Eur J Appl Physiol (1984) 53: 253–259

European Journal of Applied Physiology and Occupational Physiology
© Springer-Verlag 1984

Glucose tolerance, plasma lipoproteins and tissue lipoprotein lipase activities in body builders

H. Yki-Järvinen, VA. Koivisto, M-R. Taskinen, and EA. Nikkilä

Third Department of Medicine, University Central Hospital, Haartmaninkatu 4, SF-00290 Helsinki 29, Finland

Summary. Oral glucose tolerance, insulin binding to erythrocyte receptors, serum lipids, and lipoproteins, and lipoprotein lipase activities of adipose tissue and skeletal muscle were measured in nine body builders (relative body weight (RBW) 118 ± 4%), eight weight-matched (RBW 120 ± 5%) and seven normal-weight controls (RBW 111 ± 3%). The body builders had 50% higher relative muscle mass of body weight (% muscle) and 50% smaller relative body fat content (% fat) than the two other groups ($P < 0.005$). Maximal aerobic power was comparable in the three groups. In the oral glucose tolerance test (OGTT), blood glucose levels, and plasma insulin levels were lower ($P < 0.05$) in the body builders than in weight-matched controls. Insulin binding to erythrocytes was similar in each group. On the basis of multiple linear regression analysis, 87% of the variation in plasma insulin response could be explained by body composition (% muscle and % fat) and $V_{O_2\,max}$.

Plasma total cholesterol, low-density lipoprotein (LDL) cholesterol, and very low-density lipoprotein (VLDL) triglyceride concentrations were significantly lower in the body builders than in weight-matched controls. In comparison with the normal-weight group, the body builders had a lower total cholesterol level. High density lipoprotein (HDL) cholesterol, its subfractions (HDL_2 and HDL_3 cholesterol) and lipoprotein lipase (LPL) activities of adipose tissue and skeletal muscle were comparable in all three groups. Partial correlation analysis showed a positive relationship between plasma total triglyceride, total cholesterol and LDL cholesterol on the other hand and the % fat on the other.

The results indicate that a shift in body composition from the adipose to the muscular type is associated with 1) lower glucose and insulin levels during the OGTT and 2) decrease in total and VLDL triglyceride and in total and LDL cholesterol levels but unchanged HDL cholesterol level. Thus, body builders are characterized by some metabolic features which decrease the risk of coronary heart disease. In contrast to aerobic training, body building does not influence HDL or its subfractions.

Key words: Insulin sensitivity – Insulin receptors – Glucose tolerance – Lipoproteins – Lipoproteins – Lipoprotein lipase

Introduction

Aerobic training is associated with several alterations in both carbohydrate and lipid metabolism. Insulin response to both oral (Björntorp et al. 1972) and intravenous (Lohmann et al. 1978) glucose challenge is diminished in the face of normal glucose tolerance, indicating enhanced body sensitivity to insulin (Soman et al. 1979). Regarding lipid metabolism, serum triglyceride and total cholesterol levels are normal or reduced, while HDL cholesterol increase (for review see Dufaux et al. 1982). This is associated with elevated liprotein lipase (LPL) activity in both adipose tissue and skeletal muscle (Nikkilä et al. 1978b). Since aerobic training results not only in an increased aerobic capacity but also in a decrease of body fat and lean body mass, it is not clear whether all the changes are solely due to increased maximal aerobic power (Glueck et al. 1980; Stern et al. 1973). Body builders train primarily by heavy resistance exercise to increase their muscle mass and to decrease body fat whereas aerobic capacity remains unaltered (Pipes 1979). Studies on lipid and lipoprotein metabolism in anaerobically trained altheletes have yielded conflicting results: serum triclyceride as well as total and LDL-cholesterol levels have been reported to be either normal (Campbell 1965; Clarkson et al. 1981;

Offprint requests to: H. Yki-Järvinen at the above address

Nikkilä et al. 1978b) or decreased (Lopez-S et al. 1980). Similarly, both normal (Clarkson et al. 1981; Nikkilä et al. 1978b) or reduced (Berg et al. 1980; Lehtonen and Viikari 1980; Schnabel et al. 1980) HDL cholesterol levels have been observed. The influence of anaerobic training on tissue LPL activities has not been studied previously. Moreover, no studies are avaible on glucose metabolism in body builders.

