Androgen Use by Athletes: A Reevaluation of the Health Risks

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Abstract/Résumé

It has been estimated that 1 to 3 million male and female athletes in the United States have used androgens. Androgen use has been associated with liver dysfunction, altered blood lipids, infertility, musculotendinous injury, and psychological abnormalities. Although androgens have been available to athletes for over 50 years, there is little evidence to show that their use will cause any long-term detriment; furthermore, the use of moderate doses of androgens results in side effects that are largely benign and reversible. It is our contention that the incidence of serious health problems associated with the use of androgens by athletes has been overstated.

Selon des estimations, il y aurait entre un et trois millions d'athlètes (hommes et femmes) aux États-Unis qui consomment des androgènes. Leur usage est associé à des problèmes hépatiques, à une modification du profil sanguin lipidique, à l'infertilité, à des blessures musculotendineuses et à des troubles psychologiques. Bien que les athlètes aient accès aux androgènes depuis plus de 50 ans, il y a peu d'évidence à l'effet que leur usage soit la cause de préjudice à long terme; en outre, les effets secondaires associés à une consommation modérée d'androgènes sont généralement bénins et réversibles. Nous croyons que l'incidence de sérieux problèmes de santé associés à l'usage des androgènes est exagérée.

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Introduction

Androgens have been extensively used as an ergogenic aid by athletes (Wilson, 1988; Wilson and Griffin, 1980). Their use began in 1954 when John B. Zeigler, a physician for the U.S. weightlifting team, was told by a Russian team physician that the Russian lifters were using androgens (Cowart, 1987). In 1955, Dr. Zeigler introduced the United States weightlifting team to a modified, synthetic testosterone molecule called methandrostenolone. It was marketed by the Ciba Pharmaceutical Company under the trade name Dianabol. With this discovery, the merging of chemistry and sport had begun. By 1964, nearly all of the American-made steroids were commercially available: oxymetholone, nandrolone decanoate, fluoxymesterone, oxandrolone, stanozolol, and several testosterone esters (propionate, enanthate, undecanoate, and cypionate). Other countries soon followed suit with their own production and marketing of modified testosterone molecules, often referred to as anabolic steroids.

Among steroid users, conventional wisdom maintains that androgens differ somewhat in their effects. This has lead to the assumption that steroid use is optimized by using different combinations, referred to as *stacking*, over a course of on and off periods of use called *cycles* (Hill et al., 1983). Furthermore, many athletes self-administer levels of these drugs that are 10–200 times higher than therapeutic levels in an attempt to maximize the anabolic effects (Cohen and Hickman, 1987; Kantor et al., 1985).

Much of the literature that has reported adverse reactions associated with androgens has been primarily derived from the medical use of androgens. These findings have questionable relevance to athletes whose stacking and cycling dosage regimens differ tremendously from those employed in medical use (Kibble and Ross, 1987). The problem of studying the adverse effects of androgens in athletes is confounded by the fact that these effects depend highly on individual responses to specific dosages and drugs (Freinhar and Alvarez, 1985). Further, since these drugs are usually obtained illegally, and admitted use can cause disqualification from athletic events, the adverse effects associated with androgens may be under-reported (Kibble and Ross, 1987).

There has been little systematic investigation in women and children concerning androgen use; therefore, this review will focus primarily on the effects of androgens in adult males. We conducted a Medline computer search on scientific literature published in the English language dating back from 1966 to the present, and cross-referenced the following words: *androgen*, *anabolic steroid*, *exercise*, and *athlete*. Only those articles pertinent to the athletic use of androgens were chosen for this review.

Prevalence of Use

Presently, an estimated 1 to 3 million male and female athletes in the United States have used androgens (Goldstein, 1990; Phillips, 1991). The greatest use of androgens is not by competitive athletes, but by recreational bodybuilders who take

them for cosmetic reasons (i.e., increase lean body mass) (Lamb and Williams, 1991).

Buckley et al. (1988) investigated steroid use among male high school seniors in 46 public and private high schools from 24 states and found that ~6–7% of 12th-grade male students use or have used androgens. Pope et al. (1988) examined steroid use among 1,010 college aged males and 147 varsity athletes and found that 2% and 9.4%, respectively, had used androgens. Windsor and Dumitru (1989) found that 3% of high school students surveyed had used androgens.

Few studies have examined the prevalence of androgen use among national or world-class bodybuilders, powerlifters, or Olympic lifters. Frankle et al. (1984) conducted a study in which 250 weight lifters were interviewed from the metropolitan Chicago area. Of these, 110 admitted using androgens. McKillop (1987) surveyed 41 amateur bodybuilders in a Scottish gymnasium and found that 20% of these individuals had used androgens. Lindstrom et al. (1990) surveyed 138 Swedish male bodybuilders and found that 38% had used androgens for a median duration of 2 years. Seventy-five percent used androgens for bodybuilding competition, and the remainder used the drugs to improve their sense of well-being. Furthermore, a recently retired professional bodybuilder (Mr. Universe, Mr. Olympia runner-up) believes that 99% of male and female bodybuilders competing at the international level use androgens to enhance muscularity (L. Labrada, personal communication, July, 1995) (Table 1). Further, because these drugs are illegal, it is often impossible to get athletes to admit their use, since this may cause disqualification from athletic events and possibly prosecution for possessing a controlled substance.

