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Acute and delayed effects of prolonged exercise on serum lipoproteins

I. Composition and distribution of high density lipoprotein subfractions

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Summary. To investigate the effects of a single period of prolonged exercise on lipoprotein concentration and composition, the serum of 13 healthy, endurance-trained men was examined before and after (1 h, 20 h) a field test [running time, 130 (SD 7.4) min]. We found changes in composition of all of the lipoprotein fractions isolated. In detail, all very low density lipoprotein particle components were reduced after exercise; the most pronounced changes found were in the concentrations of phospholipids (PL) and triglycerides (TG) (PL, before vs 20 h after, $P<0.01$; TG, before vs 20 h after, $P<0.01$). The serum high density lipoprotein (HDL)-cholesterol mass was unchanged after exercise, but both HDL subfractions showed changes in composition. In HDL_3 the relative amounts of cholesterol increased (unesterified free cholesterol; FC) before vs 20 h after, $P<0.05$; cholesterylester (CE), before vs 20 h after, $P<0.01$) and TG and PL decreased (TG and PL, before vs 20 h after, $P<0.05$). The HDL_2 particles became enriched in the relative amount of CE (before vs 20 h after, $P<0.01$) and lost TG after exercise (before vs 20 h after, $P<0.01$). The observation that all the changes in lipoprotein concentration and composition reached their maximal differences compared to the pre-exercise values 20 h afterwards would support the assumption that circulating lipoproteins play an important role in the regeneration period, refilling the intramuscular triglyceride stores.

Key words: Physical activity – Lipoprotein composition – Acute and delayed effects

Introduction

The beneficial effects of physical activity on lipid and lipoprotein metabolism have been generally accepted (Berg et al. 1992). However, most of the investigations on the influence of training on lipoprotein metabolism discuss such effects of endurance training by comparing untrained and trained subjects. Such a design raises some practical problems. Because, in general, physically active subjects have a more healthy orientated life-style (no smoking, low alcohol, lower fat intake, lower body mass index) than sedentary individuals, differences between trained and untrained subjects may relate therefore not only to regular physical activity but also to accompaning factors. On the other hand, the association between energy metabolism and lipid, as well as lipoprotein metabolism is well known (Carlson and Mossfeldt 1964; Lithell et al. 1984; Dufaux et al. 1981).

Few studies have been undertaken to investigate the influence of a single period of exercise on lipoprotein metabolism in detail. These studies have consistently documented time-dependent changes in serum concentrations of triglyceride (TG, decreased) and high density lipoprotein-cholesterol (HDL-C, increased) (Dufaux et al. 1981, 1986; Cullinane et al. 1982; Kantor et al. 1984, 1987; Sady et al. 1986; Lamon-Fava et al. 1989; Davis et al. 1992). Furthermore, little information is available on the influence of a single period of prolonged exercise on the composition of lipoprotein subfractions. There is evidence that changes in composition of HDL as well as in low density lipoprotein (LDL) subclasses occur after exercise (Berg et al. 1983; Lamon-Fava et al. 1989).

The present study was undertaken to examine the effects of a single period of prolonged exercise on serum lipid and lipoprotein concentrations and on the composition of lipoprotein subclasses, in this case HDL, to elucidate the role of energy metabolism in modulating lipoprotein metabolism.

This paper is dedicated to Prof. Dr. J. Keul, Medical Director of our Department, on the occasion of his 60th birthday

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Methods

Subjects and procedure. A homogeneous group of 13 male endurance-trained volunteers [age, 31.0 (SD 7.5) years; maximal oxy-

gen uptake ($\dot{V}O_{2max}$), 59.5 (SD 2.1) ml·kg^{-1}·min^{-1} (determined in a stepwise aerobic treadmill test)] participated in a standardized endurance stress test (30 km cross-country running). The stress test was performed as a competition and each subject was encouraged to do his best. The mean running time was 130 (SD 7.4) min. According to the lactic acid concentrations in the treadmill test (maximal aerobic performance), the subjects performed the endurance stress test at a mean intensity of 78% of their $\dot{V}O_{2max}$. The $\dot{V}O_{2max}$, as an index of each individual's maximum aerobic capacity was measured with an open spirometric system (Oxyconsigma, Schröder GmbH, Iserlohn, Germany) in a maximal aerobic stress test (Berg and Keul 1981). The subjects were assessed by medical history as well as clinical, electrocardiographic and serological findings and there was no reason to suspect any chronic or metabolic diseases.

