

Anabolic-Androgenic Steroids

G. Gregory Haff, PhD, CSCS,*D, FNSCA



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Introduction

In 1974 the International Olympic Committee's (IOC) Medical Commission prohibited anabolic-androgenic steroid (AAS) use in Olympic sports, with testing of athletes commencing in 1976 (21). Even though the IOC prohibited AAS the illicit use of AAS continued (11). In fact Franke and Berendonk (11) report that in 1974 the German Democratic Republic (GDR) passed a bill that made 1) AAS use an integral part of the training process by athletes, 2) AAS use tightly controlled, centralized, and evaluated by sporting physicians, 3) AAS distribution controlled by Sportmedizinischer Dienst (Sports Medical Service), 4) research on and development of AAS a priority, 5) education about AAS use an integral part of coaching and medical education, and 6) the GDR AAS practices a state secret. Recently, the Bay Area Laboratory Cooperative (BALCO) scandal which shed light on a systemized doping program that utilized designer steroids such as tetrahydrogestrinone (THG) suggests not much has changed since the state sponsored doping programs seen in the former GDR (23, 24). To most the revelations that a subculture of athletes that are using AAS and other pharmacological aids with the explicit attempt to improve performance still exists seems shocking. With these revelations the popular media has become flooded with sensationalized information that is often based upon non-scientific information. Therefore the purpose of this brief review is to examine 1) what AAS are, 2) the types of AAS, 3) how AAS are administered, 4) the physiological effects of AAS, and 5) the potential side-effects associated with AAS.

What are Anabolic-Androgenic Steroids?

Anabolic-androgenic steroids (AAS) are synthetic compounds which are based upon the structure of testosterone (40). Testosterone is formed from cholesterol via a series of enzymatic reactions in the Leydig cells of the testes and adrenal cortex in men, while in women the primary site of formation is the adrenal cortex (14). In general, testosterone promotes the development and maintenance of secondary male sexual characteristics, which are known as androgenic effects, and the promotion of muscular growth or anabolic effects (39).

When testosterone is administered it is rapidly degraded and only small amounts reach the systemic circulation thus creating an environment in which effective levels of the drug are not maintained (47). Therefore, testosterone would have to be injected multiple times a day or week in order to be effective. Because of the limited effectiveness of testosterone, synthetic compounds must be created in order to receive the potential benefits of testosterone.

Synthetic compounds are generally created by modifying the base molecule of testosterone in order to 1) delay the degradation of the AAS in order to maintain blood levels of the drug for prolonged time periods, 2) to intensify the overall anabolic effects of the compound while limiting the androgenic effects, and 3) overpower the catabolic pathways by supplying the drug in mass quantities. When new AAS are created, in order to meet the above mentioned goals, the modifications to the base structure of testosterone result in alterations of the anabolic to androgenic ratio and this resultant change can impact the potential for side effects that can occur as a result of the drug (38). To date no AAS have been created that completely avoid the androgenic effects while magnifying the anabolic potency of the drug (26). The most commonly used AAS and their method of administration are presented in Table 1.

Types of Anabolic-Androgenic Steroids?

There are three basic modification classifications that are made to the base structure of testosterone in order to create AAS (figure #1). Each modification to testosterone results in distinct changes in the deliverability, potency, and/or adaptive responses to the AAS (17).

Class I modifications generally result in the creation of AAS that are injectable (17), but methenolone acetate and testosterone undecanoate are two Class I modifications that can be taken orally (47). This classification of modifications to testosterone are generally designed in order to cause a slower absorption rate intra-muscularly, but can result in more pronounced androgenic properties depending upon the specific chemical modifications made. Depending upon which specific chemical modifications that are made to the base molecule of testosterone the Class I AAS may only need to be injected every 2-12 weeks (3, 17). Once injected the AAS peak rapidly and then degrade gradually to baseline levels at the time of the next injection (2, 17).

Class II modification to the base structure of testosterone result in the formation of AAS which can be taken orally and have depressed hepatic (liver) degradation rates (17, 47). The oral administration of AAS can be accomplished via two mechanisms: 1) absorption from buccal mucosa (cheek area of mouth) or 2) swallowing the drug (47). Class II modified compounds are considered to be weaker than the injectable Class I AAS and more prone to causing hepatic (liver) complications (2).

