Plasma lipid, lipoprotein and apolipoprotein profiles in Nigerian university athletes and non-athletes

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The fasting plasma lipid, lipoprotein and apolipoprotein profiles were determined in 14 healthy Nigerian male athletes and controls matched for sex and anthropometric parameters. The mean levels of total cholesterol ($P < 0.05$), low-density lipoprotein (LDL) cholesterol, apolipoprotein (apo) AII and E were significantly lower ($P < 0.01$) in the athletes than in the controls. However, there were no statistically significant differences ($P > 0.05$) between the mean values of the plasma triglycerides, high-density lipoprotein (HDL), very low-density lipoprotein (VLDL) cholesterol, apo AI, B, Lp(a), LpA1 and CIII:NonB respectively for the athletes and controls. *A priori*, the potential effect on cardiovascular disease (CVD) risk was also compared using three predictor ratios – total cholesterol: HDL cholesterol (TC:HDL), LDL cholesterol: HDL cholesterol and apo B:AI. The mean of the three ratios was lower in the athletes than in the controls; however, the differences were not statistically significant ($P > 0.05$). Based on our data, exercise appears to decrease the TC:HDL ratio in the athletes by lowering LDL-cholesterol, while the HDL-cholesterol is unaffected. We conclude that physical activity has salutary effects on the lipid, lipoprotein and apolipoprotein profiles of healthy Nigerian men.

Keywords: Physical activity, lipids, lipoproteins, apolipoproteins, cardiovascular disease

Research interest in lipids and lipoprotein metabolism has increased due to the establishment of the roles played by lipids, lipoproteins and apolipoproteins in the development of cardiovascular disease (CVD)[1–5]. Plasma lipid and lipoprotein levels have been shown to be influenced by age, sex, socioeconomic status, genetics, race, diet, cigarette smoking, coffee and alcohol intake, and medication as well as habitual and leisure time physical activity[6–11].

Increased physical activity has been reported to produce favourable changes in the lipid and lipoprotein profiles[12–15]. These changes are also influenced by sex, diet, intensity of exercise, body weight and percentage body fat[8,15–19].

In black Africans, there is a relatively lower level of risk factors and incidence of CVD when compared with Caucasians and other Blacks in industrialized countries[20–22]. Less severe atherosclerotic lesions of the aorta, coronary and cerebral arteries have also been reported in black Africans[23–26]. There are presently few published reports on lipids in black athletes[27,28]. These studies examined the effect of exercise on body fats, cholesterol and triglyceride. Therefore, we examine in detail the plasma lipid, lipoprotein and apolipoprotein profiles, and three CVD predictor ratios[1–4] in some Nigerian athletes and a matched control group.

Subjects and methods

Fourteen healthy male athletes (mean(s.d.) age 22(4); range 18–28 years) at the Institute of Physical Education, Obafemi Awolowo University, Ile-Ife, Nigeria and 14 healthy non-athletes (mean age 24(5); range 18–31 years) at the same university served as controls. All participants gave their consent before the study. The athletes were all well trained, élite short/medium distance (100, 200 or 400 m) runners of the university team, and they had been running for 4–8 years. They had been undergoing moderate physical training (running 5–10 km) 3–5 days per week for about 4 weeks before the study.

The athletes and the control group were matched ($P > 0.05$) for age, weight, height, body mass index (BMI) determined by a nomograph[29], and systolic and diastolic blood pressure (*Table 1*). The subjects were all non-smokers and were not taking any medication at the time of study. They were all on the same university regular diet, and were also told to abstain from alcoholic beverages for at least 2 weeks before sampling.

Table 1. Characteristics of the athletes and controls

	Athletes	*Control*	*P*
Age (years)	22(4)	24(5)	n.s.*
Weight (kg)	65(9)	61(6)	n.s.
Height (cm)	172(9)	168(8)	n.s.
Body mass index ($kg\,m^{-2}$)	22(2)	22(3)	n.s.
Blood pressure (mmHg)			
Systolic	117(8)	120(13)	n.s.
Diastolic	79(11)	79(6)	n.s.

