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Lipid profile of body builders with and without self-administration of anabolic steroids

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Summary. Twenty-four top-level body builders [13 anabolic steroid users (A); 11 non-users (N)] and 11 performance-matched controls (C) were examined to determine the effect on lipids, lipoproteins and apolipoproteins of many years of body building with and without simultaneous intake of anabolic steroids and testosterone. After an overnight fast, triglycerides (TG), total cholesterol (TOTC), high density lipoprotein cholesterol (HDLC), low density lipoprotein cholesterol (LDLC), the HDLC subfractions HDL_2C and HDL_3C, as well as apolipoprotein A-I (Apo A-I), apolipoprotein A-II (Apo A-II) and apolipoprotein B (Apo B) were determined. Both A and N, compared to C, showed significantly lower HDLC and higher LDLC concentrations, with the differences between A and C clearly pronounced. In a subgroup of 6 body builders taking anabolic steroids at the time of the study, HDLC, HDL_2C, HDL_3C, Apo A-I and Apo A-II were all significantly lower and LDLC was significantly higher than in a second subgroup of 7 body builders who had discontinued their intake of anabolic steroids at least 4 weeks prior to the study. In some single cases HDLC was barely detectable (2–7 $mg \cdot dl^{-1}$). The TG and TOTC remained unchanged. The present findings suggest that many years of body building among top-level athletes have no beneficial effect on lipoproteins and apolipoproteins. Simultaneous use of anabolic steroids results in part in extreme alterations in lipoproteins and apolipoproteins, representing an atherogenic profile. After discontinuing the use of anabolic steroids, the changes in lipid metabolism appear to be reversible.

Key words: Body building — Anabolic steroids — Lipoproteins — Apolipoproteins

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Introduction

The positive effect of endurance training on lipid metabolism, inducing a profile suggesting reduced coronary risk, has been demonstrated by numerous studies (Berg et al. 1980a; Dufaux et al. 1982; Kullmer and Kindermann 1985; Schnabel and Kindermann 1982; Tran et al. 1983; Wood et al. 1983). In contrast, the effects of weight training on lipid metabolism still remain controversial. Reports of an improvement in the lipid profile (Goldberg et al. 1984; Hurley et al. 1984; Hurley et al. 1987; Johnson et al. 1983; Yki-Järvinen et al. 1984) have been contradicted by studies showing no change (Clarkson et al. 1981; Kokkinos et al. 1987), or even negatively altered lipid metabolism representing an increased atherogenic risk (Berg et al. 1980b; Hurley et al. 1984). These contradictory results may be due, among other reasons, to differences in the volunteer groups and the kind of weight training performed, as well as the use of anabolic substances.

Among body builders anabolic steroids are commonly used. They are often taken orally or parenterally in uncontrolled high doses, frequently with several different substances at the same time (Alèn and Rahkila 1984; Alén et al. 1985; Hurley et al. 1984; Lamb 1984; Lenders et al. 1988). Anabolic steroids cause detrimental alterations in lipid and lipoprotein profiles (Alén and Rahkila 1984; Alén et al. 1985; Haffner et al. 1983; Hazzard et al. 1984; Hurley et al. 1984;Kantor et al. 1985; Lenders et al. 1988; Webb et al. 1984). At present it is not clear to what extent regularly performed body building actually affects lipid metabolism since the effect of anabolic steroids frequently over-rides changes which are induced by the body building itself.

In the present study the effect of exercise and

that of anabolic steroids were separated and an additional comparison with a control group of the same physical performance was carried out to obtain further information about the influence of several years of body building on the lipid profile.

Material and methods

Twenty-four top-level body builders participated in the study; 13 of them were taking anabolic steroids regularly [anabolic users (A)] while the other 11 had never taken any anabolic steroids [non-users (N)]. Applying a matched pairs procedure, another 11 healthy untrained subjects with similar relative maximal oxygen uptake ($\dot{V}_{O_{2max}} \cdot kg^{-1}$) to that of N were selected [control group (C)]. The descriptive data of the three groups are presented in Table 1.

