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Regular Physical Activity and Changes in Risk Factors for Coronary Heart Disease: A Nine Months Prospective Study

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Summary: This study reports the non-acute effects of a long-term training programme of increasing intensity on some cardiovascular risk factors and the interrelation between these risk factors. Twenty sedentary men and 14 sedentary women were trained 3 to 4 times a week for nine months. After 36 weeks all individuals ran a half marathon run. The W_{max}, weight, body mass index, systolic and diastolic blood pressure were recorded. The concentrations of fibrinogen, tissue plasminogen activator, plasminogen activator inhibitor, triacylglycerols, total cholesterol, LDL cholesterol, HDL cholesterol and lipoprotein(a) were measured. The training programme induced a median increase in W_{max} of 12% in the male group (from 226 to 251.5 Watt) and of 18% in the female group (from 160 to 188.5 Watt). These increases in W_{max} did not correlate with any other property under investigation in this study. Blood pressure was not altered, but body weight and body mass index were significantly decreased in the male group (from 74.6 to 72.2 kg and from 23.1 to 22.0 kg/m^2, respectively) at the end of the training programme and decreased non-significantly in the female group (from 63.0 to 60.7 kg and from 21.6 to 21.5 kg/m^2, respectively). In the male group total cholesterol, low density lipoprotein cholesterol and triacylglycerols decreased significantly under the influence of the training sessions. Furthermore, in both groups, a great decrease in plasma plasminogen activator inhibitor concentrations was noticed: in men from $22.5 \cdot 10^3$ AU/l to $4.5 \cdot 10^3$ AU/l and in women from $18.7 \cdot 10^3$ AU/l to $5.1 \cdot 10^3$ AU/l. However, the changes in the lipid and fibrinolytic quantities were not correlated with each other. Initial total cholesterol, LDL cholesterol and triacylglycerol levels correlated significantly with systolic blood pressure, while diastolic pressure was correlated to tissue plasminogen activator. Since tissue plasminogen activator also was significantly related to triacylglycerols, a trias existed between primary risk factors like blood pressure, lipid levels and fibrinolysis. In contrast, the changes in these properties under the influence of physical training were not interrelated. Median serum lipoprotein(a) concentrations were significantly increased in both men and women five days before the half marathon run: from 32 mg/l to 39 mg/l in men, and from 65 mg/l to 125.5 mg/l in women. Concomitantly, median fibrinogen concentrations were significantly elevated in men (from 2.32 g/l to 3.10 g/l) and non-significantly in women (from 2.62 g/l to 2.93 g/l), although no correlation existed between the changes in these properties.

In conclusion, the nine months exercise programme increased the aerobic fitness in both men and women as indicated by the W_{max} increase. This improvement coincided but was not correlated with beneficial changes in several anthropometric, lipid and fibrinolytic properties. Improvement in the risk factors under investigation was more pronounced in men than in women. The changes in lipid and haemostasis properties did not correlate with each other. The increases in lipoprotein(a) and fibrinogen concentrations, both atherogenic indices, could actually present a normal physiological response to the physical strain of exercise training of increasing workload.

Introduction

Atherosclerosis leading to coronary heart disease is still the major cause of morbidity and mortality in our Western society (1). Atherosclerosis is a complex multifactorial disorder, but the pathogenesis includes a contributing role for blood lipids and haemodynamic forces (1–3). Occlusion of an atherosclerotic vessel, based on thrombus formation, links the haemostatic system with atherosclerosis at a fundamental level (2–4). Risk factors for atherosclerosis are several anthropometric determinants like sex, age, body weight and blood pressure (5–8). In addition, various biochemical quantities contribute to the formation of atherosclerotic plaques of

which hyperlipidaemia, namely an increased serum total and/or low density lipoprotein cholesterol (LDL cholesterol) is the best known (7–10). Other properties include factors of the haemostatic system, like an elevated plasma fibrinogen or plasminogen activator inhibitor concentration (11–13). Recently lipoprotein(a), an LDL-like lipoprotein with a great homology to plasminogen, a component of the haemostatic system, was designated as an independent risk factor for the genesis of artherothrombotic disease (14–17). Regular physical exercise, among other life-style habits like diet and a history of no smoking, may reduce the risk of vascular thrombotic events (6, 18–21). This protection might be mediated through the effect of exercise on the haemostatic system and the lipid metabolism, because endurance trained athletes exhibit generally more favourable values of haemostatic and lipidic variables than their non-active counterparts (22–25). The results of longitudinal studies, in which selection bias and constitutional factors have been minimized, are difficult to compare due to differences in e. g. duration and intensity of the exercise programmes (23, 26–30). Only few studies exist on the interrelation of anthropometric, lipid and haemostatic risk factors and the potentially favourable effect of long-term moderate exercise on these properties (31–33).