Values are mean(s.d.); *n.s., Not statistically significant ($P > 0.05$)

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Collection of blood samples

Fasting blood samples (10–12 h after the last meal) were collected from each subject (about 12 h after physical exercise in the athletes) into sodium-EDTA anticoagulant. The plasma was separated within 2 h of collection in a refrigerated centrifuge (Damon/IEC B-20A) precooled to 4°C at 1500 *g* for 15 min, and stored at −80°C before analysis.

Plasma lipids and lipoprotein assay

Plasma total cholesterol and HDL-cholesterol and triglycerides were analysed by an automated micro-enzymatic procedure on a Hitachi 737 chemistry analyser as described by Steiner *et al.*[30] at the Medical Research Laboratories, Cincinnati, Ohio, USA. The laboratory maintained CDC-NHLBI Part 3 Standardization[31] for all three lipid parameters. HDL was isolated using the modified heparin-2M $MnCl_2$ procedure[32]. LDL-cholesterol was calculated by the Friedewald equation[33].

Plasma apolipoprotein assay

The apolipoproteins (apo) AI, AII, B and E were analysed using competitive enzyme-linked immunoabsorbent assay (ELISA) procedures utilizing a monoclonal antibody against each apolipoprotein[34–36]. Apo Lp(a) was also determined by competitve ELISA using a monospecific rabbit antiserum to Lp(a)[37].

Plasma lipoprotein particle assay

Lipoprotein particles consisting of apo AI only (LpAI) were determined by the differential electroimmunoassay (EIA) technique[38] using a Hydragel LpAI kit (Sebia, Issy-les-Moulineaux, France). Total apo CIII was also measured by EIA[39] with a Hydragel LpCIII kit (Sebia, Issy-les-Moulineaux, France). The amount of plasma apo CIII not associated with apo B containing apoproteins (LpCIII:NonB) was measured by the same EIA for total LpCIII after the precipitation of apo B containing lipoproteins with a specific apo B antiserum. LpCIII:B was calculated as the difference between total LpCIII and CIII:NonB.

Internal quality assurance was monitored throughout the study using frozen pools at a minimum of two levels for each apolipoprotein. External quality assurance was assessed by the laboratory's participation in the CDC-IUIS apolipoprotein surveys for AI, B and Lp(a)[40, 41].

Statistical analysis

All the data are expressed as mean(s.d.). The data of the athletes and controls were compared by employing a two-tailed independent *t* test.

Results

As shown in *Table 2*, the mean plasma total cholesterol ($P < 0.05$), LDL-cholesterol, apo AII and E were significantly lower ($P < 0.01$) in the athletes than in the controls. The means of plasma CIII ($P < 0.05$) and CIII:B ($P < 0.01$) were significantly higher in the athletes than the controls, while the mean values of plasma triglycerides, HDL-cholesterol, apo AI, B, Lp(a), LpAI and CIII:NonB in the athletes were not significantly different ($P > 0.05$) from those of the controls.

Furthermore, the means of TC:HDL, LDL-cholesterol: HDL-cholesterol, and apo B:AI, respectively, were also lower than those of the controls. However, the differences were not statistically significant ($P > 0.05$).

Table 2. Fasting plasma lipid, lipoprotein and apolipoprotein levels in male athletes and controls

