Effect of Age on the Response of Blood Lipids, Body Composition, and Aerobic Power to Physical Conditioning and Deconditioning

Franco Giada, Giovanni B. Vigna, Enrico Vitale, Goretta Baldo-Enzi, Manuel Bertaglia, Rosa Crecca, and Renato Fellin

The influence of age on the response of plasma lipids, body composition, and cardiovascular performance to physical training and detraining was studied in 12 older and 12 young adult male cyclists. The athletes were first examined at the peak of their seasonal preparation and then again 2 months after its suspension. Sedentary males matched for age, weight, and height comprised the respective control groups. During training, body fat mass (BFM) was significantly lower and maximum oxygen consumption ($\dot{V}O_2max$) higher in both groups of cyclists as compared with controls. No differences in serum total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), apolipoprotein (apo) B, apo A-II, and fibrinogen were found. During the same phase, triglycerides (TG) and the LDL-C to high-density lipoprotein cholesterol (HDL-C) ratio were significantly lower and apo A-I, HDL-C, HDL_3-C, and the apo A-I/apo B ratio were significantly higher in the athletes than in their corresponding sedentary controls. After physical deconditioning, BFM increased and $\dot{V}O_2max$ decreased significantly in both groups of athletes. TG, very-low-density lipoprotein cholesterol (VLDL-C), and fibrinogen increased in young athletes while the LDL-C/HDL-C ratio increased, and apo A-I, HDL-C, HDL_2-C, and HDL_3-C decreased significantly in both young and older athletes. Thus, an aerobic training program induced an antiatherogenic lipoprotein profile and beneficial modifications in body composition and aerobic power in both older and younger subjects; a 2-month interruption in the program changed these parameters unfavorably in both groups. Age does not seem to influence significantly the plasma lipid response to physical deconditioning.

IT IS WELL KNOWN that plasma lipoprotein levels are powerful risk factors for coronary heart disease (CHD). Several studies seem to demonstrate that aerobic physical activity, when practiced regularly, is able to modify the plasma lipid pattern favorably and produce an increase in cardiovascular performance, as well as improve body composition, in young and older individuals.[1] Investigations addressing the relationship between physical exercise and the lipoprotein pattern in elderly subjects are much less frequent,[2-5] even though this group constitutes the proportion of the population with the highest incidence of cardiovascular disease. Moreover, recent studies demonstrated the importance of plasma lipids for predicting cardiovascular risk, as well as the possibility of atheromatous plaque regression following lipid-lowering therapy in elderly individuals.[6,7] However, data regarding the stability of the training-induced lipoprotein changes and the effects of exercise withdrawal are controversial.[8-10]

The purpose of this study was to evaluate the influence of age on modifications in plasma lipids and lipoproteins, body composition, and cardiovascular performance following training and detraining. We examined young and older adult male cyclists during training and after its suspension and compared findings with those observed in two groups of age-matched, healthy, sedentary male controls.

SUBJECTS AND METHODS

Subjects and Study Design

Twelve older (age range, 50 to 65 years) and 12 young (age range, 19 to 25 years) male amateur cyclists were examined at the end of the competitive season (October) and then again 2 months after the suspension of physical activity (December). During detraining, the athletes agreed not to undergo any maintenance program and limited their level of physical exercise to that determined by their working activity (sedentary in all cases). They had all been practicing road cycling, consisting mainly of aerobic physical activity, for several years (21 ± 12 and 9 ± 5 years for older and young athletes, respectively; Table 1). In the preceding 6 months, the young cyclists covered 351 ± 162 km/wk and the older athletes rode 213 ± 89 km/wk.

The control groups consisted of 12 older and 12 young adult healthy sedentary males matched for age, height, and weight who were not engaged in any physical training program. After obtaining their informed written consent, every subject underwent a general medical examination, an electrocardiogram, and routine blood tests. None of the athletes smoked; only two of the young control subjects were habitual smokers (10 cigarettes daily). During the entire study no subject was taking any medication.

