

National Athletic Trainers' Association Position Statement: Anabolic-Androgenic Steroids



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This NATA position statement was developed by the NATA Research & Education Foundation.

Objective: This manuscript summarizes the best available scholarly evidence related to anabolic-androgenic steroids (AAS) as a reference for health care professionals, including athletic trainers, educators, and interested others.

Background: Health care professionals associated with sports or exercise should understand and be prepared to educate others about AAS. These synthetic, testosterone-based derivatives are widely abused by athletes and nonathletes to gain athletic performance advantages, develop their physiques, and improve their body image. Although AAS can

be ergogenic, their abuse may lead to numerous negative health effects.

Recommendations: Abusers of AAS often rely on questionable information sources. Sports medicine professionals can therefore serve an important role by providing accurate, reliable information. The recommendations provide health care professionals with a current and accurate synopsis of the AAS-related research.

Key Words: testosterone, androgen, ergogenic aids, drugs, abuse, doping, sport, athletes

Anabolic-androgenic steroids (AAS) are synthetic testosterone analogs^{1,2} legally classified as Schedule III controlled substances.^{1,3} These hormones increase lean muscle mass and can improve athletic performance.^{1,2,4,5} Although AAS have valid medicinal uses, nontherapeutic abuse also occurs.^{1,4,5} Recent increases in androgen prescriptions are evident.^{6,7} Those involved in organized athletics and nonathletes both abuse AAS.^{1,2} Nontherapeutic users often obtain AAS information and products from a variety of dubious sources and make questionable health decisions.^{8,9} Health care professionals, including athletic trainers and educators, may interact with individuals who abuse or intend to abuse AAS. Therefore, it is imperative that such professionals understand these substances so that they can educate others using the most current and accurate evidence.

This position statement is a summation of the best available scholarly evidence with regard to AAS and integrates a Strength of Recommendation Taxonomy (SORT) criterion scale from the American Academy of

Family Physicians (Table 1).¹⁰ Recommendations are ranked as A (good-quality evidence), B (inconsistent or limited-quality or limited-quantity evidence), or C (recommendations based on consensus, usual practice, opinion, or case series). Although other frequently abused pharmaceuticals (eg, human growth hormone, insulin growth factor 1, and selective androgen receptor modulators) and nutritional supplements (eg, creatine, amino acids, protein powders) purport to promote anabolism, this position statement solely addresses AAS. For logistical and ethical reasons, few prospective, outcome-based, scholarly studies duplicated the typical nontherapeutic AAS abuse patterns often used.¹¹⁻¹³

Health care professionals owe their patients evidence-based knowledge to help in their health care decisions. Identification of the AAS abuser (or potential abuser) by a health care professional is critical to help prevent any negative consequences. Proper direction, guidance, support, and possible referral are essential in assisting AAS abusers and potential abusers.

Table 1. Strength of Recommendations Taxonomy (SORT)^{a,10}

Strength of Recommendation	Definition
A	Recommendation based upon consistent and good-quality patient-oriented evidence (morbidity, mortality, symptom improvement, cost reduction, and quality of life) ^b
B	Recommendation based on inconsistent or limited-quality patient-oriented evidence ^b
C	Recommendation based on consensus, usual practice, opinion, disease-oriented evidence (measures of intermediate, physiologic, or surrogate end points that may or may not reflect improvements in patient outcomes), or case series for studies of diagnosis, treatment, prevention, or screening

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^b Patient-oriented evidence measures outcomes that matter to patients: morbidity, mortality, symptom improvement, cost reduction, quality of life. Disease-oriented evidence measures intermediate, physiologic, or surrogate endpoints that may or may not reflect improvements in patient outcomes (ie, blood pressure, blood chemistry, physiological function, and pathological findings).

RECOMMENDATIONS

Basic Science

1. Health care professionals and educators should recall that the endocrine system synthesizes hormones that help regulate the body’s physiology. Primarily synthesized in the gonads and adrenal glands, steroid hormones are a particular class of chemical messengers that affect body tissues and have anti-inflammatory, salt-retaining, and feminizing or masculinizing properties, depending on the substance.^{1,4,7,14–16} *Evidence Category: A*
2. Health care professionals and educators should understand that testosterone, the key androgen, promotes both androgenic (masculinizing) and anabolic (tissue-building) effects.^{1,4,7,14–16} Normal testosterone levels vary with age, sex, and health status, and levels in males are significantly greater than levels in females.^{7,17–19} In males, testosterone levels normally peak during early adulthood and then decrease.^{7,18–20} Abnormal endogenous testosterone levels in adulthood appear to be associated with specific disorders or diseases.^{7,18–20} *Evidence Category: A*
3. Health care professionals and educators should appreciate that AAS are synthetic substances related to the primary male hormone, testosterone.^{1,2,4,7,14} The United States classifies the many available AAS as Schedule III controlled substances.^{1,13–15} Pharmaceutical companies and clandestine laboratories may develop various AAS to maximize anabolic effects, minimize androgenic effects, improve pharmacokinetics, increase receptor affinity, and, in some cases, avoid detection.^{14,15} The AAS are available as oral, injectable, and buccal (intraoral, next to cheek) agents, subcutaneous pellets, and transdermal patches, creams, and gels.^{14,15} *Evidence Category: A*
4. Health care professionals and educators should know that the mechanisms of action for AAS are complex and variable.^{7,14,15} The AAS appear to promote protein synthesis through increased transcription while also acting as glucocorticoid antagonists, limiting catabolism.^{14,15} They also induce various potentially ergogenic psychotropic actions, altering neurochemistry,^{11,14,21,22} and may stimulate human growth hormone and insulin growth factor 1 synthesis,¹² as well as downregulate myostatin.^{7,23,24} *Evidence Category: C*

Therapeutic AAS Use and Nontherapeutic AAS Abuse

5. Health care professionals and educators should recognize the possible therapeutic uses for AAS, which include therapy for male hypogonadism, certain rare anemias, and other medical conditions; anticatabolism for those with chronic wasting syndromes; and preserving or restoring bone health.^{6,7,18–20,24–33} Although some suggest AAS may act as “antiaging” substances, the evidence remains questionable.^{6,17,18,33} *Evidence Category: C*
6. Health care professionals and educators should appreciate that although AAS prevalence studies have limitations, the current evidence suggests that nontherapeutic AAS use is a worldwide phenomenon.^{3,8,9,34–49} Abuse of AAS occurs for performance improvement, physique development, and body-image enhancement.^{8,9,34–44,46,49} Males abuse AAS at greater frequency than do females.^{8,35,36,45,46} Those who use AAS for nontherapeutic reasons often do not participate in organized sports.^{9,41,43,45,46,49} Abusers of AAS include adolescents, collegians, professional and Olympic athletes, body builders, and recreational athletes, among others.^{8,34–37,39–47,49} The nonmedicinal abuse of AAS often begins in adolescence, sometimes as early as middle school.^{35–37,41–43,45–47} *Evidence Category: C*
7. Health care professionals and educators should realize that AAS abusers choose from many possible agents,^{11,50} including “designer steroids” unapproved by the Food and Drug Administration and veterinary-quality and black-market substances.^{1,3,13,41,49} Although AAS abusers may obtain androgens from physicians, their supplies more commonly come from other sources, such as the Internet, training partners, gymnasium owners or instructors, teammates, and coaches.^{8,9,36,37,40–42,49} Counterfeit AAS are common, leading to product quality concerns: impurities, contaminants, and inaccurate product labels.^{51–53} *Evidence Category: C*
8. Health care professionals and educators should understand that doses taken by AAS abusers are often much greater than therapeutic replacement levels.^{13,14,51,53,54} Nontherapeutic AAS users frequently “stack,” or simultaneously abuse, multiple AAS, with differences in half lives and solubilities.^{3,9,36,41,44,45} Abuse of AAS often occurs in repeated cycles of 6 to 12 weeks, followed by periods of nonuse.^{1,3,8,9,36,41} *Evidence Category: B*

AAS Efficacy

- Health care professionals and educators should respect the limitations of high-quality research designs in determining AAS effectiveness and side effects.^{11–14,22,51,55,56} Ethical issues disallow using the typically extreme dosing schedules in research studies due to the federally controlled status of AAS, as well as the reported risk profile of androgen use at nontherapeutic doses.^{11,14,51,55,56} *Evidence Category: C*
- Health care professionals and educators should realize that the efficacy of AAS as anabolic agents suggests a dose-related potential for increased relative lean body mass.^{57–61} These substances can generally act as ergogenic agents when the measure involves strength- or power-related performances.^{6,59–66} *Evidence Category: A*

AAS Abuse Health Effects

Health care professionals and educators should be aware of the following possible AAS abuse side effects on various biologic systems and organs.

