ORIGINAL ARTICLE

Aerobic exercise has beneficial impact on atherogenic index of plasma in sedentary overweigh and obese women

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Abstract

OBJECTIVE: In obese patients, we hypothesized physical exercise (PE) to affect lipids through its intrahepatic fat accumulation-lowering effect, associated with a decrease of total body fat (Fat%) and even weight (Mass).
Design and setting: Thirty seven sedentary, non-diabetic women (BMI median 34.8) from our out-patient department were tested. Elimination criteria: recent weight reduction, lipid-influencing or heart rate-modifying medication.
Participants: 50 entering, 37 finishing, 7 excluded for processing failure, 6 did not fulfill the protocol.
INTERVENTIONS: PE protocol: 60 min supervised trainings, intensity at 65% of VO_2max, modified by the clamp heart rate test. Median of total training hours was 34 during 115 days (median).
Main Outcome Measurements: an effect of PE on total cholesterol (CH), triglycerides (TG), HDL-cholesterol (HDL_C), LDL-cholesterol (LDL_C), index of atherogenity (IA), atherogenic index of plasma (AIP), maximum peak oxygen consumption (VO_2max), Mass, body mass index (BMI), waist circumference (Waist) and Fat%.
RESULTS: Statistically significant differences at start (_s) and the end (_e) of PE ($p<0.05$): AIP –0.049, Mass –3.6 (kg), BMI –1.7 (kg/m^2), Waist –2.5 (cm), Fat% –2.5, VO_2max 2.92 (L.min^{-1}kg^{-1}).
Correlation coefficients, Pearson's between Gaussian distributed (Gd-v) variables and Spearman´s (non Gd-v) statistically significant ($p<0.05$): IA and BMI, IA and Mass, IA and Waist, IA and Fat%, LDL and BMI, LDL and Mass, LDL and Fat%, LDL and Waist, IA and VO_2max, LDL_C and VO_2max.
CONCLUSIONS: PE improves lipid profile towards production of antiatherogenic particles more likely due to changes in anthropometric parameters than in improvement of physical fitness.

INTRODUCTION

Dyslipidaemia of metabolic syndrome (DMS) is a cluster of plasmatic lipid and lipoprotein abnormalities with high atherogenic potential (Ceska 2007). It is simultaneously closely related to insulin resistance and independent on presence of type 2 diabetes mellitus (Adiels *et al.* 2008). DMS is typically represented by high levels of plasma triglycerides (TG), low levels of high-density lipoprotein cholesterol (HDL_C), the appearance of small, dense, low-density lipoproteins LDL (sdLDL) and HDL (sdHDL) and an adversely high rate of apoprotein B (apoB) and apoprotein A (apoA) (Avramoglu *et al.* 2006). ApoB plasmatic levels show a strong, positive correlation to the size of sdLDL particles (Tsimihodimos *et al.* 2007). In case of HDL_C and its heterogeneity, a better indicator of its (anti)atherogenic potential is the value of atherogenic index of plasma {AIP, [log (TG/HDL_C)]}(Dobiasova 2004). Thus, the components of DMS do not represent isolated pathological findings but heterogeneous units within each subgroup, closely related to each other in many complicated metabolic pathways (Taskinen 2003). In patophysiology of DMS, the liver-overproduction of large triglyceride-rich very low density lipoproteins ($VLDL_1$) plays the key role (Adiels *et al.* 2008). Physical exercise (PE) induces a triacylglycerol-lowering effect which basically occurs in three different ways: 1/ Direct impact on key enzymes involved in patophysiology of DMS, especially on skeletal muscle lipoprotein lipase (LPL) activity, is said to be more likely a contributing TG-lowering factor (Magkos 2009). 2/ Peripheral effect: in obese people, oxidation capacity of skeletal muscle for lipids is lower than in lean subjects and inversely associated with the degree of obesity (Hulver *et al.* 2005). After massive weight loss and during exercise, this phenotype is preserved (Thyfault *et al.* 2004). 3/ Central effect: Amount of visceral fat mass seems to be the most important factor in high storage of fat in liver (Jakobsen *et al.* 2007) and directly associates with liver production of $VLDL_1$ (Fabbrini *et al.* 2008). Influx of intra-abdominal fat-originated free fatty acids (FFA) into liver is four-times higher in obese than in lean subjects (Magkos 2009). The last three years published studies aimed at the influence of PE on non-alcoholic fatty liver disease (NAFLD) differ in answering the question of how changes in basic anthropometric parameters influence DMS due to regular PE (Devries *et al.* 2008; Tsekouras *et al.* 2008). The pathophysiological mechanisms support the theory that PE can influence less insulin-resistance and beta-oxidation of FFA in skeletal muscle than high storage of fat in the liver. In case of DMS we thus hypothesised that the anti-atherogenic influence of PE occurs predominantly due to changes in body composition.

