HIGHER MUSCLE MASS BUT LOWER GYNOID FAT MASS IN ATHLETES USING ANABOLIC ANDROGENIC STEROIDS

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ABSTRACT

Nordström, A, Högström, G, Eriksson, A, Bonnerud, P, Tegner, Y, and Malm, C. Higher muscle mass but lower gynoid fat mass in athletes using anabolic androgenic steroids. J Strength Cond Res 26(1): 246–250, 2012—This study evaluated the relationship between anabolic androgenic steroid (AAS) use and body constitution. Dual-energy x-ray absorptiometry was used to measure bone mineral density (BMD, g·cm⁻²) of the total body, arms, and legs. Total gynoid and android fat mass (grams) and total lean mass (grams) were measured in 10 strength trained athletes (41.4 ± 7.9 years) who had used AASs for 5–15 years (Doped) and 7 strength trained athletes (29.4 ± 6.2 years) who had never used AASs (Clean). Seventeen sedentary men (30.3 ± 2.1 years) served as Controls. Doped athletes had significantly more lean body mass (85.5 ± 3.8 vs. 75.3 ± 2.5 vs. 60.7 ± 1.9, p < 0.001) and a greater index of fat-free/fat mass (5.8 vs. 2.6 vs. 2.5, p < 0.001) compared with Clean athletes and Controls. Doped athletes also had significantly less gynoid fat mass compared with that of Clean athletes (2.8 ± 0.4 vs. 4.8 ± 0.2 kg, p = 0.02). There were no differences in BMD between the athletes (p = 0.39–0.98), but both groups had significantly higher BMDs at all sites compared with that of Controls (p = 0.01 to <0.001). Thus, long-term AAS use seems to alter body constitution, favoring higher muscle mass and reduced gynoid fat mass without affecting BMD.

KEY WORDS male, body composition, physical activity, anabolic androgenic steroids

INTRODUCTION

Anabolic androgenic steroid (AAS) use is widely spread in both professional and recreational sports, and the potential health risks are of great concern (15). Despite the widespread usage of AAS, the knowledge of the long-term effects and side effects of these substances at high doses is incomplete.

A common sought after effect of AAS is changes in body composition, with increased muscle mass and decreased fat mass. The potential effects of AAS in different tissues are modulated at the cellular level by tissue-specific steroid converting enzymes; in adipose tissue, that is, testosterone is converted by aromatase to estrogen, whereas in human skeletal muscle, testosterone binds directly to androgen receptors (13). The few studies that have investigated the effect of AAS on body composition in strength athletes have yielded ambiguous results (5,6,9,11,14,24). The variations in measuring techniques, such as skinfold measurement, bio-impedance analyses, or underwater weighing further complicates the interpretations of the results (6,9,17).

The use of AASs has been related to impaired cardiovascular function with the potential direct effects mainly exerted on the myocardium and vasculature and indirect effect via alteration in lipids and hemorrheologic properties of the blood (3). Also, case reports have linked AAS use to acute myocardial infarction and sudden cardiac death (8,23). The clinical risk factors for cardiovascular disease (CVD) reported among AAS users includes elevations in low-density lipoprotein and reductions in high-density lipoprotein (7,15). Because fat mass and regional fat adiposity in the form of gynoid and abdominal adiposity are risk factors for CVD and not previously examined in conjunction with AAS use, it is of interest to investigate these factors. The main purpose of this study was to observe the effect of the frequent use of AASs by strength athletes on body composition such as bone mineral density (BMD), muscle mass, and general and regional fat mass.
METHODS

Experimental Approach to the Problem
It is not ethical to randomize subjects to AAS usage that reflect the AAS abuse in real life. Thus, to be able to investigate the effect of frequent AAS use by strength athletes on body composition, there is a need for an observational approach. Dual-energy x-ray absorptiometery (DEXA) was chosen for investigating body composition because it is considered to be the gold standard for measurements of body composition (10,19).