Hepatic Effects

Androgens are almost exclusively metabolized by the liver (Wilson and Griffin, 1980). It is for this reason that the liver is particularly susceptible to the toxic

Table 1 Prevalence of Androgen Use

Investigators	Subjects	% Use
Frankle et al. (1984)	250 weightlifters from Chicago area	44
McKillop (1987)	41 Scottish amateur bodybuilders	20
Buckley et al. (1988)	3,403 12th-grade males from 24 states	6.6
Pope et al. (1988)	1,010 college-age males,	2
	147 varsity athletes	9.4
Windsor & Dumitru (1989)	901 high school students from Texas	5.0 (male)
		1.4 (female)
Lindstrom et al. (1990)	138 Swedish bodybuilders	38
Labrada (1995, personal communication)	Male and female world-class bodybuilders	99

effects of both long-term and high-dose androgen administration. A strong association exists between the 17-alkylated androgens and liver function abnormalities (Friedl, 1990). The link between hepatocellular carcinoma and androgen ingestion was originally described in 1967 in a male patient with cirrhosis of the liver (Recant and Lacy, 1965). Temporary liver disturbances are common in athletes who use oral androgens (Overly et al., 1984). The most commonly seen liver abnormalities are bromsulfalein retention, increases in plasma alkaline phosphate, and conjugated bilirubin (Payne, 1975). Liver disturbances usually return to normal after the athlete stops the steroid regimen; moreover, serious hepatic effects are unusual (Wilson, 1988).

As Kibble and Ross (1987) suggested in their review of the literature, compromised liver function is often hard to diagnose in athletes since intense weight lifting alone can elevate nonspecific liver function test levels, such as aspartate aminotransferase and alanine aminotransferase. Further, these test levels can also be elevated by androgen treatment in the absence of intense exercise (Haupt and Rovere, 1984; Kibble and Ross, 1987). However, in addition to aspartate aminotransferase and alanine aminotransferase, liver dysfunction can also be shown by elevated plasma levels of bilirubin, alkaline phosphatase, and the liver-specific isoenzyme of lactate dehydrogenase (Kibble and Ross, 1987).

The majority of the adverse effects in the liver associated with androgen use, including peliosis hepatis, hepatomas and hepatic cholestasis, appear to be linked to orally active 17-alkylated androgens. Of the adverse hepatic effects, only raised liver-function tests values and cholestasis are reversible after discontinuing the drug (Frankle et al., 1988; Haupt and Rovere, 1984). Continued androgen use over prolonged periods of time may result in cholestatic jaundice, which may lead to fatal liver cirrhosis if the condition remains untreated (Limbird, 1985; Soe et al., 1992).

Among the more serious side effects of androgen use occurring in the liver is peliosis hepatis. Peliosis hepatis is characterized by localized or diffuse blood-filled lesions in the liver (Cabasso, 1994). This condition may lead to the dilatation of liver veins and sinusoids, and blood clusters between the hepatocytes. Ultimately, it may result in the rupture of hepatic cysts with haemoperitoneum and liver failure, causing the death of the patient. Peliosis hepatis can occur at any time during androgen use and, unlike most adverse effects associated with androgens, does not appear to be dose related (Soe et al., 1992).

Haupt and Rovere (1984) reported 23 cases of peliosis hepatis linked to the use of androgens. All 23 patients had been treated with androgens for medical illness. In almost every case of peliosis hepatis reported by these investigators, the disease was linked to the use of orally active androgens. However, Soe et al. (1992) reported 10 cases of peliosis hepatis due strictly to the use of non-17-alkylated androgens. There has only been one reported case of peliosis hepatis in an athlete due to androgen use. Cabasso (1994) reported that a 27-year-old male developed peliosis hepatis after using oxandrolone, methandrostenolone, nandrolone, and testosterone for a 5-week period. His pattern of androgen use prior to this 5-week cycle, however, is unclear.

In a summary of case reports of hepatocellular carcinoma, only one was reported in an athlete who ingested oxymethelone, a 17-alkylated androgen, for 60 months (Goldman, 1985). All other cases involved treatment for medical conditions such as aplastic anemia, hypogonadism, cryptorchidism, and hypopituitarism (Friedl, 1990). According to Lukas (1993), approximately half of the androgen-related hepatic tumors were not supported histologically or proved to be associated with Fanconi's anemia. Haupt and Rovere (1984) in their review list 36 cases of hepatic tumors (34 malignant) attributed to androgen use. However, of the 36, only one was an athlete, with the remainder being patients treated with androgens for medical purposes. These patients reportedly were treated with androgens for prolonged periods of time, with 23 receiving continuous treatment for over 2 years.