METHODS

Subjects

Thirty-seven women (arithmetic mean (M) age 45.0 y, standard deviation (SD) 10.1 y) recruited from our out-patients department were tested. To be eligible for participation, baseline body mass index (BMI = weight in kilograms divided by the square of height in meters) had to be between 25 and 40 kg/m^2. In addition, all participants were classified at baseline as sedentary, which was defined as reporting exercising less than 2 days a week over the previous 6 months. Participants were excluded if they met any of the following criteria: contraindications for exercise, a health condition that would limit exercise participation, aggressive weight reduction in the past two years which meant more than 10 kilograms per year, using medication that would alter the heart rate response during exercise (e.g. beta-blockers or any other antiarythmics) or medication affecting lipid metabolism or weight loss (Svacina *et al.* 2007). As subjects have not been treated for dyslipidaemia, one or a combination of more than one abnormality in lipid metabolism was observed in the cases of twenty-six subjects at baseline. These disorders included an LDL_C concentration above 4.5 mmol/L, an HDL_C concentration below 1.3 mmol/L, a TG concentration above 1.69 mmol/L, total cholesterol (CH) concentration above 5.2 mmol/L, apoprotein B (apoB) concentration above 1.7 mmol/L, index of atherogenity (IA = [CH – HDL_C/HDL_C]) above 3, or AIP above 0.11. Diabetics were not included. Ten subjects were treated for arterial hypertension before admission to the project.

Participants were asked not to change their usual eating habits. They completed a detailed medical history and physical examination. All provided written informed consent prior to initiating the study.

Design

The subjects were asked to participate in regular aerobic exercise training. A combination of cycle ergometer, treadmill, elliptical trainer or row trainer was used. One training unit (TU) took 60 min. They were recommended to exercise at least twice a week for a period of approximately 3 months (median 115 d, lower 95% confidence limit (LCL) 108 d, upper 95% confidence limit (UCL) 138 d). Number of TU should exceed 24 (median 36, LCL 34, UCL 36). All training units were to be conducted under the supervision of an experienced instructor in The Recondition Centre of the University Sports Club (RC = spaces built and equipped especially for educating and practicing exercise for overweight and obese people). Intensity of exercise was first set to 65% of peak VO_2 max and subsequently modified by CHR. Training intensity in practice was verified by heart-rate monitors (Polar Electro). Before measurements of VO_2 max, subjects were given information about the test and instructed to exercise to their

Tab. 1. Statistical characteristics of variables with Gaussian distribution.

Variable	Arithmetic mean	SEM	Significance
HDL_C_s	1.38	0.05	
HDL_C_e	1.40	0.05	NS
LDL_C_s	3.15	0.13	
LDL_C_e	3.07	0.13	NS
IA_s	2.78	0.13	
IA_e	2.65	0.11	NS
AIP_s	– 0.074	0.032	
AIP_e	– 0.123	0.033	$p<0.05$
Fat%_s	38.6	0.7	
Fat%_e	36.1	0.8	$p<0.05$
HR_max_s	167.3	2.1	
HR_max_s	168.6		NS
VO_2max_s	25.20	0.62	
VO_2max_e	28.12	0.89	$p<0.05$
Z-score_s	– 0.55	0.11	
Z-score_e	– 0.03	0.15	$p<0.05$
VO_2max%_s	89.2	2.3	
VO_2max%_e	99.8	3.2	$p<0.05$
W_s	157.8	4.3	
W_e	174.2	5.1	$p<0.05$