- Supraphysiologic AAS dosing may occasionally be associated with hypomanic or manic syndromes that are often characterized by irritable or aggressive behavior.^{21,54,67–78} Episodes of major depression may be associated with AAS withdrawal.^{79–81} Abusers of AAS may develop a dependence syndrome related to both myoactive and psychoactive effects^{82–89} and may exhibit other forms of drug dependence, such as opioids.^{83,90–93} *Evidence Category: B*
- The cardiovascular effects of therapeutic AAS remain unclear. Substantial research findings now suggest that AAS abuse negatively influences the cardiovascular system.^{25,67,94–103} The best evidence indicates that nontherapeutic AAS-related conditions include cardiomyopathy^{96,97,99,100} and the potential for atherosclerotic vascular disease caused by detrimental lipid changes, which may adversely affect one's risk for coronary artery disease.^{25,102,103} *Evidence Category: B*
- Although rare, hepatic maladies including cholestatic jaundice and peliosis hepatis might occur with the nontherapeutic abuse of AAS, especially when the oral C17 α -alkylated group of AAS is involved.^{7,11–14,22,24,51,55,104} *Evidence Category: A*
- Exogenous AAS abuse inhibits the hypothalamic-pituitary-gonadal axis, reducing the production of endogenous testosterone, luteinizing hormone, and follicle-stimulating hormone.^{7,11–14,22,51} It can also alter thyroid function^{11,12,22} and negatively affect glucose tolerance.^{12,22,105} *Evidence Category: B*
- Abuse of AAS directly affects the male reproductive system, with possible side effects including hypogonadism, decreased spermatogenesis, decreased sperm motility, erectile dysfunction, impotence, gynecomastia, and male-pattern baldness.^{7,11–14,22,51,55} Many of these conditions are reversible with cessation of AAS, although breast tissue changes and hair loss often require additional treatments, including surgery.^{7,11–13,22,51,55} *Evidence Category: B*
- Reproductive changes to females who abuse AAS generally involve virilization, including voice deepening, hirsutism, clitoral hypertrophy, breast reduction, libido

changes, menstrual dysfunction, male-pattern baldness, and acne.^{11–14,22,51} Unlike the side effects in males, many of these changes are permanent in females.^{12,14,22}

Evidence Category: B

- Skeletally immature AAS abusers might experience premature epiphyseal closure of the long bones, resulting in shortened stature.^{11,12,14,22,51} Other negative effects of AAS abuse on the musculoskeletal system may include tendinopathies (including possible rupture).^{7,11–13,22,51} Little evidence supports the therapeutic use of AAS for musculotendinous injury recovery.^{7,106} *Evidence Category: C*
- A possible immunosuppressant effect of AAS abuse may exist in humans.^{11,22,107} Abusers risk local and systemic infections (including hepatitis and human immunodeficiency virus) with unsterile syringe usage.^{11,12,14,22} *Evidence Category: B*
- High-dose AAS abuse often leads to a number of dermatologic conditions, most commonly some form of acne.^{11,12,14,22,108,109} Kidney structure and function may be at risk with supraphysiologic doses of AAS, especially when combined with use of nonsteroidal anti-inflammatory drugs, high-protein diets, certain nutritional supplements, and dehydration.^{11,22,51,55,110,111} Gingival and other oral tissues may also be affected.^{52,112} *Evidence Category: C*

AAS Abuse Prevention

- Health care professionals and educators should recognize the great variance in drug testing programs.¹¹³ Due to the small number of high-quality studies, whether such screenings significantly deter AAS abuse remains unclear.^{114,115} A need exists for prospective randomized trials to investigate the deterrent efficacy of AAS screening. *Evidence Category: C*
- Health care professionals and educators should realize that although AAS abuse education requires further study, well-designed programs might effectively inhibit such abuse in adolescents.^{114–124} Consideration for the early integration of prevention models into educational curricula and sports programs is important. *Evidence Category: C*

AAS Abuse Identification and Intervention

- Health care professionals and educators should be aware of the dynamic, social process of AAS abuse. Active monitoring for AAS abuse and maintaining an open, honest, and evidence-based dialogue with all stakeholders, including athletes, coaches, administrators, parents, advisory groups, and others, is vital.^{125,126} Athletic trainers should develop relationships with and call on other qualified health care professionals as referral resources. *Evidence Category: C*

BACKGROUND AND LITERATURE REVIEW

History of Anabolic-Androgenic Steroids

Testosterone Isolation and Development. More than 6000 years ago, herders recognized numerous changes in castrated animals.^{1,16} Centuries later, crude studies

investigating the testes' biologic role involved their transfer from castrated roosters into the abdominal cavity of hens to observe changes in the animals.^{1,16} In the mid-1800s, the effects of castration were directly related to a secreted testicular substance.^{1,16} Charles Brown-Séguard, a founder of endocrinology, published results suggesting remarkable rejuvenation effects from his self-experimentation using guinea pig and canine testicular extract.^{1,2,16} Testosterone was eventually chemically isolated^{1,2,5} and then synthetically developed.^{1,2,16} In 1935, Kockakian suggested testosterone might help stimulate anabolism and benefit certain wasting conditions.² Thereafter, sports physiology researchers began investigating the effects of testicular extracts on physical performance.^{1,2,5}

AAS Ergogenics. The 1940s brought the first published reports on the ergogenic effects of testosterone and its derivatives.² The 1945 book *The Male Hormone* may have increased athletic AAS abuse.^{1,2,7} Anecdotal reports described West Coast body builders experimenting with AAS in the late 1940s.^{1,2,16} The 1952 Helsinki Olympic Games brought about the seemingly valid US claim of hormonal manipulation by the successful Soviet weightlifters.^{4,5} Soviet physicians purportedly admitted that their athletes used synthetic hormones at the 1954 World Weightlifting Championships.⁵ This led to the development and 1958 release of the first commercially available AAS in the United States: methandrostenolone (Dianabol; Ciba Specialty Chemicals, Basel, Switzerland).^{1,4,5,7}

Abuse of AAS increased through the 1960s and 1970s,^{4,5} with evidence for abuse at every Olympic competition since 1960.⁴ The abuse spread to professional and collegiate sports,⁵ and typical dosages increased well beyond therapeutic levels.¹⁴ This era also involved large-scale, top-secret, government-supported, systemic hormonal manipulation in some Eastern bloc countries.⁴ Increased AAS abuse led to the 1967 establishment of the International Olympic Committee's Medical Commission, with the primary role of doping oversight.⁴ Urine AAS tests were first conducted at the 1974 Commonwealth Games, where more than 16% of the sampled athletes tested positive.⁵ The 1976 Montreal Games marked the first Olympic AAS tests.^{4,5} During this time, most other sport governing bodies, including colleges and professional leagues, generally did little to address AAS abuse.⁵