Eight A took anabolic steroids both parenterally and orally; the other 5 A took these substances only parenterally. Testosterone and different anabolic steroids were used. The mean daily intake was several times the common therapeutic dose. In the year prior to the study these substances had been used for an average of 11.6±4.0 weeks. With the exception of 4 body builders, all of them belonging to N, no subject performed regular endurance training of more than 2-h weekly. All body builders consumed a low-fat diet and used protein supplements with a low content of cholesterol to match their increased protein demand. There were no substantial dietary differences between A and N. In contrast, C showed the eating habits of a normal population with a higher fat and cholesterol content in their diet.

In order to investigate the effect of discontinuing the use of anabolic steroids and to rule out any influence of endurance training, the 20 body builders not performing endurance training were divided into three subgroups (for descriptive data, see Table 2): 7 body builders who were not using anabolic steroids and not performing endurance training (BO); 7 body builders who had ceased the use of anabolic steroids at least 4 weeks prior to the study (BI); 6 body builders who were taking anabolic steroids at the time of the study (BA).

All subjects performed an incremental graded cycle exercise test (with an increase of 50 W every 3 min) until subjective exhaustion. The $\dot{V}_{O_{2max}}$ was measured by an open-circuit system; $\dot{V}_{O_{2max}} \cdot kg^{-1}$ served as the criterion for physical performance. Percentage of body fat was determined from skinfold measurements at four sites (Durnin and Ramahan 1967). Lean body mass was calculated as the difference between body mass and the mass of fat.

After an overnight fast, samples of venous blood were taken from all subjects to measure in the serum the following lipids, lipoproteins and apolipoproteins: triglycerides (TG), total cholesterol (TOTC), high density lipoprotein cholesterol (HDLC), low density lipoprotein cholesterol (LDLC), the HDLC subfractions HDL_2C and HDL_3C, as well as apolipo-

Table 1. Details of the subjects. Anabolic users (A), non-users (N), control group (C)

	Age (years)	Height (cm)	Body mass (kg)	Body fat (%)	Lean body mass (kg)	$\dot{V}_{O_{2max}}$ ($ml \cdot min^{-1}$)	$\dot{V}_{O_{2max}} \cdot kg^{-1}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	Length of training (years)	Training per week (h)
A	26.5	176.5	98.9	12.2	87.0	3432	35.1	7.7	12.3
($n=13$)	±4.0	±5.6	±12.8	±2.1	±12.2	±280	±4.4	±4.5	±2.6
N	27.0	178.0	89.5	13.3	77.5	3570	40.7	7.4	10.2
($n=11$)	±7.3	±6.6	± 9.3	±2.3	± 6.4	±476	±5.1	±4.4	±3.0
C	24.5	179.0	73.1	13.8	62.6	2959	40.6	—	—
($n=11$)	±3.9	±5.6	± 9.4	±3.3	± 6.7	±280	±5.1		
Statistical comparisons									
N with C	NS	NS	***	NS	***	***	NS	—	—
N with A	NS	NS	*	NS	*	NS	**	NS	NS
A with C	NS	NS	***	NS	***	**	**	—	—

Values are means±SD; NS not significant; * $p<0.05$; ** $p<0.01$; *** $p<0.001$

Table 2. Details of the body builder subjects who did not perform endurance training

	Age (years)	Height (cm)	Body mass (kg)	Body fat (%)	Lean body mass (kg)	$\dot{V}_{O_{2max}}$ ($ml \cdot min^{-1}$)	$\dot{V}_{O_{2max}} \cdot kg^{-1}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	Length of training (years)	Training per week (h)
BA	26.5	176.7	100.7	12.6	88.2	3422	34.7	7.8	10.7 ┐
($n=6$)	±5.2	±4.9	±14.4	±1.7	±13.7	±157	±5.6	±5.1	±1.4 *
BI	26.4	176.3	97.6	11.8	86.1	3440	35.5	7.6	13.8 ┘
($n=7$)	±3.1	±6.5	±12.0	±2.6	±11.7	±369	±3.4	±4.3	±2.6
BO	26.0	180.0	90.1	13.6	77.7	3386	39.0	9.0	9.6
($n=7$)	±5.0	±5.3	± 8.6	±2.5	± 5.1	±358	±3.2	±4.8	±3.3