Body Composition

The subjects were weighed to the nearest 0.1 kg, and their height was measured to the nearest 0.5 cm. Bicipital, tricipital, subscapular, and suprailiac skinfold thicknesses were measured on the right side of the body using a Harpenden caliper; three consecutive measurements were taken for the mean.[11] The percentage of body weight consisting of adipose tissue (body fat mass [BFM]), and the fat-free body mass ([FFM] in kilograms) were estimated by bioelectrical impedance analysis (BIA 109, RJL Akern, Firenze, Italy).[12-14]

Diet

Information regarding diet was obtained using a 7-day dietary record. Participants were asked not to modify their usual food habits during this period, and to record their daily intake on a standard form that was distributed by a dietician along with a detailed list of instructions. Food values were calculated using a table of food contents.[15]

From the Division of Internal Medicine, Hospital Umberto I, Mestre; the Institute of Medical Pathology, University of Ferrara, Ferrara; and the Institute of Internal Medicine, University of Padua, Padua, Italy.

Submitted August 30, 1993; accepted May 18, 1994.

Address reprint requests to Renato Fellin, MD, Institute of Medical Pathology, University of Ferrara, via Savonarola n.9, 44100 Ferrara, Italy.

0026-0495/95/4402-0006$03.00/0

Table 1. Anthropometric Characteristics (mean ± SD)

Subjects	Age (yr)	BMI (kg/m²)	BFM (%)	FFM (kg)	Skinfold Thickness (mm) BIC	TRIC	SCAP	ILIAC	$\dot{V}O_2$max (mL/min/kg)	Training (km/wk)
Older										
Cyclists (n = 12)										
Training	55 ± 5	26.8 ± 2.4	15 ± 3*†	64 ± 5	5 ± 2	11 ± 3	15 ± 4*†	12 ± 4*†	43 ± 7*†§	213 ± 89
Detraining	—	27.1 ± 2.3	17 ± 4	63 ± 5	6 ± 3	12 ± 3	18 ± 7	23 ± 11	36 ± 7‡	—
Sedentary controls (n = 12)	58 ± 6	26.5 ± 2.6	21 ± 3	63 ± 5	8 ± 3	13 ± 4	23 ± 6	25 ± 7	23 ± 3‖	—
Young										
Cyclists (n = 12)										
Training	24 ± 6	22.9 ± 1.9	15 ± 3*†	61 ± 6	3 ± 1	6 ± 3	11 ± 3*†	12 ± 5*†	59 ± 10*†	351 ± 162
Detraining	—	23.8 ± 1.6	19 ± 4	59 ± 5	4 ± 2	9 ± 3	13 ± 5	16 ± 7	49 ± 9‡	—
Sedentary controls (n = 12)	23 ± 2	23.6 ± 3.0	21 ± 6	58 ± 7	6 ± 3	14 ± 7	21 ± 7	24 ± 7	35 ± 6	—

Abbreviations: BMI, body mass index; BIC, biceps; TRIC, triceps; SCAP, subscapular; ILIAC, suprailiac.

*$P < .05$, training *v* detraining.

†$P < .05$, training *v* controls.

‡$P < .05$, detraining *v* controls.

§$P < .05$, older cyclists training *v* young controls.

‖$P < .05$, older sedentary controls *v* young sedentary controls.

Measurement of Maximum Aerobic Power

Maximum oxygen consumption ($\dot{V}O_2$max) was determined during a maximal exercise test using a cycle ergometer by analyzing expired air.[16]

Lipid Analysis and Lipoprotein Isolation

A venous blood sample was collected in the morning following a 12-hour fast and an interval of at least 36 hours since the last training session. Total cholesterol (TC) in serum and in lipoprotein fractions (very–low-density lipoprotein cholesterol [VLDL-C], low-density lipoprotein cholesterol [LDL-C], and high-density lipoprotein cholesterol [HDL-C]) was determined enzymatically.[17] Serum triglycerides (TG) were evaluated according to the method of Wahlefeld.[18] Apolipoprotein (apo) A-I, apo A-II, apo B, and fibrinogen were assayed by means of radial immunodiffusion using NOR-Partigen monoclonal kits (Behringwerke, Marburg/Lahn, Germany). Lipoprotein fractions were isolated by sequential preparative ultracentrifugation according to the method of Havel et al.[19] Cholesterol levels in HDL subfractions (HDL_2-C and HDL_3-C) were determined according to the method of Gidez et al.[20]