SEM = standard error of mean
suffix _s means before exercise (at the start of the trial)
sufix_e means after exercise (at the end of the trial)
NS = non-significant
p = *p*-value
HDL_C = high-density lipoprotein cholesterol [mmol/L]
LDL_C = low-density lipoprotein cholesterol [mmol/L]
IA = index of atherogenity [CH – HDL_C/HDL_C]
AIP = Atherogenic Index of Plasma [log (TG/HDL-C)]
Fat% = total body fat [%]
HR = maximum reached heart rate (beats per minute)
VO_2max = maximum peak oxygen consumption ($L.min^{-1}kg^{-1}$)
Z-score = multiple of normalized standard deviation of VO_2max%
VO_2max% = percentage of predicted (optimal) VO_2max value age- and gender-matched due to Selliger´s standards created on the Czech population
W = peak power output (watt)

Tab. 2. Statistical characteristics of variables with non-Gaussian distribution.

Variable	Median	95% LCL	95% UCL	Significance
CH_s	4.88	4.70	5.42	
CH_e	5.05	4.69	5.39	NS
TG_s	1.09	0.99	1.20	
TG_e	1.03	0.83	1.31	NS
TG_HDL_s	0.80	0.72	0.94	
TG_HDL_e	0.73	0.58	0.93	NS
apoB_s	0.91	0.87	1.03	
apoB_e	0.94	0.84	1.03	NS
Mass_s	96.5	89.5	105.2	
Mass_e	92.9	87.7	97.0	$p<0.05$
Waist_s	103.5	99.0	106.0	
Waist_e	101.0	95.0	105.0	$p<0.05$
BMI_s	34.8	32.1	37.3	
BMI_e	33.1	31.0	35.9	$p<0.05$
AST_s	0.44	0.39	0.48	
AST_e	0.40	0.35	0.43	NS
GGT_s	0.32	0.27	0.40	
GGT_e	0.29	0.23	0.40	NS
ALT_s	0.42	0.39	0.48	
ALT_e	0.40	0.36	0.49	NS

suffix _s means before exercise (at the start of the trial)
sufix_e means after exercise (at the end of the trial)
95% LCL = 95% lower confidence limit
95% UCL = 95% upper confidence limit
NS = non-significant
p = *p*-value
CH = total cholesterol [mmol/L]
TG = triglycerides [mmol/L]
TG_HDL = ratio TG/HDL_C [mmol/L]
apoB = apolipoprotein B [mmol/L]
Mass = total body mass [kg]
Waist = waist circumference [cm]
BMI = Body Mass Index [kg/m^2]
AST = aspartat aminotranspherase [mmol/L]
GGT = gamma-glutamyl thanspherase [mmol/L]
ALT = alanine aminotransferase [mmol/L]

maximum limit. Reaching oxygen uptake via increased workload and a respiratory exchange ratio > 1.00 was used as criteria for the true VO_2 max and this was achieved in all individuals in the present study. Oxycon Delta Jaeger (ergometer Ergoline 900) was used. Nutrient intakes were followed by evaluating a questionnaire once a week so we could see that none of the subjects were on a reduction diet (defined as less than 1790 kcal per day). For all outcomes, the subjects were tested before entering the programme and after finishing it (= after completing at least 24 TU in the RC). Primary outcomes were differences in values of CH, HDL-C, LDL-C, TG, IA, AIP, apoB, aspartat aminotranspherase (AST), alanine aminotransferase (ALT) and gamma-glutamyl thanspherase (GGT). Fasting plasma samples were performed with standard local procedures. Secondary outcomes were changes in total body mass in kg (Mass), BMI, percentage of total body fat (Fat%), waist circumference in cm (Waist). Weight was measured on medical scales to the nearest 0.1 kg. Participants were weighed in undergarments. Height was measured with a wall-mounted stadiometer to the nearest 0.5 cm. BMI was calculated [mass (kg)/height^2 (m^2)]. Fat% was measured by bioimpedance (Tanita Body Fat Monitor/Scale

TBF-551). Waist was measured by tape measure to the nearest 0.5 cm. Tertiary outcome for assessing change in fitness was peak maximal oxygen consumption (VO_2 max [$L.kg^{-1}.min^{-1}$]) corresponding Z-score, percentage of its physiological value and peak power output quantified in watts (W).