Values are mean±SD; * $p<0.05$; BA=current users of anabolic steroids; BI=body builders who had ceased using anabolic steroids at least 4 weeks prior to the present study; BO=non-users of anabolic steroids

Table 3a. Triglycerides (TG), total cholesterol (TOTC) and high and low density lipoprotein cholesterol and subfractions (HDLC, LDLC, HDL_2C and HDL_3C) of groups A, N and C (for definitions see Table 1)

	TG ($mg \cdot dl^{-1}$)	TOTC ($mg \cdot dl^{-1}$)	HDLC ($mg \cdot dl^{-1}$)	HDLC:TOTC	LDLC ($mg \cdot dl^{-1}$)	LDLC:TOTC	HDL_2C ($mg \cdot dl^{-1}$)	HDL_3C ($mg \cdot dl^{-1}$)
A	104	186	23	0.14	154	0.81	6.5	16.8
($n=13$)	±35	±52	±16	±0.10	±58	±0.11	±4.8	±12.3
N	90	167	34	0.20	121	0.72	8.5	25.2
($n=11$)	±23	±25	± 7	±0.04	±22	±0.05	±3.6	± 8.0
C	94	172	46	0.27	111	0.64	—	—
($n=11$)	±32	±32	±12	±0.04	±23	±0.05	—	—
Statistical comparisons								
N with C	NS	NS	**	**	NS	**	—	—
N with A	NS	NS	*	*	NS	*	NS	*
A with C	NS	NS	**	***	*	***	—	—

Values are means ± SD; NS not significant; * $p<0.05$; ** $p<0.01$; *** $p<0.001$

Table 3b. Apolipoproteins (Apo A-I, Apo A-II, Apo B) of groups A, N, C (for definitions see Table 1)

	Apo A-I ($mg \cdot dl^{-1}$)	Apo A-II ($mg \cdot dl^{-1}$)	Apo B ($mg \cdot dl^{-1}$)	Apo B / Apo A-I
A	99	45	113	1.7
($n=13$)	±59	±26	±44	±2.0
N	128	54	88	0.8
($n=11$)	±32	±11	±17	±0.3
C	120	40	74	0.6
($n=11$)	±29	± 6	±22	±0.2
Statistical comparison see Table 1				
N with C	NS	**	*	NS
N with A	NS	NS	NS	NS
A with C	NS	NS	**	NS

Values are mean ± SD; NS = not significant; * $p<0.05$, ** $p<0.01$

protein A-I (Apo A-I), apolipoprotein A-II (Apo A-II) and apolipoprotein B (Apo B). In addition, the following ratios were calculated: HDLC:TOTC, LDLC:TOTC and Apo B: Apo A-I.

The TG and TOTC were determined enzymatically (test kit Boehringer, Mannheim). Lipoproteins were measured by quantitative electrophoresis using a commercially available system (Lipidophor All in 12, Immuno, Heidelberg), from which results HDLC and LDLC were calculated (Wieland and Seidel 1978). The HDL_2C and HDL_3C were determined using a precipitation test (Quantolip, Immuno, Heidelberg). Apolipoproteins were measured by radial immunodiffusion (Combi RID, Immuno, Heidelberg). Concentrations of all parameters of lipid metabolism are given in $mg \cdot dl^{-1}$.

Mean values and standard deviations were calculated ($\bar{x} \pm SD$). For statistical analysis the Mann-Whitney *U*-test was employed with $p<0.05$ as level of significance.

Results

Lean body mass was higher in A compared to N and clearly lower in C than in both body builder groups. The $\dot{V}_{O_2max} \cdot kg^{-1}$ was identical in N and C, but lower in A (Table 1).