Statistical Analysis

All data are expressed as the mean ± SD. The four groups were compared using one-way ANOVA (program P2V, BMDP statistical package) with adjustment according to Bonferroni probabilities; "pooled variance" was selected to evaluate the level of significance.[21] Student's *t* test for paired data was used to compare data obtained during and after training. Correlations between different variables were determined by linear regression analysis. *P* values less than .05 were considered significant.

RESULTS

Anthropometric characteristics of all study subjects are reported in Table 1. No differences in body mass index (kg/m²), bicipital and tricipital skinfold thicknesses, and FFM were found among the groups. However, during training BFM and subscapular and suprailiac skinfold thicknesses were significantly lower and $\dot{V}O_2$max was significantly higher in both groups of athletes as compared with the corresponding control groups. After physical deconditioning, BFM and subscapular and suprailiac skinfold thicknesses increased and $\dot{V}O_2$max decreased significantly in both young and older athletes. During training, $\dot{V}O_2$max was higher in older athletes as compared with young controls. Daily caloric intake was greater in athletes as compared with controls and greater during training as compared with detraining (Table 2). No differences were observed in macronutrient intakes.

Serum lipid levels and cholesterol concentrations in lipoprotein fractions are reported in Tables 3 and 4, respectively. During training, neither young nor older athletes showed differences in TC, VLDL-C, LDL-C, HDL_2-C, apo B, apo A-II, and fibrinogen as compared with their corresponding controls, whereas TG and the LDL-C/HDL-C ratio were significantly lower and apo A-I, HDL-C, HDL_3-C, and the apo A-I/apo B ratio were significantly higher. Following training suspension, VLDL-C and fibrinogen increased significantly only in young athletes, whereas TG and the LDL-C/HDL-C ratio increased and apo A-I, HDL-C, HDL_2-C, and HDL_3-C decreased significantly in both groups of athletes. The only parameters that remained significantly different as compared with values in sedentary controls were the apo A-I/apo B ratio and HDL_3-C in older athletes and the LDL-C/HDL-C ratio and HDL_3-C in the younger ones.

Older control subjects showed higher levels of TC, TG, LDL-C, apo B, and HDL-C as compared with young controls. In contrast, TG and LDL-C levels in older athletes during training overlapped those of young sedentary controls, whereas TC, apo B, HDL-C, apo A-I, and HDL_3-C were higher.

In both young and older athletes, TG, HDL-C, and apo A-I levels showed no significant correlations with BFM, training distance, or $\dot{V}O_2$max. Similarly, $\dot{V}O_2$max did not correlate with BFM or training distance.

Table 2. Diet Composition and Mean Daily Nutrient Intake (mean ± SD)

	Total kcal	% of Total kcal			Alcohol (g)	Cholesterol (mg)	P/S
		Protein	Fat	Carbohydrate			
Older							
Cyclists (n = 12)							
Training	3,120 ± 225*†‡	13.0 ± 1.3	29.4 ± 5.9	59.0 ± 5.2	45 ± 25	397 ± 139	0.34 ± 0.20
Detraining	2,846 ± 697	14.8 ± 1.9	29.3 ± 5.6	57.2 ± 5.7	40 ± 25	326 ± 151	0.32 ± 0.30
Sedentary controls							
(n = 12)	2,772 ± 522	13.0 ± 2.4	27.0 ± 3.7	60.0 ± 3.0	47 ± 19	260 ± 84	0.24 ± 0.26
Young							
Cyclists (n = 12)							
Training	3,579 ± 1,067*†	14.8 ± 1.4	30.0 ± 2.7	56.2 ± 5.0	26 ± 26	406 ± 124	0.30 ± 0.20
Detraining	3,328 ± 851	15.6 ± 1.8	30.1 ± 2.6	55.2 ± 4.5	18 ± 20	466 ± 245	0.30 ± 0.22
Sedentary controls							
(n = 12)	2,700 ± 610	15.0 ± 2.0	29.0 ± 4.7	56.4 ± 5.0	47 ± 41	327 ± 136	0.46 ± 0.20

Abbreviation: P/S, polyunsaturated to saturated fat ratio.
*$P < .05$, training *v* detraining.
†$P < .05$, training *v* controls.
‡$P < .05$, older cyclists training *v* young controls.