The present study was designed to investigate the non-acute effect of long-term physical conditioning on anthropometric and biochemical quantities in previously sedentary men and women and to evaluate the relationship between the studied components.

Materials and Methods

Subjects

Three hundred seventy people replied to advertisements in two local newspapers and on a local radio station. Those who participated in any sports such as running or jogging or who were active for more than one hour per week in other recreational sports were excluded. From the remaining group, twenty males aged from 32 to 49 years (median: 39 years) and 14 females aged from 27–41 years (median: 35 years) were aselectly chosen to participate in the study. No lipid lowering or antihypertensive medication was used by any of the volunteers. Among the participants were three female and six male cigarette smokers (maximum of ten cigarettes a day), who continued to smoke during the test period. All individuals kept their diet and other living habits, like the use of alcohol, as constant as possible during the study. After two information sessions all subjects gave their written informed consent.

Training programme

After a medical examination and pre-exercise control measurements, all volunteers participated in a 9 months endurance training programme preparing for a half marathon run (21 km). The training period lasted 9 months. After 24 and 36 weeks of training all subjects ran a 15 km and 21 km race, respectively. During the general preparation period the volunteers trained 3–4 times a week; 6 weeks before each contest the training programme was intensified to 5–6 times a week. Beside the attention paid to style and technique of running, stretching, speed, intervals, warming-up and cooling-down, the training included three elements: long-distance running, running at high speed and interval training. The elements followed each other naturally as far as the intensity is concerned. The intensity was for long-distance runs 70–80% of maximal heart rate, for running at a fixed pace 80–85% of maximal heart rate (over 200–2000 m depending on the training status) and for intervals over shorter distances (200–400 m) 95–100% of maximal heart rate. By putting these three elements together (in relation of 70 : 20 : 10 of the distances per week) a gradual undulating increase of the amount of training can be accomplished by training to a maximum of 60 min per training session in the first part of the study and to a maximum of 100 min per training session in the second part (34).

Sampling

All blood samples were drawn between 8.00 and 9.00 a. m. All subjects were seated and had not eaten or exercised during the preceding ten hours before phlebotomy. Samples were taken before the start of the training programme and, in order to avoid acute effects, five days before both races. Non-traumatic venipuncture was performed by trained technicians. Within one hour from sample collection serum was separated from blood, snap-frozen in aliquots, stored at −70 °C in plastic tubes and thawed at 37 °C immediately prior to serial analysis.

Blood pressure

Blood pressure was determined using a standard auscultatory method by a skilled technician, after a five-minutes rest period of the subjects in supine position. For the systolic blood pressure the first *Korotkoff* sounds were noted, while the diastolic blood pressure was measured at the disappearance of the *Korotkoff* sounds.

Graded maximal exercise tolerance test

Exercise tolerance test was conducted using a Lode bicycle ergometer. The test started at a workload of 50 Watt. After a warming-up period of 10 minutes, the exercise intensity was increased by 50 Watt every 4 minutes until exhaustion. During the test, subjects cycled at a rate of approximately 80 min^{-1} and heart rate was monitored continuously. W_{max}[1]) was calculated as the power step at which was cycled for the complete 4 minutes, plus the fraction of power cycled in the final intensity step.

Laboratory procedures

Fibrinogen was determined according to the *Clauss* method (intra-assay CV 3.2%; (35)). Reference values, determined in plasma from 50 healthy volunteers ranged from 2.0 to 4.0 g/l. The antigen concentrations of the following properties were determined using an ELISA test method: Tissue plasminogen activator (Kabi Vitrum Diagnostica, Molndal, Sweden, intra-assay CV 4.9%) and lipoprotein(a) (Biopool AB, Umea, Sweden) (36, 37). Reference range for tissue plasminogen activator, determined in plasma of 50 healthy volunteers (males and females) was: 0.9–12.1 µg/l. The lipoprotein(a) assay included ready to use micro-test plates containing affinity purified sheep anti-apolipoprotein(a) Ig and sheep anti-apolipoprotein(a) peroxidase conjugated Ig (37). Reference range, determined in serum from 50 healthy volunteers was 0–300 mg/l. The intra-assay coefficient of variation was 3.1% at a concentration of 150 mg/l Lp(a) and 4.8% at a concentration of 65 mg/l Lp(a). Plasminogen activator inhibitor activity was analyzed using an excess single chain tissue plasmogen activator and S-2251 as a chromogenic substrate for plasmin (Kabi Vitrum Diagnostica, intra-assay CV 9.5%; (38)). Reference ranges determined in 50 healthy volunteers were: $10.7–32.7 \cdot 10^3$ AU/l[1]). Serum triacylglycerols (reference range: 0.8–2.0 mmol/l) and total cholesterol (reference