To assess habitual food intake, 7-day records of diet were kept by the subjects. The records were analysed by the PRODI III plus software package (Kluthe 1989). Daily total energy intake [2917 (SD 564) kcal, 12222 (SD 2363) kJ] and the mean percentages of energy intake for carbohydrate [50.3 (SD 6.2)%], fat [32.4 (SD 4.2)%], protein [13.7 (SD 2.1)%] and alcohol [3.5 (SD 3.5)%] were calculated. All the participants were nonsmokers.

Time schedule. Fasting blood samples were drawn between 7.30 a.m. and 8.30 a.m. The subjects were then given a low energy carbohydrate-rich mixed meal. The test started at 10.00 a.m., the outdoor temperature was 18° C. In contrast to any other energy intake, fluid intake during and after the test was allowed ad libitum; 1 h after running was over the second blood samples were drawn. A third blood sample was taken the next day between 7.30 a.m. and 8.30 a.m., which was 20 h after the end of the test, the subjects again being in a fasted state.

Blood analysis. The spontaneously coagulated blood samples were centrifuged twice; serum aliquots were stored at 4° C and −35° C. Immediately after blood collection HDL-, LDL- and very low density lipoprotein-cholesterol (VLDL-C) concentrations were determined by lipoprotein electrophoresis (Wieland and Seidel 1978) and lipoprotein fractions and subfractions of HDL (HDL_2, 1.063–1.125 g·ml^{-1}; HDL_3, 1.125–1.210 g·ml^{-1}) were prepared by ultracentrifugation under standard conditions (Lindgren et al. 1972; Lindgren 1975). In each subfraction the concentrations of TG, total cholesterol (TC) and unesterified free cholesterol (FC) as well as phospholipids (PL) were measured using commercial kits (Boehringer, Mannheim, Germany; bio Merieux, Nürtingen, Germany). Concentrations of cholesterylester (CE) were calculated as molar difference between TC and FC. Apolipoprotein concentrations (apoA-I, apoA-II, apoB) were determined by nephelometry using specific human antisera from Beckman (Munich, Germany) and Behring (Marburg, Germany; apoA-II).

To document exercise-induced changes in plasma volume packed cell volume was determined.

Statistics. Differences between before and after values were tested for significance using Friedmans two-way ANOVA. Univariate comparisons (before vs 1 h, before vs 20 h) were performed by Wilcoxon matched-pairs signed ranks test using SPSS/PC+ (1983).

Results

As shown in Table 1, there were only small differences in serum lipid- and apolipoprotein concentrations between before- and 1-h after exercise values. But 20 h after the field test, differences in TC, TG and apolipoprotein concentrations became more distinct and reached significance levels.

In Table 2a acute and delayed changes in the concentrations of the VLDL components are shown. All VLDL components were reduced after exercise, suggesting a reduction in the number of VLDL particles. We found the lowest concentrations 20 h after the field test. On the other hand, we found changes in composition, in that VLDL particles lost FC and PL after exercise (Table 2b). Exercise-induced changes in the concentrations of the components of HDL and HDL subfractions were very small (Table 3).

Table 4 shows exercise-induced changes in the relative composition of HDL_3 and HDL_2 subfractions. In both HDL subfractions we found a typical shift in lipid components. After exercise, the relative amounts of cholesterol (FC, CE) were significantly increased in HDL_3, whereas the contents of TG and PL were significantly decreased. In this subfraction the relative amount of apoA-I remained unchanged, but apoA-II content was significantly increased 1 h after exercise. The HDL_2 particles became significantly enriched in the relative amount of CE but lost TG after exercise. The relative amount of apolipoproteins remained unchanged within these subfractions.

All of the observed changes showed their maximal differences to the pre-exercise values 20 h after the endurance stress test.