Although AAS abusers learned about the efficacy of these substances through trial and error, many medical professionals and scientists erroneously suggested that these drugs were ineffective based on laboratory studies that failed to duplicate the doses and training conditions experienced by actual AAS abusers in the field.^{4,5,14,51} This discrepancy led to distrust of the science and those reporting it.^{5,51} Drug testing and the knowledge gap pushed AAS abuse further underground.^{4,5} Many trade publications promoting AAS abuse became available in the 1970s and 1980s.^{4,5} Ben Johnson's apparent gold medal and world record at the 1988 Seoul Olympics highlighted the enormity of AAS abuse.⁴ The 1990 Anabolic Steroid Control Act reclassified many AAS as Schedule III controlled substances.¹ A follow-up statute—the Anabolic Steroid Act of 1994—added more AAS and some testosterone precursors to the Schedule III drug list.¹

So-called designer steroids became known⁴ when a contaminated needle was anonymously sent to the US

Anti-Doping Agency in 2003. The substance was a new AAS not approved by the US Food and Drug Administration and since named tetrahydrogestrinone.⁴ These findings led to the Bay Area Laboratory Cooperative (BALCO) scandal that involved many high-profile athletes.^{4,127} The US Congress held hearings concerning AAS abuse, leading to the 2004 Anabolic Steroid Control Act, which reclassified many anabolic-related pharmaceuticals as Schedule III controlled substances.^{53,107}

In 2004, the World Anti-Doping Agency began AAS oversight for all international competitions involving Olympic sports.⁴ Many collegiate sport organizations, as well as US professional sport leagues, now include AAS testing, although the rigor of these programs varies greatly.¹¹³ Some modern competitors continue to seek increasingly sophisticated, illegal, and unethical ways to maximize their performance or physique (or both) with AAS.⁴ The most common source of positive doping tests by the World Anti-Doping Agency is AAS.^{4,51}

Epidemiology of AAS Abuse

Although AAS have legal therapeutic uses for specific medical disorders, healthy persons also abuse them to enhance physical performance or physique (or both).^{1,2,4,5,7,14} Researchers have most frequently concentrated on the prevalence of AAS abuse but have also investigated commonly abused AAS, abuser characteristics, patterns of abuse, and AAS sources.^{3,8,9,35–46} These abuser profiles are important to understand before educational and preventive initiatives are devised.

Prevalence of Nontherapeutic AAS Abuse. Most authors of prevalence studies used anonymous, direct survey methods, typically within specific populations, such as adolescents, collegians, elite athletes, or gym attendees. Many published reports with various research designs detailed AAS prevalence rates (Table 2).^{3,8,9,34–47,49,128} Anonymous survey research, although valuable, involves limitations, and interpreting results requires caution. For example, respondents may falsely indicate they used steroids when they actually used only corticosteroids or dietary supplements.¹²⁹ Alternatively, those who complete such questionnaires may be less likely to abuse drugs. Given these possibilities, the research results presented here generally include those studies with the strongest sampling techniques, sample sizes, survey administrations, and reporting methods, as well as documentation of instrument validity and reliability.^{3,8,9,34–47}

Adolescent AAS Abuse. Adolescents are the most studied population for the prevalence of AAS abuse (Table 2). The first national high school survey (1988) suggested AAS abuse by 6.6% of male high school seniors.⁴¹ A survey of male Indiana high school athletes in 1999 indicated a 6.3% lifetime prevalence of AAS abuse.⁴² In 2002, others³⁴ suggested that 0.7% of sport participants aged 10 to 15 years from 34 states abused AAS. A 2006 US study of junior high and high school students reflected an AAS abuse prevalence of 1.3%, including 2.7% of males and 0.4% of females.⁴⁶ Results from a 2007 study of Minnesota students in grades 7 through 12 indicated AAS abuse rates of 1.7% and 1.4% in male and female adolescents, respectively.⁴³ The 2009 Centers for Disease Control and Prevention Youth Risk

Table 2. Anabolic-Androgenic Steroid Abuse Prevalence Studies Implementing the Highest-Quality Research Techniques

AAS Abusers	Author(s) (Year)	Total Participants, Males, Females, n		Sampling Technique	Group Description	Age/Grade	Location	Total Abusers		Male Abusers		Female Abusers	
		N	n					(n)	(%)	(n)	(%)	(n)	(%)
Adolescents	Buckley et al (1988) ⁴¹	3403	3403	0 Stratified random cluster	Students at 46 high schools	Grade 12	United States	226	6.6	226	6.6	0	0.0
	Dodge and Jaccard (2006) ⁴⁶	14 322	6759	7563 Representative sample	Students at junior high and high schools	Grades 7–12	United States	183	1.3*	153	2.7	30	0.4
	European School Survey Project on Alcohol and Other Drugs (2007) ⁴⁷	104 828	51 249	53 579 Populations or representative clusters	Students (1991 births)	Average, 15.8 y	Europe (35 countries)	NA	NA	NA	2.0	NA	1.0
	Hoffman et al (2007) ⁵³	3248	1559	1689 NA	Students	Grades 8–12	United States	NA	1.6	NA	2.4	NA	0.8
	Kokkevi et al (2008) ³⁶	18 430	NA	NA Cluster	Students at high schools	Average, 16 y	Europe (6 countries)	388	2.1	NA	NA	NA	NA
	Stigler and Yesalis (1999) ⁴²	873	873	0 Random cluster	Football athletes at 347 high schools	NA	Indiana	55	6.3	55	6.3	0	0.0
	vandenBerg et al (2007) ⁴³	2516	1130	1386 Selected	Students at 31 junior high and high schools	Grades 7–12	St Paul, MN	38*	1.5*	19	1.7	19	1.4
	Wroble et al (2002) ³⁴	1553	1079	474 Cluster	Athletes in youth sports	Range, 10–15 years	United States (34 states)	11*	0.7	10*	0.9	1*	0.2
Collegians	Centers for Disease Control and Prevention (2009) ³⁵	16 410	NA	NA 2-Stage cluster sample	Students at high schools	Grades 9–12	United States	NA	3.3	NA	4.3	NA	2.2
	Kersey (1996) ⁸	1185	833	352 Stratified random cluster	Athletes at ten 2-year colleges	Average, 19.6 y	California	38	3.3	34	4.2	4	1.2
	McCabe et al (2007) ³	54 567	NA	NA Random cluster	Students at 119 4-year colleges	About 21 y	United States	NA	1.0	NA	NA	NA	NA
	National Collegiate Athletic Association (2012) ³⁷	20 474	NA	NA NA	Collegiate athletes	NA	United States	NA	0.8	NA	NA	NA	NA
Elite athletes	Horn et al (2009) ³⁹	2552	2552	0 Population	Retired National Football League players	Average, 53.8 y	United States	233	9.1	233	9.1	0	0.0
	Tricker et al (1989) ³⁸	176	108	68 Population	National Physique Committee registered bodybuilders	Average, 26.7 y	Kansas and Missouri	66	37.5	59	54.6	7	10.3
	Wagman et al (1995) ⁴⁰	15	15	0 Population	Elite powerlifters	Range, 20–45 y	United States	10	66.7	10	66.7	NA	NA
Gym attendees	Baker and Graham (2006) ⁴⁵	146	136	10 Convenience	Clients at "hardcore" gyms	Average, 33.6 y	South Wales, UK	102	69.9	NA	NA	NA	NA
	Bolding et al (2002) ⁴⁴	772	772	0 Convenience	Gay male attendees at 6 gyms	Median, 35 y	London, UK	117	15.2	117	15.2	0	0.0
	Kanayama et al (2001) ⁴⁸	511	334	177 Convenience	Clients at 5 gyms	Range, 14–70 y	Boston, MA	18	3.5	18	5.4	0	0.0
	Perry et al (2005) ⁹	260	NA	NA Self-selected via Internet	Weight lifters and bodybuilders	NA	United States	207	79.6	NA	NA	NA	NA
	Santos et al (2010) ⁴⁹	123	123	0 Convenience	Bodybuilders	Range, 18–50 years	Brazil	41	33.3	41	33.3	0	0.0

Abbreviations: AAS, anabolic-androgenic steroids; *, numbers were calculated by present authors, not directly stated by original authors; NA, not available.