Table 3. Serum Lipid, Apolipoprotein, and Fibrinogen Levels (mg/dL, mean ± SD)

	TC	TG	Apo A-I	Apo A-II	Apo B	Apo A-I/Apo B	Fibrinogen
Older							
Cyclists (n = 12)							
Training	242 ± 33§	90 ± 36*	185 ± 21*†§	38 ± 8	134 ± 21§	1.4 ± 0.2†	373 ± 62
Detraining	241 ± 38	110 ± 35	169 ± 25	37 ± 7	130 ± 20	1.3 ± 0.3‡	412 ± 102
Sedentary controls							
(n = 12)	231 ± 20‖	98 ± 32‖	142 ± 23	34 ± 5	148 ± 15‖	0.9 ± 0.2	371 ± 61
Young							
Cyclists (n = 12)							
Training	159 ± 23	55 ± 25*†	155 ± 13*†	33 ± 5	96 ± 19	1.7 ± 0.4†	345 ± 44*
Detraining	163 ± 24	63 ± 28	142 ± 13	32 ± 5	91 ± 16	1.6 ± 0.4	363 ± 47
Sedentary controls							
(n = 12)	172 ± 15	73 ± 14	132 ± 8	32 ± 4	100 ± 14	1.3 ± 0.2	340 ± 28

*$P < .05$, training *v* detraining.
†$P < .05$, training *v* controls.
‡$P < .05$, detraining *v* controls.
§$P < .05$, older cyclists training *v* young controls.
‖$P < .05$, older sedentary controls *v* young sedentary controls.

Table 4. Cholesterol Levels in Lipoprotein Fractions (mg/dL, mean ± SD)

	VLDL-C	LDL-C	HDL-C	HDL_2-C	HDL_3-C	LDL-C/HDL-C
Older						
Cyclists (n = 12)						
Training	8.4 ± 5.9	127 ± 27	69 ± 15*†§	25 ± 15*	44 ± 3*†§	1.9 ± 0.4*†
Detraining	9.7 ± 4.6	140 ± 35	63 ± 18	22 ± 15	42 ± 7‡	2.4 ± 0.7
Sedentary controls						
(n = 12)	9.8 ± 5.4	146 ± 17‖	57 ± 12‖	25 ± 7	32 ± 6	2.6 ± 0.5
Young						
Cyclists (n = 12)						
Training	4.7 ± 4.2*	81 ± 18	59 ± 8*†	19 ± 6*	39 ± 4*†	1.4 ± 0.4*†
Detraining	6.3 ± 4.3	88 ± 19	53 ± 10	17 ± 7	36 ± 4‡	1.7 ± 0.4‡
Sedentary controls						
(n = 12)	14.7 ± 2.9	110 ± 13	47 ± 2	16 ± 3	30 ± 3	2.3 ± 0.3

*$P < .05$, training *v* detraining.
†$P < .05$, training *v* controls.
‡$P < .05$, detraining *v* controls.
§$P < .05$, older cyclists training *v* young controls.
‖$P < .05$, older sedentary controls *v* young sedentary controls.

DISCUSSION

Studies on the primary and secondary prevention of cardiovascular disease have demonstrated a direct relationship between inactivity and CHD; it was also shown that physical activity is inversely associated with overall mortality and that due to CHD itself in both healthy and postinfarct individuals.[22,23] One factor responsible for this protective effect could be the change in the plasma lipoprotein pattern induced by physical exercise. Accordingly, several transversal surveys found improved lipoprotein levels (ie, an increase in HDL-C and apo A-I and a reduction in TG and the LDL-C/HDL-C ratio) in young and middle-aged athletes as compared with age-matched sedentary controls.[24,25] On the other hand, prospective studies in healthy sedentary subjects undergoing exercise training produced controversial results.[26] This variability could be related at least in part to the age and basal lipoprotein levels of the examined subjects, their diet, and the BFM changes due to the training and the intensity and duration of the training itself. It is therefore of fundamental importance to know just how stable the training-induced lipid modifications are over time.