The present study was carried out to characterize the insulin response to oral glucose as well as lipid and lipoprotein metabolism in well-trained body builders with enlarged muscle mass but normal aerobic capacity.

Subjects and methods

Three groups of subject were studied. The first consisted of nine body builders, whose relative body weight (Table 1) varied between 101% and 133% of ideal. Each of them had been engaged in body building for more than 2 years at least four to five times per week. The second group (weight-matched controls) consisted of eight men matched for relative body weight and maximal aerobic power ($V_{O_2\,max}$, Table 1). A third group of normal-weight controls (IBW < 120%) matched for height and $V_{O_2\,max}$ was also studied (normal-weight controls). None of the subjects was taking any medications at least 3 week before the studies. None of the body builders had ever previously used anabolic steroids. The body builders had a low carbohydrate (10–20%), high protein (40–50%) diet with reduced salt intake for 2–3 months prior to competition. Following the competition, they changed gradually to normal diet consisting of 40–45% carbohydrate, 15–20% protein and 40–45% fat. Each of the subject had ingested the normal diet for at least 2 months before the study. Drinking and smoking habits were comparable between the groups. For 2 days before the study, the subjects ingested a weight-mainting diet containing 200–250 g carbohydrate per day. The nature, purpose, and possible risks of the study were explained to all subjects before they gave their voluntary consent to participate. The subjects were studied on 3 separate days. On the 2 first days they were studied after a 12-h overnight fast. On the first visit, and oral glucose tolerance test and anthropometric measurements were performed and blood was withdrawn for insulin binding studies. On the second morning, blood was taken for lipid analyses. Thereafter, needle biopsy specimens of adipose tissue and skeletal muscle were taken for the measurement of LPL activities. Maximal aerobic power ($V_{O_2\,max}$) was measured on the third study occasion.

Oral glucose tolerance test. Oral glucose (75 g) tolerance test was performed according to standard recommendations (WHO Expert Committee on Diabetes Mellitus, 1980). Blood samples for the determination of blood glucose and plasma insulin levels were withdrawn at 0, 15, 30, 60, 90, and 120 min.

Insulin binding to red cells. Insulin binding to erythrocytes was determined by the method of Gambhir et al. (1977). Blood was withdrawn in a heparinized tube and processed immediately for binding studies. The uppermost 5–10% of the erythrocyte pellet containing some granulocytes and reticulocytes was removed after centrifugations in the Ficoll-Paque (Pharacia, Uppsala, Sweden) gradient. The final erythrocyte suspension contained less than 0.2% of reticulocytes. The red cells were incubated for 210 min at 15° C in Hepes buffer (50 mmol · l^{-1}, pH 8.0) with A_{14}-labeled (^{125}I)-insulin (SA 1,200 μCi · $nmol^{-1}$, Novo Research Institute, Copenhagen, Denmark) at a final concentration of 0.035 nmol · l^{-1} with and without increasing amounts of unlabeled insulin. Nonspecific binding was determined as the amount of (^{125}I)-insulin bound in the presence of 17 μmol · l^{-1} unlabeled insulin. Insulin binding to erythrocytes was expressed as the percentage of specific binding per $3.52 \times 10^9 \pm 0.1 \times 10^9$ cells/ml.

Separation of lipoproteins. The lipoproteins were isolated by sequential flotation in a Beckman Model L 70 ultracentrifuge (Beckman Instruments, Inc., Palo Alto, CA, USA) using type 50.3 Ti Beckman rotor. VLDL was separated by spinning serum at a density of 1.006 g · ml^{-1} for 18 h at 38,000 RPM. The top layer (= VLDL) was removed by tube slicing and density of the infranatant was adjusted to 1.063 g · ml^{-1} with a KBr (354 g · l^{-1}) + NaCl (153 g · l^{-1}) solution and the LDL was flotated to surface by spinning for 24 h at 38,000 RPM. The density of the LDL infranatant was raised to 1.125 g · ml^{-1} with the same KBr-NaCl mixture and the HDL_2 was separated by centrifugation at 38,000 RPM for 65 h. After tube slicing the density of the bottom fraction was adjusted to 1.210 g · ml^{-1} with the KBr-NaCl mixture and the HDL_3 was flotated to surface by spinning for 65 h