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Table 3. Available Anabolic-Androgenic Steroids, Including Veterinary Pharmaceuticals

Generic Name	Trade Names	Formula	Administration
Testosterone			
Gel	AndroGel, Axiron, Testim, Fortigel	C ₁₉ H ₂₈ O ₂	Transdermal
Patches	Testoderm, Androderm	C ₁₉ H ₂₈ O ₂	Transdermal
Pellets	Testopel	C ₁₉ H ₂₈ O ₂	Subcutaneous
Buccal system	Striant	C ₁₉ H ₂₈ O ₂	Buccal
Suspension	Testosus, Aquaviron, Univet Uni-test	C ₁₉ H ₂₈ O ₂	Injection
Testosterone propionate	Testrex, Androgeston, Testogen	C ₂₂ H ₃₂ O ₃	Injection
Testosterone enanthate	Delatestryl, Primotestone, Testinon	C ₂₆ H ₄₀ O ₃	Injection
Testosterone cypionate	Depo-Testosterone, Durandro	C ₂₇ H ₄₀ O ₃	Injection
Testosterone undecanoate	Andriol, Undestor, Nebido	C ₃₀ H ₄₈ O ₃	Injection
Nandrolone decanoate	Deca-Durabolin, Anabolin	C ₂₈ H ₄₄ O ₃	Injection
Boldenone undecylenate	Equipoise, Ganadol, Equigon	C ₃₀ H ₄₄ O ₃	Injection
Methenolone enanthate	Primobolan depot, Delapromor	C ₂₇ H ₄₂ O ₃	Injection
Oxandrolone	Anavar, Oxandrin, Vasorome	C ₁₉ H ₃₀ O ₃	Oral
Methandrostenolone	Dianabol, Anabol, Refovit	C ₂₀ H ₂₈ O ₂	Oral
Fluoxymesterone	Halotestin, Fluotestin, Ora-Testryl	C ₂₀ H ₂₉ FO ₃	Oral
Oxymetholone	Anadrol, Roboral, Anasteron	C ₂₁ H ₃₂ O ₃	Oral
Methyltestosterone	Android, Testred, Methitest	C ₂₀ H ₃₀ O ₂	Oral, buccal, sublingual
Trenbolone acetate	Finaplix H, Finaplix S, Parabolan	C ₂₀ H ₂₄ O ₃	Oral, injection, pellets
Stanozolol	Winstrol, Stromba, Winstrol V	C ₂₁ H ₃₂ N ₂ O	Oral/injection

Behavior Surveillance System³⁵ reported that national AAS abuse rates were 4.3% and 2.2% for males and females, respectively (3.3% overall). Results from the 2007 European School Survey Project on Alcohol and Other Drugs involving 35 European nations noted an AAS abuse prevalence of 2% for males and 1% for females.⁴⁷ A 2008 six-country European study reflected an overall AAS abuse prevalence of 2.1% among high school students.³⁶

Collegiate AAS Abuse. The National Collegiate Athletic Association (NCAA), among others, studied AAS abuse among collegiate student-athletes every 4 years since 1985. The most recent (2009) findings (Table 2) indicated an overall lifetime AAS abuse prevalence of 0.8%.³⁷ A summation of 4 independent studies at 119 US universities indicated that 0.9% of all college students abused AAS.³ Only one author⁸ investigated AAS abuse among community college student-athletes and reported a prevalence of 3.3% (4.2% of men, 1.2% of women).

Elite Athlete AAS Abuse. The prevalence of AAS abuse by elite athletes has been examined in relatively few high-quality studies (Table 2).³⁸⁻⁴⁰ In a 1989 study,³⁸ 37.5% of competitive body builders admitted AAS abuse, whereas a 1995 investigation revealed that 66.7% of competitive power lifters abused AAS.⁴⁰ A 2009 paper³⁹ indicated that 9.1% of retired National Football League athletes admitted using AAS during their professional careers. Prevalence was highest among offensive (16.3%) and defensive (14.8%) linemen.

Gym Attendee AAS Abuse. A few well-designed studies described AAS abuse by gym attendees (Table 2). A 5.4% reported lifetime prevalence of AAS abuse was demonstrated among 334 men in 5 Boston-area gyms, but no cases of AAS use among 177 women were noted.⁴⁸ In a high-quality British study,⁴⁴ AAS abuse was found in 15.2% of gay male gym attendees. In body-building gyms, AAS abuse prevalence rates were 33.3% in Brazil⁴⁹ and 69.9% in the United Kingdom.⁴⁵

Commonly Abused AAS. Options for AAS abusers are many,¹³⁻¹⁵ including pharmaceutical-grade, veterinary-quality, and unapproved AAS.^{1,4,14,49,127} Abuse of

“designer” AAS, such as tetrahydrogestrinone and others, developed outside the normal US Food and Drug Administration approval process and is a recent and apparently growing phenomenon.^{1,4,14,127} The most common administration routes are oral and parenteral, although topical and transdermal routes are also options.^{1,4,14,127} Examples of commonly abused AAS are shown in Table 3.^{8,9,38,45,49}

Patterns of AAS Abuse. Unique patterns of AAS abuse evolved through anecdotal evidence, as well as trial and error. Research^{14,54,127} suggests that most AAS abusers administer doses well beyond medicinal levels. These doses vary depending on the desired outcomes, but weekly totals in excess of 1000 mg are not uncommon¹²⁷; typical medicinal doses for hypogonadal males are 35 to 70 mg per week.⁷ Additionally, AAS abusers frequently “stack” (simultaneously administer) multiple AAS.^{1,4,8,9,41,45} Abuse often occurs in cycles, or periods of use followed by nonuse, typically lasting 6 to 12 weeks.^{1,4,8,9,41} The efficacy of these abuse patterns is unconfirmed by research, but extreme athletic performances and physiques may indicate that practice is ahead of science.^{1,127}

AAS Abuser Characteristics. Many AAS prevalence studies also gathered related demographic information that helped to define the AAS abuser.^{3,8,35-37,46} A 2009 national Centers for Disease Control and Prevention study of adolescents found that male teens (4.3%) were more likely to abuse AAS than female teens (2.2%).³⁵ Hispanics, whites, and blacks were the most likely AAS abusers.³⁵ In another investigation,⁴⁶ an association was suggested between sport participation and AAS abuse, especially with males. Other authors³⁶ reported a similar association between AAS abuse and physical exercise in European adolescents. Some researchers⁴⁶ found a strong association between the use of legal performance-enhancing substances and AAS, whereas others³⁶ suggested a similar association for recent alcohol abuse and lifetime abuse of sedatives or cannabis.

