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Original article

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Running title: Maximal vs. submaximal exertion and CHD

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Abstract

Background: Decisions about fat consumption and levels of physical activity are among the everyday choices we make in life and risk of coronary heart disease (CHD) can be affected by those choices.

Objective: The purpose of this study was to investigate the influence of a standard fat load combined with physical exertion of different intensities on the plasma lipid profile of CHD patients and CHD-free individuals.

Methods: This study looked at the influence of different intensities of physical exercise on postprandial lipid metabolism in 20 healthy men and 36 men with diagnosis of coronary heart disease (CHD). Venous blood samples were obtained after overnight fasting, 3 hours after standard fat load (before the physical load) and immediately after maximal or submaximal physical exercise on bicycle ergometer.

Results: After fat load total cholesterol (TC) concentration did not change in either group. However, after the addition of maximal exercise, TC, triglycerides (TG), low density lipoprotein cholesterol (LDL-C), and apolipoprotein (Apo) B increased significantly ($p<0.01$) in both groups. After fat load and maximal exercise, there was no change in high density lipoprotein cholesterol (HDL-C) in healthy men, but in men with CHD, HDL-C fell significantly ($p<0.01$); and Apo AI rose in healthy men ($p<0.01$) but dropped significantly ($p<0.01$) in men with CHD. Submaximal physical exercise (60% of max $V0_2$ load for 40 min) after fat load decreased TG level in CHD patients ($p<0.01$), and improved other lipid parameters in both groups significantly (↓LDL-C, ↑HDL-C, ↑ Apo AI, ↓ Apo B, $p<0.01$). We observed a worsening of physical work capacity (PWC) in men with CHD (significant reduction of duration and total amount of work performed, maximal $V0_2$, oxygen pulse), during maximal stress test performed 3 hours after fat load. There was a doubling of the number of abnormal stress test results ($p<0.01$). Healthy persons showed an increase in respiratory parameters (ventilation, $C0_2$ production, maximal $V0_2$ and oxygen pulse), but no significant change was found in work capacity. Thus, maximal physical exercise produced atherogenic blood lipid changes (increased TC, increased LDL-C, increased TG, increased Apo

B, p <0.01) in men with CHD and in healthy men; however, individuals with CHD also demonstrated a significant decrease in HDL-C and Apo AI (p <0.01). In contrast, the submaximal physical load improved postprandial lipid changes in both healthy men and men with CHD.

Conclusions: This study demonstrates that moderate exercise is beneficial in improving postprandial lipid abnormalities in both CHD and CHD-free subjects after fatty meal preload. In addition, maximal exercise demonstrated evidence of increase of lipid abnormalities in both CHD and CHD-free individuals under similar conditions of fatty meal preload.

KEYWORDS*:* Coronary heart disease (CHD); Combined fat and physical load; Physical work capacity; Apolipoproteins AI and B

Introduction

Decisions about fat consumption and levels of physical activity are among the everyday choices we make in life and risk of CHD can clearly be affected by those choices. Dieary factors, especially cholesterol and saturated fat consumption, have been demonstrated to be associated with increased risk of CHD.[1-3] Evidence suggests that not only the long- term effect of dietary habits effect CHD risk, but even the fatty content of a single meal may effect risk.[4,5] Zilversmit [6] has advanced the hypothesis of «postprandial phenomena», according to which the postprandial triglyceride-rich chylomicrons play an important role in atherogenesis. It is widely appreciated that regular occupational and recreational physical activity may reduce the risk of CHD. However, in prescribing physical activity, especially to CHD patients, it is advisable to specify the intensity of activity, since there are data suggesting that very strenuous occupational or leisure time physical effort is associated with a higher risk of CHD.[7-9] The purpose of this study was to investigate the influence of a standard fat load combined with physical exertion of different intensities on the plasma lipid profile of CHD patients and CHD-free individuals.

Material and methods

Study participants

Fifty six untrained adult men ranging in age from 33 to 56 were enrolled in the study. There were two groups. Group I was comprised of 20 CHD-free men with a mean age of 43 ± 1.2 years and no evidence of CHD after selective coronary angiography (performed because of suspicion of CHD), maximal bicycle stress test and clinical examination. Group II was made up of 36 CHD patients with a mean age of 47 ± 2.1 years and a functional class I after the same type of evaluation.