Overly et al. (1984) reported a male bodybuilder who had used several different oral and injectable androgens for 4 straight years before dying of hepatocellular carcinoma and hepatic cholangiocarcinoma. In addition, Creagh et al. (1988) recounted the case of a 27-year-old bodybuilder who died from a ruptured liver tumor. He reportedly had been taking androgens for at least 3 years; however, the dosages used and the pattern of use were not reported (Table 2).

In a controlled study in which the androgens were prescribed by medical doctors, the side effects of these compounds were transient (Millar, 1994). A total of 169 athletes received the androgens for a 3- to 5-year period and were monitored for side effects during the course of administration. All athletes were first put on a 7-week steroid program that called for the ingestion of 140 mg per week of methenolone acetate. After the initial course of steroids, a variety of androgens were prescribed: oxymetholone, testosterone undecanoate, nandrolone decanoate, testosterone esters, and testosterone ester/nandrolone decanoate. There were no significant changes in liver function as determined by lactate dehydrogenase or alkaline phosphatase levels. High-density lipoprotein (HDL) levels and total cholesterol levels were also affected, but returned to normal 6 to 12 weeks after use. Changes in libido were variable; however, 3 of the subjects fathered children during the study. Gynecomastia was reported in 3 subjects, but they were successfully treated with tamoxifen.

Reproductive/Endocrine Effects

The effect of androgens on the reproductive system is highly variable. Several investigators have reported various effects on the male reproductive system, including oligospermia, azoospermia, testicular atrophy, and reductions in endogenous testosterone and gonadotrophic hormones (Alen and Souminen, 1984; Schurmeyer et al., 1984).

The World Health Organization Task Force on Methods for the Regulation of Male Fertility (WHO, 1990) investigated the use of androgens as a form of male contraception. A multicenter study was conducted in seven countries on 271 healthy fertile men. Each subject received 200 mg of testosterone enanthate weekly by intramuscular injection for approximately one year. Subjects experienced azoospermia and an increase in body weight. The study concluded that testosterone enanthate

Table 2 Serious Side Effects Associated With Androgen Use in Athletes

Author	Subject	Condition	Drug(s)/(Duration of use)	Comments
			Hepatic	
Overly et al. (1984)	26 yr BB	HCC	methd, oxa,sta/ (4 yr) ND, meth	Pattern of use unknown; patient refused chemotherapy and died at 27.
Goldman (1985)	37 yr BB	HCA	oxy/ (5 yr)	Survived following surgical resection of adenoma.
Creagh et al. (1988)	27 yr BB	HCA	Many androgens/ (3 yr)	Type of androgen used and pattern of use unknown; fatal rupture of hepatic tumor resulting in death.
Cabasso (1994)	27 yr BB	PH	oxa, methd, ND, T/ (8 yr)	Chronic intermittent user according to authors; doses used unknown; patient recovered completely.
			Cardiopulmonary	
McNutt et al. (1988)	22 yr WL	MI	Oral and injectable androgens	Pattern, dose, and duration of use unknown; patient weighed 330lbs and dietary factors may have predisposed him to cardiovascular disease.
Frankle et al. (1988)	34 yr BB	Stroke	Androgen/ (4 yr)	Steroids used and pattern of use unknown; patient recovered with mild motor deficits.
Luke et al. (1990)	21 yr BB	MI	T, ND, TC	Pattern of use unknown; he had injected ND one week prior to death.
Ferenchick & Adelman	37 yr WL	MI	ND, bol, TC, sta, oxa/ (7 yr)	Intermittent use over 7 yr consisting of 16-wk (1992) cycles.
Montine & Gaede (1992)	36 yr PL	Pulmonary embolus	androgens	Only reported case of androgen-associated death via pulmonary embolism; drugs used, pattern and length of use unknown; patient was a Type II diabetic.
Robinson & White (1993)	26 yr BB	Pulmonary embolus	metandi/ (6 months)	Subject had taken metandi every week for 6 months prior to incident; previous intake of androgens unknown.