The most recent NCAA study indicated collegians typically begin their AAS abuse under age 20, with some

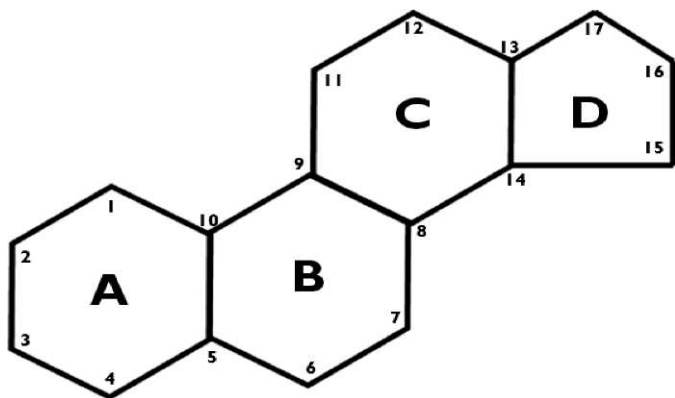


Figure 1. Chemical structure of steroid hormones with three 6-carbon rings and one 5-carbon ring.

declaring their first-time abuse occurring between ages 12 and 13.³⁷ Most collegians claimed to abuse AAS to improve athletic performance and for reasons unrelated to sports, with fewer using AAS to treat injury.³⁷ Males were more likely than females to utilize nontherapeutic AAS within the past 12 months.³⁷ Other findings³ suggested that collegiate AAS abusers tended to be married, male, student-athletes, and older than 23 years of age. Although students at commuter colleges had a higher prevalence of AAS abuse, geographic region, NCAA division, institutional status (private versus public), and size were not associated with the nontherapeutic use of AAS.³ These authors reported a significant association between AAS abuse and cigarette smoking, binge drinking, illicit drug abuse (marijuana and cocaine), and other risky behaviors.³

Sources of Nontherapeutic AAS. The United States classifies AAS as Schedule III controlled substances, which require a prescription.^{1,3} Research indicates that AAS abusers obtain their drugs through many venues.^{8,37,40,41,49} Among male high school senior AAS abusers, 60.5% obtained their drugs via black-market sources, with about 20% obtained from physicians, pharmacists, and veterinarians.⁴¹ Similarly, 59% of community college abusers obtained their AAS through black-market sources.⁸ Physicians who provided AAS with a prescription (26.5%) and without a prescription (8.8%) were the next most common sources.⁸ Only 10% of AAS-abusing power lifters obtained their drugs from physicians or pharmacists.⁴⁰ The 2009 NCAA study revealed the most common AAS source for collegiate abusers was a teammate, friend, or family member.³⁷ About 14% acquired their drugs via the Internet, whereas physicians, coaches, and athletic trainers were the declared sources for 13%, 7%, and 6%, respectively.³⁷ With many of these drugs coming from nonmedicinal sources, one must always consider the likelihood of contamination, as well as the accuracy of the actual drug amount.^{52,127}

AAS Pharmacology

Hormones are compounds produced by the endocrine system that chemically regulate specific body functions. Steroid hormones have a biochemically unique 4-ring structure, including three 6-carbon rings and one 5-carbon ring (Figure 1).¹⁴ Androgens are steroid hormones

synthesized primarily in the gonads and adrenal glands.^{1,4,7,14–16,51,127} Dihydrotestosterone, dehydroepiandrosterone, androstenedione, androstenediol, and testosterone are the human androgens (Figure 2).¹⁵ Testosterone (Figure 3) is the key androgen promoting masculine (androgenic) characteristics, as well as maintaining nitrogen balance and facilitating protein synthesis (anabolism).¹⁴ Males synthesize 2.5 to 11 mg of testosterone per day, whereas females produce about 0.25 mg daily.⁷ Although both males and females synthesize testosterone, serum concentrations in males (300 to 1000 ng/dL) are significantly greater than in females (15 to 65 ng/dL).⁷ In addition to individual differences and sex variability, testosterone levels generally decline in older men and vary with certain disease states (eg, adrenal gland disorders in males and females, as well as polycystic ovary syndrome and rare ovarian cancers in women).^{7,17–20}

Throughout history, pharmaceutical companies and clandestine laboratories developed scores of unique derivatives of the endogenous androgenic hormones, including testosterone (Table 3).^{13–15} New AAS innovations seek to enhance or minimize certain effects, improve administration or absorption, improve receptor affinity, and, more recently, avoid detection.^{14,15} Frequently, efforts are focused on facilitating anabolism and inhibiting androgenesis, although to date no AAS are solely anabolic.^{7,14}

Pharmacokinetics. Unaltered oral testosterone is readily metabolized by the liver, so pharmaceutical companies modify the molecule or the delivery system to enhance its bioavailability, change its rate of absorption, and enhance or diminish its various characteristics.¹⁴ Traditionally, AAS administration routes included oral or parenteral routes and subcutaneous pellets, but buccal and transdermal methods are now also available.^{14,15} Testosterone and its derivatives are often alkylated at the 17 β -hydroxy position to decrease first-pass liver metabolism when administered orally.^{14,15} Esterification at the 17 β -hydroxy site makes AAS molecules less polar and more soluble as an oil-based injectable.^{14,15}

Once absorbed into the blood, AAS pass throughout the circulatory system, generally bound to either sex-hormone-binding globulin or more loosely to the plasma protein albumin.^{7,14,15} Less than 2% of testosterone remains unbound and freely available.^{7,9} Commonly metabolized by the liver, AAS molecules convert to androsterone and etiocholanolone.^{7,15} These biologically inert compounds undergo glucuronidation or sulfation and are primarily excreted through the urinary system.¹⁴

Pharmacodynamics. The AAS are exogenous synthetic derivatives of, and generally function as agonists for, the androgen hormones.^{1,2,4,7,14} The AAS mechanism of action occurs at the cellular level and requires a variety of specific enzymes in concert with the many androgen receptors.^{14,15} The best current evidence suggests that AAS pharmacodynamics mechanisms on muscle tissue are numerous and varied.^{7,14,15}

The primary AAS mechanism of action involves the stimulus for DNA transcription.^{14,15} Once they are available at the cellular level, the lipid-soluble AAS molecules diffuse across the cell membrane into the cytoplasm, where they bind with the intracellular androgen receptors.^{14,15} These receptor-bound compounds enter the cell nucleus to bind with DNA, which promotes transcription, thus