[1]) W_{max} = maximum work load
AU = arbitrary units

range: 4.0–7.5 mmol/l) were determined using enzymatic assays (Roche Diagnostica, Basel, Switzerland and Boehringer Mannheim, respectively). High density lipoprotein cholesterol (HDL cholesterol fraction was isolated from serum by the phosphostungstic acid/magnesium chloride precipitant and determined with a test kit of Boehringer (39), reference range: 0.9–1.7 mmol/l. Low density lipoprotein cholesterol (LDL cholesterol) was calculated according to the *Friedewald* equation (40), reference range : 3.0–5.0 mmol/l. For all assays of lipids and lipoproteins the intra-assay coefficients of variation was less than 5%.

Statistics

All statistic computations were done with SPSS/PC+ Statistics 4.0 computer package (SPSS Inc., Chicago, USA). *Wilcoxon* signed rank test was used to compare differences in pre- and post-exercise plasma samples. The *Mann-Whitney* test was used to compare the male and female population. All data are given as medians and interquartile ranges. Correlations between changes in lipids, lipoproteins and haemostatic factors and changes in anthropometric values were calculated according to the method of *Spearman* (stepwise). Multiple regression was carried out to find independent determinants for the variations in the increase of W_{max}.

Results

Effect of training on maximal workload and the risk factor profiles

Median (interquartile range) initial anthropometric determinants, values of the lipid metabolism, the haemostatic and fibrinolytic system and the changes in these properties after nine months of training are summarized in table 1. In both men and women a nine month training programme produced a significant increase in W_{max}.

In men this increase was 24 Watt = 12% ($p < 0.001$) (median change from 226 to 251.5 Watt) and in women 25 Watt = 18% (n. s.) (median change from 160 to 188.5 Watt). In men the median diastolic blood pressure at rest before and after the training programme was 80 mm Hg. In women median diastolic pressure was shifted from 72.5 to 77.5 mm Hg (n. s.). In men the median systolic blood pressure at rest before the training programme was 132.5 mm Hg and 9 months later 130 mm Hg (n. s.). In women the systolic blood pressure shifted from 120 mm Hg to 125 mm Hg (n. s.). The median (interquartile range) body weight shifted in the male population from 74.6 (69.1–79.8) kg at start to 72.2 (64.8–77.7) kg ($p < 0.01$) at the end of the training programme. In the female population from 63.0 (57.0–65.8) kg to 60.7 (55.0–63.4) kg (n. s.). The median (interquartile range) body mass index shifted in the male group from 23.1 (21.6–25.5) kg/m^2 to 22.0 (21.1–24.4) kg/m^2 ($p < 0.01$) and in the female group from 21.6 (21.3–22.6) kg/m^2 to 21.5 (21.3–22.3) kg/m^2 (n. s.). The lipids showed a significant ($p < 0.01$) decrease in

Tab. 1 Median (interquartile range) initial anthropometric characteristics and variables of the lipid metabolism and haemostatic system in men and women and the changes in six months (pre–post) for these properties.

Men

	n	Initial	Change	p-value
W_{max} (Watt)	20	226 (203–266)	24.0 (8.5–42.0)	$p < 0.001$
Weight (kg)	20	74.6 (69.1–79.8)	− 1.6 (−3.0–(0.2))	$p < 0.001$
Body mass index (kg/m^2)	20	23.1 (21.6–25.5)	− 0.5 (−0.9–0.0)	$p < 0.001$
Diastolic blood pressure (mm Hg)	20	80 (80–81)	0.0 (−5.0–0.0)	$p = 0.27$
Systolic blood pressure (mm Hg)	20	133 (129–146)	− 7.5 (−15.0–0.0)	$p = 0.06$
Fibrinogen (g/l)	18	2.32 (1.97–2.48)	0.78 (0.50–1.02)	$p < 0.001$
Tissue plasminogen activator (µg/l)	19	5.8 (4.7–8.4)	0.10 (−2.10–(−0.60))	$p = 0.48$
Plasminogen activator inhibitor (10^3 AU/l)	17	22.5 (16.0–26.8)	−13,5 (−17.5–(−7.0))	$p = 0.001$
Triacylglycerols (mmol/l)	20	1.1 (0.9–1.5)	− 0.33 (−0.40–(−0.07))	$p = 0.001$
Total cholesterol (mmol/l)	19	5.8 (5.0–6.4)	− 0.45 (−1.15–(−0.2))	$p < 0.001$
LDL cholesterol (mmol/l)	19	4.0 (3.3–4.8)	− 0.50 (−1.01–(−0.28))	$p = 0.001$
HDL cholesterol (mmol/l)	19	1.0 (0.9–.13)	0.0 (−0.10–0.10)	$p = 0.75$
Lipoprotein(a) (mg/l)	20	32 (11–63)	39.0 (25.0–68.0)	$p < 0.001$