Kennedy et al. (1993)	27 yr BB	MI & CH	Many androgens/ (6 yr)	Prior to his fatal MI, he had sustained an uncomplicated MI 10 months earlier; patient resumed lifting and begin injecting androgens at 8 months; he died 2 months later.
Jaillard et al. (1994)	31 yr BB	Cerebral venous thrombosis	T,meth,tre/ (5 yr)	Ingested ~175 mg/week; continuous use for 5 yr; patient recovered completely in 15 days.
Huie (1994)	25 yr BB	MI	ND/ (16 wk)	Father died of an MI at 40 yr; uncle survived an MI at ~40 yr; patient recovered; remains off androgens.
Appleby et al. (1994)	31 yr BB	MI & HK,	Androgens, amphet Frumil, Potassium/ (5–10 yr)	Patient smoked 10–20 cigarettes/day for 10 yr; he refused to accept that he had an MI and eventually discharged himself.
Dickerman et al. (1995)	20 yr BB	Sudden cardiac death bilateral pulmonary hemorrhage	methd, TE, NL	Subject had been self administering androgens prior to death, up to 700 mg/wk for a 16 wk cycle; no family history of heart disease; chronic pattern of use unknown.
		Musculotendinous		
Hill et al. (1983)	24 yr PL	Left distal RF tear, right QF tear	TP, methd,TC, HCG Furosemide	Average ~1,000 mg steroid for a 10-wk cycle prior to rupture; had used steroids for several years.
Kramhoft & Solgaard (1986)	42 yr BB	EPL tendon rupture	eth, NP, ND/ (4 months)	Subject claims to have only used androgens for 4 months, 200–300 mg/wk.
Bach et al. (1987)	33 yr WL	TB rupture	ND,methd	Subject had used androgens for 4 months prior to rupture, 500-700mg/day; long-term pattern of use unknown.
Herrick & Herrick (1987)	32 yr PL	TB rupture	androgens	Subject admitted to using androgens; pattern of use was not revealed.
Stannard & Bucknell (1993)	35 yr BB	TB tendon rupture	TC,ND,meth/ (5 yr)	Subject had used an average of 1,700 mg androgen for a 12-wk cycle, 3–4 times/yr.

(continued)

Table 2 *(continued)*

Author	Subject	Condition	Drug(s)/(Duration of use)	Comments
Visuri & Lindolm (1994)	23 yr BB	Bilateral BB distal tendon avulsion	oxa,oxy,sta,TE/ (6 yr) meth,methd, HCG, tamox	Subject used ~1,500–2,000 mg/wk; cycle length unknown.
Liow & Tavares (1995)	29 yr BB	Bilateral rupture QF tendon	sta,ND,methd,clen	Pattern of use unknown; had been experiencing pain in QF prior to injury.
David et al. (1995)	32 yr BB	Bilateral rupture QF	androgens/ (10 yr)	Drugs used and pattern of use unknown.
Freeman & Rooker (1995)	22 yr BB	ACL rupture	oxy	Subject had just finished an 8-wk cycle (200 mg day); long-term pattern of use unknown.
			Other	
Roberts & Essenhigh (1986)	40 yr BB	Prostate cancer	methd, ND, sta, flu/ (20 yr) TU,GH,mes	Admitted to ~15 cycles of 4–12 weeks.
Winwood et al. (1990)	30 yr BB	Bleeding esophageal varices	sta,metandi,oxa/ (1.5 yr)	Ingested ~260 mg/wk; recovered completely in 2 wk.

Note. Serious side effects were those requiring immediate medical attention. Type of athlete: BB = bodybuilder; PL = powerlifter; WL = weightlifter. Condition: HCC = hepatocellular carcinoma; HCA = hepatocellular adenoma; PH = peliosis hepatis; MI = myocardial infarction; CH = cerebral hemorrhage; HK = hyperkalemia. Muscles/Ligament: ACL = anterior cruciate ligament; BB = biceps brachii; EPL = extensor pollicis longus; QF = quadriceps femoris; RF = rectus femoris; TB = triceps brachii. Androgens/drugs: amphetamines (amphet); boldenone (bol); clenbuterol (clen); ethylestrenol (eth); fluoxymesterone (flu); growth hormone (GH); human chorionic gonadotropin (HCG); mesterelone (mes); metandienone (metandi); methandrostenelone (methd); methenelone (meth); nandrolone decanoate (ND); nandrolone laurate (NL); nandrolone phenpropionate (NP); oxandrolone (oxa); oxymethelone (oxy); stanazolol(sta); taxmoxifen (tamox); testosterone (T); testosterone cypionate (TC); testosterone enanthate (TE); testosterone propionate (TP); trenbelone (tre).

can provide highly effective, sustained, and reversible male contraception with minimal side effects.

Schurmeyer et al. (1984) investigated the efficacy of esterified 19-nortestosterone as an agent for producing reversible azoospermia. The drug was administered intramuscularly to 5 healthy male subjects in doses of 100 mg/week for 3 weeks followed by 200 mg/week for an additional 10 weeks. The results showed that azoospermia occurred 7 to 13 weeks after the introduction of steroid and continued for 4 to 14 weeks after the last injection. No serious side effects were noted. A study by Holma (1977) investigated the effects of the oral steroid metandienone on spermatogenesis and noted that the effect of androgens on spermatogenesis were completely reversible following discontinuation of their use.