In our study, the Canadian Cardiovascular Society grading of angina pectoris (sometimes referred to as the CCS Functional Classification of Angina)[10,11] was used to grade the severity of angina pectoris.

Individuals in group 2 had demonstrated evidence of stenosis on coronary angiograms or 2-mm or more of ischemic type depression of S-T segment during maximal bicycle stress test with or without angina pain. Only 9 of 25 patients of the group 2 had stenosis (less that 50% of diameter; non-left main coronary artery). Ischemic changes in ECG were seen on the peak of maximal stress test, according to the age.[12] It is commonly accepted that these patients do not need any type revascularization (as there are no anatomic or physiologic criteria for revascularization).[13] Patients with diabetes mellitus, or having Quetelet body mass index > 29, liver, kidney, or bronchopulmonary dysfunction were excluded from the study. All subjects were free of any medication.

The study was conducted in accordance with the Helsinki Declaration and informed consent was obtained from the study participants. The study was approved by the ethics committees of the National Research Center for Preventive Medicine, Moscow, and Institute for Atherosclerosis, Moscow, Russian Federation.

Analyzed parameters

Standard fat load

Standard fat load: the standard fat load consisted of emulsified 20% milk cream (65 g of fat for 1 sq m of body surface, 639 ± 7 ml) and 50 g of wheat bread as described by Patsch *et al.*[14] This breakfast contained an average 510 mg of cholesterol, 130 g of fat (saturated fat 77.6g. (59.7%)) and 23.4 g of carbohydrates for approximately total 1300 ccal.

Stress tests

a) The maximal stress test was a continuous gradual step-wise bicycle exercise in a sitting position. At the first stage, the load was 300 kgm/min. Each successive load increased by this value. Each work load was sustained for 3 minutes. The interruption criteria included the attainment of full fatigue, or maximal heart rate (HR), blood pressure increase to 230/120 mm Hg or greater, provocation of angina pectoris, ischemic ST segment

displacement greater than or equal to 2 mm. Mean duration of maximal stress test was 582 ± 20 sec for group I and 461 ± 31 sec for group II. The amount of energy expended were 59.14 KJ and 41.75 KJ, respectively.

Simultaneously, electrocardiogram (ECG) monitoring with an oscilloscope and ECG in 12 leads was performed before exercise, at the end of every minute during exercise, and at 30 seconds, 1, 2, 3, 5, 7, and 10 minutes after exercise. During this time, blood pressure (BP) was measured according to the Korotkoff method using a mercury sphygmomanometer.

Maximal physical workload test according to the method, described above, was performed twice: first time - for diagnostic reason without drawing blood for lipids, second time - 3 hours after the standard fat load. Blood samples were drawn from the cubital vein for the measurement of lipid and apolipoprotein levels in the morning at fasting condition before the fat load and 3 hours after the fat load. Immediately after drawing the blood a patient performed the maximal physical workload test, after which the third blood sample was drawn for lipids tests (immediately after the test was stopped).

b) 3 days after the first combined fat load and physical workload, a patient performed physical exercise on a bicycle ergometer for 40 minutes, power being equal to 60% from that found during the earlier individual exercise test: as the combined fat and maximal physical workload test, this test was performed 3 hours after the standard fat load. Blood collection for lipids was done before meal, then, 3 hours after the standard fat load before the submaximal physical workload, which lasted 40 minutes and then, immediately after it was stopped. The amounts of energy expended were 233.01 KJ and 203.36 KJ for group.

In Table 1 there are figures on the amount of work and energy expenditure of the performed tests.

Oxygen consumption, carbon dioxide production, minute ventilation during the physical exercise and alimentary load

Oxygen consumption, carbon dioxide production, minute ventilation during the physical exercise and alimentary load were determined with a gas autoanalyzer “Horizon” (“Beckman”, Austria).

Parameters of blood, analyzed in the study

Venous blood samples were taken after overnight fasting, 3 hours after alimentary load (before physical exercise) and immediately after completion of maximal or submaximal physical exercise.

The following parameters of blood were assayed: The concentration of serum cholesterol (CH) and triglycerides (TG) were determined by the enzymatic method on "Centrifixem-600" autoanalyzer. The very low density lipoprotein cholesterol and chylomicrons were isolated by ultracentrifugation of serum for 18 hr at 13° C by 3600 rev/min, rotor - 40.3 (by Lingren,[15]).