Discussion

The present data underline the observation that acute prolonged exercise affects lipid and lipoprotein meta-

Table 1. Exercise-induced changes in total cholesterol (TC), very low density lipoprotein-cholesterol (VLDL-C), low density lipoprotein-cholesterol (LDL-C), high density lipoprotein-cholesterol (HDL-C), triglyceride (TG), and apolipoprotein concentrations (apoB, apoA-I, apoA-II) and packed cell volume

	TC		TG		VLDL-C		LDL-C		HDL-C		apoB		apoA-I		apoA-II		PCV	
	(mmol·l^{-1})										(mg·dl^{-1})						(%)	
	mean	SD	mean	SD	mean	SD	mean	SD	mean	SD	mean	SD	mean	SD	mean	SD	mean	SD
Before exercise	4.53	0.78	1.10	0.31	0.31	0.14	2.78	0.58	1.44	0.29	68.6	13	159	29	48	8	43.1	4.3
1 h after	4.64	0.80	0.92	0.30	0.26	0.14	2.91	0.52	1.47	0.43	69.6	16	150	33*	51	10	44.9	4.9[a]
20 h after	4.21	0.64**	0.76	0.20**	0.25	0.09	2.53	0.63*	1.44	0.49	63.6	12**	134	23**	49	7	42.8	4.6

$n=13$. Significant differences versus pre-exercise values: [a] $P<0.01$ (Friedmann ANOVA NS; Wilcoxon sign test); * $P<0.05$, ** $P<0.01$ (Friedmann ANOVA and Wilcoxon sign test)

Table 2a. Exercise-induced changes in concentrations of phospholipids (PL), unesterified cholesterol (FC), cholesterylester (CE), triglycerides (TG) and apolipoprotein B in very low density lipoprotein

	PL		FC		CE		TG		ApoB	
	(mmol·l^{-1})								(mg·dl^{-1})	
	mean	SD	mean	SD	mean	SD	mean	SD	mean	SD
Before exercise	0.201	0.097	0.108	0.068	0.183	0.124	0.590	0.245	4.4	2.2
1 h after	0.158	0.102*	0.086	0.070*	0.159	0.116	0.449	0.243	4.0	2.0
20 h after	0.127	0.059**	0.056	0.045**	0.099	0.059*	0.381	0.146**	2.9	0.8*

$n = 13$. Significant differences versus pre-exercise values:
* $P < 0.05$, ** $P < 0.01$ (Friedmann ANOVA and Wilcoxon sign test)

Table 2b. Exercise-induced changes in the number of lipid molecules per very low density lipoprotein particle (calculated as molar ratio of lipid per apoB)

	PL		FC		CE		TG	
	mean	SD	mean	SD	mean	SD	mean	SD
Before exercise	2395	567	1230	286	2019	476	7444	2929
1 h after	1905	437[a]	971	337*	1850	478	5671	1299[a]
20 h after	2158	649	898	516*	1673	630	6752	2343

$n = 13$. Significant differences versus pre-exercise values:
[a] $P < 0.05$ (Friedmann ANOVA NS; Wilcoxon sign test);
* $P < 0.05$ (Friedmann ANOVA and Wilcoxon sign test)

bolism in a characteristic way. As confirmed by subsequent studies, increased TG utilisation has to be regarded as a key event (Dufaux et al. 1981; Cullinane et al. 1982; Sady et al. 1986). We found lowered TG concentrations in serum and VLDL as well as in HDL and LDL subfractions (for LDL see part II of this study, Baumstark et al. (1993)). Both, enhanced peripheral clearance of plasma TG and decreased liver secretion via VLDL have to be considered as possible explanations for the lowered serum TG concentrations. It is well-known that during prolonged exercise an increasing demand for fatty acids as energy substrates is provided via enhanced lipolysis of TG and TG-rich lipoproteins (Keul 1975; Paul 1975; Berg et al. 1989). The TG catabolism has been shown to be mediated by the activity of lipoprotein lipase (LPL; Taskinen et al. 1980). The LPL has been shown to be synthesized in adipocytes and muscle cells, later released and transported to the capillary endothelium where it binds to the luminal surface (Nilsson-Ehle et al. 1980; Saxena et al. 1991). When the muscle TG-stores are mobilized, the synthesis of LPL in the muscle endothelial bed is increased and the activities of adipose tissue LPL and postheparin (plasma) LPL have been found to be elevated (Kantor et al. 1984, 1987; Sady et al. 1986; Savard et al. 1987) leading to increased catabolism of TG and TG-rich lipoproteins.

