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Reversibility of the effects on blood cells, lipids, liver function and hormones in former anabolic–androgenic steroid abusers☆

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Abstract

Background: In contrast to the acute effects of anabolic–androgenic steroid (AAS) abuse, the long-term risk profile of former long-term abusers (ExA) is less clear.

Methods: Blood parameters of 32 male bodybuilders and powerlifters were studied. Fifteen ExA had not been abusing AAS for at least 12–43 months on average (mean dosage 700 mg for 26 weeks per year over 9 years), 17 athletes (A) were still abusing AAS (750 mg for 33 weeks per 8 years).

Findings: Hemoglobin (+5%), leucocytes (+33%) and platelets (+38%) were significantly higher in A. Alanine aminotransferase (ALT) and aspartate aminotransferase (AST) were higher, cholinesterase activity (CHE) lower in A (65 ± 55, 38 ± 27 and 3719 ± 1528 U/l) compared to ExA (24 ± 10, 18 ± 11 and 6345 ± 975 U/l; each $P < 0.001$) with normal values for gamma-glutamyl transpeptidase (gamma-GT) and bilirubin. ALT, AST and CHE correlated significantly with the extent (duration and weekly dosage, expressed as a point score) of AAS abuse in A ($r = 0.68$, 0.57 and -0.62; each $P < 0.01$). Total and LDL-cholesterol were similar, HDL-cholesterol was distinctly lower in A than in ExA (17 ± 11 and 43 ± 11 mg/dl; $P < 0.001$) and correlated negatively with the extent of AAS abuse ($r = -0.50$; $P < 0.05$). Testosterone and estradiol were significantly higher, while LH, FSH and the sexual-hormone-binding (SHB) protein were lower in A than in ExA (each $P < 0.001$). Two ExA had testosterone levels below the normal range.

Interpretation: The alterations in cell counts, HDL-cholesterol, liver function and most hormones of the pituitary–testicular axis induced by a long-term abuse of AAS were reversible after stopping the medication for over 1 year. In some ExA, an increased ALT activity and a depressed testosterone synthesis were found.

Keywords: Anabolic–androgenic steroids; Bodybuilders; Blood lipids; Doping; Hormones; Liver enzymes

1. Introduction

An uncontrolled non-medical usage of anabolic–androgenic steroids (AAS) is not only described in competitive strength athletes not systematically subject to doping controls, but increasingly reported in leisure time athletes [1]. Irrespective of whether AAS are taken in cycles or over a period of several years, these substances show a considerable potential of side effects. The short-term side effects of AAS, such as especially a disturbed liver function, an atherogenic blood lipid profile, an impaired gonadal function as well as dermatological alterations, have already been documented in detail. However, except for the irreversible clinical signs of virilization in females and the gynecomastia in males, there is a clear deficit of data regarding the long-term effects of former self-administered AAS on health after withdrawal of these substances.

In most of the examinations on humans, the so-called former users were examined only after a break of intake of several weeks to few months. An examination in five former AAS abusers who had been steroid-free for at least 5 years, in most cases, showed reversible peculiarities of liver enzymes, lipoprotein changes and of the testosterone level in comparison with current abusers [2]. In a long-term study with a 6-month administration of AAS in mice, which would correspond to a period of approximately 15 years in humans, 1 year after administration 35% of the animals compared with 12% of the controls had deceased [3]. Beside these toxic and hormonal effects, cardiac complications are reported in animal studies as well as in strength athletes abusing AAS [4], too, which mainly include a concentric left ventricular hypertrophy with possibly impaired diastolic

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relaxation [5] and in single cases also a precipitation of dilated cardiomyopathy [6].

The purpose of the present study was therefore to examine for the first time whether, one to several years after discontinuing the massive intake of AAS, changes of laboratory parameters were still detectable in male strength athletes. These ex-abusers were compared with a group of current AAS abusers.

2. Material and methods

2.1. Subjects

Thirty-two male bodybuilders and powerlifters participated voluntarily in the study approved by the review committee of the German National Institute of Sports Sciences and signed an informed consent. They were expressly guaranteed absolute anonymity.