In this study, we were interested to investigate the dynamics of the main atherogenic lipoproteins (LDL) and antiatherogenic lipoprotein (HDL), as well as apolipoproteins B and AI in postprandial and postexercise periods. TG are major lipids of these both fractions of chylomicrons and VLDL (TG-rich lipoproteins); and thus, the level of TG was thought to represent a key characteristic of these fractions.

Concentration of HDL-C in infranatant was measured after heparin manganese precipitation of Apo B - containing lipoproteins using a "Technicon AA-L" autoanalyzer. Concentration of LDL-C was calculated as the difference between the TC of infranatant and HDL-C. Apolipoproteins AI and B were measured by "rocket" electroimmunoassay method.

All fasting and postprandial lipid and apolipoprotein parameters had analysis repeated to confirm measurements and they proved consistently accurate with no significant difference between measurements ($p>0.05$).

Statistical analysis

The data obtained were processed by variational techniques of statistics using an SAS software package. The variation difference was determined by the Student's and Wilkoson's t test and chi-square. The differences were considered statistically significant if $p<0.05$.

Results

Table 2 shows the parameters of PWC of both subject groups before and after the standard fat load. All PWC parameters of healthy persons except the oxygen pulse were higher than those of CHD patients. Nevertheless, CHD patients had a sufficiently high $V0_2$ (27.8 ± 1.2 ml/kg.$^{-1}$ min^{-1} or 7.6 ± 0.4 MET's) indicating that they were functional class I. We specifically selected CHD patients with a relatively high level physical functioning so that they would be able to perform at maximal or near maximal physical effort.

After a standard fat load there were no significant changes in duration or total amount of performed work in healthy persons, but there were significant reductions in these parameters observed in CHD patients ($p<0.05$ and $p<0.01$ respectively).

In spite of performing approximately the same amount of physical work both with and without the fatty meal preload load the $V0_2$ max, metabolic equivalent units (MET's) and oxygen pulse in group I increased significantly ($p<0.01$) after fat load. This was demonstrated by the intensification of pulmonary ventilation (increase of minute ventilation ($p<0.05$), and $C0_2$ production ($p<0.01$).

In contrast, subjects with CHD showed the opposite effect after fat load. Their $V0_2$ max, MET's, oxygen pulse, minute ventilation and $C0_2$ production were all significantly reduced ($p<0.01$). Correspondingly the double product was also lower ($p<0.05$).

Maximal physical exertion in healthy persons did not produce positive electrocardiographic stress test results either with or without fat load. However, in the CHD group the number of abnormal or positive stress test results after fat load increased significantly as evidenced by increased incidence of angina pain, ischemic ST segment depression and ventricular extrasystoles (Table 3).

The changes in lipoproteins concentration after fat load and combined fat and maximal physical load are presented in Table 4. There were no changes of total cholesterol level in both group I and group II after fat load As expected, fat load produced an increase of TG ($p<0.05$) in both group I and II. Fat load caused the level of LDL-C to decrease in group I ($p<0.01$), but no changes were observed in group II. However, the HDL-C level decreased in both groups ($p<0.01$).

Comparing the apolipoproteins in each group after fat load we observed the increase of Apo AI ($p<0.01$) and decrease of ratio Apo B/Apo AI ($p<0.01$) in group I, but decrease of Apo AI ($p<0.01$) and increase of Apo B ($p<0.01$) in group II with a subsequent increase in atherogenicity as judged by the increase of the ratio Apo B/ Apo AI ($p<0.01$).

The addition of a maximal physical load to the fat load dramatically effected lipids and lipoproteins. All lipid parameters were significantly worsened in group II ($p<0.01$) shifting to a more atherogenic profile. Similarly, in group I with maximal physical load TC, TG, LDL-C and Apo B increased significantly ($p<0.01$). However, in group I Apo AI increased ($p<0.01$) and HDL-C and the Apo B/Apo AI remained essentially un-changed.

The influence of submaximal physical exercise for 40 min. on lipids is presented in Table 5. Submaximal physical load reduced postprandial high level of TG, LDL-C, Apo B induced by fat load and increased the level of HDL-C, Apo AI in both groups (except the level of TG in group I). The Apo B/Apo AI ratio also decreased in both groups ($p<0.01$).