Between A and N significant differences were found for HDLC, HDL_3C, HDLC:TOTC and LDLC:TOTC. Most of the lipoprotein and apolipoprotein parameters in C were significantly different compared to A and N, while the apolipoproteins showed no differences between the groups A and N (Table 3a, b).

The comparisons of the body-builder subgroups selected by different use of anabolic steroids demonstrated trends but not significant differences for lean body mass (BA > BO) and $\dot{V}_{O_2max} \cdot kg^{-1}$ (BO > BA; BO > BI). In BI the duration of weekly weight training was significantly longer than in BA and BO (Table 2).

Lipids, lipoprotein cholesterol and apolipoproteins yielded no differences between BO and BI. In contrast, the values for HDLC, HDLC:TOTC, HDL_2C, HDL_3C, Apo A-I and Apo A-II were significantly lower in BA than in BI and in BO; LDLC values, the LDLC:TOTC and Apo B:Apo A-I ratios were significantly higher in BA than in the other two groups. As a result of the great range of variation, the difference for Apo B was not statistically significant (Fig. 1a, b).

Five body builders showed extremely low values for HDLC between 2 and 11 $mg \cdot dl^{-1}$ (Table 4a), with the HDL fraction in lipoprotein electrophoresis in some single cases barely detectable (Fig. 2). Four of these body builders were taking anabolic steroids at the time of the study, while body builder no. 3 (Table 4a, b) had discontinued use of anabolic steroids 6 weeks prior to the study. Parallel to the lower HDLC values, HDL_2C and Apo A-I especially were clearly below the