Table 1. Physical characteristics of the subjects

	Body builders	Weight-matched controls	Normal weight controls
Number of subjects	9	8	8
Age (years)	24 ± 2	27 ± 2	25 ± 2
Weight (kg)	83 ± 3*	85 ± 5	76 ± 2
Height (cm)	179 ± 3	179 ± 2	177 ± 1
Relative body weight (%)**	118 ± 4	120 ± 5	111 ± 3
Absolute muscle mass (kg)	51 ± 4***	34 ± 2	30 ± 1
% Muscle	61 ± 3***	41 ± 1	39 ± 1
Absolute fat mass (kg)	4 ± 1****	9 ± 1	8 ± 1
% Fat	5 ± 1***	10 ± 1	10 ± 1
$V_{O_2\,max}$ (ml · kg^{-1} · min^{-1})	53 ± 3	52 ± 6	54 ± 3

* $P < 0{,}05$ versus normal weight controls
** Metropolitan Life Insurance Tables, 1958. Documenta Geigy
*** $P < 0.005$ versus both control groups
**** $P < 0.005$ versus weight-matched controls; $P < 0.05$ versus normal-weight controls

at 35,000 RPM. After isolation the four lipoprotein fractions were dialyzed overnight against 0.15 M NaCl at + 4° C and their cholesterol and triglyceride content were analyzed. The recovery of cholesterol and triglyceride in isolated lipoprotein fractions averaged 92 ± 2% and 97 ± 2% (mean ± SEM), respectively. The concentration of cholesterol and triglyceride in lipoprotein fractions were corrected according to recovery percent.

Assay of lipoprotein lipase activity. Heparin releasable LPL activity was measured from needle biopsy tissue specimens. Subcutaneous adipose tissue was taken by aspiration from the gluteal region. Skeletal muscle was biopsied at about the middle point of lateral part of *M. quadriceps* using a Tru-Cut (Travenol) needle. The skin at both biopsy sites was anesthetized with 1% lidocain. The specimens were taken into saline at room temperature, washed, blotted, weighed, and immediately used for assay of LPL activity. Enzyme activity was measured from heparin eluates of tissue labeled triolein emulsion as substrate (Taskinen et al. 1980). The activity is expressed as micromoles of NEFA released from the substrate in 1 h.

Estimation of body muscle mass. The muscle area of the upper extremity (AMA) was estimated from measures of upper arm circumference (AC, mm) and triceps skinfold (TSF, mm). Skinfold thicknesses were determined using Harpenden calipers (M-13, John Bull, Britsh Indicators Ltd., United Kingdom). All measurements were done by the same person in triplicate on the dominant side of the body. AMA was calculated according to the formula of Jelliffe et al. (1969) as follows: AMA (cm^2), for men $=(AC\text{-}\pi TSF)^2 \cdot (4\pi)^{-1}\text{-}10$. Total body muscle mass was estimated according to the formula of Heymsfield et al. (1982): total body muscle mass (TBM, kg) = height (0.0264 + 0.0029 × AMA). The formula is based on correlation between AMA and 24-h urinary creatinine excretion [14]. Percentage of body weight (BW) made up of muscle tissue (% muscle) was calculated by dividing TBM by BW (× 100).

Estimation of body fat. Percentage of BW made up of fat tissue (% fat) was estimated from the sum of six skinfolds measured from triceps. Subscapular, pectoral, umbilical, suprailiac, and thigh regions. The values of six skinfolds were summed up and 8 mm was subtracted to correct for the nonadipose content of the skinfolds. The corrected sum of skinfolds was divided by BW to obtain skinfold/BW ratio (R). The relative amount of fat was calculated from the formula: % fat = 11.543 × R-0.2838 (Anayan 1978).

Maximal aerobic power ($V_{O_2\,max}$). The maximal aerobic power ($V_{O_2\,max}$) was determined directly by a standard incremental exercise test on a cycle ergometer (Saltin and Åstrand 1967).