Fifteen athletes (ex-abusers; age 38.0 ± 7.0 years, height 176 ± 6 cm, body mass 89.5 ± 10.0 kg) had stopped their self-administered abuse of AAS at least 1 year ago (mean value 43 months, median 24 months, minimum 12 months, maximum 10 years); their mean dosage had amounted to 720 mg per week for 26 weeks per year over 9 years. Seventeen athletes (abusers; age 30.5 ± 5.0 years, height 177 ± 8 cm, body mass 96.0 ± 10.0 kg) were current AAS abusers at a mean dosage of 1030 mg per week (median 750 mg) for 33 weeks per year over 8 years. Thus, in both groups, the amount considerably exceeded the one usually prescribed for clinical indications.

The AAS administered by intramuscular injections were boldenone, drostanolone, formebolone, metenolone, nandrolone, stanozolol, different esters of testosterone and trenbolone. The orally taken substances included 4-dehydrochlormethyltestosterone, fluoxymesterone, mesterolone, metenolone, metandienone, oxandrolone, oxymetholone and stanozolol. All except two ex-abusers used combinations of both oral and injectable substances. Five ex-abusers and 6 abusers had sporadic (<1 year; 2–16 I.U. daily) experiences with growth hormone, 9 ex-abusers and 15 abusers took clenbuterol (3–20 times of the therapeutic dosage). Nine ex-abusers and 14 abusers had used anti-oestrogenes. No subject had taken diuretics nor restricted his fluid intake at the moment of the blood sampling. Six ex-abusers and 4 abusers were cigarette smokers, 9 ex-abusers and 11 abusers were non-smokers, and 2 abusers had stopped 1–2 years previously. None of the subjects were in the habit of excessive alcohol consumption.

Ex-abusers had been training for 14.0±4.5 years for 6.0± 2.0 h per week, abusers for 11.0 ± 5.0 years for 6.0 ± 1.0 h per week (no statistical difference).

2.2. Anabolic–androgenic-score

In order to obtain an estimate of the extent of AAS abuse, a point score was established. 1–4 points were given in each of the three categories “years of administration” (2.5–4.0/5.0–6.0/6.5–10.0 and >10 years), “weeks of AAS abuse per year” (<20/21–30/31–37 and >38 weeks) and “weekly dosage in mg” (<500/550–750/800–1250 and >1250 mg). The limits of the categories were chosen in order to include 6–10 athletes in each of the categories; if other anabolic substances such as growth hormone and clenbuterol were also used, an additional point was accorded, respectively. The maximal point score available was therefore 14, the minimal 3. The anabolic–androgenic-score (AAS-score) of the abusers (9.2 ± 2.7) was not significantly higher in comparison with ex-abusers (7.8 ± 3.2).

2.3. Liver sonography

The examination was made by using a 5 MHz sector scan (GE Ultrasound). The liver parenchyma and bile ducts were examined visually.

2.4. Blood parameters

After an overnight fast, between 8 and 10 o’clock in the morning venous blood samplings were taken from the right arm after 10 min of rest in a supine position. The sedimentation rate was taken after 1 h (Sarstedt). The blood cells were measured by Cellcounter K1000 (Sysmex). Serum aspartate aminotransferase (AST), alanine aminotransferase (ALT), gamma-glutamyl transpeptidase (gamma-GT), glutamate dehydrogenase (GLDH), alkaline phosphatase, creatine kinase (CK) and cholinesterase were measured by using kinetic UV-tests with a photometer (testkits, Merck), amylase by the Reflotron system (Boehringer Mannheim), all enzymes being measured at 25 °C. Sodium and potassium were measured by an ionselective electrode (Bayer Diagnostics), magnesium, calcium and iron photometrically by testkits from Fa. Merck. Urea was measured by urease (GLDH-method, enzymatic UV-Test, Merck), creatinine with enzymatic PAP-method (Merck), uric acid, total cholesterol, triglycerides and bilirubin by color test (Merck and Boehringer Mannheim). The lipoprotein subfractions were analyzed by lipid electrophoresis (Baxter). Blood glucose was measured photometrically by UV-test (glucose-dehydrogenase, Merck). HbA1 was measured according to the separation method in fast ion exchange (Human).