Class III modifications have been used to create both oral and injectable formulations (47). Generally, the modification of the base testosterone molecule is undertaken in order to accomplish: 1) a slowed rate of inactivation, 2) enhanced drug potency, or 3) a change in the drugs metabolism (47). One example of a class III modification results in an AAS with similar properties to that of Class II AAS, but results in a decreased or nonexistent hepatic effect (17).

How are Anabolic-Androgenic Steroids Administered?

Generally, AAS can be administered in several ways 1) oral consumption, 2) intramuscular injection (oil or water based), 3) transdermal (patches, gels and or creams), 4) nasal sprays, and 5) implantable pellets (2, 17). Regardless of the delivery mechanism AAS users tend to use dosages which can exceed the typical medical dosages of 6-10 mg.d-1 by 40-100 or more times (17, 38). It is not uncommon for athletes to take an average of five different AAS, both oral and injectable drugs, at the same time (38).

When AAS users take multiple drugs at the same time (also known as “Stacking”), they develop complex multi-drug regimes in which multiple forms of AAS with various methods of administration are used in order to utilize each drugs unique absorption, metabolic, and excretion properties in an attempt to magnify the effectiveness of each of the drugs administered (2, 17, 20, 38). Conceptually, the idea behind “stacking” several AAS is done in order to maximize the binding and activation of multiple steroid receptor sites (17). It is important to note that no scientific evidence has been reported that supports this concept (38). It is also important to note that AAS users also engage in administering AAS in a cyclical fashion (17, 38). Cyclic drug administration is marked by varying periods of drug administration followed by periods of none use or periods of staggered AAS administration (38).

The cycling of AAS is generally undertaken in order to avoid a “plateau” or tolerance to a specific drug (17, 38). In some instances athletes will employ an administration protocol which utilizes a pyramidal drug schedule where the dosages of AAS are increased across the drug cycle (12). The pyramid drug schedule can also be “stacked”, with several drugs being utilized with varying dosages across the drug cycle (1). After completing the ascending portion of the pyramid, AAS users may go through period of 1-12 months of what is termed a “drug free holiday” (17). This “drug free holiday” is undertaken for 1-12 months in an attempt to reduce the potential for side-effect, promote recuperation of various hormonal systems, or avoid drug detection during competition (30).

What are the Physiological Effects of Anabolic-Androgenic Steroids?

The administration of testosterone or AAS to hypogonadal men (i.e. someone with low testosterone levels) (5, 7, 22, 35, 46), HIV patients (32), and dosages that typically exceed those that are typically recommended to eugonadal men (i.e. someone with normal testosterone levels) (4, 9, 10, 15) can result in increases in fat-free mass, muscular size, and muscular strength (Table 2). When testosterone or AAS administration is coupled with resistance training these effects are magnified (4).

In the seminal study performed by Dr. Shalender Bhasin and colleagues (4) high dosages of testosterone enanthate were administered to forty health eugonadal men who ranged in age from 19-40 years. The truly unique aspect of this study is that the 40 men were randomly divided into four equal groups: 1) administered AAS and resistance trained; 2) administered AAS and no resistance training, 3) administered a placebo and resistance trained, and 4) administered a placebo and no resistance training. Each of the subjects in the groups that were given AAS received a weekly intramuscular injection of 600 mg of testosterone enanthate while the subjects in the other groups received a weekly injection of a placebo across the 10 weeks of the study. The men who were administered testosterone enanthate and resistance trained experienced significantly greater increases in body mass (+6 kg), fat free mass (+6.1 kg), quadriceps area (+1174 mm²), triceps area (+501 mm²), 1-RM bench press (+22kg), and 1-RM back squat (+38 kg) when compared to the other groups. Interestingly, the group that received AAS and did no resistance training exhibited increases in body mass (+3.5 kg), fat free mass (+3.2 kg), 1-RM bench press (+12 kg), and 1-RM back squat (+13 kg). Bhasin et al. (4) reported that no side-effects were noted across the duration of this investigation. This investigation suggests that high dosages of testosterone enanthate stimulate significant positive alterations in fat-free mass, muscle size, and muscular strength especially when taken in conjunction with a progressive resistance training program.