Subjects
Seventeen strength trained elite athletes were recruited through personal contacts. The subjects were active in sports such as weight lifting, strong men competitions, and bodybuilding at national and international levels and had trained regularly between 4 and 6 times per week for at least 5 years. This was not an intervention study, and no actions were taken to influence the participating subjects’ exercise regime, diet, or other factors, which may all influence performance and body composition. However, it must be emphasized that this is the only ethically feasible approach to study long-term effects of AAS abuse among athletes.

All subjects were individually interviewed regarding doping substances. Seven subjects were assigned to the Clean group. These subjects had signed a contract with their local club and Swedish power lifting federation that committed them never to use any drugs, under severe monetary punishment. All athletes were highly competitive and participated regularly in national or international competitions in power events. They trained regularly 4–6 times a week, 2–3 hours per session. The sessions consisted of 4–7 sets of exercises and 3–12 repetitions per set.

Intake pattern was examined through extensive interviews and questionnaires. Ten of the athletes had used various AAS substances such as testosterone (oral and injectable anabolic steroids [250–2,000 mg wk⁻¹]), dianabol (Methandrostenolone, oral anabolic steroid [50–350 mg wk⁻¹]), deca-durabolin (Nandrolone Decanoate, injectable anabolic steroid [200–7,000 mg wk⁻¹]), boldenone (Boldenone undecylenated, injectable anabolic steroid [500–1,000 mg wk⁻¹]), and anadrol (Oxymetholone, oral anabolic steroid [175 mg wk⁻¹]) and also substances such as insulin (injected [10–12 IU d⁻¹]), insulin-like growth factor-1 (injectable growth protein [50 mg d⁻¹]), GH (Growth Hormone, injected [4–6 IU d⁻¹; 6 d wk⁻¹]), Ephedrine (oral appetite suppressor [60 mg d⁻¹]) and HCG (human chorionic gonadotropin, injectable glycoprotein hormone

<table>
<thead>
<tr>
<th>Table 1. Data for age and anthropometry.*</th>
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<tbody>
<tr>
<td>Doped (N = 10)</td>
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<td>-----------------</td>
</tr>
<tr>
<td>Age (y)</td>
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<tr>
<td>Height (cm)</td>
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<tr>
<td>Weight (kg)</td>
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*Values are given as mean ± SD.  
†Doped significantly greater than controls.  
‡Doped significantly greater than clean.  
§Clean significantly greater than controls.

<table>
<thead>
<tr>
<th>Table 2. Data for bone mass measurements.*</th>
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<tr>
<td>Bone mineral density (g cm⁻²)</td>
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<td>-----------------------------</td>
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<tr>
<td>Total</td>
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<tr>
<td>Arms</td>
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<td>Spine</td>
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<td>Pelvis</td>
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<td>Legs</td>
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*Values are given as mean ± SD.  
†Doped significantly greater than controls.  
‡Clean significantly greater than controls.  
§Data missing.
AAS Usage and Body Constitution

**Table 3.** Fat mass and fat-free mass measurements in 10 AAS using doped athletes, 7 clean athletes, and 17 controls.*

<table>
<thead>
<tr>
<th>Mass (kg)</th>
<th>Doped (N = 10)</th>
<th>Clean (N = 7)</th>
<th>Controls (N = 17)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total fat</td>
<td>18.1 ± 2.6</td>
<td>29.6 ± 2.6</td>
<td>25.4 ± 1.6</td>
<td>0.04 (ANOVA)</td>
</tr>
<tr>
<td>Android fat</td>
<td>1.9 ± 0.3</td>
<td>3.0 ± 0.3</td>
<td></td>
<td>0.17 (t-Test)</td>
</tr>
<tr>
<td>Gynoid fat</td>
<td>2.8 ± 0.4</td>
<td>4.8 ± 0.2</td>
<td></td>
<td>0.02 (t-Test)</td>
</tr>
<tr>
<td>Fat-free (muscle)</td>
<td>85.5 ± 3.8§</td>
<td>75.3 ± 2.5§</td>
<td>60.7 ± 1.9†</td>
<td>&lt;0.001 (t-Test)</td>
</tr>
<tr>
<td>Ratio fat-free mass/fat mass</td>
<td>5.8§</td>
<td>2.6§</td>
<td>2.5‡</td>
<td>&lt;0.001 (t-Test)</td>
</tr>
</tbody>
</table>

*AAS = anabolic androgenic steroid; ANOVA = analysis of variance.
†Values are given as mean ± SD.
§Doped significantly greater than controls.
||Doped significantly greater than clean.
||Clean significantly greater than controls.