All hormones were measured within the same assay after storage at −70 °C for a maximum of 6 months. The thyroid-stimulating hormone (TSH; intra-assay variability 4.6%), the luteinizing hormone (LH; intra-assay variability 4.7%) and the follicle-stimulating hormone (FSH; intra-assay variability 6.0%) were assessed by chemiluminescence immunoassay (Nichols). Lipoprotein (a) was determined nephelometrically, sexual-hormone-binding globulin (SHBG), testosterone (intra-assay variability 4.5%) and estradiol (intra-assay variability 3.5%) by radioimmunoassay (CIS bio international, France).

2.5. Statistics

Data are expressed as means ± standard deviation (S.D.). In the tables, the reference values of the laboratory are also shown, respectively. Comparisons of normally distributed (tested by using the Kolmogoroff–Smirnoff test) variables between ex-abusers and abusers were calculated using the *t*-test for unpaired data, not-normally distributed variables were compared by using the Mann–Whitney *U*-test. Relationships between selected measures and the ASS-score were calculated by a Spearman rank correlation. The level of statistical significance was set at $P < 0.05$.

2.5.1. Role of the funding source

The funding source had no involvement in study design, in the collection, analysis, and interpretation of data, in the writing of the report nor in the decision to submit the paper for publication.

3. Results

Nine ex-abusers and 12 abusers indicated that, in the past, they had suffered from gynecomastia at least temporarily; out of them, 5 and 7, respectively, had undergone a subcutaneous mastectomy.

3.1. Liver sonography

In three ex-abusers and two abusers, the liver sonography showed an unspecifically increased echodensity of the liver parenchyma. Liver enlargements or pathological findings were not to be found.

3.2. Blood count (Table 1)

Hemoglobin (+5%), hematocrit (+9%), erythrocytes (+8%) as well as leucocytes (+33%) and thrombocytes (+38%) were increased in abusers compared to ex-abusers. Five abusers and one ex-user showed hemoglobin values of >17 g/dl, three abusers had numbers of leucocytes >10,000/μl, nine abusers and one ex-user numbers thrombocytes >300,000/μl. The leucocytes correlated significantly with the time interval since the intake of AAS in the group of ex-abusers (Fig. 1).

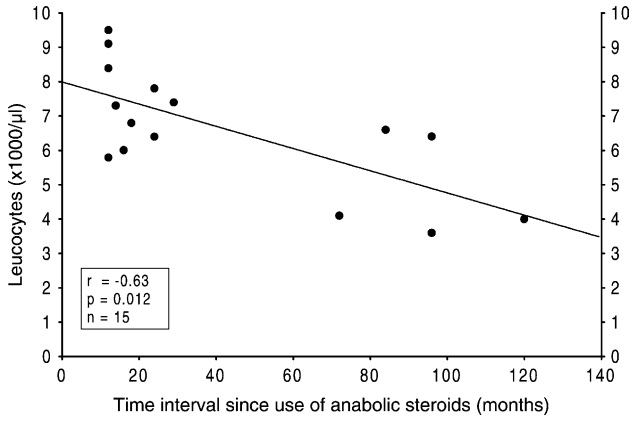

Fig. 1. Linear correlation between the blood leucocyte count and the time interval since stopping anabolic steroid abuse in the group of the ex-abusers.

3.3. Liver function and CK activity (Table 2)

All but one abusers showed increased values for ALT and AST above the upper limit of reference with higher values for ALT than AST in each case. The values in ex-abusers were increased above the upper limit of reference in six (ALT) and three (AST) subjects, respectively, with all but one (with considerably increased CK activity of 1747 U/l) showing higher ALT values than AST. The gamma-GT in ex-abusers was significantly higher than in abusers with values above the upper limit of reference in one ex-user (43 U/l) and one user (37 U/l). The GLDH was increased above the upper limit of reference in four abusers and three ex-abusers (among them one ex-user chronically taking non-steroid anti-rheumatics after a road accident). The cholinesterase in abusers was, to a high degree, significantly lower than in ex-abusers. Decreased values (below the limit of reference of <3000 U/l) were to be found in five abusers, but not in any ex-user. There was a significant correlation of ALT with GLDH and cholinesterase ($r = 0.64$ and -0.93, $P < 0.01$ and 0.001) in abusers. Total bilirubin, alkaline phosphatase and amylase (not shown) were within the normal range of reference and did not differ between the groups.