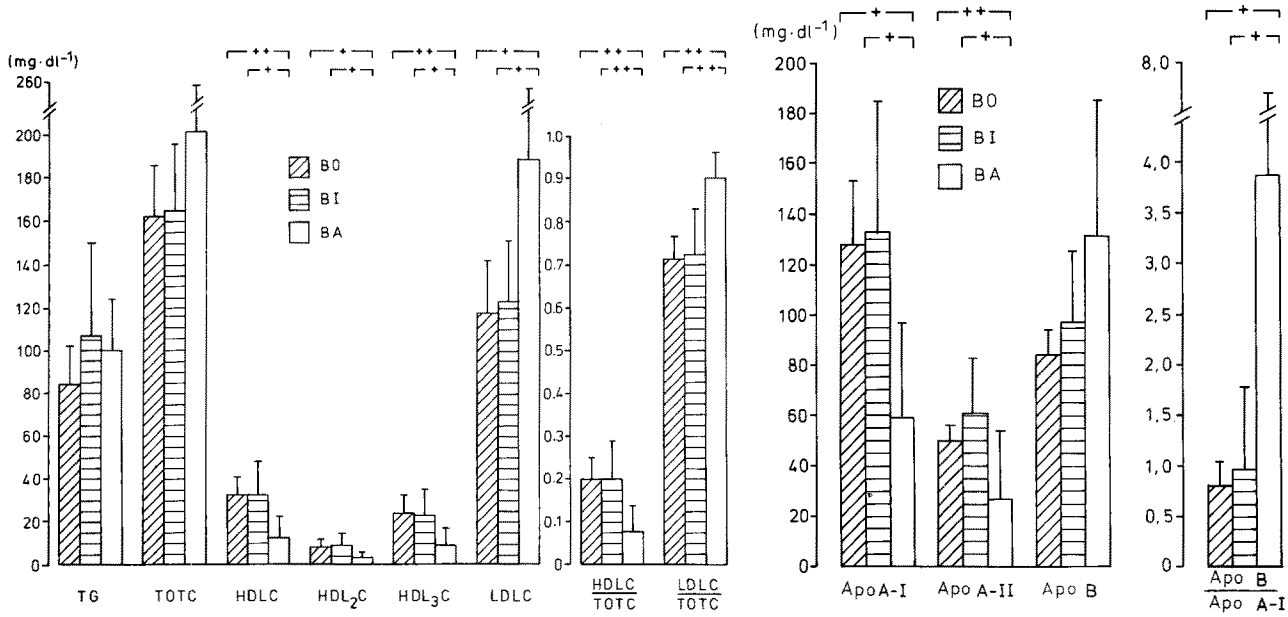

Fig. 1a, b. Triglycerides (*TG*), total cholesterol (*TOTC*), high and low density lipoprotein cholesterol and subfractions (HDLC, HDL_2C, HDL_3C, LDLC) and apolipoproteins (Apo A-I, Apo A-II and Apo B) of group *BO, BI* and *BA* (for definitions see Table 2); + $p<0.05$, + + $p<0.01$

normal values. In all of the 5 body builders at least 91% of TOTC was represented by LDLC (LDLC:TOTC > 0.91; Table 4a, b).

Discussion

The object of the present study was to investigate whether regularly performed body-building training affects lipids, lipoproteins and apolipoproteins and to what degree these parameters are quantitatively changed by additional use of anabolic steroids. The comparison of N with C yielded some statistically significant differences in lipoprotein cholesterol and some apolipoproteins in favour of C. Similar findings of unchanged or even negative quantitative changes in lipoproteins and apolipoproteins after many years of weight training have already been reported extensively in the literature (Berg et al. 1980b; Clarkson et al. 1981; Hurley et al. 1984). On the other hand, there have also been findings which suggest a positive effect on the lipid profile of many years of body building (Hurley et al. 1984; Yki-Järvinen et al. 1984). The question here is whether additional endurance exercise was performed, particularly since in those studies maximal aerobic capacity was above normal values for sedentary persons and clearly higher than in the subjects taking part in the present investigation. Additionally, it must be taken into account that this study covered only top-level body builders with several previous years of training. Dietary differences rather than the performed body building training do not explain the alterations found in lipid profile. Since the body builders consumed a diet with less content of fat and cholesterol than the controls, changes of lipoprotein, cholesterol and apolipoproteins could have been expected that were opposite to those actually seen in this study.

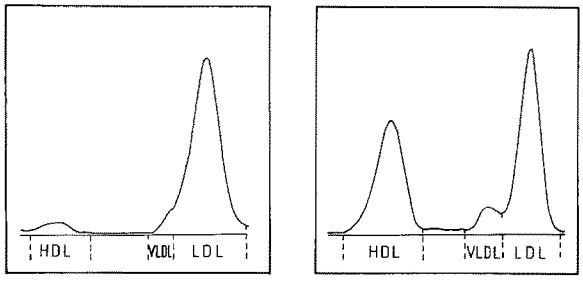

Fig. 2. Lipoprotein electrophoresis of two body builders; right = normal high density lipoprotein (*HDL*)-band without use of anabolic steroids; left = *HDL*-band barely detectable during intake of anabolic steroids

Extreme endurance exercise and extreme weight training produce different effects on the cardio-circulatory system. Endurance exercise regularly induces enlargement of the heart (an athlete's heart); weight training causes no dimensional changes in the heart (Kindermann et al.

Table 4a. Triglycerides (TG), total cholesterol (TOTC) and high and low density lipoprotein cholesterol and subfractions (HDLC, LDLC, HDL_2C and HDL_3C) of 5 body builders with low HDLC between 2 and 11 $mg \cdot dl^{-1}$

Subject no.	TG ($mg \cdot dl^{-1}$)	TOTC ($mg \cdot dl^{-1}$)	HDLC ($mg \cdot dl^{-1}$)	HDLC:TOTC	LDLC ($mg \cdot dl^{-1}$)	LDLC:TOTC	HDL_2C ($mg \cdot dl^{-1}$)	HDL_3C ($mg \cdot dl^{-1}$)
1	116	219	2	0.01	205	0.94	0.7	1.3
2	130	289	7	0.02	274	0.95	3.8	3.2
3	92	139	5	0.04	127	0.91	2.2	2.8
4	120	280	11	0.04	257	0.92	3.7	7.3
5	75	141	7	0.05	131	0.93	1.2	5.8