Analytic procedures. Blood glucose was determined by the O-toluidine method (Hyvärinen and Nikkilä 1962) and plasma insulin by radioimmunoassay (Desbuquois and Aurbach 1971). The concentration of cholesterol was determined by an enzymatic method using a commercial reagent kit (187313 of Boehringer Diagnostica, Mannheim GmbH, FRG). Triglycerides were measured by Autoanalyzer using kit 297771 of Boehringer Diagnostica (Kessler and Lederer 1965).

Data analysis. Statistical comparisons of the groups were calculated by analysis of variance and Student's unpaired *t*-test using Biomedical Data Processing (BMDP) programs 7D and 3D, respectively (Dixon 1981). Multiple linear regression analysis, simple, and partial correlation coefficients, and coefficients of variation were calculated using BMDP computer programs 1R, 8D and 2R. All data are expressed as mean ± SEM.

Results

Oral glucose tolerance

Both blood glucose and plasma insulin responses during the OGTT were lower in the body builders than in their weight-matched controls (Figs. 1A and 2A). Compared to the normal-weight group the body builders showed a lower plasma insulin level at 60 min after glucose ingestion (Fig. 2B). To evaluate the separate contributions of the % body fat, the % body muscle and the $V_{O_2\,max}$ on glucose and insulin responses, partial correlation coefficients were calculated. This analysis indicated that only $V_{O_2\,max}$ was significantly related to the plasma insulin response (Table 2). When the combined effects of the % fat, % muscle, and $V_{O_2\,max}$ were analyzed by multiple linear regression analysis, it appeared that these three

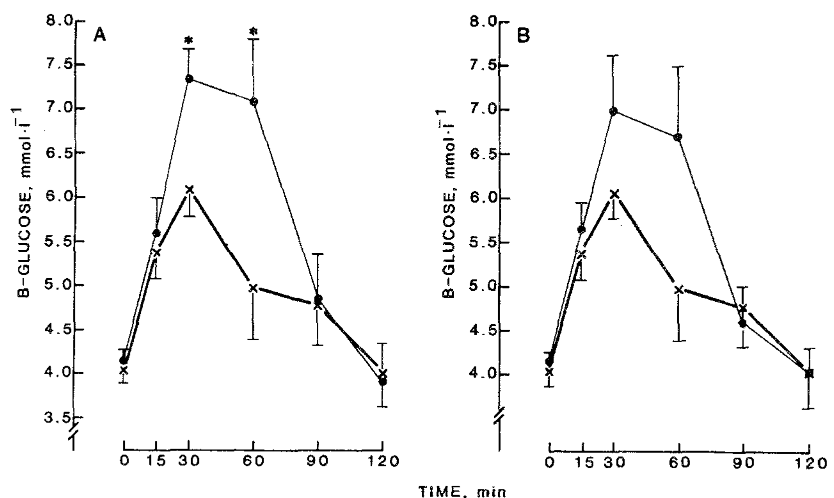

Fig. 1. Blood glucose values during the OGTT in : **A** Body builders (×) vs weight-matched controls (●), **B** body builders (×) vs normal-weight controls (●). * $P < 0.05$

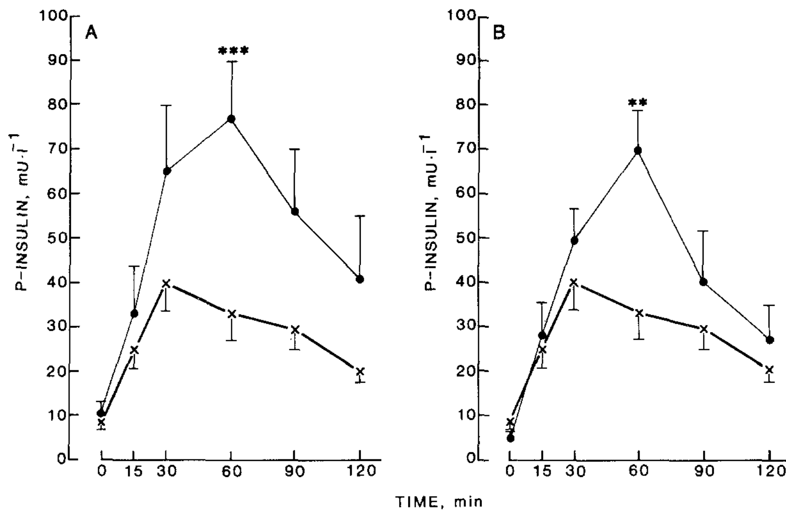

Fig. 2. Plama insulin values during the OGTT in: **A** Body builders (×) vs weight-matched controls (●), **B** body builders (×) vs normal-weight controls (●). ** $P < 0.01$ *** $P < 0.001$