Table 1
Blood count in former (ex-abusers; age 38.0 ± 7.0 years) and current (abusers; age 30.5 ± 5.0 years) anabolic steroid users

	Range of reference	Ex-abusers ($n = 15$)	Abusers ($n = 17$)	*P*-value (ex-A/A)
Sedimentation rate (mm first hour)	<10	2.5 ± 1.5 (1–5)	2.5 ± 3.5 (1–15)	NS
Hemoglobin (g/dl)	14.0–17.5	15.8 ± 1.0 (13.5–17.2)	16.6 ± 0.8 (15.0–17.7)	<0.05
Hematocrit (%)	42–52	44 ± 3 (38–49)	48 ± 2 (43–51)	<0.001
Erythrocytes (ml/μl)	4.6–6.2	5.13 ± 0.37 (4.49–5.66)	5.55 ± 0.41 (4.83–6.44)	<0.01
Leucocytes (cells/μl)	3500–11000	6600 ± 1800 (3600–9500)	8800 ± 1700 (5700–11400)	<0.01
Thrombocytes (1000 cells/μl)	140–336	220 ± 72 (117–433)	303 ± 58 (188–385)	<0.001

Values are expressed as mean (minimum–maximum) ± S.D.

Table 2
Enzymes and bilirubine in former (ex-abusers; age 38.0 ± 7.0 years) and current (abusers; age 30.5 ± 5.0 years) anabolic steroid users

	Range of reference	Ex-abusers ($n = 15$)	Abusers ($n = 17$)	P-value (ex-A/A)
Alkaline phosphatase (U/l)	60–170	125 ± 29 (87–170)	110 ± 33 (56–166)	NS
Gamma-glutamyl-transferase (U/l)	4–28	18 ± 9 (7–43)	12 ± 8 (6–37)	<0.05
Cholinesterase (U/l)	3000–9000	6345 ± 975 (5096–8006)	3719 ± 1528 (1120–6178)	<0.001
Creatinkinase (U/l)	<81	290 ± 468 (11–1747)	624 ± 321 (166–1445)	<0.01
Creatinkinase (U/l) isoenzyme MB	<5	3 ± 4 (0–15)	9 ± 7 (0–27)	<0.01
Glutamate-dehydrogenase (U/l)	<4.0	3.3 ± 2.6 (0.8–9.5)	6.5 ± 10.6 (1.4–45.9)	NS
Bilirubine (μmol/l [mg/dl])	<17 (1.0)	13.0 ± 3.8 (8.5–20.2) [0.76 ± 0.22; (0.50–1.19)]	12.8 ± 2.7 (8.7–21.1) [0.75 ± 0.16; 0.51–1.24)]	NS
Alanine amino-transferase (U/l)	≤22	24 ± 10 (5–39)	65 ± 55 (17–143)	<0.001
Aspartate amino-transferase (U/l)	≤18	18 ± 11 (11–54)	38 ± 27 (16–133)	<0.001

Values are expressed as mean (minimum–maximum) ± S.D.

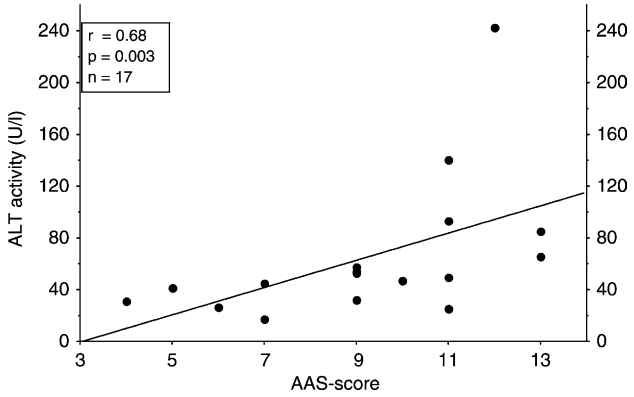

Fig. 2. Linear correlation between the blood ALT activity and the point score estimating the abuse of anabolic–androgenic steroids (AAS-score) in the group of the abusers.