[10,000 IU total]). The substances had been used in different combinations and doses for a period of 5–15 years; thus, the exact doses are not possible to determine.

The steroid regimen included both "stacking" (simultaneous use of several types of AAS) and "cycling" (a drug-free period followed by times when doses and types of drugs taken were initiated or increased). Intake usually follows a pyramid schedule with increased intake over time to avoid equation of AAS levels.

The control group’s weight-bearing physical activity consisted of playing soccer and floor ball, distance running, and some weight training. An inclusion criterion for the controls was that their total average amount of weight-bearing physical activity was estimated to <4 h wk⁻¹. None of these subjects participated in any regular or organized training. The participants all gave informed written consent, and the Ethics Committee of the Medical Faculty, Umeå University, Umeå, Sweden, approved the protocol.

**Measurements**

**Anthropometry.** Weight and height were measured in light clothing without shoes. Weight was measured to the nearest 0.1 kg with a digital scale (Seca, Ergonordic AB, Bromma, Sweden) and height to the nearest centimeter with a wall-mounted scale.

**Physical Activity of Athletes.** Physical activity was evaluated by using a questionnaire and was defined as the self-reported mean hours of training each week during the past year. These 2 groups’ physical activity consisted exclusively of weight training. The questionnaire used included questions on smoking habits, known illnesses, and medication intake.

**Body Composition.** The total body mass density (BMD; grams per centimeter squared) and total bone mineral content (BMC; grams), total fat mass (grams) gynoid and android fat mass (grams), and total muscle mass (grams) were measured using a Lunar Prodigy or a Lunar DPX-L (GE-Lunar, GE Healthcare) dual-energy x-ray absorptiometer and software version 4.6e. Dual-energy x-ray absorptiometry has previously been validated in adults and in the elderly and considered to be a reliable and valid method for measuring fat and lean mass (10,19) and can be considered the gold standard for measurements of body composition. The coefficient of variation was determined to be 0.7–1.2% by scanning 1 person 5 times on the same day, with repositioning between each scan. The equipment was calibrated each day by using a standardized phantom to detect drifts in the measurements and to test machine functions. The equipment was also evaluated regularly during the study using a spine phantom. No drifts were detected.

**Statistical Analyses**

Differences among the 3 groups were investigated using univariate analysis of variance with adjustment for age. SPPS for Mac (version 18.0) was used for statistical analyses, and analyzing differences between 2 groups was done by an independent T-test with Bonferroni’s post hoc test. A p value ≤0.05 was considered significant.

**Results**

**Physical Characteristics**

Data for age and anthropometric data are presented in Table 1. Because the 3 groups Doped, Clean, and Control differed in age (41.4, 29.4, and 30.3 years, respectively), all data are adjusted for age. There were no differences in height between the groups. Both groups of strength athletes had a higher body weight compared with that of Controls (Doped 107.4 ± 5.5, Clean 108.6 ± 4.7, and Controls 89.9 ± 3.2 kg; p = 0.002). There were no major differences in training characteristics between the 2 groups of strength athletes, but the Doped group consisted mostly of strong men and body builders and the Clean group of power lifters.

**Body Composition**

Data on BMD are presented in Table 2. There were no differences in BMD at any measured sites between the Doped
and Clean athletes, but both groups had a higher BMD at all measured sites compared with Controls ($p < 0.01$).