Women

	n	Initial	Change	p-value
W_{max} (Watt)	14	160 (152–175)	25.0 (17.5–38.5)	$p = 0.016$
Weight (kg)	14	63.0 (57.0–65.8)	− 0.7 (−2.0–0.5)	$p = 0.10$
Body mass index (kg/m^2)	14	21.6 (21.3–22.6)	− 0.2 (−0.7–0.2)	$p = 0.14$
Diastolic blood pressure (mm Hg)	14	73 (70–80)	0.0 (−5.0–10.0)	$p = 0.35$
Systolic blood pressure (mm Hg)	14	120 (115–130)	5.0 (−2.5–10.0)	$p = 0.25$
Fibrinogen (g/l)	14	2.62 (2.13–3.01)	0.31 (−0.21–0.72)	$p = 0.06$
Tissue plasminogen activator (µg/l)	14	3.7 (2.7–7.0)	0.45 (−1.70–2.60)	$p = 0.57$
Plasminogen activator inhibitor (10^3AU/l)	11	18.7 (16.0–25.5)	−12.5 (−15.6–(−9.6))	$p = 0.003$
Triacylglycerols (mmol/l)	14	0.9 (0.7–1.1)	− 0.10 (−0.24–0.10)	$p = 0.28$
Total cholesterol (mmol/l)	14	5.3 (3.9–6.0)	− 0.40 (−1.13–0.08)	$p = 0.16$
LDL cholesterol (mmol/l)	14	3.2 (2.5–3.9)	− 0.24 (−0.79–0.10)	$p = 0.22$
HDL cholesterol (mmol/l)	14	1.5 (1.1–1.9)	− 0.10 (−0.25–0.10)	$p = 0.18$
Lipoprotein(a) (mg/l)	14	65 (23–199)	65.5 (31.8–283.3)	$p < 0.001$

triacylglycerols, total cholesterol, and low density lipoprotein cholesterol (LDL cholesterol) in the male group, but not in the female population. In contrast, Lp(a) concentrations were significantly ($p < 0.01$) higher in both men and women after the completion of the training programme. In the male group, fibrinogen concentrations were significantly ($p < 0.01$) raised after nine months, while in the female group the rise in median fibrinogen concentration was not significantly different from pretraining levels. Finally, in both men and women a large and highly significant ($p < 0.001$) decrease in median plasminogen activator inhibitor levels was observed after nine months of training.

Correlation analysis

Table 2 shows the correlation (*Spearman*) between the change in W_{max} and the change in the studied properties after nine months of training in men and women. No significant relation existed between the increase in maximal workload on a cycle ergometer and the change in atherogenic determinants. Table 3 shows the correlations between initial anthropometric and biochemical quantities for the total group. Diastolic blood pressure was significantly correlated with tissue plasminogen activator concentration in serum ($r = 0.46$, $p < 0.01$) while systolic blood pressure related significantly to total cholesterol ($r = 0.55$, $p < 0.001$), LDL cholesterol ($r = 0.58$, $p < 0.001$) and triacylglycerols ($r = 0.60$, $p < 0.001$). Triacylglycerols also correlated significantly with body mass index ($r = 0.46$, $p < 0.01$). Correlation analysis between initial values of the haemostatic and fibrinolytic system and lipid metabolism showed a significant relation between tissue plasminogen activator and serum triacylglycerols ($r = 0.60$, $p < 0.001$). Finally, the correlation (*Spearman*) between changes in lipid and lipoprotein metabolism with changes in the haemostatic and fibrinolytic system for the total group are shown in table 4. No significant relation was found between changes in these biochemical quantities.