We found that physical activity in both age groups was associated with a favorable body composition characterized by a BFM decrease and a higher FFM/BFM ratio. Analysis of skinfold thickness evidenced a reduction in the subscapular and suprailiac skinfolds during the training period; these skinfolds are considered indices of central distribution of fatty tissue, and hence their reduction during the training period may be evaluated favorably, given the relationship between central obesity and risk of CHD.[27] Our observations agree with the results obtained by Schwartz et al[2,3] in groups of young and elderly subjects undergoing exercise training; they reported an increase in $\dot{V}O_2$max and a decrease in BFM and the waist to hip ratio, another index of central distribution of fatty tissue.

In agreement with other investigators,[2-5] we observed antiatherogenic modifications during training in the lipid profiles of both young and older athletes, consisting of a TG decrease and an apo A-I, HDL-C, and HDL_3-C increase. However, no differences were found in apo B and apo A-II levels, as also reported by Berg et al[28] in young adult athletes.

Fibrinogen, which is an independent factor of coronary risk,[29] showed no differences between athletes and controls; it increased after detraining in both groups of cyclists, but only in young athletes was the difference statistically significant. In studying the effects of physical exercise on coagulation parameters in diabetic and healthy control subjects, other researchers found no variation in plasma fibrinogen levels after 6 weeks of training.[30]

During adulthood, TC, TG, and LDL-C increase progressively and reach their highest values (in males) at approximately 55 years of age[31]; HDL-C has been reported to increase,[32] undergo no changes, or decrease.[33,34] In our study, the lipoprotein pattern in older athletes was similar in part to that of the younger ones. Indeed, like their respective sedentary controls, they had higher TC, HDL-C, apo A-I, and apo B levels as compared with the younger group, but they showed no increase in TG and LDL-C. As Frey et al[35] also advanced, this suggests that the physical inactivity typical of elderly persons might well be one of the factors in age-associated lipoprotein changes. The lipoprotein pattern modifications observed during the training period in both groups of athletes were reversible following suspension of physical activity.

No changes in diet composition were observed during detraining, with the exception of a decrease in daily caloric intake. This suggests a minor direct effect of dietary modification on the unfavorable metabolic outcome of reducing physical activity. On the other hand, the increase in BFM might well have some importance, given its known inverse and direct relationships with HDL-C and TG, respectively.[36] No further investigations on plasma lipoprotein changes and physical deconditioning in elderly athletes are currently available; however, the behavior of lipid parameters we observed after detraining agrees with the results of Nikkilä et al,[8] who found that the inactivity in persons immobilized for orthopedic problems was related to a reduction in HDL-C and apo A-I plasma levels. In a similar manner, Wood et al[37] concluded that to improve the plasma lipid pattern, physical exercise should be continued in time. Unlike our results, Thompson et al[9] observed no reduction in HDL-C, HDL_2-C, HDL_3-C, apo A-I, and TG levels or changes in $\dot{V}O_2$max or BFM in a group of athletes 6 weeks after detraining. We interpret these findings to indicate that for lipid changes to take place, detraining should be sufficiently long and complete to bring about a significant reduction in aerobic power and an increase in BFM. The plasma lipoprotein response to physical deconditioning might also depend on the athlete's training level at the moment training is interrupted. In a similar study, Sutherland et al[10] examined lipid-parameter behavior in a group of runners who decreased their training intensity over a period of 2 months, and observed an increase in LDL-C and VLDL-C, without any change in HDL-C; however, in this study detraining was only partial, $\dot{V}O_2$max was not monitored, and other factors interfering with lipid metabolism (such as diet during deconditioning and BFM behavior) were not taken into account.

