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Early markers of endothelial dysfunction in young professional athletes

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Abstract

Objective. *To identify early markers of endothelial dysfunction (ED) in professional athletes depending on risk factors (RF) and genetic polymorphisms.*

Materials and methods. *The study included 78 athletes aged from 18 to 45 years old involved in various professional sports: athletics, soccer, volleyball, boxing, Greco-Roman wrestling, mixed martial arts. The analysis of physical activity was based on the Mitchell's sports classification (2005). The anonymous survey was performed to assess the presence of the main cardiovascular (CV) RF. All study participants, along with a physical examination, underwent blood pressure (BP) assessment and volumetric sphygmography. The association between gene polymorphisms (ACE, ADD1, AGT, AGTR1, AGTR2, CYP11B2, GNB3, NOS3), arterial wall parameters and CV RF was studied.*

Results. *Elevated BP was registered in 25 athletes (32.1%) with average systolic blood pressure (SBP) — 146.3±4.3 mmHg, diastolic blood pressure (DBP) — 83.8±7.43 mmHg. At the same time, differences in BP between various types of sports were not statistically significant (χ^2=1.67, df=1, p=0.196). The level of SBP was associated with pulse wave velocity (PWV) (r=0.463, p<0.05), the risk of ED development correlated with SBP level, expressed through PWV (χ^2=19.940, p<0.001; OR=44.6, 95% CI: 5.0–392.8).*

Conclusion. *Professional athletes with the presence of RF (grade 1 and stage I arterial hypertension) showed increased PWV values that reflected ED, which can indicate the presence of early-stage vascular remodeling, in case when the influence of genetic polymorphisms of the renin-angiotensin-aldosterone system was excluded.*

Keywords: *endothelial dysfunction, risk factors, physical activity.*

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Introduction
Nowadays, much attention is paid to the field of preventive medicine, and to one of the most significant risk factors (RF) for the development of cardiovascular diseases (CVD) is the level of adherence to physical activity (PA). The adverse effect of low PA on an organism has already been proven, especially in young people [1].

At the same time, intense PA in professional sports can also be considered as a risk factor that contributes to early development of CVD. Intense PA that exceeds body capabilities and does not consider morphological adaptation process, increases the risk of endothelial dysfunction (ED) and atherosclerosis [2].

Modern achievements of scientific technology have revealed new cellular and molecular elements of atherosclerosis, such as ED and vascular wall stiffness, which play the key role in its pathogenesis. At the same time, the influence of allelic variations in gene expression and its association with subclinical changes of vascular wall have not been studied enough yet. At the moment, it has been established that some genetic polymorphisms of the renin-angiotensin-aldosterone system (RAAS) can aggravate the prognosis of patients, for instance, they can play a pivotal role in the development of several CVDs, such as arterial hypertension (AH) that often causes structural changes in the arterial wall, and, eventually leads to ED.

Decreased nitric oxide synthase (NOS) activity lowers the bioavailability of NO that causes ED and triggers further vascular wall remodeling.

Endothelial function impairment can be used as early preclinical marker of vascular lesion assessed with new innovational low invasive methods that are used in clinical practice. For example, vascular stiffness parameters can be assessed with volumetric sphygmography [3].

Timely detection of vascular wall changes, especially in professional athletes, is an important issue of modern scientific research.

The objective of this study was to identify early markers of ED in professional athletes, depending on the presence of RF and genetic polymorphisms.

Materials and methods
The study included patients who have constant intense PA and are involved in professional sports.

This cross-sectional study was based on the Republican Medical and Sports Dispensary of Saransk and included 78 athletes involved in various professional sports (athletics, soccer, volleyball, boxing, Greco-Roman wrestling, mixed martial arts) aged from 18 to 35 years old who underwent a periodic medical examination. PA was assessed according the Mitchell's sports classification (2005). The sports experience of the participants ranged from 5 to 27 years.

The main exclusion criteria for the study were: acute stages of were chronic diseases, postoperative period and secondary symptomatic hypertension.

The study was conducted in accordance with the standards of good clinical practice and the principles of the Declaration of Helsinki. The study protocol was

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approved by the Ethics Committee of Moscow State University named after N.P. Ogaryova on January 29, 2019, protocol No. 71.