Our current knowledge suggests that there are at least two separate metabolic processes that link HDL to TG-rich lipoproteins. Firstly, HDL acts as an acceptor for material released from VLDL during their lipolyses and, secondly, there is an exchange of lipids between HDL and VLDL mediated by specific lipid transfer proteins. As the present data show, the decrease in VLDL concentration was accompanied by changes in the relative composition of HDL subfrac-

Table 3. Exercise-induced changes in the concentrations of phospholipids (PL), unesterified free cholesterol (FC), cholesterylester (CE), triglycerides (TG) and apolipoprotein A-I and apoA-II in high density lipoprotein subfractions

	PL		FC		CE		TG		apoA-I		apoA-II	
	(mmol·l^{-1})								(mg·dl^{-1})			
HDL	mean	SD	mean	SD	mean	SD	mean	SD	mean	SD	mean	SD
Before exercise	1.1913	0.2116	0.2320	0.0994	1.2564	0.2812	0.1231	0.0399	115.92	28.62	42.58	5.94
1 h after	1.1894	0.2128	0.2669	0.0763*	1.3163	0.2785[b]	0.0990	0.0279*	116.09	25.38	44.73	6.51
20 h after	1.1520	0.2186	0.2624	0.0799*	1.2337	0.2808	0.0832	0.0257**	104.05	20.74[a]	43.57	7.79
HDL_3												
Before exercise	0.3902	0.0529	0.0568	0.0115	0.3984	0.0739	0.0293	0.0089	64.31	10.56	22.41	3.86
1 h after	0.3891	0.0478	0.0632	0.0099[a]	0.4368	0.0713	0.0250	0.0069	61.08	8.37	24.28	3.04
20 h after	0.3653	0.0480	0.0609	0.0099	0.4112	0.0592	0.0226	0.0086[a]	59.95	10.21	21.34	3.22
HDL_2												
Before exercise	0.7328	0.1977	0.1673	0.0644	0.7626	0.2092	0.0751	0.0262	55.94	16.59	17.26	3.15
1 h after	0.7366	0.2033	0.1806	0.0642	0.8148	0.2283	0.0622	0.0182	57.91	20.11	17.43	4.10
20 h after	0.7512	0.2233	0.1834	0.0739	0.8022	0.2416	0.0549	0.0191**	55.60	17.80	17.05	4.86

$n = 13$. Significant differences versus pre-exercise values:
[a] $P < 0.05$ (Friedmann ANOVA NS; Wilcoxon sign test)
* $P < 0.05$, ** $P < 0.01$ (Friedmann ANOVA and Wilcoxon sign test)
[b] $P > 0.05$ (Friedmann ANOVA; Wilcoxon sign tests NS)

Table 4. Exercise-induced changes in the relative composition of high density lipoprotein-subfractions. Phospholipids (PL), unesterified free cholesterol (FC), cholesterylester (CE), triglycerides (TG), apolipoprotein A-I and apoA-II

	PL		FC		CE		TG		apoA-I		apoA-II	
	(% mass)											
HDL_3												
Before exercise	20.6	2.7	1.5	0.2	17.4	1.4	1.8	0.7	43.5	3.5	15.1	1.3
1 h after	20.4	1.1	1.6	0.2*	19.0	1.3*	1.5	0.5	41.1	2.3	16.4	0.6[a]
20 h after	20.3	1.9[a]	1.7	0.2*	18.9	1.4**	1.4	0.5*	42.5	1.5	15.2	1.2
HDL_2												
Before exercise	29.4	0.9	3.3	0.5	25.7	2.0	3.5	1.3	28.8	2.1	9.3	1.6
1 h after	28.9	0.9	3.5	0.3[b]	26.8	1.3	2.9	1.0*	28.8	2.4	9.0	1.8
20 h after	29.8	0.6	3.5	0.5	26.8	1.3**	2.6	0.9**	28.3	2.3	8.9	1.7