Although the mechanism responsible for the lipoprotein changes observed during physical inactivity is not yet known, as advanced by Nikkilä et al[8] it is plausible that a reduction in tissue and plasma lipolytic activity takes place, with a reduced TG-rich lipoprotein turnover and a decrease in HDL formation. These changes would occur in a direction opposite to that during training.[38]

Conclusions

In both older and young individuals, an aerobic training program such as amateur cycling induces beneficial effects on the body composition, cardiovascular performance, and lipoprotein profile. Physical exercise seems able to reduce at least in part the differences existing between young and elderly subjects regarding the lipoprotein pattern. However, these effects are reversible, and the complete suspension of physical activity brings about unfavorable modifications in body composition and aerobic power in both age groups. Moreover, the lipid pattern is not stable, and a

2-month detraining period such as that followed at intervals by some athletes or dictated by disease or accident leads to a rapid regression of the training-induced favorable effects. The age of the subjects does not seem to influence the lipid response to physical deconditioning. The findings of the present study suggest that a long-term commitment to a training program is necessary to maintain an antiatherogenic lipoprotein profile over time.

REFERENCES

1. Goldberg L, Elliot DL: The effect of exercise on lipid metabolism in men and women. Sports Med 4:307-321, 1987
2. Schwartz RS: Effect of exercise training on high-density lipoproteins and apolipoprotein A-I in old and young men. Metabolism 37:1128-1133, 1988
3. Schwartz RS, Cain KC, Shuman WP, et al: Effect of intensive endurance training on lipoprotein profiles in young and older men. Metabolism 41:649-654, 1992
4. Seals DR, Hagberg JM, Hurley BF, et al: Effects of endurance training on glucose tolerance and plasma lipid levels in older men and women. JAMA 252:645-649, 1984
5. Weber F, Barnard RJ, Roy D: Effects of a high–complex-carbohydrate, low-fat diet and daily exercise on individuals 70 years of age and older. J Gerontol 38:155-161, 1983
6. Benfante R, Reed D: Is elevated serum cholesterol level a risk factor for coronary heart disease in the elderly? JAMA 263:393-402, 1990
7. Brown G, Albers JJ, Fisher LD, et al: Regression of coronary artery disease as result of intensive lipid-lowering therapy in men with high levels of apolipoprotein B. N Engl J Med 323:1289-1298, 1990
8. Nikkilä EA, Kuusi T, Myllynen P: High density lipoprotein and apolipoprotein A-I during physical inactivity. Atherosclerosis 37:457-462, 1980
9. Thompson CE, Thomas TR, Araujo J, et al: Response of HDL cholesterol, apoprotein A-I, and LCAT to exercise withdrawal. Atherosclerosis 54:65-73, 1985
10. Sutherland WHF, Woodhouse SP, Williamson S, et al: Decreased and continued physical activity and plasma lipoprotein lipids in previously trained men. Atherosclerosis 39:307-311, 1981
11. Durnin JVGA, Rahaman MM: The assessment of the amount of fat in the human body from measurements of skinfold thickness. Br J Nutr 21:681-689, 1967
12. Kishner RF, Schoeller DA: Estimation of total water by bioelectrical impedance analysis. Am J Clin Nutr 44:417-424, 1986
13. Van Loan MD: Bioelectrical impedance analysis to determine fat-free mass, total body water and body fat. Sports Med 10:205-217, 1990
14. Martin AD, Drinkwater DT: Variability in the measurement of body fat. Assumptions or technique? Sports Med 11:277-288, 1991
15. Fidanza F, Liguori G: Nutrizione umana. Naples, Italy, Ildelson, 1984, pp 677-732
16. Saltin B, Astrand PO: Maximal oxygen uptake in athletes. J Appl Physiol 23:353-358, 1967