The 2 groups of athletes had more fat-free mass compared with that of Controls (Doped $85.5 \pm 3.8$, Clean $75.3 \pm 2.5$, and Controls $60.7 \pm 1.9$ kg; $p = 0.002$). Doped athletes also had a higher percent fat-free mass ($85.5 \pm 3.8\%$ vs. $75.3 \pm 2.5\%$; $p = 0.002$), lower percent gynoid fat ($2.8 \pm 0.4\%$ vs. $4.8 \pm 0.2\%$; $p = 0.02$), and a higher fat-free mass/fat mass index ($5.82 \pm 2.91$ vs. $2.53 \pm 0.62$; $p < 0.001$) compared with that of Clean athletes.

**DISCUSSION**

The novel finding in our study is the clear difference in lean and fat mass ratio seen between Doped and Clean athletes and Controls. Furthermore, there was also a trend toward less total fat mass in Doped athletes compared with that in Clean strength athletes ($p = 0.04$). The effects of Doping were separated from the effects of strength training based on the lean to fat ratio. Because there was no difference between Clean and Controls, but a higher ratio in Doped, AAS use seems to alter the ratio between muscle and fat mass favoring a greater muscle mass or lower fat mass (Table 3).

In general, there seems to be an alteration in fat to lean body mass ratio with long-term supraphysiological doses of AAS, favoring lean body mass. Our results are supported by experimental research that suggests that androgens can affect body composition in 2 distinct ways, by increasing fractional muscle protein synthesis and both type 1 and 2 muscle fibers and also by mechanisms directing the pluripotent mesenchymal stem cell toward myogenic lineage rather than toward adipogenic lineage (4).

To our knowledge, this is the first observational study investigating the effects of AAS abuse on body composition compared with 2 sets of controls, both nondoped athletes and sedentary individuals. Our study shows higher levels of lean mass in Doped athletes, which corroborate data in a previous study using the DEXA technique by Van Marken Lichtenbelt et al. (18). An increase in lean body mass with AAS has been demonstrated using indirect measurements; the results have, however, been indefinite (5,6,9,11,12,14,24).

Studies have indicated that anthropometric measures of regional fat distribution may be a better predictor of both the risk of myocardial infarction (25) and stroke (22) than BMI, suggesting that the distribution of fat rather than general obesity should be considered as a risk factor. Furthermore, previously, we have found that gynoid fat mass is negatively associated with both myocardial infarction and cardiovascular risk factors (20,21).

Thus, the lower gynoid fat mass in Doped strength athletes could be one of the underlying mechanisms of the higher incidence of CVD previously found in doped athletes (1,2,16).

A limitation in our study is its observational nature. The subject’s use of AAS did not follow any medical protocol, and the subjects admitted using a mixture of AAS with varying regimens, both oral and injections. Because no designed study can reflect the use of AAS in real life, our study more adequately reflects the actual effects of illegal usage of AAS better than a designed longitudinal study does. Another drawback of this type of studies is the fact that because the AASs were purchased illegally and were unavailable for analysis, it was not possible to determine the actual doses the subjects were exposed to. This is however the reality of AAS abuse in both professional and recreational sports, and the design can thus also be viewed as strength.

It is clear that the use of AASs will alter body composition beyond what is achieved by strength training alone, mainly by increasing fat-free (muscle) mass. The observed decrease in gynoid fat, a known risk for cardiovascular disease, indicates that the mechanism behind the known increased risk of CVD with AASs may be related to fat redistribution.

**PRACTICAL APPLICATIONS**

Increased percent lean body mass and decreased adipose tissue appear to be a result of long-term AAS abuse. This is advantageous in several sports spanning from bodybuilding to distance running. The use of AAS can be expected also in sports not aimed at maximizing muscle strength and mass, and doping controls should be appropriately executed and analyses for AAS included also for endurance sports. Coaches should be aware that rapid increases in muscle-to-fat ratio despite a maintained training regimen may indicate AAS use.

It is apparent that many AAS users are never caught in doping controls today, mainly because of the development of novel, to date, untraceable substances and novel ways of administration. Thus, the need to investigate both direct and indirect measurements for AAS use should be considered. One such noninvasive method, demonstrating the effects of doping, is altered muscle-to-fat ratio.

**REFERENCES**