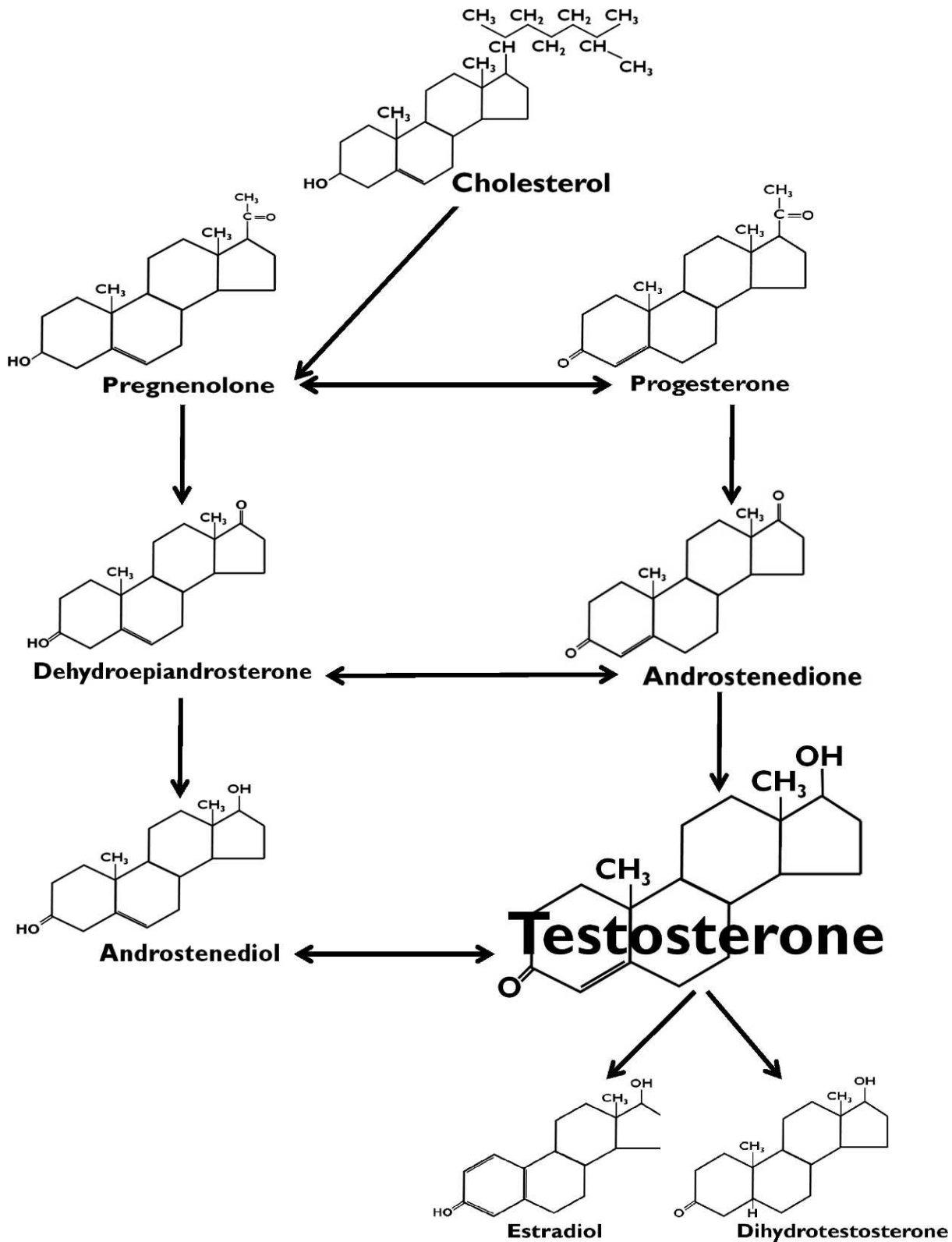


Figure 2. Biochemical pathways of the human androgens: androstenediol, androstenedione, dehydroepiandrosterone, dihydrotestosterone, and testosterone.

stimulating protein synthesis.^{14,15} The AAS also may work as glucocorticoid antagonists and displace these catabolic compounds from their receptor sites, thereby limiting the possible effects of cortisol and related compounds.¹⁴ Some authors^{14,22} also suggest a possible ergogenic effect of AAS

through psychotropic actions. Preliminary research indicates that other possible ergogenic actions of AAS require further study,¹⁴ including the downregulation of myostatin,^{7,23,24} and the possible stimulus to synthesize human growth hormone and insulin growth factor 1.¹²

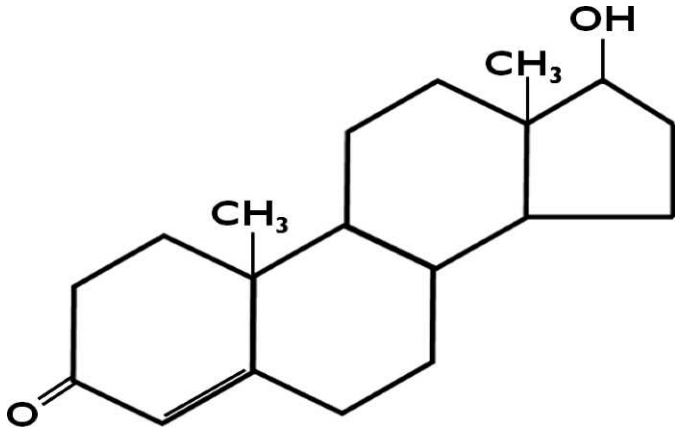


Figure 3. Chemical structure of testosterone.

Therapeutic Uses of AAS

Certain medicinal uses for AAS are accepted; other possible health benefits are not currently acknowledged or accepted by the medical community.^{1,2,6,7,14,17–19,24,26–33} Pharmacologic uses of AAS for the treatment of diseases and disorders continue to evolve.^{1,2,7,14,18,19} Although any potential therapeutic benefits must not be exaggerated, it is just as important to not overstate the health risks.^{14,56}

Reproductive Health. Testosterone and its synthetic analogs have long been considered possible treatment agents for various male reproductive disorders.^{1,2,7,16–19,26} Two meta-analyses^{18,19} suggested that AAS treatment may be associated with benefits for male sexual function but cautioned about the somewhat weak results and cited a need for more well-designed, long-term studies. Additionally, the authors²⁶ of a systematic review reported that no AAS-based male contraceptive was ready for therapeutic use but encouraged continued research.

Anticatabolism. The AAS have been clinically used to treat catabolic conditions since the 1940s.⁷ Generally, the strongest evidence for anticatabolic AAS efficacy involves the treatment of severe burn patients.^{7,24} High-quality systematic reviews and meta-analyses that addressed chronic catabolism with AIDS tend to show positive outcomes for AAS therapy.^{7,24,27,28} Equivocal results were demonstrated for AAS use in other wasting conditions, including chronic obstructive pulmonary disease,^{7,30,32} wound healing and postoperative recovery,¹⁵ and alcohol-related liver disease.^{7,24,31} To solidify the efficacy and safety of therapeutic AAS use for anticatabolism, more well-designed studies are needed.^{7,24,28–30}

Bone Health. Based on a 2006 systematic review and meta-analysis²⁰ on testosterone and bone health, intramuscular AAS injections moderately improved lumbar bone density, but the effects on femoral-neck density were inconclusive. The clinical efficacy of testosterone to prevent or treat osteoporosis was weak, although the authors encouraged further study of fractures and testosterone.

Anti-Aging. With advancing age, male endogenous testosterone production declines,^{7,18–20} which has been suggested by some to underlie many health maladies.^{17,18} Therapeutic AAS use has increased in middle-aged to older men as an anti-aging therapy while spurring reviews of efficacy and safety.^{6,17,18} Additionally, recent concerns

about female hormone replacement therapy (HRT) contribute to the controversial use of AAS for “andropause.”⁶ A number of systematic reviews and meta-analyses^{6,17,18,33} indicate possible benefits, but the findings remain inconclusive. Lifestyle, including increased activity, may limit some age-related negative hormonal changes without the negative pharmacologic side effects.⁶² Further study is required before definitive statements can be made.¹⁸

Other Medicinal Purposes. Although treatment efficacy remains uncertain, AAS also have been considered and used in the therapeutic treatment of some cancers (eg, certain breast cancers) and chronic renal failure, among other conditions.^{2,7,16}

Ergogenic Effects of AAS

A number of high-quality scientific papers^{57–66,130–132} addressed the efficacy of AAS on mass, body composition, and physical performance (Table 4). Many issues confound AAS efficacy study designs, thus yielding diverse results.^{11,13,22,51,52,55,56} Ethical issues aside, institutional review boards generally do not allow researchers to duplicate real-world supraphysiologic, polypharmaceutical dosing regimens.^{11–13,51,52,55,56} Furthermore, the effectiveness of participant blinding when studying AAS is questionable.⁵⁹ Efficacy studies involve many different research factors, including numerous AAS, varied dosages, assorted training protocols, dissimilar periods, genetically diverse participants, dietary variances, and numerous physical performance measures.^{11–13,20,51,55,59} The complexity of AAS efficacy studies makes comparisons and conclusions difficult.^{11,20,22,51,55,56,59} Well-designed studies are prospective, placebo controlled, and double blind in nature.^{20,51} Protocols also must involve random assignment and control diet and training.^{20,51} Sample size, participant background, performance measures, and a crossover design also affect a study’s overall quality.^{20,51} After evaluating scores of AAS efficacy studies according to these criteria, we provide the following examples of high-quality studies.