In submaximal stress tests (60% of peak power) we did not find ST depression, arrhythmias or angina pain in any patient with CHD.

Discussion

This study demonstrates interesting and clinically significant differences in fat metabolism and lipid transport in persons with and without CHD when challenged with a fatty meal and maximal physical exertion. As

interesting is the apparent therapeutic benefit in both groups when challenged with submaximal exertion after the fatty meal preload.

Hypertriglyceridemia induced by fat load in healthy persons was accompanied by the decrease of LDL-C levels; presumably the result of physiological activation of Apo B and Apo E receptors. Subjects with CHD also had an increase in TG but showed no change in LDL-C after fat load, suggesting blunted receptor sensitivity associated with their CHD.

Fat load in the group of CHD-free subjects led to decrease of HDL-C; this observation has been reported by others.[16,17] In these same subjects the increase in Apo AI accompanied by the decrease of Apo B/Apo AI ratio could be interpreted as evidence of activation of reverse cholesterol transport from the tissues to the liver in response to the fat and cholesterol absorbed from the gastrointestinal tract. In contrast, the decrease of Apo AI, and increase in the Apo B/Apo AI ratio in CHD patients observed could be considered evidence of an atherogenic shift in postprandial lipid transport system.

Maximal physical effort led to increased Apo B - containing lipoproteins (CH, TG, LDL-C) in both groups of subjects, especially in CHD patients, but the changes of Apo AI were dramatically different. Maximal physical effort led to additional significant increase of Apo AI in healthy individuals; however, in CHD patients maximal physical effort further reduced the decrease in Apo AI, observed after fatty meal aggravating an already impaired lipid transport system. The combination of the two factors, fat load and maximal physical efforts, appear to compound disturbances in the lipid transport system of persons with CHD.

The demands on the cardiorespiratory system were increased in postprandial state in both healthy persons and CHD subjects. After a fat load the body requires additional oxygen for the oxidation of fatty food. Strenuous physical work in postprandial state produces further oxygen demand. In healthy subjects this increased demand is responded to by taxing available cardiopulmonary reserve. This was evidenced by increase of minute ventilation, $V0_2$ max, and number of MET's. Thus, healthy persons are able to perform the same amount of physical work after fat load without evidence of myocardial ischemia. It is known that double product indirectly reflects the myocardial oxygen supply.[18] For healthy subjects in this study the double product value after fat load and maximal stress was the same in spite of the increase in $V0_2$ max and MET's.

The increased $V0_2$ max and MET's coupled with the unchanged value of double product after combined fat and maximal physical load indirectly confirms the supposition that the myocardial oxygen supply with the combined load was the same as during isolated physical load (without fat load). The excess oxygen consumption had been used for fat metabolism (oxidation). The demand of additional oxygen supply for fat metabolism requires more intensive function of cardiorespiratory system and an increased cardiac output.

Individuals with CHD may not be able to easily meet the demands for increased cardiorespiratory work induced by postprandial hyperlipidemia. Increased oxygen consumption with heavy physical work added to the metabolic demands of the postprandial hyperlipidemia further compromises the ability of the person with CHD to accomplish both tasks. As might be expected in CHD subjects we observed both a reduced PWC and a deterioration of the lipid profile with an increase of free fatty acids and remnants in blood left unmetabolized.

Strenuous exercise after a fatty meal in CHD patients led to a decrease of $V0_2$ max, the number of MET's, and the double product. These changes indicate a decrease in myocardial oxygen supply. These subjects were not able to maintain adequate cardiopulmonary function resulting in the accumulation of $C0_2$ and the development of metabolic acidosis. Under these circumstances the anaerobic pathway of energy supply is stimulated. Although the anaerobic pathway may allow the CHD subject to continue some exercise it is not at the same capacity or duration as noted prior to fat load and it is associated with increased stress test evidence of ischemia.