It appears that the magnitude of these positive adaptations has a dose response relationship (6). Bhasin et al. (6) administered a long-acting gonadotropin-releasing hormone (GnRH) agonist monthly for 20 weeks in order to suppress endogenous testosterone production in 61 eugonadal men. The 61 men were randomly assigned to one of five treatment groups in which they received a weekly intramuscular injection of 25, 50, 125, 300 or 600 mg of testosterone enanthate. The results of this investigation suggest that the greatest changes in fat free mass and muscular strength occurred in the treatment groups receiving higher dosages of testosterone enanthate. However, higher dosages were correlated to greater decreases in high density lipoprotein cholesterol and overall fat mass.

Based upon the contemporary body of scientific literature it appears the ergogenic effects associated with AAS are magnified when higher dosages are administered in conjunction with a progressive resistance training program (4, 6).

What are the Common Side-Effects Associated with Anabolic-Androgenic Steroids?

When looking at the adverse effects of AAS, it is very difficult to define these occurrences with any precision because of a lack of clinical trials that actually mimic what athletes are actually using (17). Sturmi and Diorio (38) suggest that the vast majority of side effects associated with AAS use are overstated and in most incidences would be considered minor and reversible following the cessation of drug use (table 3). However, it is important to note that the effects of long term use of excessively high dosages of AAS are virtually unknown.

Generally, the physiological side-effects associated with AAS use can be broken down into four distinct categories: 1) Cardiovascular, 2) Liver, 3) Reproductive, and 4) Dermatologic

Cardiovascular: When looking at the cardiovascular system AAS use appears to promote an unfavorable lipid profile in which low density lipoproteins (LDL)(bad cholesterol) are elevated while high density lipoproteins are suppressed (HDL) (good cholesterol) (17, 18). The decline in HDL's are most evident with the use of oral administration of AAS with drugs such as stanozolol, oxymetholone, and metandienone (18, 41) and can often be seen in a few days after the initiation of AAS use (41). These adverse effects to the lipoprotein profiles of AAS may result in an increased exposure to cardiovascular maladaptations but further investigation is warranted in order to fully understand this relationship.

Another potential cardiovascular side-effect associated with AAS is a more pronounced increase in left ventricular hypertrophy when compared to non-AAS users (18, 41, 45). Urhausen et al. (45) have demonstrated that ex –AAS users and current AAS users have increased left ventricular mass and wall thickness. Interestingly these effects seen in current AAS users do not disappear after a minimum of 1 year of the cessation of AAS use (45). These alterations in the cardiac tissue also appear to be related to an increased risk of arrhythmias (i.e. irregular heart beats) (8). Legros et al. (25) suggest that some of the maladaptations to the cardiac tissue may result from an increased stiffness which may be a result of increased collagen cross links. Based upon this body of evidence it is often suggested that AAS use increases the risk of stroke and myocardial infarction (42).

Liver: Typically liver enzymes, such as alanine and aspartate-aminotransferase, are altered as a result of AAS use. These maladies are usually seen with oral AAS use (8). In extreme cases liver failure (8), peliosis hepatitis (blood filled cystic lesion in the liver) (19), hepatocellular carcinoma (malignant liver tumors) (27), and hepatocellular adenoma (abnormal tissue growth in the liver) (17) have been noted with the use of AAS.

Reproductive: When looking at the reproductive function of men the most striking side-effect to excessively high dosages of AAS is a reduced quantity and quality of semen production which could be related to infertility (43, 44). Long-term AAS administration could potentially result in testicular atrophy (i.e. decreased size) and other effects associated with hy-

gonadotrophic hypogonadism (a decreased hormone release from the gonads) (18, 28). Additionally, when excessively high dosages of AAS are taken by men they can develop excessive breast tissue growth (termed gynaecomastia) as a result of the increase conversion of the AAS to estrogen (31). Typically, tamoxifen is taken by athletes in an attempt to prevent the occurrence of excessive breast tissue growth even though no research data exists to support the efficacy of this practice (18). Interestingly, many of these reproductive maladies can take more than a year to recover from (18).

Very little research exploring the effects of AAS on the reproductive function of women exists. However, the administration of AAS to female body builders has been linked to clitoral hypertrophy, menstrual irregularities, and a reduction of the breasts (18). Unlike their male counterparts, it appears that many of the effects seen in women are not reversible.