Table 2. Partial correlation coefficients between physical characteristics (% fat, % muscle, $V_{O_2\,max}$) and variables of glucose, lipid, and lipoprotein metabolism in the whole group ($n = 25$)

Variable	% fat	% Muscle	$V_{O_2\,max}$	Multiple R	R^2
Blood glucose (at 60 min)	0.514	−0.319	0.190	0.747	56%
Plasma insulin (at 60 min)	0.459	−0.290	−0.421*	0.930***	87%
Total triglyceride	0.965**	0.483	0.200	0.574	33%
VLDL triglyceride	0.795	0.272	0.182	0.543	30%
Total cholesterol	0.949**	0.341	−0.196	0.822***	68%
LDL cholesterol	0.855*	0.282	−0.280	0.821***	67%

* $P < 0.05$; ** $P < 0.01$; *** $P < 0.005$; **** $P < 0.001$

factors explained 87% of the variation in the plasma insulin response ($P < 0.001$).

Insulin binding to erythrocytes

The percentage of ^{125}-insulin bound to erythrocytes was similar in all groups at each insulin concentration. The percentage of insulin bound at insulin tracer concentration averaged $23 \pm 1\%$, $24 \pm 3\%$, and $24 \pm 3\%$ in the body builders, weight-matched controls, and normal-weight controls, respectively.

Serum lipids and lipoproteins

Total triglyceride was lowest in the group of body builders (Table 3). The difference was mainly due to VLDL triglycerides which were 58% ($P < 0.02$) and 41% ($P < 0.05$) lower in body builders than in weight-matched controls and normal-weight controls, respectively. Partial correlation analysis indicated that total and VLDL triglyceride were related to the % fat but not to the % muscle or the $V_{O_2\,max}$ (Table 2). Total cholesterol was reduced by 24% ($P < 0.02$) and 21% ($P < 0.02$) in the body builders as compared to the weight-matched controls and normal-weight controls, respectively. This difference was most pronounced in LDL-cholesterol (Table 3). Both total and LDL cholesterol were related to the % fat but not to the % muscle or to $V_{O_2\,max}$ (Table 2). No significant differences were found in the cholesterol levels or in its subfraction HDL_2 and HDL_3 cholesterol, between the three groups (Table 3). Correlation analysis of fasting and post-glucose insulin levels and serum lipoproteins indicated that VLDL-triglyceride was directly related to plasma insulin levels at 60 min ($r = 0.47$, $P < 0.05$).

Tissue lipoprotein lipase activities

No significant differences in adipose tissue LPL activities between the three groups were found when the activity was expressed per tissue weight or per total body fat (Table 3). Similarly, LPL activities in skeletal muscle were comparable when calculated per tissue weight or per total body muscle (Table 3).

Table 3. Triglyceride and cholesterol concentrations in whole serum and in lipoprotein fractions

	Body builders	Weight-matched controls	Normal weight controls
Triglyceride ($mmol \cdot l^{-1}$)			
Total	1.01 ± 0.13	1.54 ± 0.35	1.23 ± 0.13
VLDL	0.39 ± 0.07*	0.92 ± 0.28	0.66 ± 0.14
LDL	0.27 ± 0.05	0.31 ± 0.03	0.30 ± 0.02
HDL	0.36 ± 0.04	0.31 ± 0.03	0.27 ± 0.02
HDL_2	0.18 ± 0.02	0.16 ± 0.03	0.13 ± 0.02
HDL_3	0.18 ± 0.02	0.15 ± 0.02	0.14 ± 0.02
Cholesterol ($mmol \cdot l^{-1}$)			
Total	3.69 ± 0.27**	4.84 ± 0.40	4.65 ± 0.18
VLDL	0.19 ± 0.04*	0.41 ± 0.11	0.28 ± 0.05
LDL	2.48 ± 0.23	3.27 ± 0.34	3.28 ± 0.05
HDL	1.02 ± 0.07	1.16 ± 0.07	1.09 ± 0.09
HDL_2	0.46 ± 0.08	0.56 ± 0.06	0.54 ± 0.06
HDL_3	0.56 ± 0.06	0.60 ± 0.07	0.55 ± 0.05