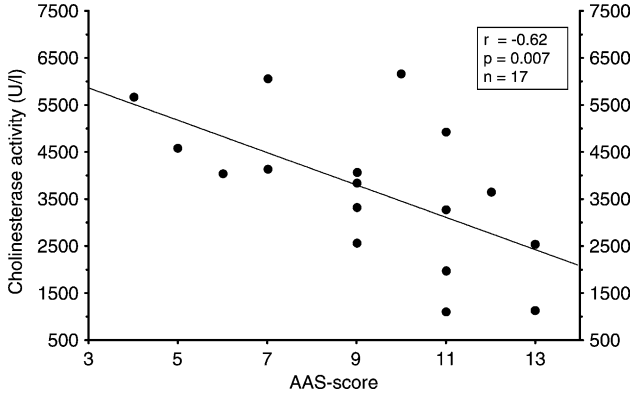

Fig. 3. Linear correlation between the blood cholinesterase activity and the point score estimating the abuse of anabolic–androgenic steroids (AAS-score) in the group of the abusers.

In abusers, a significant correlation of AST ($r = 0.57$, $P = 0.016$), ALT and cholinesterase activity (CHE) with the AAS-score was to be found (Figs. 2 and 3).

The activity of total CK and CKMB was significantly higher in abusers in comparison with ex-abusers. There was a significant correlation between total CK activity and AST or ALT ($r = 0.87$ and 0.67, both $P < 0.001$).

3.4. Blood lipids (Table 3)

HDL-cholesterol was considerably lower in abusers than in ex-abusers. Fifteen out of 17 abusers showed a HDL-cholesterol concentration <0.90 mmol/l (lowest value 0.06 mmol/l), as well as 2 ex-abusers (lowest value 0.65 mmol/l). Lipoprotein (a) was <0.43 μmol/l in all abusers and in 10 ex-abusers. In abusers, HDL-cholesterol correlated significantly with the ASS-score ($r = -0.50$; $P < 0.05$).

3.5. Hormones (Table 4)

Abusers showed a significantly higher testosterone (by 290%) and estradiol (by 400%) level as well as lower concentrations for SHBG (by 84%), LH (by 94%) and

Table 3
Blood lipids in former (ex-abusers; age 38.0 ± 7.0 years) and current (abusers; age 30.5 ± 5.0 years) anabolic steroid users

	Range of reference	Ex-abusers ($n = 15$)	Abusers ($n = 17$)	P-value (ex-A/A)
Total cholesterol (mmol/l [mg/dl])	<5.18 [200]	4.58 ± 0.96 (3.26–6.14) [177 ± 37; (126–237)]	4.30 ± 1.01 (2.64–6.03) [166 ± 39; (102–233)]	NS
Triglycerides (mmol/l [mg/dl])	<1.71 [150]	1.54 ± 0.66 (0.78–3.12) [135 ± 58; (68–274)]	1.15 ± 68 (0.54–3.40) [101 ± 60; (47–298)]	<0.05
VLDL-cholesterol (mmol/l [mg/dl])	–	0.39 ± 0.18 (0.08–0.78) [15 ± 7; (3–30)]	0.28 ± 0.13 (0.08–0.49) [11 ± 5; (3–19)]	NS
LDL-cholesterol (mmol/l [mg/dl])	<3.89 [150]	3.08 ± 0.78 (2.02–4.43) [119 ± 30; (78–171)]	3.6 ± 0.96 (1.94–5.39) [139 ± 37; (75–208)]	NS
HDL-cholesterol (mmol/l [mg/dl])	>0.91 [35]	1.11 ± 0.28 (0.65–1.61) [43 ± 11; (25–62)]	0.44 ± 0.28 (0.06–1.06) [17 ± 11; (2.5–41)]	<0.001

Values are expressed as mean (minimum–maximum) ± S.D.