AAS Effects on Body Composition. In one early study¹³⁰ involving 28 moderately active college-aged males, participants were randomly assigned to the group receiving 1.0 mg/kg of nandrolone decanoate in weeks 2, 5, and 8 or a placebo group. After 9 weeks of progressive strength training without dietary controls, no changes were noted in body mass or composition. Another group¹³¹ completed a double-blind, placebo-controlled, random-assignment project in which participants received either a placebo or 75 mg/d (increased to 150 mg/d after 4 weeks) of mesterolone. All 21 healthy young male participants completed a high-intensity, progressive resistive training program throughout the 8-week study. No changes were seen in body mass for either group, although thigh circumference increased for the AAS group.¹³¹ In a 4-week, double-blind, placebo-controlled 1971 study,⁶³ 20 collegiate weightlifters were randomly assigned to ingest 10 mg/d of methandrostenolone or a placebo. A significant increase in total body mass occurred, with caliper measurements suggesting an increase in lean tissue. Others⁶⁴ reported that healthy males given daily 5-mg doses of methandrostenolone increased muscle size (circumference) and total weight and decreased

Table 4. Selected High-Quality Studies on Anabolic-Androgenic Steroid Efficacy

Author(s) (Year)	Diet Controlled?	Participants, N	Age, y	Training	Study Groups	Outcomes
Baume et al (2006) ¹³²	No	25	20–30	7 d/wk × 4 wk (endurance)	300 mg/wk 19-nor-4-androstenedione 240 mg/wk testosterone undecanoate 900 mg/wk placebo	<ul style="list-style-type: none"> ∅ Serum: AST, creatine kinase, urea ∅ Urine: adrenaline, noradrenaline, dopamine ∅ Treadmill running: maximum, submaximum ↑ Leg press (1RM) ↑ Lean mass, triceps and quadriceps size ↑ Testosterone (free, total) ↓ FSH, LH
Bhasin et al (2005) ⁵⁸	Yes	52	60–75	20 wk	25 mg/wk testosterone enanthate 50 mg/wk testosterone enanthate 125 mg/wk testosterone enanthate 300 mg/wk testosterone enanthate 600 mg/wk testosterone enanthate	<ul style="list-style-type: none"> ↑ Bench press, squat (1RM) ↑ Fat-free mass, triceps and quadriceps size ↑ Testosterone (free) ↓ FSH, LH, SHBG ∅ Body composition, anthropometrics ∅ Bench press, dead lift (1RM) ∅ Maximal aerobic capacity, peak work ∅ Bench press (1RM), mass, lean mass ↑ Arm girth, rectus femoris circumference ↓ Abdominal skinfold thickness ↑ Mass, lean mass, limb circumferences, muscle widths
Bhasin et al (1996) ⁶⁰	Yes	40	19–40	3 d/wk × 10 wk	600 mg/wk placebo w/o exercise 600 mg/wk placebo w/exercise 600 mg/wk testosterone enanthate w/o exercise 600 mg/wk testosterone enanthate w/exercise	<ul style="list-style-type: none"> ↑ Total potassium, total nitrogen, leg strength ∅ Arm or hand strength, fat and subcutaneous fat ↑ Dynamic and static strength, mass, limb size ∅ V_{O2}max, sperm count, subcutaneous fat ↑ Mass (total, fat free), body mass index
Fahey and Brown (1973) ¹³⁰	No	28	College	3 d/wk × 8 wk	1 mg/kg × 3 wk nandrolone decanoate 1 mg/kg × 3 wk placebo	<ul style="list-style-type: none"> ↑ Mass, squat, bench press (1RM) ↑ Cycle sprint (peak power, total work) ∅ Muscle fatigability, specific tension
Giorgi et al (1999) ⁶¹	No	21	19–45	4 d/wk × 12 wk	3.5 mg/kg/wk testosterone enanthate 3.5 mg/kg/wk placebo	<ul style="list-style-type: none"> ↑ Thigh circumference ∅ Maximal strength, mass, V_{O2}max, free testosterone ↓ Testosterone (plasma-bound, total)
Hervey et al (1981) ⁶⁵	No	7	25–38	Variable for 6 wk	100 mg/d methandienone 100 mg/d placebo	<ul style="list-style-type: none"> ∅ Serum: AST, creatine kinase, urea ∅ Urine: adrenaline, noradrenaline, dopamine ∅ Treadmill running: maximum, submaximum ↑ Leg press (1RM) ↑ Lean mass, triceps and quadriceps size ↑ Testosterone (free) ↓ FSH, LH, SHBG ∅ Body composition, anthropometrics ∅ Bench press, dead lift (1RM) ∅ Maximal aerobic capacity, peak work ∅ Bench press (1RM), mass, lean mass ↑ Arm girth, rectus femoris circumference ↓ Abdominal skinfold thickness ↑ Mass, lean mass, limb circumferences, muscle widths ↑ Total potassium, total nitrogen, leg strength ∅ Arm or hand strength, fat and subcutaneous fat ↑ Dynamic and static strength, mass, limb size ∅ V_{O2}max, sperm count, subcutaneous fat ↑ Mass (total, fat free), body mass index
Johnson et al (1972) ⁶⁴	Protein intake only	31	18–38	3 d/wk × 3 wk	10 mg/d methandrostenolone 10 mg/d placebo control	<ul style="list-style-type: none"> ↑ Mass, squat, bench press (1RM) ↑ Mass, leg press, bench press (1RM) ↑ Cycle sprint (peak power, total work) ∅ Dose-dependent leg press, leg power (1RM) ∅ Muscle fatigability, specific tension
Lichtenbelt et al (2004) ⁵⁷	Yes	16	19–44	8 wk	200 mg/wk nandrolone decanoate 200 mg/wk placebo	<ul style="list-style-type: none"> ↑ Thigh circumference ∅ Maximal strength, mass, V_{O2}max, free testosterone ↓ Testosterone (plasma-bound, total)
O'Shea (1971) ⁶³	Protein intake only	20	19–26	3 d/wk × 4 wk	10 mg/d methandrostenolone 10 mg/d placebo	<ul style="list-style-type: none"> ∅ Serum: AST, creatine kinase, urea ∅ Urine: adrenaline, noradrenaline, dopamine ∅ Treadmill running: maximum, submaximum ↑ Leg press (1RM) ↑ Lean mass, triceps and quadriceps size ↑ Testosterone (free, total) ↓ FSH, LH ∅ Body composition, anthropometrics ∅ Bench press, dead lift (1RM) ∅ Maximal aerobic capacity, peak work ∅ Bench press (1RM), mass, lean mass ↑ Arm girth, rectus femoris circumference ↓ Abdominal skinfold thickness ↑ Mass, lean mass, limb circumferences, muscle widths ↑ Total potassium, total nitrogen, leg strength ∅ Arm or hand strength, fat and subcutaneous fat ↑ Dynamic and static strength, mass, limb size ∅ V_{O2}max, sperm count, subcutaneous fat ↑ Mass (total, fat free), body mass index
Rogerson et al (2007) ⁵⁹	Yes	16	21–35	2–3 d/wk × 6 wk	3.5 mg/kg/wk testosterone enanthate 3.5 mg/kg/wk placebo	<ul style="list-style-type: none"> ↑ Mass, leg press, bench press (1RM) ↑ Cycle sprint (peak power, total work) ∅ Dose-dependent leg press, leg power (1RM) ∅ Muscle fatigability, specific tension
Storer et al (2003) ⁶⁶	Yes	54	18–35	None	25 mg/wk testosterone enanthate 50 mg/wk testosterone enanthate 125 mg/wk testosterone enanthate 300 mg/wk testosterone enanthate 600 mg/wk testosterone enanthate	<ul style="list-style-type: none"> ↑ Thigh circumference ∅ Maximal strength, mass, V_{O2}max, free testosterone ↓ Testosterone (plasma-bound, total)
Stromme et al (1974) ¹³¹	Protein intake only	21	21–27	3 d/wk × 8 wk	75 mg/d × 4 wk to 150 mg/d × 4 wk mesterolone 75 mg/d × 4 wk to 150mg/d × 4 wk placebo	<ul style="list-style-type: none"> ∅ Serum: AST, creatine kinase, urea ∅ Urine: adrenaline, noradrenaline, dopamine ∅ Treadmill running: maximum, submaximum ↑ Leg press (1RM) ↑ Lean mass, triceps and quadriceps size ↑ Testosterone (free, total) ↓ FSH, LH ∅ Body composition, anthropometrics ∅ Bench press, dead lift (1RM) ∅ Maximal aerobic capacity, peak work ∅ Bench press (1RM), mass, lean mass ↑ Arm girth, rectus femoris circumference ↓ Abdominal skinfold thickness ↑ Mass, lean mass, limb circumferences, muscle widths ↑ Total potassium, total nitrogen, leg strength ∅ Arm or hand strength, fat and subcutaneous fat ↑ Dynamic and static strength, mass, limb size ∅ V_{O2}max, sperm count, subcutaneous fat ↑ Mass (total, fat free), body mass index

Abbreviations: ∅, no changes; ↑ increase; ↓, decrease; 1RM, 1-repetition maximum; AST, aspartate aminotransferase; FSH, follicle-stimulating hormone; LH, luteinizing hormone; SHGB, sex-hormone-binding globulin; w/o, without.

subcutaneous adipose tissue compared with the placebo group. Authors of a 1981 study⁶⁵ reported the effects of 100 mg/d of methandienone on 7 male weightlifters. The double-blind, placebo-controlled study involved a crossover design, including two 6-week periods with a 6-week washout. Participants gained weight during the AAS periods, although body fat did not vary.