All study participants were enlightened with the study design and signed voluntary informed consent prior to the participation. RF assessment was carried out by face-to-face interviews and questionnaires in order to identify specific signs of cardiovascular pathology such as CVD in first-degree relatives and episodes of increased blood pressure (BP).

The questionnaire was developed at the Department of Internal Medicine of the Moscow State University named after N.P. Ogaryova. Physical examination was performed with the assessment of height, body weight and BP measurement.

Height (cm) was measured using mechanical stadiometer RP (Russia) in a standing position. Body weight (kg) was assessed with mechanical floor scale (Momert 5200, Hungary). Body mass index (BMI) was calculated using the following formula: BMI = body weight (kg)/height (m^2).

The measurement of "office" BP was performed by auscultatory method using a professional mechanical tonometer (Little Doctor LD-71, Singapore) while patient was sitting three times with 1–2 minutes interval. Volumetric sphygmography using the "VaSera VS-1500" device (Fukuda Denshi, Japan) recorded the level of BP in upper and lower extremities, and a visual assessment of the BP balance in four vascular beds was carried out.

The stage of arterial hypertension was assessed according to the clinical guidelines for arterial hypertension in adults (Russian Society of Cardiology, 2020) [4].

All athletes with elevated BP (over 140/90 mmHg) underwent additional examination: electrocardiogram (ECG) (Med-Mos ECG300G, China), echocardiography (Echo-CG) and carotid artery duplex scan (Toshiba Xario SSA-660A, Japan), urinalysis with albumin / creatinine ratio assessment (iChem Velocity, Beckman Coulter, USA), blood test with lipid profile assessment, serum creatinine level (Architect c8000, Abbott, USA) and calculation of glomerular filtration rate (GFR) in ml/min/1.73 m^2 using the Chronic Kidney Disease Epidemiology formula.

To identify the signs of ED, a complex examination was performed and included the assessment of parameters that reflect the vascular wall stiffness. These parameters were assessed by volumetric sphygmography using the VaSera VS-1500 device (Fukuda Denshi, Japan) and included: ankle-brachial index (ABI), pulse wave velocity (PWV) and cardio-ankle vascular index (CAVI). The evaluation of indicators was performed in accordance with the guidelines for the use of the apparatus.

Genetic testing included the analysis of the presence of RAAS gene polymorphisms that are involved in the development of CVD, and may also contribute to vascular wall remodeling.

Molecular genetics analysis was carried out in the central research laboratory of the Penza Institute for Postgraduate Medical Education. Genetic polymorphisms were studied using a real-time polymerase chain reaction and a set of reagents "CardioGenetics Hypertension" by "DNA-Technology" (Russia) (Table 1).

The distribution of genotype frequencies of the studied genes corresponded to the Hardy-Weinberg equilibrium. Calculations were performed using the online calculator available by the following link: https://wpcalc.com/en/equilibrium-hardy-weinberg/.

Statistical analysis was performed using the Statistica v.10.0 program (StatSoft, USA). The normality of the distribution was assessed using the Kolmogorov–Smirnov test, and also considered the indicators of kurtosis and symmetry. For normally distributed parameters, the arithmetic mean and standard deviation (M±SD) were calculated. Categorical

Table 1. **Genetic polymorphisms characteristics**

Gene	Protein	Genetic marker	RS	Genotype variants
ACE	angiotensin converting enzyme	ALU INS/DEL	4646994	I/I; I/D; D/D
ADD1	α–adductin	G1378T	4961	G/G; G/T; T/T
AGT	angiotensinogen	T704C	699	T/T; T/C; C/C
		C521T	4762	C/C; C/T; T/T
AGTR1	angiotensin II receptor type 1	A1166 C	5186	A/A; A/C; C/C
AGTR2	angiotensin II receptor type 2	G1675A	1403543	G/G; G/A; A/A
CYP11B2	cytochrome 11b2 — aldosterone synthase	C344T	1799998	C/C; C/T; T/T
GNB3	G Protein Subunit Beta 3 — guanine — binding protein	C825T	5443	C/C; C/T; T/T
NOS3	nitric oxide synthase	T786C	2070744	T/T; T/C; C/C
		G894T	1799983	G/G; G/T; T/T

data are presented in absolute values and percentages (%). Comparative analysis of quantitative variables was performed using the Mann–Whitney test (U-test), for qualitative variables — chi-square test (χ^2). In addition, the odds ratio (OR) with 95% confidence interval (CI) were calculated. To determine the association between quantitative parameters, linear regression and correlation analysis Pearson correlation coefficient or Spearman's Rank correlation coefficient (r) were used. The level of statistical significance was set as $p<0.05$.

