej, wykazała istotny związek między liczbą minut spędzanych tygodniowo na intensywnym i umiarkowanym wysiłku oraz wskaźnikiem aktywności fizycznej ocenianym na podstawie kwestionariusza a zużyciem tlenu w punkcie beztlenowym i na szczycie wysiłku. Stwierdzono odwrotną korelację między spoczynkowym stężeniem IL-6 a zużyciem tlenu na kilogram masy ciała na szczycie wysiłku (p = 0,035), jak również odwrotną korelację między spoczynkowym stężeniem IL-6 i CD40L a liczbą minut spędzanych tygodniowo na wysiłku intensywnym (odpowiednio p = 0,028 i p = 0,006) i umiarkowanym (odpowiednio p = 0,035 i p = 0,022) oraz wskaźnikiem aktywności fizycznej (odpowiednio p = 0,02 i p = 0,004). W wieloczynnikowej analizie regresji wykazano, że zmiennymi wpływającymi na stężenie ligandu CD40 przed wysiłkiem jest wskaźnik aktywności fizycznej (p = 0,007).

Wnioski: Stwierdzono wpływ intensywnego wysiłku i wysokiej wydolności fizycznej organizmu na zmniejszenie stężeń IL-6 i CD40L w surowicy. Tego rodzaju aktywność fizyczna może mieć znaczenie w profilaktyce miażdżycy.

Słowa kluczowe: aktywność fizyczna, biomarkery, zapalenie, śródbłonek

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