Dermatologic: When looking at the common dermatological side-effects acne cutaneous striae (stretch marks), oily skin, alopecia (complete loss of hair), and male pattern baldness are commonly noted with AAS use (29). High dosages of AAS increase skin surface lipids and the cutaneous population of propionibacteria acnes (is a type of bacteria) which results in an increase occurrence of acne (33). The occurrence of cutaneous striae (stretch marks) most likely is a result of a reduction of skin elasticity which does not allow for the skin to stretch as rapidly as the increases in body mass. Another potential dermatologic side-effect seen in women who use AAS is the occurrence of hirsutism (excessive and abnormal hair growth) (29). Hirsutism is marked by increased facial and body hair growth (8).

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Table 1 • Generic and Trade Names for Common Anabolic Steroids • Note: adapted (13, 16, 17, 36)

Generic Name	Trade Names	Means of Administration
Danazol	Danocrine	Oral
Ethylestrenol	Maxibolin	Oral
Fluoxymesterone	Halostestin	Oral
	Ultandren	Oral
	Android-F	Oral
Mesterolone	Proviron	Oral
	Mestranum	Oral
Methandienone, Methandrostenolone	Dianabol	Oral
Methyltestosterone	Oreton Methyl	Oral
	Android	Oral
	Testered	Oral
	Virolin	Oral
Minolone	Cheque, Drops	Oral
Oxandrolone	Anavar	Oral
	Oxandrin	Oral
Oxymetholone	Anadrol-50	Oral
	Hemogenin	Oral
Stanozolol	Winstrol	Oral
Boldenone Undecylenate	Parenabol	Injectable
	Equipoise	Injectable
Dromostanolone	Drolban	Injectable
Methanolone Enanthate	Primoblan-Depot	Injectable
Nandrolone Decanoate	Anabol	Injectable
	Deca Durabolin	Injectable
Nandrolone Phenpropionate	Durabolin	Injectable
Nandrolone Undecanoate	Dynabolan	Injectable
Stanozolol	Winstrol-V	Injectable
Testosterone Cypionate	Depo-Testosterone	Injectable
Testosterone Enanthate	Delatestryl	Injectable
	Sustanon	Injectable
Testosterone Esters Blends	Sustanon	Injectable
	Sten	Injectable
Testosterone Propionate	Oreton	Injectable
	Testoviron	Injectable
	Androlan	Injectable
Testosterone Undecanoate	Andriol	Injectable
	Restandol	Injectable
Trenbolone Acetate	Prabolan	Injectable
Trenbolone Hexahydrobenzylcarbonate	Parabolan	Injectable
Testosterone	Androderm	Transdermal
	Androgel	Transdermal
	Testim	Transdermal
	Testoderm	Transdermal

Table 2 • Effects of Increasing Testosterone Levels with Anabolic-Androgenic Steroids. (4, 9, 10, 15, 34)

Potential Positive Effects
Fat Free Mass
Muscular Strength
Fat Mass
Hypertrophy of Type II Fibers
Hypertrophy of Type I Fibers
in Type II Myonuclear Number

Note: adapted from (4, 9, 10, 15, 34)

Table 3 • Potential Side-Effects from Anabolic Androgenic Steroid Use

General Side Effects	
Acne Cutaneous Striae (stretch marks)	Infertility
Aggression	Left Ventricular Hypertrophy
Alopecia (Hair Loss)	LDL Cholesterol
Blood Pressure	Libido Changes (Altered sex drive)
Cardiac Arrhythmias (abnormal heart beat)	Myocardial Elasticity (the heart muscle in its elasticity)
HDL Cholesterol	Myocardial Hypertrophy (heart size)
Hepatocellular Adenoma (abnormal tissue growth in the liver)	Peliosis hepatitis (blood filled cystic lesions in the liver)
Hepatocellular Carcinoma (malignant liver tumors)	Risk of Stroke
Hypothyroidism (thyroid hormone production)	Risk of thrombosis (clot formation)
Side Effects Specific to Men	
Gyneocomastia (breast tissue growth)	
Sperm Production	
Prostate Hypertrophy (prostate size)	
Testicular Atrophy (testicle size)	
Side Effects Specific to Women	
Altered Menstruation	Deepening of voice
Breast Size	Hirsutism (excessive abnormal hair growth)
Clitoromegaly (clitoral size)	Masculinization

Note: Adapted from (8, 17, 25, 37, 45)

Figure 1 • Three Types of Modifications of Testosterone used To Produce AAS.

Note: Adapted from Wilson (47) and Haff (16)