High-dose androgen treatment is often used during puberty to reduce the predicted adult height in excessively tall boys. In a recent study, a group of German scientists looked retrospectively at tall men who had been treated for one year with testosterone (500 mg testosterone enanthate injected every 2 weeks) starting at age 14.8 years (Lemcke et al., 1996). They were examined again at age 26 and compared to a control population who had not been administered androgens. There were no differences in sperm concentration, sperm count, sperm morphology, testicular volume, and follicle-stimulating hormone between the two groups. Thus, reproductive capacity is unaltered, and testicular size does not diminish with long-term androgen use as a teenager. There is, however, one case of prostate cancer reported in a 40-year-old English bodybuilder with a history of androgen use (Roberts and Essenhigh, 1986). The patient had reported using numerous androgens as well as somatotropin for approximately 20 years.

Cohen and Hickman (1987) found that glucose tolerance in powerlifters who used androgens was diminished when compared to non-steroid-using powerlifters and sedentary nonobese men. The steroid users in this study had used androgens for 3 to 7 years and had no history of steroid-related disorders; however, glucose tolerance in the steroid users was still classified as normal according to the criteria of the National Diabetes Data Group. On the other hand, Hobbs et al. (1996) found that 6 weeks of testosterone enanthate treatment (300 mg/week) did not adversely affect glucose disposal, and nandrolone decanoate (300 mg/week) enhanced non-insulin-dependent glucose disposal. The authors postulate that this is due to the fact that nandrolone decanoate is a non-aromatizable androgen.

Dessig and Weissel (1993) investigated 5 athletes who had been using androgens for 6 or more weeks prior to the study, all of whom had demonstrated normal thyroid function. They found that thyroid function may be slightly impaired due to the use of androgens, but it is impaired only to a level that is not clinically detectable and probably not relevant.

Cardiopulmonary Effects

The effect of exogenous administration of androgens on the lipid profile has been well documented (Alen and Rahkila, 1984; Hurley et al., 1984; Webb et al., 1984). Androgen use by 8 bodybuilders and 4 powerlifters lowered HDL levels by 55%

and raised low-density lipoprotein (LDL) values by 61% (Hurley et al. 1984). Webb et al. (1984) demonstrated that the self-administration by bodybuilders and powerlifters of ~825 mg of androgens (i.e., methandrostenelone, nandrolone, decanoate, and testosterone esters) severely depressed HDL and increased LDL levels; however, cholesterol levels were reversible after androgen use was discontinued.

A study by O'Connor et al. (1990) on the blood chemistry of current and previous users of androgens supports the findings of the Webb study. They demonstrated that while current users of androgens had altered LDL and HDL levels, individuals who had previously used androgens over periods ranging between 10 and 14 years, showed cholesterol levels well within the normal range.

However, not all androgens have the same effect on blood lipids. For example, the 17-alkylated androgens (e.g., stanozolol, oxandrolone) can reduce HDL cholesterol, whereas nandrolone and testosterone esters have little effect (Friedl, 1990). Kantor et al. (1985) suggested that hepatic triglyceride lipase may play an important role in HDL metabolism, possibly by removing circulating HDL_2 subfraction particles, thus decreasing HDL levels in androgen users.

Additionally, elevated levels of LDLs have been shown to increase platelet sensitivity, which could theoretically induce thrombosis (Ferenchick, 1991). Several studies in animals have established a connection between androgens and thrombosis. Although no direct evidence exists that androgens are thrombogenic in humans, indirect experimental data indicate that androgens affect coagulation proteins, platelet aggregation, and the vascular system in ways that could promote thrombosis. This idea is supported by Frankle et al. (1988) who state that oral androgens such as methenolone acetate have been linked to an increase of clotting factors V, X, and prothrombin. Frankle et al. (1988) also offered the idea of an increased risk of thrombosis associated with androgen use might be due to the ability of androgens to aromatize to estrogen, which has previously been associated with a hypercoagulable state.

Several cases of myocardial infarctions associated with androgen use have been documented, and a few have resulted in death (Dickerman et al., 1995; Kennedy et al., 1993; Luke et al., 1990). Huie (1994) reported the case of a 25-year-old male who had been taking nandrolone decanoate (100–200 mg/week) for 16 weeks prior to suffering an acute myocardial infarction. The actual cause of the myocardial infarction was not determined, but Huie suggested the patient's use of androgens may have predisposed him to platelet hyperaggregation. McNutt et al. (1988) reported a similar case of an acute nonfatal myocardial infarction occurring in a 22-year-old powerlifter who had been taking oral and injectable androgens for 6 weeks before the incident occurred. This individual weighed 330 pounds and had extremely elevated cholesterol levels (596 mg/dl). Also, Robinson and White (1993) tell of the case of a 26-year-old bodybuilder who suffered pulmonary emboli after taking metandienone for 6 months. It should be noted, however, that in many of these case studies involving athletes, it is unknown or unreported as to the types of androgens used, as well as the pattern of use or abuse that may have predisposed these individuals to these ill effects. Furthermore, the

contribution of other risk factors to cardiovascular disease in these individuals is unclear.