$n=13$. Significant differences versus pre-exercise values: [a] $P<0.05$ (Friedmann ANOVA NS, Wilcoxon sign test); * $P<0.05$, ** $P<0.01$ (Friedman ANOVA and Wilcoxon sign test) [b] $P<0.05$ (Friedmann ANOVA; Wilcoxon sign test NS)

tions. The differences in HDL subfraction composition were small 1 h after the field-test and became more pronounced in the early regeneration period. At this time postexercise HDL particles showed a changed lipid pattern with elevated concentrations of cholesterol (FC, CE) and lowered concentrations of PL and TG. A more or less pronounced increase in concentration of HDL-C within the two HDL subfractions following acute exercise has been described earlier (Berg et al. 1983; Kantor et al. 1984; Kuusi et al. 1984; Dufaux et al. 1986; Davis et al. 1992). The observed cholesterol enrichment of HDL particles might either have been a consequence of the incorporation of surface components of VLDL remnants, or a consequence of the uptake of peripheral cholesterol. In both cases small HDL precursors will have been converted to cholesterol-laden HDL_3 particles. It is well-known that cholesterol-enriched HDL_3 particles are a preferred substrate for the enzyme lecithin:cholesterol acyltransferase (LCAT) (Fielding and Fielding 1971). Under the action of LCAT, unesterified cholesterol located in the particle surface is esterified and afterwards displaced to the lipophilic core of the lipoprotein particle, a process which is regarded to be the initial event in the conversion of HDL_3 to cholesterol-rich HDL_2 particles. As has been documented by a few studies, LCAT activity is elevated following physical activity (Dufaux et al. 1986; Frey et al. 1991). The increased amounts of CE in HDL_2 particles we found after exercise might therefore have been a result of an exercise-induced increase in LCAT activity.

On the other hand, lipid transfer processes may also have played a role in inducing changes in particle composition, but very little is known about the influence of exercise on the activity of lipid transfer proteins.

Few data have been published concerning exercise-induced changes in apolipoprotein concentrations. After exercise most authors have found concentrations of apoA-I and apoA-II either unchanged or just slightly altered (increased apoA-I, decreased apoA-II; Kantor et al. 1984; Sady et al. 1986; Lamon-Fava et al. 1989; Dufaux et al. 1986; Frey et al. 1991). We found serum apoA-I concentrations reduced after exercise, reaching their lowest concentrations 20 h later (-16%, $P<0.01$), confirming data published by Lithell et al. (1984). The reduction in apoA-I concentration was less pronounced in HDL (-10%, $P<0.05$). Within HDL_3 we found apoA-I concentrations reduced after exercise by about 7% (NS), in HDL_2 apoA-I concentrations were unchanged after exercise, leading to the assumption that the exercise-induced changes in apoA-I concentration were partly due to variations in apoA-I not associated with HDL particles.

All of the observed changes in lipoprotein concentration and composition showed a characteristic time course, with maximal differences compared to pre-exercise values in the early regeneration period, agreeing with the data obtained in earlier studies (Dufaux et al. 1981, 1986; Cullinane et al. 1982; Lithell et al. 1984; Kantor et al. 1984, 1987; Lamon-Fava et al. 1989). The low concentrations of TG (in serum and lipoproteins), even 20 h after exercise agree with the observation that muscle LPL activity is still elevated in the regeneration phase (Lithell et al. 1984; Sady et al. 1986; Kantor et al. 1987), leading to the assumption that the circulating TG-rich lipoproteins may play a role in replenishing the intramuscular TG stores during the regeneration period.

In conclusion, our data would confirm that a single period of prolonged exercise induces a marked decrease in TG in serum and lipoprotein fractions. This effect is accompanied by an increased cholesterol content in HDL_3 and HDL_2 subfractions. As concentrations of apoA-I in HDL_2 remained unchanged after exercise, we would suggest that the HDL_2 subfraction serves as an effective lipid shuttle, taking up remnants of catabolized TG-rich lipoproteins during and after exercise as well as in the regeneration period.

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