Study results

The study included 78 athletes with average age of 24.6±5.3 years old, and average sports experience of 11.6±6.5 years. The majority of the participants were male (91%) and 9% — female. Average age did not differ significantly between men and women (24.7±5.4 years for males and 23.7±4.7 years for females), (p = 0.731). Sports experience was also comparable between men and women (11.6±6.6 years and 11.6±6.0 years, respectively), (p = 0.889).

The analysis of family history and questionnaire results revealed that 22 (28.2%) athletes were predisposed to CVDs. Most often, athletes had relatives with elevated BP. Family history of AH was detected in 16 (20.5%) athletes, MI in 3 (3.8%) athletes, and pulmonary embolism in 1 (1.3%) athlete. The presence of CVD was on the mother's side in 15 (68.2%) athletes, on the father's side — in 3 (13.6%) athletes, and on both sides — in 4 (18.2%) athletes. Family history of CVD was more common in male athletes (86.4%).

The anthropometric data revealed that men were taller (180.9±7.9 cm) than women (164.0±5.7 cm) and had higher average body mass (in men 78.4±12.4 kg, in women 54, 4±5.2 kg) ($p<0.001$). The average BMI was 23.7±3.8 kg/m^2 in men that was significantly higher value (24.0±3.8 kg/m^2) compared with women (20.2±1.5 kg/m^2) ($p < 0.001$).

According to the office BP assessment, the average level of systolic BP (SBP) was 133.2±11.2 mm Hg, diastolic BP (DBP) — 78.7±6.9 mm Hg. Mean SBP in women was 124.3±8.8 mmHg, DBP — 75.0±5.8 mm Hg, mean SBP in men — 134.1±11.1 mm Hg, DBP — 79.1±7.0 mmHg. SBP was significantly lower in women (p=0.032), and DBP did not differ significantly (p=0.115).

According to three-time simultaneous and dynamic (within 1 week) BP assessment by volumetric sphygmography and within the classification of AH [4], 32 (41%) athletes, showed BP within reference values. In 9 athletes both SBP and DBP levels corresponded to optimal BP, and in 23 — to normal BP. High normal blood pressure was detected in 21 (26.9%) athletes with the mean SBP of 134.5±2.9 mmHg, DBP — 78.4±5.2 mm Hg. Elevated BP ⩾140/90 mm Hg was revealed in 25 (32.1%) athletes that corresponded to stage 1 AH with mean SBP — 146.3±4.3 mm Hg, DBP — 83.8±7.43 mm Hg. There was no statistically significant difference of BP values depending on the type of sport (χ^2=1.67, df=1, p=0.196).

The combination of family history of CVD and elevated BP was seen in 10.2% athletes.

Thus, we identified athletes with RF (family history of CVD and/or elevated BP) and without RF. Subsequently, all the athletes were divided into 2 groups.

Group 1 consisted of athletes without RF (n=31), group 2 included athletes (n=47) with RF (with AH and/or family history of CVD).

The characteristics of study groups are presented in Table 2. The groups were comparable by gender, sports experience, age and anthropometric data.

The main difference between groups was the level of BP. All athletes with BP values exceeding normal values according to AH classification [4] underwent additional examination in order to detect hypertensive target organ damage (TOM). According to ECG and ECHO-CG, there were no signs of left ventricular hypertrophy. Carotid artery duplex scan did not reveal any atherosclerotic plaques. Blood creatinine level and estimated GFR were within reference values. The albumin-creatinine ratio in a single urine portion was normal. The lipid profile of all study participants remained normal.

Considering the formation of study groups depending on the presence of RF, the assessment of vascular wall stiffness parameters was carried out using volumetric sphygmography in order to identify the subclinical signs of ED.

The parameters of vascular wall stiffness were comparable between groups regardless of the presence of RF (table 3). Despite the fact that PWV parameters were significantly higher in athletes with RF compared with the group without RF (table 3), revealed parameters of arterial stiffness were within reference values.

The analysis of PWV indicators between the groups showed ambiguous results and, therefore, they were divided into subgroups depending on the obtained PWV values (normal/above normal) (table 4).