Other cardiovascular effects induced by steroid administration include left ventricular hypertrophy and elevated blood pressure (Kantor et al., 1985). However, Thompson et al. (1992) found that in a study of 12 androgen users, steroid use was not associated with left ventricular hypertrophy or clinically detectable systolic and diastolic dysfunction.

Musculotendinous Effects

It has been suggested that androgen use in conjunction with intense weight training results in muscular adaptations that outpace the adaptive response of the connective tissue (i.e., tendons). In their review, Laseter and Russell (1991) suggested that androgens combined with exercise may induce tendon pathology. This evidence is derived from rodent studies in which abnormalities were found in the ultrastructure of collagen fibrils following the administration of androgens (Michna, 1987). The muscles most commonly injured are the quadriceps femoris and triceps brachii (Table 2). Hill et al. (1983) reported on a powerlifter who ruptured the left rectus femoris at its patellar insertion and had a minor rupture of the right quadriceps femoris muscle while doing a 750-lb squat. Bilateral distal tendon avulsions were reported in a 23-year-old male bodybuilder who had used androgens since age 17 (Visuri and Lindolm, 1994). Liow and Tavares (1995) reported on a bodybuilder who suffered a bilateral rupture of the quadriceps femoris tendon following a jump from a height of 15 feet, landing on both feet with flexed knees. The authors suggested that androgen use may have played a major role in his injury; however, one cannot ignore the role that the actual jump may have played in this injury.

Kramhoft and Solgaard (1986) reported on a bodybuilder who ruptured his extensor pollicis tendon. He had been taking androgens for four months. Furthermore, there have been cases of anterior cruciate ligament rupture in a 22-year-old bodybuilder (Freeman and Rooker, 1995) and triceps rupture in a competitive weightlifter (Bach et al., 1987), national class powerlifter (Herrick and Herrick, 1987), and bodybuilder (Stannard and Bucknell, 1993). They each had prior history of androgen use.

It would be difficult to demonstrate a direct causation of androgen use and musculotendinous injury. Anecdotally, it does seem that bodybuilders and powerlifters suffer from tendon avulsions more so than other athletes. However, the degree of musculoskeletal stress imposed on these athletes far exceeds what any normal sedentary individual would encounter. Are these tendon avulsions simply the result of a well-motivated individuals subjecting their bodies to stresses beyond their capacity? Indeed, the number androgen users is plentiful, yet the incidence of muscle tendon ruptures is actually quite rare. Moreover, tendon ruptures also occur in athletes not using steroids (Laseter and Russell, 1991). In some individuals though, androgen use in combination with heavy training may result in an increased risk of injury.

Psychological Effects

Abnormal aggression, mood swings, and psychiatric dysfunction have been associated with androgen use (Strauss et al., 1985). Annitto and Layman (1980) reported a case of acute schizophrenia in a 17-year-old weightlifter using methandienone. During the course of therapy the subject reported feelings of depression, nervousness, and sensory hyperawareness. He also reported that his symptoms began about 6 months after the initiation of his steroid regimen, and ceased after the anabolic agent was discontinued.

Freinhar and Alvarez (1985) presented a case study of a 27-year-old bodybuilder with oxandrolone-induced hypomania. The patient self-administered oxandrolone (6 mg/day). Two days later he reported feelings of irritability, hyperphagia, hyperactivity, and hypersexuality. The steroid was discontinued and within 3 to 4 days the patient's symptoms had stopped. One week later the patient began another regimen of steroids, self-administering oxymetholone. His symptoms reappeared, and he was treated accordingly. The researchers concluded that the steroid brought out this psychologic abnormality and the possibility that synthetic androgens can induce hypomania in some individuals was proposed. Uzych (1992) listed numerous psychiatric effects associated with androgens, including euphoria, irritability, anxiety, hypomania, mania, dysthymia, depression, suicidal ideation, intolerance, and abuse.

Pope and Katz (1988) studied the effects of androgen self-administration and psychiatric abnormalities in male and female gymnasium users. The subjects were 39 males and 2 females. A wide range of psychiatric symptoms were reported, including auditory hallucinations, paranoid delusions, delusions of reference, manic episodes, and depression. It is interesting to note that no subjects reported having manic episodes when not on steroids, and several subjects suffered from depression upon withdrawal of the drug. The methodology of this study has been questioned by experts in that Pope and Katz used the same subjects in the experimental and control group. The use of subjects as a source for control data might have contaminated the results. Problems might have arisen if subjects had mental health problems when they were not using steroids and knowingly or unknowingly failed to convey this information to the researchers.