STATISTICAL METHODS AND RESULTS

For all statistical calculations NCSS 2007 (Hintze 2007) was used. Twenty paired variables were analyzed in this study. At the very beginning a distribution of each variable was tested for normality. H_0 could not be rejected in 10 variables; in the 10 variables H_0 was rejected. For the variables with Gaussian distribution of differences an arithmetic mean was used as a measure of location and standard error of mean (SEM) as a measure of variability. For the variables with non-Gaussian distribution median was used as a measure of location. Lower confidence limit (LCL) and upper confidence limit (UCL) were used as measures of variability. Values at the end of study were tested against the same variables values at the start of the study. Depending on the distribution, either paired *t*-test or Mann-Whitney test were used.

Tab. 3. A: Correlations between anthropometric parameters and the parameters of lipid metabolism and AST, GGT and ALT at the start of the trial.

	AIP_s	LDL_s	IA_s	CH_s	AST_s	GGT_s	ALT_s
Mass_s	0.072436	0.365979 Δ	0.393138 Δ	0.127097 Δ	0.412831	0.158113	0.23924
***p*-value**	0.670071	0.025902 *	0.01608 *	0.453487	0.011105 *	0.349967	0.153839
BMI_s	0.009249	0.493477	0.446293	0.31525	0.477544	0.241514	0.266002
***p*-value**	0.956673	0.001912 *	0.005627 *	0.057361 *	0.0028 *	0.149848	0.11154
Fat%_s	– 0.047074	0.451648 Δ	0.352132 Δ	0.326822 Δ	0.359494	0.218431	0.054922
***p*-value**	0.782036	0.005016 *	0.032565 *	0.048343 *	0.028866 *	0.194009	0.746804
Waist_s	0.106194	0.41773	0.415136	0.260872	0.389751	0.26002	0.346266
***p*-value**	0.531608	0.010095 *	0.01062 *	0.118877	0.0171 *	0.120131	0.035782 *

Tab. 3. B: Correlation between anthropometric parameters and the parameters of lipid metabolism and AST, GGT and ALT.

	AIP_e	LDL_e	IA_e	CH_e	AST_e	GGT_e	ALT_e
Mass_e	0.285443	0.340637Δ	0.530532 Δ	0.203426 Δ	0.262371	0.384698	0.062411
***p*-value**	0.0868	0.039108 *	0.000731 *	0.227202	0.116697	0.018724 *	0.713649
BMI_e	0.253468	0.50913	0.580206	0.390242	0.237776	0.428639	– 0.012519
***p*-value**	0.1301	0.00129 *	0.000167 *	0.016949 *	0.15645	0.008122 *	0.941376
Fat%_e	0.31094	0.511051 Δ	0.684909 Δ	0.396995 Δ	0.106844	0.496351	0.004689
***p*-value**	0.061042	0.001228 *	0.000003 *	0.01498 *	0.529083	0.001781 *	0.978028
Waist_e	0.270296	0.332602	0.497059	0.206796	0.347878	0.419263	0.179973
***p*-value**	0.105664	0.044286 *	0.00175 *	0.21943	0.034873 *	0.009795 *	0.286473

suffix _s means before exercise (at the start of the trial)
sufix_e means after exercise (at the end of the trial)
symbol * shows statistically significant values at least at 5% probability
symbol Δ shows Pearson´s correlation coefficient between variables with Gaussian distribution (the rest are the Spearman´s correlation coefficients)
Mass = total body mass [kg]
BMI = Body Mass Index [kg/m^2]
Fat% = total body fat [%]
Waist = waist circumference [cm]
AIP = Atherogenic Index of Plasma [log (TG/HDL-C)]
LDL = low-density lipoproteins [mmol/L]
IA = index of atherogenity [CH – HDL_C/HDL_C]
CH = total cholesterol [mmol/L]
HDL_C = HDL cholesterol [mmol/L]
AST = aspartat aminotranspherase [mmol/L]
ALT = alanine aminotransferase [mmol/L]
GGT = gamma-glutamyl thanspherase [mmol/L]

Tab. 4. A: Spearman´s correlation coefficients between the parameters of lipid metabolism and AST, GGT and ALT at the start of the trial.