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Table 2. **Clinical characteristics of study participants depending of the presence of RF**

Parameter		Data presentation	Group 1 (without risk factors) n=31	Group 2 (with risk factors) n=47	p
Gender	Male	ABS. (%)	28 (90.3)	43 (91.5)	0.810
	Female		3 (9.7)	4 (8.5)	
Sports experience, years		M ± SD	10.6±7.0	12.3±6.1	0.192
		95% CI	8.1–13.2	10.5–14.1	
Age, years		M ± SD	24.5±5.6	24.7±5.1	0.914
		95% CI	22.5–25.6	23.1–26.2	
Height, cm		M ± SD	178.3±8.0	180.0±9.7	0.339
		95% CI	175.4–181.2	177.2–182.9	
Body mass, kg		M ± SD	73.4±10.3	78.2±15.4	0.244
		95% CI	69.6–77.1	73.7–82.7	
BMI, kg/m²		M ± SD	23.0±2.5	24.1±4.5	0.500
		95% CI	22.1–23.9	22.8–25.4	
SBP, mmHg		M ± SD	124.9±8.2	138.7±9.4	0.001
		95% CI	122.0–128.0	135.9–141.4	
DBP, mmHg		M ± SD	75.1±5.3	81.1±6.9	0.001
		95% CI	73.1–77.0	79.1–83.1	
Pulse BP, mmHg		M ± SD	49.9±7.1	57.6±8.7	0.001
		95% CI	47.3–52.3	55.0–60.1	
Mean BP, mmHg		M ± SD	92.1±6.2	100.8±7.2	0.001
		95% CI	89.8–94.4	98.7–102.9	
Heart rate, beats/min		M ± SD	60.5±14.0	59.9±10.6	0.744
		95% CI	55.4–65.6	56.8–63.0	

Table 3. **Vascular wall stiffness parameters that reflect endothelial dysfunction in athletes**

Parameter	Group 1 (without risk factors) n=31	Group 2 (with risk factors) n=47	p
R-CAVI	5.6±0.7	5.8±0.7	0.211
L–CAVI	5.8±0.7	6.0±0.6	0.345
R-ABI	1.1±0.1	1.1±0.1	0.159
L-ABI	1.1±0.1	1.1±0.1	0.140
PWV	4.7±0.8	6.5±2.9	0.002

Table 4. **Vascular wall stiffness parameters that reflect endothelial dysfunction in athletes**

Parameter	Group 2 (with risk factors) n=47		p
	Normal PWV (n=29)	Above normal PWV (n=18)	
R-CAVI	5.9±0.7	5.8±0.8	0.792
L–CAVI	6.0±0.6	5.9±0.7	0.704
R-ABI	1.1±0.1	1.1±0.1	0.616
L-ABI	1.1±0.1	1.0±0.1	0.275
PWV	4.8±1.1	9.6±2.7	0.001

The results of PWV indicators analysis in the subgroups attracts attention to the ratio of athletes with family history of CVD as well as with elevated BP. Thus, in the subgroup with normal PWV values, 5 athletes had a combination of RF (family history of CVD and elevated BP), 21 had family history of CVD, and 3 — elevated BP.

In the subgroup with high PWV, 3 athletes had combination of RF (family history of CVD and elevated BP), 1 athlete had family history of CVD and 14 athletes had elevated BP.

The correlation analysis between SBP and PWV indicators was performed in order to find the cause of PWV elevation in the group with RF (figure 1). The parameters showed statistically significant positive correlation ($r=0.463$, at $p<0.05$).

The odds ratio (OR) was also calculated in order to identify the risk of ED development by the PWV and BP values elevation (table 5). Elevated BP increased the risk of ED development by 44.6 times that was estimated by PWV. At the same time, multiple regression analysis did not reveal statistically significant association between PWV and gender, age, anthropometric parameters (height, body mass, BMI), sports experience, DBP, pulse and mean BP, heart rate, and family history of CVD ($p>0.05$).

Considering the role of genetics in the development of CVD, especially those genes that are associated with the RAAS system, we also performed the screening for 10 genes of the RAAS system in all athletes, depending on the presence of RF (Table 6).