Table 4
Hormonal concentrations in former (ex-abusers; age 38.0 ± 7.0 years) and current (abusers; age 30.5 ± 5.0 years) anabolic steroid users

	Range of reference	Ex-abusers ($n = 14$)	Abusers ($n = 17$)	P-value (ex-A/A)
Testosterone (nmol/l)	12–30	14.6 ± 4.2 (6.6–22.2)	56.9 ± 34.7 (0.3–113)	<0.001
Sexual-hormone-binding globulin (nmol/l)	16–52	32.0 ± 14.8 (17.0–76.9)	5.2 ± 4.7 (1.0–18.5)	<0.001
Estradiol (pmol/l)	0–162	90.3 ± 30.5 (24.2–147)	455 ± 335 (0.0–1154)	<0.001
Luteinizing hormone (IU/l)	2–10	3.24 ± 1.34 (0.11–5.39)	0.21 ± 0.15 (0.06–0.62)	<0.001
Follicle-stimulating hormone (IU/l)	1–7	5.08 ± 1.86 (2.95–8.24)	0.45 ± 1.13 (0.0–4.76)	<0.001
Thyroid-stimulating hormone (mU/l)	0.5–4.6	1.66 ± 1.03 (0.32–4.15)	1.90 ± 1.11 (0.74–4.49)	NS

Values are expressed as mean (minimum–maximum) ± S.D.

FSH (by 91%) compared with ex-abusers with distinct inter-individual variations in abusers. Thirteen ex-abusers were found in the lower 20% of the range of reference for testosterone, two ex-abusers were found below the normal range with 6.6 and 9.0 nmol/l, respectively. The other hormones were normal in the group of ex-abusers. Concerning estradiol 13 abusers were partly far above the upper range of reference for men, with a maximal value of 1154 pmol/l, which is even above the upper limit value for women.

3.6. Other substrates

Serum electrolytes (sodium, potassium, magnesium, calcium), iron, urea, creatinine, uric acid, glucose and HBA1 did not show significant differences between the groups.

4. Discussion

Long-term studies about the side effects of AAS are of great importance particularly with regard to an improved health education and more effective prevention strategies. In the present study, it was the first time that a large number of strength athletes who had discontinued their formerly massive intake of AAS for more than 1 year were examined and compared with current abusers. Both ex-abusers and abusers of this study used self-administered AAS in a clearly supratherapeutic dosage over a period of several years.

The higher values with regard to the red and white blood count in abusers correspond to the stimulation of the erythropoiesis [7] and granulopoiesis [8] by androgenes with a reversible increase in the level of erythropoietin in the organism [9]. The same applies to the increase in thrombocytes in abusers in our study. Similar findings were reported by Alen [10] in strength athletes with AAS. A slight increase in white blood cells was also to be shown when administering 200 mg testosterone per week [11]. An increase in hematocrit and thrombocytes is to be regarded as critical since increased hematocrit values are correlated with an increase in the cardiovascular risk and total mortality [12] and the tendency of thrombocytes to aggregation increases in connection with testosterone [13]. In literature, there are several case reports of thrombosis in young strength athletes [14,15]. Sixteen weeks after discontinuing the intake of AAS, a hemoglobin concentration that was still above the original value was to be found [9], in another study the hemoglobin value returned to its original level after 5–6 months [15]. In our investigation, the red blood count and thrombocytes in ex-abusers had returned to normal after at least 1 year with a time-dependent decrease in leucocytes.

Toxic effects of AAS on the liver primarily due to 17-α-alkylated steroid abuse [16] have often been described and include increased enzyme activities, a cholestasis and peliosis hepatis adenoma and even unconfirmed case reports of carcinoma [3,15,17–20]. However, morphological alterations could not be diagnosed under sonography in either of our subjects.