	AST_s	GGT_s	ALT_s
CH_s	0.080779	0.28257	0.157189
***p*-value**	0.634591	0.090162	0.352828
HDL_s	– 0.00101	0.353193	– 0.07036
***p*-value**	0.995266	0.032009 *	0.679016
LDL_s	0.156232	0.147383	0.169081
***p*-value**	0.355804	0.384029	0.317114
TG_s	0.214816	0.209162	0.325299
***p*-value**	0.201674	0.214085	0.04946 *
AIP_s	0.195403	0.069526	0.316021
***p*-value**	0.246451	0.68262	0.056721

Tab. 4. B: Correlation between the parameters of lipid metabolism and AST, GGT and ALT at the end of the trial.

	AST_e	GGT_e	ALT_e
CH_e	0.141221	0.38128	0.066627
***p*-value**	0.404438	0.019893 *	0.695204
HDL_e	0.08453	0.140695	-0.224181
***p*-value**	0.618891	0.406207	0.182241
LDL_e	0.101056	0.334263	0.149401
***p*-value**	0.551756	0.043173 *	0.377482
TG_e	0.071348	0.513796	0.1463
***p*-value**	0.674754	0.001143 *	0.387575
AIP_e	0.018868	0.365362	0.207641
***p*-value**	0.911743	0.026173 *	0.21751

suffix _s means before exercise (at the start of the trial)
sufix_e means after exercise (at the end of the trial)
symbol * shows statistically significant correlations at least at 5% probability
AIP = Atherogenic Index of Plasma [log (TG/HDL-C)]
LDL = low-density lipoproteins [mmol/L]
CH = total cholesterol [mmol/L]
HDL_C = HDL cholesterol [mmol/L]
AST = aspartat aminotranspherase [mmol/L]
ALT = alanine aminotransferase [mmol/L]
GGT = gamma-glutamyl thanspherase [mmol/L]

Measures of location and variability for which differences were significant at least at 5% probability level are given in Tables 1 and 2. Tables 3 to 5 show Pearson's correlation coefficients between variables with Gaussian distribution and Spearman's correlation coefficients between variables with non-Gaussian distribution.

DISCUSSION

To affect lipid metabolism and especially plasmatic levels of HDL-C, high intensity exercise was said to be the dominant factor (Tjonna *et al.* 2008). The conclusion of (Kraus *et al.* 2002) showed that it was not high intensity but rather the amount of PE that is the key factor to improve lipids in obese subjects with dyslipidaemia at baseline. In both publications, the improvement in lipids was independently followed by significant weight reduction. The last recommendation of the American College of Sports Medicine (Donnelly *et al.* 2009) allows that there is a dose-response effect of PE on weight loss and the amount of PE between 150 and 225 minutes per week can be followed by modest weight loss of approximately 2 to 3 kg. The design of a PE programme was driven by the idea that we have worked with overweight and obese subjects who have led a sedentary lifestyle their entire lives to date (none of our thirty-seven participants have ever done regular sport or exercise of however racing or recreational level which correlates with below-average % values of VO_2max due to Seliger´s standards assessing a Czech population (Macek *et al.* 1979)). We tried to create a programme that complies with safety criteria which limits both intensity and weekly amount of exercise. Our participants did not have an initial period of several months to adjust to certain higher levels of exercise, as for example in the study of (Kraus *et al.* 2002). However, all of them continued to engage in regular exercise in RC, even after completing the project (further follow-up is not a subject of this study). This fact leads us to assess the protocol as being sufficiently motivational. Our premise was that the exercise intensities based on heart rate from spiroergometry could not often have been reached and maintained in practice. Each investigation on spiroergometer was then followed by CHR test to regulate the real training heart-rate value. We assumed that due to often high initial motivation of the investigated subjects, better outcomes from spiroergometry are reached. Furthermore, the final heart rate-values recommended for the training programme are too high and unachievable for sedentary and even advanced obese patients to sustain for the first few weeks of exercise. On the other hand frequent indisposition, such as polyarthrosis, muscle weakness, or low fitness per se, could lead to an insufficient performance on spiroergometry so the heart rate recommended for training sessions touches lower limits and the subjects would have been recommended to exercise at lower intensities unnecessarily. CHR represents in fact a test of cardiovascular efficiency based on reversed regulation of work rate intensity by a defined heart rate. The advantage of this method is that an examinate does not have to reach maximum performance, which makes CHR safer for our type of patients. The subjects could be observed for 40 minutes of exercise in contrast to about 10–15 minutes in the case of spiroergometry.