Results are expressed as mean ± SEM. * $P < 0.05$ versus weight-matched controls, ** $P < 0.02$ versus weight-matched controls; $P < 0.05$ versus normal weight controls

Table 4. Heparin-releasable lipoprotein lipase activities of adipose tissue and skeletal muscle in body builders and controls

	Body builders	Weight-matched controls	Normal weight controls
Adipose tissue LPL activity			
Per weight (μmol NEFA $\cdot g^{-1} \cdot h^{-1}$)	1.39 ± 0.29	0.85 ± 0.3	1.31 ± 0.34
Per total body fat (mmol NEFA $\cdot h^{-1}$)	5.8 ± 0.9	8.8 ± 2.6	10.3 ± 3.0
Skeletal muscle LPL activity			
Per weight (μmol NEFA $\cdot g^{-1} \cdot h^{-1}$)	0.40 ± 0.06	0.39 ± 0.07	0.56 ± 0.10
Per total body muscle (mmol NEFA $\cdot h^{-1}$)	18.6 ± 3.6	12.7 ± 2.8	19.5 ± 4.0

Results are expressed as mean ± SEM

Discussion

In this study, some aspects of carbohydrate and lipid metabolism were determined in relation to body composition in a group of body builders and in two control groups. The first control group was selected to match for total body weight and for $V_{O2\,max}$. Since this group included some overweight subjects, a second control group was studied to abolish the possible interference of obesity on the results.

In the oral glucose tolerance test, both glucose and insulin responses were lower in the body builders than in their weight-matched controls. When compared to the normal-weight group, only the insulin response was lower in the body builders. The glucose levels tended to be also in the normal-weight group, but the difference was not statistically significant. Regarding the mechanism of the lower insulin response in body builders, several possibilities should be considered. First, hepatic uptake of glucose could be greater in body builders as compared to controls. This would result in a lower blood glucose response and a reduced stimulus for insulin secretion. We are not aware of any data on hepatic glucose uptake in body builders and since we did not measure glucose kinetics, this possibility cannot be excluded. Second, peripheral glucose uptake could be increased in body builders. Recent data indicate that 60–75% of oral glucose is primarily taken up by peripheral tissue, mainly muscle (Katz et al. 1983). The absolute muscle mass was 1.5–1.7 fold greater in the body builders than in the two controls groups. Consequently, the lower glucose levels could be, at least partly, due to greater peripheral glucose uptake by an increased mass of muscle tissue. Whether there are qualitative differences in the muscle tissue to accelerate glucose uptake, has not been measured directly. However, ultrastructural studies indicate reduced rather than increased oxidative capacity of muscle fibers in body builders (MacDougall et al. 1982; Prince et al. 1976), and in contrast to aerobically trained athletes in whom insulin binding to blood cells is increased (Soman et al. 1979), insulin binding in body builders was not different from that in controls. Furthermore, we have previously shown that during intravenously maintained hyperinsulinemia, the rate of glucose

disposal is enhanced in aerobically but not in anaerobically trained subjects when expressed per muscle weight (Yki-Järvinen and Koivisto 1983). These observations make qualitative differences in muscle tissue unlikely. The lower glucose level could, in turn, result in a reduced insulin response.

VLDL triglyceride was significantly reduced in the body builders as compared to the weight-matched control group. This was an expected finding since both plasma total and VLDL triglyceride levels are commonly elevated in obese overweight subjects and their concentration is directly related to adiposity (Salans and Wise 1970; Olefsky et al. 1974). In accordance with this, both total and VLDL triglyceride were directly related to the % body fat also in this study. The rate of hepatic triglyceride synthesis increases in response to an elevated insulin concentration (Olefsky et al. 1974). In the present group of subjects, a positive relationship was noted between the plasma insulin response and the level of VLDL triglycerides. These data suggest that the decrease in VLDL triglyceride in the body builders could be mediated by decreased hepatic triglyceride synthesis due to low plasma insulin concentration. Decreased VLDL production with normal VLDL conversion to LDL could also partly account for the reduction in total and LDL cholesterol in the body builders.