As for the evaluation of increased transaminases in athletes, muscularly induced increases in enzymes caused by training have to be taken into consideration [19,21]. The increased CK values in abusers as compared to ex-abusers correspond to earlier findings in strength athletes [20,22]. An increased myocyte membrane permeability [22], a higher training intensity or muscle mass [23] and intramuscular injections could play a role. A rhabdomyolysis with CK values of up to 30,000 U/l [24] may be triggered by an extremely intensive bodybuilding training in combination with a simultaneous intake of AAS. As is the case in other studies with sport induced increases in enzyme activity [21], the CK-MB values of none of our subjects were higher than 6% of total CK, so that a damage to the heart muscle cannot be deduced.

Muscularly caused increases in transaminases, however, usually show an AST/ALT-ratio >1.0 [21,26], which was the case in only one ex-user in the present study. Other authors [19], however, describe that in strength athletes with or without anabolic steroid consumption ALT-values are on average higher than AST values, which was in contrast to a group of sports students showing higher AST concentrations. In our investigation, the correlation between the ALT activity of abusers and GLDH as well as with cholinesterase, which are both not influenced by physical training [21], leads to the assumption that the higher transaminase values of abusers might primarily be the manifestation of an impairment of the liver function induced by AAS.

Four of our 15 ex-abusers also showed increased ALT values above the upper limit of reference, which might be interpreted as late sequelae of the intake of AAS because these elevations could not be explained by preceding

physical exercise. The clinical importance, however, of these elevated ALT concentrations finally remains unclear. In another study, 12–16 weeks after discontinuing the intake of the preparations, the increased ALT values of AAS abusers had returned to normal compared with a steroid-free control group of strength athletes [15]. In contrast with our study, the enzyme activities, however, still remained within the clinical normal range during the intake, which possibly resulted from the distinctly lower dosages. In the study of O'Connor et al. [2], ALT and AST were within the normal range 5 years after discontinuing the intake of AAS; in this context, however, it must be considered that the last-mentioned did not train any more. As in the case of other authors [19,20], our abusers showed slightly but significantly lower gamma-GT values than the ex-abusers who, however, with the exception of one athlete, were in the normal range. We do not have an explanation for this phenomenon.

A decrease in HDL-cholesterol with distinct increase in hepatic triglyceridlipase as well as decrease in lipoprotein (a) are one of the most frequently documented side effects of an AAS [3,25–27], with individually very low concentrations of 0.05 mmol/l [28] as in one of our abusers. As regards the behavior of total, LDL-cholesterol as well as triglycerides, the results in literature are inconsistent. The administration only of testosterone does not induce a significant increase in this enzyme, while 17-α-alkylated AAS as well as not or only little aromatizable anabolics show the strongest effect [27].

The data of various authors suggest that the alterations in the lipid profile are reversible after some weeks to 3–5 months [25–30]. In our ex-abusers, the HDL-cholesterol concentrations were within the normal range at least 1 year after the discontinuation, with the exception of one athlete, which might have been genetically caused.

A frequent side effect of the intake of some AAS is the transformation into estrogene compounds in the fat tissue and other body compartments. The resulting increase in the blood estradiol level [31] may also cause gynecomastia [20,26], which was the case in two-third of our subjects.

The carrier protein SHBG also considerably decreased in our abusers was within the normal range again in ex-abusers. Sixteen weeks after discontinuing the intake of AAS, SHBG-values not having returned to normal are reported [32]. After the discontinuation of AAS or after a therapy with testosterone enanthate for the inhibition of growth in juveniles, there seems to be a phase of transient hypogonadism of several months [32] or even years [32,33], while the gonadotropin values were within the normal range [33,34].

It should be considered that statements regarding the reversibility of medication effects are less valid in the case of a cross-sectional study. However, longitudinal studies have not been performed up to now. The AAS-score can only be the attempt to quantify the anabolic substances taken to make at least vague statements regarding a dosage–effect-correlation. A clear transfer from damage potential to single substances is not possible.

It is concluded that at least 1 year after discontinuation, the negative effects of a massive consumption of AAS on blood count, lipid metabolism, most liver function and hormones returned to normal. In some ex-abusers, however, increased ALT activities were to be found whose importance is still unclear as well as decreased testosterone concentrations. Future longitudinal studies should especially investigate the clinical significance of repeatedly impaired blood lipid profiles in AAS abusers as well as the long-term toxic and hormonal effects in subjects using lower dosages of AAS.

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