Tab. 2 *Spearman*'s rank coefficient of correlation (r) between the change in W_{max} and the change in the variables of the risk factor profile after nine months of training.

Change in	Men		Women	
	N	r	N	r
Weight (kg)	20	0.04	14	0.48
Body mass index (kg/m^2)	20	0.04	14	0.42
Diastolic blood pressure (mm Hg)	20	−0.12	14	−0.32
Systolic blood pressure (mm Hg)	20	0.15	14	0.03
Fibrinogen (g/l)	18	0.07	14	0.15
Tissue plasminogen activator (µg/l)	19	−0.21	14	−0.16
Plasminogen activator inhibitor (10^3 AU/l)	16	−0.01	10	−0.57
Triacylglycerols (mmol/l)	19	−0.47	14	0.24
Total cholesterol (mmol/l)	19	−0.04	14	−0.36
LDL cholesterol (mmol/l)	19	−0.15	14	−0.24
HDL cholesterol (mmol/l)	19	0.36	13	−0.02
Lipoprotein(a) (mg/l)	20	0.005	14	0.18

Multiple regression

In the stepwise multiple regression model, dependent variable was the change in W_{max} after nine months (ΔW_{max}). Independent variables for ΔW_{max} were initial body weight, body mass index, diastolic and systolic blood pressure. Using these independent variables, the regression equation had an R^2 value of 0.33 ($N = 34$). In the stepwise multiple regression model however, no independent variables were selected for ΔW_{max}.

Discussion

Several epidemiological studies have revealed that a sedentary lifestyle is an independent risk factor for coronary heart disease, while habitual physical activity may reduce the individual risk for this condition (6, 18–21). The protective effect of exercise seems to be mediated through favourable influences on several established anthropometric and biochemical risk factors (19, 27, 33).

Tab. 3 *Spearman*'s rank coefficient of correlation between initial anthropometric variables and initial blood properties in the total group ($N = 34$).

	Age (a)	W_{max} (Watt)	Weight (kg)	Body mass index (kg/m^2)	Diastolic blood pressure (mm Hg)	Systolic blood pressure (mm Hg)
Fibrinogen (g/l)	0.02	−0.39	−0.18	0.12	0.09	0.01
Tissue plasminogen activator (µg/l)	0.29	0.13	0.34	0.32	0.46[a]	0.39
Plasminogen activator inhibitor (10^3 AU/l)	−0.10	0.07	0.36	0.21	0.08	0.03
Triacylglycerols (mmol/l)	0.19	0.17	0.36	0.46[a]	0.32	0.60[b]
Total cholesterol (mmol/l)	0.32	−0.02	0.05	0.27	0.19	0.55[b]
LDL cholesterol (mmol/l)	0.41	0.14	0.20	0.36	0.28	0.58[b]
HDL cholesterol (mmol/l)	0.13	−0.37	−0.39	−0.12	−0.25	0.02
Lipoprotein(a) (mg/l)	−0.09	−0.17	−0.15	0.05	−0.12	0.14

[a] $P < 0.01$; [b] $p < 0.001$

Tab. 4 *Spearman*'s rank coefficient of correlation between changes (Δ) in quantities of the lipid metabolism and changes (Δ) in factors of the haemostatic and fibrinolytic system after nine months of training in the total group (N = 34).

	Δ Triacylglyerols (mmol/l)	Δ Total cholesterol (mmol/l)	Δ Low density lipoprotein cholesterol (mmol/l)	Δ High density lipoprotein cholesterol (mmol/l)	Δ Lipoprotein(a) (mg/l)
Δ Fibrinogen (g/l)	0.24	−0.13	−0.13	0.29	−0.19
Δ Tissue plasminogen activator (μg/l)	0.09	−0.04	0.28	−0.16	−0.03
Δ Plasminogen activator inhibitor (10^3 AU/l)	−0.15	−0.06	0.08	0.15	0.10

Vigourous physical activity may directly or indirectly influence body weight, blood pressure and the lipid status (22, 24). Furthermore, beneficial effects of intensive exercise on the haemostatic system are described (23, 25). Nowadays, whether the intensity of exercise training, attainable for the majority of people, will provoke similar results, is a topic of investigation (6, 10, 28, 32, 41, 42).