	Athletes	*Controls*	*P*
Lipids and lipoproteins (mg dl^{-1})			
Total cholesterol	107(20.3)	129(24.1)	<0.05
Triglyceride	66(13.7)	58(24.1)	n.s.*
HDL-cholesterol	44(13.7)	45(11.0)	n.s.
VLDL-cholesterol	13(2.7)	12(4.8)	n.s.
LDL-cholesterol	49(20.0)	73(22.0)	<0.01
Apolipoproteins (mg dl^{-1})			
AI	149(44.1)	130(23.4)	n.s.
AII	20(6.5)	38(9.8)	<0.01
B	69(12.8)	70(13.4)	n.s.
Lp(a)	12(6.7)	15(10.0)	n.s.
E	2(1.3)	5(2.1)	<0.01
LpAI	43(11.0)	38(5.9)	n.s.
CIII	20(5.9)	16(3.9)	<0.05
CIII:B	11(5.6)	6(2.6)	<0.01
CIII:NonB	9(5.6)	10(4.1)	n.s.
CVD Risk indices			
Total cholesterol: HDL-cholesterol	2.61(0.78)	3.05(0.95)	n.s.
LDL-cholesterol: HDL-cholesterol	1.28(0.70)	1.76(0.78)	n.s.
Apo B:AI	0.49(0.15)	0.56(0.15)	n.s.

Values are mean(s.d.); *n.s., Not statistically significant ($P > 0.05$)

Discussion

The roles played by the various lipid, lipoprotein and apolipoprotein fractions in the development of coronary heart disease have been documented in the literature. However, few reports[27, 28] are available on this subject in black athletes. The present study is, therefore, important. It shows that the levels of antiatherogenic apo AI and LpAI, and the atherogenic lipids total and LDL-cholesterol, apo B and Lp(a) were favourable in the athletes, an indication that physical activity may be associated with favourable changes in the lipid and apolipoprotein profiles of young and healthy Nigerian athletes. This observation is similar to an earlier finding in male untrained Nigerian university students[28] and some other reports on Caucasians[8, 12–14, 20].

In people engaged in training programmes, both acute responses and chronic adaptation may contribute to the respective lipoprotein profiles[42]. Physical training has been shown to produce favourable changes in the lipid and lipoprotein profiles[12–15]; some reports, however, did not show apparent changes, most especially in women[16, 17, 43]. This has been attributed to changes in endogenous sex

hormones during the exercise training periods and also probably because premenopausal women start with a higher pretraining HDL cholesterol level than men[44].

The inverse association between physical activity and CVD incidence appears to relate at least in part to the effect on HDL cholesterol[8, 13, 16, 20, 46]. In men, increased physical activity usually results in an increase in HDL level[16, 20, 45, 46], some researchers, however, found no change in HDL level[47, 48]. This may be due to changes in the distribution of HDL subfractions without alteration of total HDL concentration, or to differences in experimental design. In this study, there was also no apparent difference between the mean HDL level in the male athletes when compared with the controls. This observation may be due to the fact that the athletes are mostly involved in anaerobic sports. The LDL-cholesterol fraction was, however, significantly lower ($P < 0.01$) in the athletes.

The mean total triglycerides in athletes in this study tended to be higher, although non-significantly, than the control value. This may also be due to the reported acute nature of the triglyceride lowering effect of exercise, moreover, the pre-exercise triglyceride level tends to influence its response to exercise[8].

Overall, it is expected that the athletes will be more physically fit than the controls. Using the CVD predictor ratios TC:HDL, LDL-cholesterol: HDL-cholesterol, and apo B:AI, the athletes showed a favourable, but statistically insignificant, decreased risk of CVD when compared with the controls. It thus appears that exercise decreases TC:HDL ratios in Nigerians by lowering LDL-cholesterol. In Caucasians, exercise tends to lower TC:HDL ratios by raising HDL-cholesterol[13, 15, 20, 45, 46], as well as lowering LDL-cholesterol[9, 12, 16].

While a cause and effect conclusion cannot be drawn from our present data, our findings revealed that regular exercise may be associated with desirable lipid, lipoprotein and apolipoprotein profiles in Nigerian athletes. The antiatherogenic apo AI and LpAI and the atherogenic total and LDL-cholesterol, apo B, Lp(a) were favourably affected. Furthermore, the CVD risk predictor ratios: TC:HDL, LDL-cholesterol:HDL-cholesterol, and apo B:AI showed an insignificant decrease in the athletes. Based on our findings, we conclude that increased physical activity level may reduce CVD risk in healthy Nigerians.

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