Perhaps, the most interesting and clinically significant findings from this study are the contrasts between the hazard of maximal exercise and the general improvements with the performance of submaximal physical exercise of the lipid profiles of both CHD and CHD-free subjects after the fatty meal preload. The relationship between physical activity and CHD has been well studied. It is established that persons with physically active occupations or regularly performing physical or recreational exercise have a lower TC/HDL-C ratio, lower triglycerides, higher HDL-C and a lower rate of CHD and mortality than those who are sedentary.[19-22] A lower level of physical fitness is associated with a higher risk of death from cardiovascular diseases and all causes.[23,24]

While physical activity and fitness is generally beneficial, it is necessary to differentiate the intensity of physical exercise. The Harvard Alumni Health Study [25] showed that the age-standardized mortality rate (deaths/10,000) was the highest (103.78) among those who expended less than 4200 kJ/wk on all activities. The

mortality rate declined when the energy expended increased to 14700 kJ/wk (88.05 death/10,000), but had a tendency to increase when energy exceeded 14700 kJ/wk (90.76). Similarly, the US Railroad Study noted that the death rate (deaths/100) for rate for those who spent less than 250 kcal/wk total leisure time physical activity was 29.8, for those who spent 1000 - 1999 kcal 24.7 and for those who spent 2000 kcal/wk or more 26.2.[26] It has been shown that the risk of primary cardiac arrest is increased during vigorous exercise, whereas moderate regular physical exercise is associated with an overall decreased risk of primary cardiac arrest.[121, 27-29]

The results of our study support these observations and may partly explain the reasons for these phenomena. Elevated lipid levels associated with strenuous work appears to be an adaptive response to provide energy-rich fuel to the large muscle groups. One gram of fat provides 9.461 kcal of energy or approximately twice more than other energy sources. Fat mobilization during strenuous muscle effort is initiated by the adrenals with the release of the norepinephrine and corticosteroids.[30,31] This contributes to the release of free fatty acids and cholesterol in the blood and activates TG synthesis in the liver. At the same time lipoprotein lipase activity is blocked and delayed metabolism of chylomicron remnants and LDL-C occurs. As a result of all these changes HDL-C levels also decrease in the blood.[16,32]

Fat metabolism for energy requires a large amount of oxygen. Metabolic conversion of one gram of fat requires 2019.3 ml oxygen, whereas one gram of carbohydrate requires only 828.6 ml of oxygen.

Thus, while fats are energy rich, they place increased demands on the cardiorespiratory system if they are going to be used as the primary source of fuel for physical work. The stepwise increase in work in a maximal physical stress test fairly quickly leads to a significant oxygen debt and a switch to an anaerobic type of energy supply. In this situation fats cannot be oxidatively metabolized. So, increased levels remain in the circulation until the eventual transport of lipids to the liver can be accomplished.

In this study moderate exercise was shown to improve postprandial dyslipidemias. This is consistent with other research that has demonstrated that systematic physical training improves lipid disturbances, physical fitness and the clinical symptoms of CHD and can even retard progression of coronary artery disease [33-37]. Comparisons of different intensities of physical training on CHD patients show the beneficial effects of moderate intensity training on physical fitness and the clinical manifestation of CHD.[38-42]

What is the mechanism of lipid improvement with submaximal exercise as seen in this study? Possibly, the level of exercise combines optimally the lipolysis of fats from their depots and their oxidative metabolism under the conditions of sustained aerobic work. The decrease of triglycerides and LDL-C during the non-strenuous physical work may be linked to their increased removal by lipoprotein lipase in the expanded capillary circulation in muscles and fat depots. This level of physical activity also appears to increase HDL-C (especially HDL_2).[43-46] During lipolysis HDL_3 under the influence of lecithin- cholesterol acetyl transferase becomes HDL_2 releasing free cholesterol into the plasma. Apo AI, for reasons that are unclear, appears to undergo increased synthesis in healthy persons with both moderate and intensive exercise but that response was observed in CHD subjects only with moderate exercise.

Another benefit reported with moderate exercise but not seen with vigorous exercise is a favorable effect on fibrinolytic activity as evidence by increased levels of tissue plasminogen activator.[47-50] Inhibition of secondary platelet adhesiveness has also been observed in men who exercise regularly,[50-52] platelet adhesiveness and platelet aggregation have been shown to increase with strenuous exercise whereas both are decreased by moderate exercise.[53,54] It is unknown whether these observations have any relationship to the findings reported in our study, but they do add to the strength of a clinical recommendation for regular moderate exercise.

Conclusion

In conclusion, this study demonstrates that moderate exercise is beneficial in improving postprandial lipid abnormalities in both CHD and CHD-free subjects after fatty meal preload. In addition, maximal exercise demonstrated evidence of increase of lipid abnormalities in both CHD and CHD-free subjects under similar conditions of fatty meal preload.