In our study a nine months exercise programme of moderate intensity resulted in a median W_{max} increase of 24 Watt (12%) in men and 25 Watt (18%) in women. Although aerobic fitness is commonly expressed as maximal oxygen uptake ($VO_{2_{max}}$), W_{max} is a more stable determinant for aerobic endurance capacity and easier to assess in the laboratory (43). Results of other studies, in which previously sedentary individuals participated in an aerobic training programme of moderate intensity, showed increases in $VO_{2_{max}}$ of 10−20% (30, 41, 42). These results are comparable with the results of the present study, since $VO_{2_{max}}$ and W_{max} are linearly interrelated (43, 44). The nine months training programme provoked, besides increases in W_{max} at all participants, several changes in selected risk factors. However, the change in aerobic endurance capacity was not significantly correlated with changes in anthropometric variables or changes in lipid and haemostatic properties. Furthermore, in the stepwise multiple regression model, no initial anthropometric variables were independent predictors for the changes of W_{max}. So, an active life style and physical conditioning by itself influences risk factors, irrespective of anthropometric characteristics or the outcome of the training sessions (10, 12, 20, 27, 45). Regular physical activity can lower mean resting blood pressure in moderately hypertensive patients, although this finding is not universal (46−48). In our study, no significant changes were found in diastolic or systolic blood pressure at the end of the training programme in either men or women. However, one can doubt whether a fall in blood pressure in healthy normotensive subjects is likely to be expected (41, 42, 43, 49). The nine months training programme induced a significant decrease in median body weight and body mass index in the male group, as well as a non-significant reduction of body weight and body mass index in the female group. This finding can be explained by the increased energy expenditure of the individuals under investigation as a result of the regular training sessions (24, 33). Before the training programme, initial total cholesterol, LDL cholesterol and triacylglycerol levels correlated significantly with the systolic blood pressure in the total group, while the diastolic blood pressure was significantly correlated to tissue plasminogen activator. Furthermore, a significant relation between tissue plasminogen activator and triacylglycerols was found. Hypofibrinolysis in patients with hyperlipidaemia has been frequently reported (50−53). Also a high blood pressure seems to influence fibrinolysis (54, 55). The results of the present study focus on the important interplay between primary risk factors, like blood pressure and lipid levels with fibrinolysis (54). The changes in the haemostatic, fibrinolytic and lipid status as a result of long-term training programme has been separately discussed before (56−59). The fall in plasma plasminogen activator inhibitor levels, observed in both men and women and the significant decrease in total cholesterol, LDL cholesterol and triacylglycerols in the group of men after nine months of training are known favourable effects of physical conditioning (26, 27, 30, 33, 63). The changes in these variables were not interrelated, stressing the fact that these properties as risk factors for atherosclerotic disease behave independently (61). Lipoprotein(a), an LDL like lipoprotein, competes with plasminogen for fibrin binding in vitro, and therefore may relate atherosclerosis to thrombosis (14−17). Although some studies report an association between lipoprotein(a) levels and fibrinogen concentrations, most investigators failed to find any relation between lipoprotein(a) and factors of the haemostatic or fibrinolytic system (62−64). Also in the present study, the increases in lipoprotein(a) levels in both groups and the increases in fibrinogen levels in the male group at the end of the training programme were not correlated. Lipoprotein(a) levels are often increased in patients suffering from coronary heart disease (16, 65). Recently lipoprotein(a) levels in sera of healthy physically active individuals were found to be raised, which parallels the results of the present study (66). Therefore, lipoprotein(a) may play a physiological role as an acute phase reactant in tissue repair, making its function not solely restricted to be an atherogenic determinant (66,

67). In the present study, the training sessions in preparation for the half marathon run probably have caused physical stress on the body of the participants, inducing a rise in lipoprotein(a) and also in fibrinogen as acute phase reactants (66–69). Finally, the present study was restricted to a selected group of healthy volunteers and most pretraining metabolic properties of the individuals were in the normal reference range. Therefore the impact of the results of this training programme could only be limited. Taking this in consideration, the effect of physical conditioning on the risk factor profile should be more pronounced in individuals with slight to moderate increased risk to cardiovascular disease. Further research on this subject should therefore include patients with an elevated risk factor profile. Furthermore, longer exercise programmes and larger study groups may be needed to get effects comparable with cross-sectional studies.

In conclusion, the results of the present study show that regular physical conditioning of moderate intensity influences coronary risk factors at rest. These changes are more pronounced in men than in women. Improvement of anthropometric factors, lipid profile and fibrinolytic potential are important favourable effects. Supposed adverse effects of exercising, like increases in lipoprotein(a) and fibrinogen, could actually be a normal physiological response to the physical strain of the exercise programme of increasing workload.

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