The comparison analysis did not reveal any significant differences between groups depending on the

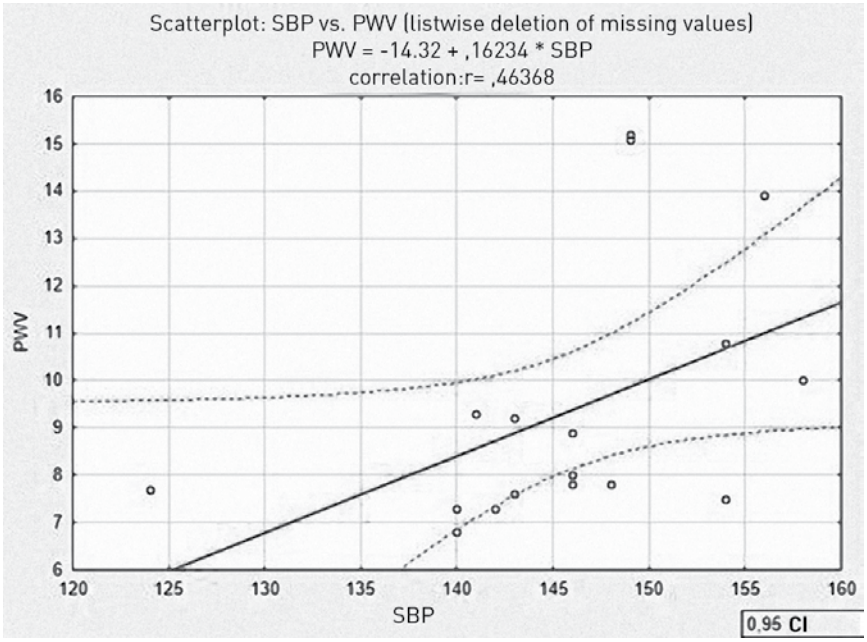

Figure 1. The association between PWV and SBP

Table 5. **Relative risk of PWV increase in athletes with elevated SBP**

BP	Above normal PWV (n=18)	Normal PWV (n=29)	Pearson's chi-squared test	OR (95% CI)
	%	%		
SBP ⩾ 140 mmHg (n=25)	94.4	27.6	19.940 P<0.001	44.625 (5.0–392.8)
SBP < 140 mmHg (n=22)	5.6	72.4		

presence of RF. The analysis of genetic polymorphisms of the RAAS system, including endothelial nitric oxide synthase (eNOS), did not reveal any statistically significant difference (p>0.05) depending the PWV value.

Discussion

In the last years excessive PA in professional sports has been discussed as the factor that can lead to the development of AH, atherosclerosis and sudden cardiac death.

The results of the studies dedicated to the investigation of AH prevalence showed ambiguous results on the frequency of elevated BP depending on the PA type.

According to the data of foreign literature review, the prevalence of AH in athletes involved in weightlifting and American football ranged from 8.8 to 25.6%, which is higher than in athletes involved in other sports [5].

The data on the AH prevalence in domestic studies are also noteworthy. Thus, elevated BP was diagnosed in 37% of athletes in heavyweight categories that include athletes involved in such sports as bodybuilding, powerlifting, weightlifting [6].

The results of our study showed that athletes with elevated BP were commonly involved in dynamic and static exercising (χ^2=1.67, df=1, p=0.196). Similar data were obtained in the Yakut population, where there was also no relationship between AH, type of sport and sportsmanship (χ^2=3.48, df=1, p=0.062) [7].

At the same time, it has been established that high-intensity exercising accelerated the development of ED and atherosclerosis.

Thus, Smirnov I. E. et al. [8] performed quantitive analysis of blood levels of angiogenin, vascular endothelial growth factor, homocysteine, endothelin and nitric oxide in young athletes that showed the change of ED parameters during myocardial overload due to physical activity.

Nowadays, ED can be interpreted as universal mechanism that implements the action of all CVD RF. Clinical trial [9] and, in particular, the Rotterdam study [10], showed that early signs of an atherosclerosis can be detected with PWV that is a local indicator of elastic properties of the arteries and can be used as early a marker of ED, and elevated BP is the factor of microvascular lesion.