In aeribically trained athletes, LPL activity in skeletal muscle and in adipose tissue is increased (Nikkilä et al. 1978b). The increase in HDL cholesterol which occurs mainly in the HDL_2, subfraction by endurance training (Krauss et al. 1977), is thought to be the consequence of an adaptive increase in LPL, which generates HDL_2 cholesterol by hydrolyzing triglyceride-rich lipoproteins (Nikkilä et al. 1978a). In the present study, HDL cholesterol as well as HDL_2 and HDL_3 cholesterol levels were unaltered in body builders similarly as reported by others for HDL cholesterol (Clarkson et al. 1981; Nikkilä et al. 1978b). Berg et al. (1980) found lower HDL cholesterol levels in anaerobically trained subjects but this could have been due to use of anabolic steroids which decrease HDL cholesterol (Solyom 1972). In keeping with normal HDL cholesterol levels, LPL activities in both skeletal muscle and adipose tissue were unchanged in the body builders as compared to the control groups.

In conclusion, plasma glucose and insulin levels after oral glucose are reduced in body builders as compared to weight-matched controls. This is most likely a consequence of increased muscle mass. Reduced insulinemia and a low relative amount of fat in body builders are associated with a decrease in VLDL triglyceride and LDL cholesterol. Thus, body builders are characterized by some metabolic features known to decrease the risk for coronary heart disease. However, anaerobic exercise dose not affect the antiatherogenic HDL_2 cholesterol subfraction or the enzyme catalyzing its formation in peripheral tissues.

Acknowledgements. The help of Sirkka-Liisa Karonen, Ph. D. for insulin determinations; Risto Elovainio, M. D. for the measurement of $V_{O_2\,max}$ and the skillful technical assistance of Miss Anne Salo, Miss Paula Teräväinen and Mrs Sirkka-Liisa Runeberg are gratefully acknowledged. This study was supported by grants from the Finnish State Medical Research Council (Academy of Finland), the Nordisk Insulinfond (Gentofte, Denmark), the Finnish Ministry of Education and the Finnish Diabetes Association.

References

Anyan WR, Jr (1978) Adolescent medicine in Primary care. John Wiley et Sons, New York, p 137

Berg A, Keul J, Ringwald G, Deus B, Wybitul K (1980) Physical performance and serum cholesterol fractions in healthy young men. Clin Chim Acta 106: 325–330

Björntorp P, Fahlen M, Grimby G, Gustafson A, Holm J, Renström P, Schersten T (1972) Carbohydrate and lipid metabolism in middle-aged physically well-trained men. Metabolism 21: 1037–1044

Campbell DE (1965) Influence of several physical activities on serum cholesterol concentrations in young men. Lipid Res 6: 478–480

Clarkson PM, Hintermeister R, Fillyow M, Stylos L (1981) High-density lipoprotein cholesterol in young adult weight lifters, runners and untrained subjects. Human Biol 53: 251–257

Desbuquois B, Aurbach GD (1971) Use of polyethylene glycol to separate free and antibody-bound peptide hormones in radioimmunoassay. J Clin Endocrinol Metab 33: 732–738

Dixon WJ (1981) BMDP Statistical software. University of California Press, Berkeley

Dufaux B, Assmann G, Hollmann W (1982) Plasma lipoproteins and physical activity: A review. Int J Sports Med 3: 123–136

Gambhir KK, Archer JA, Carter L (1977) Insulin radioreceptor assay for human erythrocytes. Clin Chem 23: 1590–1595

Glueck CJ, Tylor HL, Jacobs D, Morrison JA, Breaglehole R, Williams OD (1980) Plasma high density lipoprotein cholesterol: association with measurements of body mass. Circulation 62: 62–69