17. Allain CC, Poon LS, Chan CSG, et al: Enzymatic determination of total serum cholesterol. Clin Chem 20:470-475, 1974
18. Wahlefeld AW: Triglycerides determination after enzymatic hydrolysis, in Bergmeyer HU (ed): Methods of Enzymatic Analysis. Weinheim, Germany, Verlag Chemie, 1974, pp 1831-1835
19. Havel RJ, Eder HA, Bragdon JH: The distribution and chemical composition of ultracentrifugally separated lipoproteins in human serum. J Clin Invest 34:1345-1353, 1955
20. Gidez LI, Miller GJ, Burstein M, et al: Separation and quantitation of subclasses of human plasma high density lipoproteins by a simple precipitation procedure. J Lipid Res 23:1206-1223, 1982
21. Jenrich R, Sampson P, Frane J: Analysis of variance and covariance including repeated measures, in Dixon W, Brown M, Engelman L, et al (eds): BMDP Statistical Software. Berkeley, CA, University of California, 1981, pp 359-387
22. Oldrige NB, Guyat GH, Fischer ME, et al: Cardiac rehabilitation after myocardial infarction: Combined experience of randomized clinical trials. JAMA 260:945-950, 1988
23. Powell KE, Thompson PD, Caspersen CJ, et al: Physical activity and the incidence of coronary heart disease. Annu Rev Public Health 8:253-287, 1987
24. Giada F, Baldo-Enzi G, Baiocchi MR, et al: Heparin-released plasma lipase activities, lipoprotein and apolipoprotein levels in young adult cyclists and sedentary men. Int J Sports Med 9:270-274, 1988
25. Giada F, Baldo-Enzi G, Baiocchi MR, et al: Specialized physical training programs: Effects on serum lipoprotein cholesterol, apoproteins A-I and B and lipolytic enzyme activities. J Sport Med Phys Fitness 31:196-203, 1991
26. Tran ZV, Weltman A, Glazes G: The effect of exercise on blood lipids and lipoproteins: A meta analysis of studies. Med Sci Sports Exerc 15:393-402, 1983
27. Donahue RP, Abbot RD, Bloom E, et al: Central obesity and coronary heart disease in men. Lancet 1:821-824, 1987
28. Berg A, Frey I, Keul J: Apolipoprotein profile in healthy males and its relation to maximum aerobic capacity (MAC). Clin Chim Acta 161:165-171, 1986
29. Di Minno G, Mancini M: Measuring plasma fibrinogen to predict stroke and myocardial infarction. Arteriosclerosis 10:1-7, 1990
30. Schneider S, Kim HC, Khachadurian AK, et al: Impaired fibrinolytic response to exercise in type II diabetes: Effects of exercise and physical training. Metabolism 37:924-929, 1988
31. Heiss G, Tamir I, Davis CE, et al: Lipoprotein cholesterol distributions in selected North America populations: The Lipid Research Clinics Program Prevalence Study. Circulation 61:302-315, 1980
32. Dedonder-Decoopman E, Fievei-Desreumaux L, Campos E, et al: Plasma levels of VLDL- and LDL-cholesterol, HDL-cholesterol, triglycerides and apoproteins B and A-I in an healthy population. Atherosclerosis 37:559-568, 1980
33. Takeuchi N, Matsumoto A, Katayama Y, et al: Changes with ageing in serum lipoproteins and apolipoprotein C subclasses. Arch Gerontol Geriatr 2:41-48, 1983
34. Williams P, Robinson D, Baily A: High density lipoprotein and coronary risk factors in normal men. Lancet 1:72-75, 1979
35. Frey I, Berg A, Baumstark MW, et al: Effects of age and physical performance on distribution and composition of high density lipoprotein subfractions in men. Eur J Appl Physiol 60:441-444, 1990
36. Deprès JP, Allard C, Trembay A, et al: Evidence for a regional component of body fatness in the association with serum lipids in men and women. Metabolism 34:967-973, 1985
37. Wood PD, Haskell W, Blair S, et al: Increased exercise level and plasma lipoprotein concentrations. Metabolism 32:31-39, 1983
38. Nikkilä EA, Taskinen MR, Rehnnen S, et al: Lipoprotein lipase activity in adipose tissue and skeletal muscles of runners: Relations to serum lipoproteins. Metabolism 27:1661-1671, 1978