Subjects with CHD also demonstrated significant deterioration of work performance and increased abnormalities on stress testing when maximally exercised after a fatty meal preload. Although clinical generalizations from this study should be limited to untrained middle-aged men, the findings strongly support the value of specific exercise prescriptions for optimal health maintenance. Even persons without CHD did not

appear to benefit from strenuous exercise and clearly persons with CHD appear to be put at immediate increased risk. Since most untrained middle-aged men don't have a clear idea of what their maximal or submaximal exercise levels are, it would appear advisable when recommending an exercise program, to start with a formal exercise assessment especially if other CHD risk factors are present. A moderate exercise prescription could then be provided including metabolic equivalents in a variety of different or seasonally specific recreational activities.

Authors' contribution

David M. Aronov contributed to the design of the study, participated in the collection and interpretation of the obtained data as well as in writing of the manuscript.

Marina G. Bubnova contributed to the design of the study, participated in the collection and interpretation of the obtained data as well as in writing of the manuscript.

Natalia V. Perova contributed to the design of the study, participated in the collection and interpretation of the obtained data as well as in writing of the manuscript.

Alexander N. Orekhov contributed to the design of the study, participated in the interpretation of the obtained data as well as in writing of the manuscript.

Yuri V. Bobryshev contributed to the design of the study, participated in the interpretation of the obtained data as well as in writing of the manuscript.

All authors have approved the final version of the manuscript.

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Conflict of interests

The authors declare no conflicts of interest.

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Table 1. The amount of work and energy expenditure in tests

Work Variables	Maximal load		Submaximal load	
	group I	group II	group I	group II
Amount of work performed (kgm)	6030	4257	23760	20736
Energy expended (KJ)	59.14	41.75	233.01	203.36

Table 2. Physical work capacity parameters before and after fat load

Parameters		Groups		P
		I	II	
Max physical load 1 (kgm/min)		1050 ± 32	864 ± 24	<0.01
2		990 ± 30	824 ± 36	<0.01
Duration of physical	1	582 ± 20	461 ± 31	<0.01
load (sec.)	2	555 ± 23	431 ± 15*	<0.01
Total amount of	1	6078 ± 62	4236 ± 43	<0.01
performed work (kgm)	2	6030 ± 63	3654 ± 40**	<0.01
max $V0_2$ (ml/kg. min)	1	32.4 ± 1.4	27.8 ± 1.2	<0.05
	2	36.3 ± 1.4**	25.8 ± 1.2**	<0.01
Oxygen pulse (ml/beats)	1	15.0 ± 0.6	14.9 ± 0.7	NS
	2	17.0 ± 0.5**	13.7 ± 0.7**	<0.01
MET's	1	9.3 ± 0.4	7.6 ± 0.4	<0.01
	2	10.5 ± 0.4**	6.5 ± 0.4**	<0.01
Ventilation (l/min)	1	76.0 ± 4.3	61.8 ± 4.2	<0.01
	2	78.0 ± 5.0*	55.3 ± 3.2**	<0.01

Note: 1-without fat load

2 - 3 hour after fat load * $p<0.05$ ** $p<0.01$

NS - non significant

Table 3. Results of maximal stress test in patients with manifested CHD

Results	Without Fat Load		After Fat Load		P
	n	%	n	%	
Anginal pain	7	18.8	11	31.3	<0.01
Depression of ST- segment 1 mm or more	9	25	18	50	<0.01
Arrhythmias (ventricular extrasys toles)	-	0	7	18.7	<0.01

Table 4. Changes of lipids and apolipoproteins after combined standard fat and maximal physical loads