Ratings of subjects´ perceived exertion were obtained using the 20-point Borg scale. To reach better results we proposed to shorten the resting pauses to a maximum of 15 days (Slentz *et al.* 2007) and prolong the testing interval to at least 3 months, which, based on current evidence, is the minimum time necessary for a potentially successful lipid-lowering effect of PE (Kraus *et al.* 2002; Tjonna *et al.* 2008; Jakicic *et al.* 2003).

CONCLUSIONS

We have showed that regular aerobic PE significantly decreases AIP. Although there is no significantly lowering effect on HDL_C, we established a significantly positive correlation between IA and all investigated anthropometric characteristics (Mass, BMI, Waist, Fat%) at the beginning, and at the end of the project this relationship becomes stronger. In association with significant changes of body composition after exercise including Mass, we showed a positive correlation between IA and VO_2max at the end of the project. Although the difference between ALT, AST and GGT before and after PA was not statistically significant, significantly positive correlation between GGT and Mass, GGT and BMI, GGT and Waist and GGT and Fat% after PA and also positive correlation between ALT and Waist before entering the protocol, similarly to study (Devries *et al.* 2008) showing a positive correlation between Waist and both GGT and ALT in relationship to positive correlation of GGT and ALT to hepatic lipid content can allow us to consider the lowering effect of PE on AIP to be a result of a reduced intrahepatic-lipid accumulation.

Competing interests

The authors declare that they have no competing interests.

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Tab. 5. A: Correlation between parameters of lipid metabolism and the parameters of physical fitness at the start of the trial.

	VO_2max_p_s	W_s
CH_s	– 0.455287 Δ	– 0.491028 Δ
***p*-value**	0.004633 *	0.00203 *
LDL_s	– 0.504977 Δ	– 0.446066 Δ
***p*-value**	0.001435 *	0.005655 *
IA_s	– 0.323499 Δ	– 0.167957 Δ
***p*-value**	0.050807 *	0.320389
AIP_s	0.04909	0.006449
***p*-value**	0.772943	0.969782

Tab. 5. B: Correlation between parameters of lipid metabolism and the parameters of physical fitness at the end of the trial.

	VO_2max_p_e	W_e
CH_e	– 0.381805 Δ	– 0.439716 Δ
***p*-value**	0.019709 *	0.006466 *
LDL_e	– 0.48453 Δ	– 0.49262 Δ
***p*-value**	0.002374 *	0.001952 *
IA_e	– 0.575729 Δ	– 0.219895 Δ
***p*-value**	0.000193 *	0.190963
AIP_e	– 0.329935	0.040981
***p*-value**	0.046123 *	0.809693

suffix _s means before exercise (at the start of the trial)
sufix_e means after exercise (at the end of the trial)
symbol * shows statistically significant correlations at least at 5% probability
symbol Δ shows Pearson´s correlation coefficient between variables with Gaussian distribution (the rest are the Spearman´s correlation coefficients)
AIP = Atherogenic Index of Plasma [log (TG/HDL-C)]
LDL = low-density lipoproteins [mmol/L]
IA = index of atherogenity [CH – HDL_C/HDL_C]
CH = total cholesterol [mmol/L]
VO_2max = peak maximal oxygen consumption per kilogram body weight [$L.min^{-1}.kg^{-1}$]

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