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Table 6. **Genetic polymorphisms frequency in groups depending on the RF**

Genetic polymorphism	Genotype	Group 1 (without risk factors) n=31	Group 2 (with risk factors) n=47	p
ACE: Alu Ins/Del	I/I	63.2	57.9	0.159
	I/D	36.8	42.1	0.306
	D/D	0	0	0
ADD1: G1378T	G/G	56.5	72.2	0.215
	G/T	34.8	27.8	0.569
	T/T	8.7	0	0.072
AGT: T704C	T/T	34.8	16.7	0.111
	T/C	52.2	52.8	0.964
	C/C	13.0	30.6	0.124
AGT: C521T	C/C	73.9	73.5	0.975
	C/T	26.1	23.5	0.826
	T/T	0	3.0	0.383
AGTR1: A1166C	A/A	45.5	65.7	0.132
	A/C	50.0	31.4	0.161
	C/C	4.5	2.9	0.736
AGTR2: G1675A	G/G	47.8	50.0	0.873
	G/A	8.7	2.9	0.340
	A/A	43.5	47.1	0.791
CYP11B2: C344T	C/C	30.5	22.9	0.520
	C/T	47.8	51.4	0.789
	T/T	21.7	25.7	0.730
GNB3: C825T	C/C	46.7	39.4	0.636
	C/T	33.3	54.5	0.116
	T/T	20	6.1	0.143
NOS3: T786C	T/T	43.5	38.9	0.727
	T/C	39.1	44.4	0.688
	C/C	17.4	16.7	0.943
NOS3: G894T	G/G	69.9	51.4	0.171
	G/T	21.7	42.9	0.098
	T/T	8.7	5.7	0.662

There is a fairy significant evidence base on the effect of AH on the parameters of vascular wall stiffness both in general population and in professional athletes. It also has been shown that the values of vascular wall stiffness correlated with high SBP at the 5th stage of the test with dosed PA in young athletes [11].

The analysis of BP parameters in our study revealed grade 1 AH in the examined athletes without target organ damage with the increase of PWV parameters. Elevated BP and PWV positively correlated with each other ($r=0.46$ at $p<0.05$). It has also been shown that grade 1, stage 1 AH increased the risk of PWV elevation by 44.6 times (CI — 5.0–392.8). The analysis of the association between elevated PWV and the following RF: age, types of PA sports experience, BMI, did not reveal any significant correlations. Thus, the revealed grade 1 stage 1 AH can be considered as the RF, and PWV — as an early marker of ED.

It should also be noted that ED can be genetically determined. However, the role of genetic screening in atherosclerosis prediction is controversial. In this regard, several studies on the potential association between the allelic variations of genes and subclinical changes of the arteries are currently underway.

Akopyan A. A. et al.[12] was the first one who have found that the presence of the D allele of the ACE gene increased the risk of arterial stiffness by 1.89 times (95 % CI: 1.16–3.12), and the presence of the DD genotype — by 3.42 times (95 % CI: 1.39–9.36). The association between ED and the c.894G>T polymorphism of the NOS3 gene have also been established. The presence of the GG genotype increased the risk of ED by 2.65 times (95 % CI: 1.26–5.72), according to the literature, the TT genotype of the NOS3 gene polymorphism was also associated with atherosclerosis. Lack of consistent results in numerous studies, including investigations in different populations, makes uncertain the role of genetic polymorphisms in the pathogenesis of atherosclerosis.

We have selected allelic variants of the RAAS genes (ACE, ADD1, AGT, AGTR1, AGTR2, CYP11B2, GNB3, NOS3), that could be possibly associated with

the parameters of arterial wall and identified CVD RF. The results of comparative analysis between groups of athletes depending on the presence of RF and PWV and carriage of genetic polymorphisms have not found any significant differences.

This may be explained by little effect of the individual genetic variants on the risk of vascular remodeling, atherosclerotic changes in particular. Its effects are enhanced in synergy with other genetic and behavioral factors.

In addition, the absence of genetic predisposition to CVD in athletes can be explained by the selection of practically healthy young people for professional sports.

Conclusion

Thus, in young professional athletes aged from 18 to 35 years old with elevated BP (grade 1, stage 1 AH) that was considered as the risk factor, PWV was increased. PWV is included in the criteria that determine the stages of vascular wall stiffness and can be used as early marker of ED.

No association between PWV and RAAS gene polymorphisms in this study can be explained by the features of study sample — young professional athletes.

Considering the fact that traditional scales for the assessment of cardiovascular risk may underestimate the occurrence of cardiovascular complications, the determination of vascular wall stiffness by volumetric sphygmography and PWV indicators in particular, has high prognostic value. This method of examination is especially relevant for people with intense PA that may "mask" subjective sensations.

Genetic screening can be recommended for individuals with family history of AH.

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