Male hormones have long been thought to induce aggressive behavior. Androgens were reportedly first used during World War II when they were given to German troops to increase aggressiveness (Wade, 1972). Ehrenkranz et al. (1974) studied plasma testosterone levels and aggressive behavior in 36 males from the general inmate population of a correctional facility and found that aggressive subjects had a significantly higher mean level of testosterone than those who were not aggressive. It is interesting to note that Ehrenkranz et al. (1974) suggested that high levels of testosterone may cause those individuals to develop a muscular build, which may in turn allow them to be physically domineering; however, aggressive subjects in the study did not have the outward appearance of a muscular build, and there were no significant differences in the height and weight of subjects. A confounding variable in the Ehrenkranz study was that subjects

were taking various tranquilizing medications, including benzodiazepine and phenothiazine.

Strauss et al. (1983) found subjectively perceived increased irritability and aggression in 56% of 32 male steroid users surveyed. Olweus et al. (1980) studied behaviors relating to aggression and testosterone levels in adolescent males. They found no relationship between testosterone and antisocial behavior; however, there was a correlation between response to provocation and high levels of testosterone. Bahrke et al. (1992), in a study of 30 current male steroid users observed no significant differences between androgen users and population norms in regards to either aggression/hostility or tension/anxiety. They further showed no correlation between either total weekly steroid dose or length of cycle and an increase in any of the four standardized psychological inventories used in the study.

In direct disagreement with the Bahrke et al. (1992), Yates et al. (1992) examined 12 androgen users who reported mood, anxiety, and psychomotor symptoms attributed to their steroid use. According to Yates et al. (1992), the discrepancy in these studies could be explained by differences in sample selection, sample reliability, androgen drug type or dose, or the timing of the assessment compared to the administration of steroids. Bahrke et al. (1992) noted in their study that steroid users did report steroid-associated changes in enthusiasm, aggression, irritability, insomnia, and libido during interview sessions; however, the psychological inventories used were unable to confirm these findings.

On the other hand, Wang et al. (1995) suggested that testosterone enanthate supplementation may actually decrease irritability, anger, sadness, tiredness, and nervousness while increasing energy levels, friendliness, and good feelings in hypogonadal men; however, once a minimally sufficient level of serum testosterone was achieved, further increases in serum testosterone did not further enhance feelings of well-being.

It should be noted that many studies that have examined the psychological effects of androgen use lack scientific rigor. Most studies are retrospective and rely solely on the subject's self-reporting. In addition, most of these studies provide no past medical or psychiatric history and no control populations were used (Windsor and Dumitru, 1988).

It is our speculation that in certain individuals androgen use may enhance aggression; however, it would be difficult to determine if this aggression is a direct result of the drug, or is due to a change in how the individual perceives him- or herself. That is, individuals who use steroids concurrent with heavy resistance training will accrue significant gains in muscle strength and mass. Consequently, because of this enhanced strength and size, these individuals can "act" aggressively toward other people, knowing clearly that their size alone may intimidate other, smaller individuals.

Also, some behavioral changes due to androgen use may actually be viewed as beneficial. Many in the athletic community believe that these behavioral changes, when properly directed, may increase motivation and therefore enhance training intensity and athletic performance (Bahrke et al., 1992). Thus, it is unclear how androgens affect behavior. It is plausible that in certain individuals, androgens

may induce aggressive behavior as well as other psychologic correlates. However, these behavior changes do not occur invariably and would therefore be difficult to predict for any given individual.

Summary

Side effects resulting in serious harm or death to athletes from androgen self-administration are exceedingly rare (Table 2). Furthermore, it is difficult to demonstrate that androgen use per se caused these maladies. The pattern of androgen use among many of these individuals is unknown. Also, it is unclear what role heredity, diet, diabetes, smoking, percent body fat, and the use of nonandrogenic drugs may have had on the few individuals who have suffered an androgen-associated death.

There is only one study in the scientific literature that has examined the long-term impact of androgen use in former athletes (Silvester, 1995). This study determined the effect of chronic androgen use through personal interviews of 22 former athletes. These athletes had used androgens for an average of 7.82 years at a mean daily dose of 31.95 mg. Nineteen of these athletes reported no long-term physiological side effects. One reported an atrophied testicle, one had an unspecified negative effect, and another believed that androgen use in conjunction with Olympic weightlifting caused spinal spurs. Concerning long-range psychological effects, 2 reported that they had increased aggression or irritability, and the other 20 had none. However, it should be noted that no physiological variables were actually measured in this study, and one can question the accuracy and veracity of data gathered via interviews.

Serious side effects in the liver are quite rare in athletes who self-administer androgens. In fact, most of these harmful effects are associated with use of the orally active 17-alkylated androgens (Table 3), and these have been reported in individuals who were chronically administered androgens for various medical reasons. It is clear that there are potential ill-effects of androgen use, especially the orally active 17-alkylated androgens, but the testosterone esters and nandrolones seem to have fewer side effects. Perhaps the use of injectable androgens would alleviate any potential harm to the liver.