Heymsfield SB, McManus C, Smith J, Stevens V, Nixon DW (1982) Anthropometric measurement of muscle mass: revised equations for calculating bone-free arm-muscle area. Am J Clin Nutr 36: 680–690

Hyvärinen A, Nikkilä EA (1962) Specific determination of blood glucose with o-toluidine. Clin Chim Acta 7: 140–143

Jelliffe EFB, Jelliffe DB (1969) The arm circumference as a public health index of protein calorie malnutrition in early childhood. J Trop Pediatr 15: 177–260

Katz LD, Glickman MG, Rapaport S, Ferrannini E, DeFronzo RA (1983) Splanchnic and peripheral disposal of glucose in man. Diabetes 32: 675–679

Kessler G, Lederer H (1965) Fluorometric measurements of triglycerides. In: Skeggs LT (ed) Automation in analytical chemistry. Mediad, New York, p 345

Krauss RM, Lindgren FT, Wood PD, Haskell WL, Albers JJ, Cheung MC (1977) Differential increases in plasma high density lipoprotein subfractions and apoliproteins (Apo-Lp) in runners. Circulation [Suppl III] 56: 4

Lehtonen A, Viikari J (1980) Serum lipids in soccer and ice-hockey players. Metabolism 29: 36–39

Lohmann D, Liebold F, Heilmann W, Senger H, Pohl A (1978) Diminished insulin response in highly trained athletes. Metabolism 27: 521–524

Lopez-S A, Stone MH, Johnson C, Bell L (1980) Effect of resistive training on serum lipids and lipoproteins of middle age sedentary men (Abstract) Am J Clin Nutr 33: 939

MacDougall JD, Sale DG, Elder GCB, Sutton JR (1982) Muscle ultrastructural characteristics of elite powerlifters and bodybuilders. Eur J Applied Physiol 48: 117–126

Nikkilä EA, Taskinen M-R, Kekki M (1978a) Relation of plasma high-density lipoprotein cholesterol to lipoprotein-lipase activity in adipose tissue and skeletal muscle of man. Atheroclerosis 29: 497–501

Nikkilä EA, Taskinen M-R, Rehunen S, Härkönen M (1978b) Lipoprotein lipase activity in adipose tissue and skeletal muscle of runners: relation to serum lipoproteins. Metabolism 27: 1661–1671

Olefsky J, Reaven GM, Farquhar JW (1974) Effects of weight reduction on obesity. Studies of lipid and carbohydrate metabolism in normal and hyperlipoproteinemic subjects. J Clin Invest 53: 64–76

Pipes TV (1979) Physiological characteristics of elite body builders. Physician Sportsmed 7: 116–122

Prince FP, Hikida RS, Hagerman FC (1976) Human muscle fiber types in power lifters, distance runners and untrained subjects. Pflügers Arch 363: 19–26

Salans LB and Wise JK (1970) Metabolic studies in obesity. Med Clin North Am 54: 1533–1542

Saltin B, Åstrand P-O (1967) Maximal oxygen uptake in athletes. J Applied Physiol 23: 353–358

Schnabel A, Kindermann W (1982) Effect of maximal oxygen uptake and different forms of physical training on serum lipoproteins. Eur J Applied Physiol 48: 263–277

Solyom A (1972) Effect of androgens on serum lipids and lipoproteins. Lipids 7: 100–105

Soman VR, Koivisto VA, Deibert D, Felig P, DeFronzo RA (1979) Increased insulin sensitivity and insulin binding to monocytes after physical training. N Engl J Med 301: 1200–1204

Stern MP, Olefsky J, Farquhar JW, Reaven GM (1973) Relationship between fasting plasma lipids levels and adipose tissue morphology. Metabolism 22: 1311–1317

Taskinen M-R, Nikkilä EA, Huttunen JK, Hilden H (1980) A micromethod for the assay of lipoprotein lipase activity in needle biopsy samples of human adipose tissue and skeletal muscle. Clin Chim Acta 104: 107–117

WHO Expert Committee on Diabetes Mellitus (1980) WHO Technical Report Series NO 646

Yki-Järvinen H, Koivisto VA (1983) Effects of body composition on insulin sensitivity. Diabetes 32: 965–969

Accepted September 19, 1984