Parameters		Groups		P
		I	II	
Total cholesterol (mg/dL)	1	217 ± 8.0	233 ± 7.0	NS
	2	211 ± 8.0	240 ± 7.3	<0.02
	3	233 ± 9.0^{abb}	256 ± 8.0^{aabb}	<0.05
TG (mg/dL)	1	121 ± 4.2	128 ± 14.7	NS
	2	264 ± 24.2^{a}	224 ± 23.9^{aa}	NS
	3	312 ± 32.9^{aabb}	272 ± 35.4^{aabb}	NS
LDL-C (mg/dL)	1	146 ± 7.4	167 ± 7.0	<0.05
	2	138 ± 8.3^{aa}	170 ± 6.9	<0.01
	3	152 ± 7.3^{abb}	189 ± 7.2^{aabb}	<0.01
HDL-C (mg/dL)	1	45 ± 1.9	38 ± 1.7	<0.01
	2	43 ± 1.8^{aa}	33 ± 2.2^{aa}	<0.01
	3	42 ± 2.0 ^{aa}	30 ± 2.1^{aabb}	<0.01
Apo AI (mg/dL)	1	142 ± 2.8	134 ± 2.6	<0.05
	2	149 ± 3.0^{aa}	128 ± 2.1 ^{aa}	<0.01
	3	158 ± 3.8^{aabb}	121 ± 3.3^{aabb}	<0.01
Apo B (mg/dL)	1	111 ± 3.7	128 ± 2.6	<0.01
	2	108 ± 3.8	134 ± 3.3^{aa}	<0.01
	3	116 ± 4.0^{aabb}	140 ± 3.3^{aabb}	<0.01
Apo B/AI	1	0.79 ± 0.03	0.98 ± 0.02	<0.01
	2	0.72 ± 0.03 ^{aa}	1.05 ± 0.03^{aa}	<0.01
	3	0.74 ± 0.03 ^{aa}	1.16 ± 0.24^{aabb}	<0.01

Note: 1 - fasting parameters; 2 - 3 hour after fat load; 3 - 10 min later on physical load

a - $p<0.05$; aa - $p<0.01$ comparing with fasting state;

b - $p<0.05$; bb- $p<0.01$ comparing with after fat load; NS - non significant

Table 5. Changes of lipids and apolipoproteins after combined standard fat and submaximal physical loads

Parameters		Groups		P
		I	II	
Total cholesterol (mg/dL)	1	218 ± 7.1	224 ± 7.0	NS
	2	207 ± 8.0[a]	238 ± 7.1	<0.02
	3	210 ± 7.5[a]	240 ± 7.4	<0.02
TG (mg/dL)	1	113 ± 14.2	125 ± 13.8	NS
	2	235 ± 26.2[aa]	231 ± 26.5 [aa]	NS
	3	217 ± 26.9[aa]	200 ± 26.3[aabb]	NS
LDL-C (mg/dL)	1	150 ± 6.7	160 ± 6.9	<0.05
	2	133 ± 7.0[aa]	166 ± 7.2	<0.01
	3	123 ± 7.1[aabb]	145 ± 7.2[aabb]	<0.01
HDL-C (mg/dL)	1	45 ± 2.0	38 ± 2.1	<0.01
	2	42 ± 1.8[aa]	32 ± 2.0[aa]	<0.01
	3	52 ± 1.7[aabb]	47 ± 2.7[aabb]	NS
Apo Al (mg/dL)	1	143 ± 2.8	133 ± 2.8	<0.05
	2	148 ± 2.8[aa]	127 ± 3.0 [aa]	<0.01
	3	161 ± 3.2[aabb]	142 ± 3.0[aabb]	<0.01
Apo B (mg/dL)	1	113 ± 3.4	126 ± 3.1	<0.01
	2	111 ± 3.3	133 ± 3.1[aa]	<0.01
	3	105 ± 4.0[aabb]	119 ± 3.7[aabb]	<0.05
Apo B/AI	1	0.81 ± 0.03	0.94 ± 0.03	<0.01
	2	0.75 ± 0.03[aa]	1.01 ± 0.04[aa]	<0.01
	3	0.66 ± 0.03[Mbb]	0.84 ± 0.03[aabb]	<0.01

Note: 1 - fasting parameters; 2 - 3 hour after fat load; ; 3 - after completion of the work within 3-5 min;

a- $p<0.05$; aa - $p<0.01$ comparing with fasting state;

b- $p<0.05$; bb- $p<0.01$ comparing with after fat load; NS - non significant

Highlights:

- Influence of a standard fat load combined with physical exertion was investigated
- Plasma lipid profile of CHD patients and CHD-free individuals were analyzed
- Physical exercise produced atherogenic blood lipid changes in both groups
- Moderate exercise is beneficial in improving postprandial lipid abnormalities
- Maximal exercise demonstrated evidence of increase of lipid abnormalities