The effects on endocrine/reproductive function include a reduction in endogenous testosterone and gonadotrophic hormone production, testicular atrophy, oligo- or azoospermia, and altered libido, yet there is no evidence for any long-term detriment to the reproductive system. The oral administration of the 17-alkylated androgens has been shown to decrease insulin sensitivity; however, testosterone enanthate does not negatively affect glucose metabolism while nandrolone may improve glucose disposal (Hobbs et al., 1996).

An elevation of LDL cholesterol and a depression of HDL-cholesterol are also a consistent pattern seen in androgen users; however, the 17-alkylated androgens have a more negative impact on blood lipids than nandrolone or testosterone enanthate (Friedl et al., 1990). One would surmise that the use of certain androgens would predispose users to cardiovascular disease; however, the intermittent nature

Table 3 Androgens Commonly Used by Athletes

Generic name	Various brand names
Oral	
Danazol[a]	Cyclomen, Danol, Danocrine
Ethylestrenol[a]	Maxibolin, Orabolin
Fluoxymesterone[a]	Halotestin, Stennox, Android-F, Ultandren
Mesterolone	Mestoranum, Proviron
Metandienone[a]	Danabol, Lanabolin, Dianabol
Methandrostenelone[a]	Metandionona-5, Metanabol, Andoredan
Methenelone acetate	Primobolan tabs
Methyltestosterone[a]	Metandren, Oreton Methyl, Testred
Mibolerone[a]	Cheque Drops[b]
Norethandrolone[a]	Nilevar
Oxandrolone[a]	Anavar, Lonavar, Oxandrin
Oxymesterone[a]	Anamidol, Balnimax, Oranabol
Oxymethelone[a]	Anadrol-50, Anapolon, Androyd
Stanozolol[a]	Winstrol, Stromba
Injectable	
Boldenone undecylenate	Equipoise[b], Maxigan[b]
Methenelone enanthate	Primobolan Depot, Primobolan S, Primonabol
Nandrolone decanoate	Deca-Durabolin, Norandren[b], Durabolin
Nandrolone laurate	Laurabolin[b]
Nandrolone phenpropionate	Durabolin
Stanozolol	Winstrol-V[b], Strombaject
Testosterone blend	Deposterona[b], Sten, Testoprim-D
Testosterone cypionate	Depo-testosterone, Testa-C, Andronate, Depoandro, Duratest, Testred cypionate
Testosterone enanthate	Delatestryl, Primoteston, Testone L.A., Testrin-P.A. Testosterona 200[b]
Testosterone nicotinate	Bolfortan
Testosterone propionate	Oreton, Testex, Malogen, Virormone
Testosterone undecanoate	Andriol, Restandol
Trenbolone acetate	Finajet[b], Parabolin, FinaPlix[b]

Note. This information is taken from LaBree (1991) and Wilson (1988).
[a]17-alkylated androgens. [b]Veterinary compound.

in which most athletes use steroids may preclude this. Furthermore, lipid profiles return to normal following the cessation of steroid use. There have been few instances of myocardial infarction in athletes who have self-administered androgens, but it is not clear what other risk factors were involved. Tendon avulsions have been also reported in athletes who have used androgens, yet for the most part, this is extremely rare.

Perhaps the most controversial aspect of androgen use is its purported effects on behavior. According to Yates et al. (1992), it is impossible to determine whether aggression is due to androgen use or whether it was an existing condition prior to use. Many in the medical community and in the media have been convinced (based on no scientific evidence) that bodybuilders undergo a "roid rage" (i.e., uncontrollably aggressive/violent behavior) when using these drugs.

Conclusion

It is our contention that the incidence of serious health problems associated with the use of androgens by athletes has been exaggerated. Longitudinal studies by the WHO (1990) and Millar (1994) have demonstrated that moderate androgen use (140–200 mg/week) produce minor but reversible side effects. If androgens are so dangerous, one would expect an abundance of maladies befalling the over 300,000 individuals who used androgens in 1991 (Yesalis et al., 1993). Androgens have been used by athletes, especially in the strength sports, since the mid-1950s. Professional bodybuilders, perhaps the population most likely to use steroids, had used these drugs legally until the mid-1980s, and currently use them illegally. Although more than one million individuals are current or former steroid users, there is no evidence to suggest that they suffer from hepatic or cardiovascular disease more so than the non-androgen-using population. Based on the available evidence, we would posit that the administration of moderate doses (200–300 mg/week for 6 to 12 weeks once per year) of an injectable androgen, such as testosterone enanthate or nandrolone decanoate, in healthy adult males could induce positive changes in body composition and athletic performance with little or no side effects (Lemcke et al., 1996; Millar, 1994; WHO, 1990). On the other hand, some athletes, especially bodybuilders, often take much higher doses for longer periods of time. It could be speculated that such high doses might lead to more serious health problems, but at this point, there is no evidence to support such a contention.

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