Table 4b. Apolipoproteins (Apo A-I, Apo A-II and Apo B) of 5 body builders with low high density lipoprotein cholesterol between 2 and 11 $mg \cdot dl^{-1}$

Subject no.	Apo A-I ($mg \cdot dl^{-1}$)	Apo A-II ($mg \cdot dl^{-1}$)	Apo B ($mg \cdot dl^{-1}$)	Apo B / Apo A-I
1	21	10	165	7.8
2	21	10	205	9.7
3	39	21	112	2.8
4	53	29	165	3.1
5	52	25	80	1.5

1974; Rost and Hollmann 1983). By analogy, endurance and weight training also seem to have different effects on lipid metabolism: endurance-trained athletes have increased lipoprotein lipase activity in the skeletal muscle and fatty tissue (Nikkilä et al. 1978), which leads to quantitatively positive changes in the lipid lipoprotein profile (Berg et al. 1980a; Dufaux et al. 1982; Kullmer and Kindermann 1985; Tran et al. 1983; Wood et al. 1983). In comparison to a control group, Yki-Järvinen et al. (1984) found the lipoprotein lipase activity in the skeletal muscle and fatty tissue of body builders remained unchanged. Thus, body building alone does not appear to alter lipid metabolism to any considerable extent. In particular, training effects which cause changes representing a non-atherogenic profile are not to be expected.

Anabolic steroids and testosterone applied in high doses caused, in part, massive changes in lipid metabolism among the body builders investigated. By analogy to earlier studies, both lipoproteins and apolipoproteins were affected negatively, whereas triglycerides and cholesterol showed no substantial quantitative changes (Alén et al. 1985; Hurley et al. 1984; Kantor et al. 1985; Lenders et al. 1988). The decrease in HDL cholesterol is extreme in some cases, showing a barely detectable HDL fraction in lipoprotein electrophoresis (Table 4a and Fig. 2). No other reports have yet been published of similarly low concentrations in athletes using anabolic steroids. The exact mechanism causing the detrimental changes in lipoproteins and apolipoproteins during intake of anabolic steroids and testosterone is not yet known. One cause is assumed to be an accelerated HDL catabolism, since an increase in the hepatic triglyceride lipase activity through anabolic steroids has been described (Applebaum-Bowden 1984; Haffner et al. 1983; Hazzard et al. 1984; Kantor et al. 1985; Lenders et al. 1988). As there were no substantial differences among the body builders in their daily food intake, dietary effects on these changes in lipid metabolism can be ruled out.

The almost identical values of the parameters of lipid metabolism in BO and BI suggest that the changes are reversible. Different periods of time required for altered levels to return to normal are found in the literature (Alén et al. 1985; Lenders et al. 1988; Webb et al. 1984). Even 6 weeks after discontinuation of the use of anabolic steroids the lipoproteins may still remain extremely altered, as demonstrated in Table 4, body builder no. 3. At present, we can only speculate whether the changes in lipid metabolism induced by anabolic steroids indicate an increased atherogenic risk. It is theoretically conceivable that after long-term use of anabolic steroids and testosterone, lipoproteins and apolipoproteins become quantitatively permanently altered, if the intervals of discontinuation are relatively brief. Only long-term observations can determine whether that represents a similar atherogenic risk as demonstrated in epidemiological studies for lipid metabolism (Castelli et al. 1975; Kannel et al. 1979; Lipid Research Clinics Program 1984).

In summary, many years of body building in top-level athletes have no beneficial effects on lipoproteins and apolipoproteins. For the benefit of their health, body builders should, therefore, perform additional endurance training. Simultaneous use of anabolic steroids causes detrimental changes in lipid metabolism, sometimes with an extreme decrease in HDL-cholesterol. A conclu-

sive evaluation of potential health risks is not possible at the present time.

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