A well-designed and well-controlled 1996 study⁶⁰ investigated the effects of a 600-mg weekly dose of testosterone enanthate or placebo on body mass and composition. A total of 40 young, healthy participants completed the 30-week study, which consisted of 4 weeks of a control period, 10 weeks of treatment, and 16 weeks of recovery. This double-blind study involved 4 randomly determined groups: placebo without training, placebo with training, AAS without training, and AAS with training. The researchers also controlled protein and total caloric intake. Mean weight in the placebo groups did not change but increased in both AAS groups; the AAS-with-training group gained significantly more weight than the AAS-without-training group. Additionally, except for the placebo-without-training group, all groups gained lean mass. Finally, muscle cross-sectional area increased for both AAS groups; the AAS-with-training group achieved the greatest gains.⁶⁰

A 2004 double-blind, randomized study⁵⁷ of 16 experienced bodybuilders involved weekly injections of nandrolone decanoate or a placebo (200 mg) for 8 weeks. Participants continued training at their normal levels. The AAS group increased total body mass and relative lean tissue compared with the placebo group.

Authors of a 1999 study⁶¹ reported weight and size (ie, girth) increases for strength-training participants given testosterone enanthate compared with a placebo. After random assignment, 21 young, healthy males completed 12 weeks of double-blind drug or placebo (weekly doses of 3.5 mg/kg) and strength-training intervention. Weight and size increases reverted to normal 12 weeks after the drug intervention.⁶¹

One 2005 paper⁵⁸ detailed a 36-week prospective study of testosterone enanthate and gonadotropin-releasing hormone agonists on 60 healthy, older (60–75 years) men. During the 20-week treatment period, participants received weekly injections of doses ranging from 25 to 600 mg, depending on their random group assignment. Participants did not strength train or complete moderate to intense aerobic exercise during the study. A positive, dose-related response with regard to lean mass increases and an inverse dose-response relationship to fat mass were observed.

Finally, 16 healthy collegiate men enrolled in a 2007 placebo-controlled, double-blind study⁵⁹ to investigate, in part, the effects of AAS on body mass and composition. Researchers randomly assigned participants to receive weekly injections of either testosterone enanthate or placebo. All trained for muscle hypertrophy during the trial. The AAS group gained more mass than the placebo group.⁵⁹

Overall, the best science related to the effects of exogenous, nontherapeutic abuse of AAS on body composition suggests a dose-related potential for increased relative lean body mass.^{57–61}

AAS Effects on Physical Performance. Even though evidence suggests possible increased lean mass with AAS

abuse,^{57–61,63–65} more muscle does not always equal improved performance. In some endeavors, such as marathons and triathlons, increased mass (lean or otherwise) may lead to a performance decrease. Considering the known association between muscle mass and force development, a relationship between AAS abusers and strength or power activities, although not necessarily causal, is not unexpected.

The authors of an early study⁶³ investigated methandrostenolone use at 10 mg/d and resistance training by university students over 4 weeks. All participants ingested a high-protein supplement. The protocol involved a randomized, double-blind, placebo-controlled design. Training with AAS increased performance in the 1-repetition maximum (1RM) bench press and squat. Others⁶⁴ studied the use of 10 mg/d of methandrostenolone and a protein supplement by college students. This randomized, double-blind, placebo-controlled study lasted 7 weeks, including the 3-week treatment phase. Participants trained 3 times per week at increasing intensities. The authors reported increases in 1RM bench press and squats by the treatment group over both the placebo and control groups.

A 1973 paper involved a randomized, double-blind, placebo-controlled study on AAS efficacy in 28 healthy males.¹³⁰ Throughout the 9-week study, participants strength trained 3 days per week using progressive resistance. Participants received 1.0 mg of nandrolone decanoate or saline solution per kilogram of body weight during weeks 2, 5, and 8. This study did not control for diet, nor did it supplement with additional protein. No significant gains were seen in the AAS group over the placebo group for any performance indicators, including 1RM bench press and dead lift, as well as all isokinetic peak torques for elbow, shoulder, and knee flexion-extension movements.

Other investigators¹³¹ designed a double-blind, placebo-controlled prospective study involving 21 healthy college students. Random assignment was to either the mesterolone or placebo group. Drug dosage increased from 75 mg/d to 150 mg/d midway through the 8-week study. The authors controlled a progressively increasing, high-intensity training program. Dietary protein (30 g/d) was supplied to both groups throughout the study. Performance measures included maximal isometric force for 4 motions (elbow flexion, trunk flexion, trunk extension, and knee flexion-extension). No differences were seen between the AAS and placebo groups.

A 1981 prospective, randomized, double-blinded, placebo-controlled, and crossover study consisted of 7 healthy, young weight lifters.⁶⁵ Two treatment periods (100 mg/d of methandrostenolone) of 6 weeks were separated by 6 weeks of drug washout. Progressive resistance training occurred over the full 18 weeks. Gains were observed in leg strength (isometric dynamometry similar to the dead lift) but not with the isometric shoulder press or hand grip.

More recently, a few well-designed AAS efficacy studies were conducted in healthy, eugonadal participants.^{59–61,66,132} One group⁶⁰ examined the effects of 600 mg/wk of testosterone enanthate on 40 experienced male weight lifters, ages 19 to 41 years. Participants were assigned to 1 of 4 groups: testosterone with exercise, testosterone without exercise, placebo with exercise, and placebo without exercise. The authors controlled diets, and all participants consumed 1.5 g of protein per kilogram of body weight.

Training consisted of supervised, moderate-intensity weight training 3 days per week for 10 weeks. Participants in the testosterone-with-exercise group experienced the greatest performance gains, as measured by 1RM bench press and squat. The testosterone-without-exercise and placebo-with-exercise groups also gained strength when compared with the placebo-without-exercise group.⁶⁰

A 1999 study⁶¹ prospectively investigated the effects of 3.5 mg/kg of testosterone enanthate over 12 weeks of strength training by 21 healthy, active males in a double-blind trial. After random assignment, participants completed a 4-d/wk, periodized, whole-body strength-training program determined by their individual baselines. Gains occurred in the 1RM bench press for those receiving the testosterone injections compared with the placebo group. Twelve weeks of follow-up training without injections resulted in a return of the measure to that of the placebo group.⁶¹

In a prospective, 20-week 2003 study,⁶⁶ 61 young, healthy males with prior resistance training experience were randomly assigned to 1 of 5 treatment groups receiving weekly testosterone enanthate injections: 25 mg, 50 mg, 150 mg, 300 mg, or 600 mg. They did not train during the treatment period. Daily energy intake involved 150 kJ·kg⁻¹·d⁻¹, including 1.3 g/kg of protein. A dose-dependent increase in leg-press strength and power was observed.

Authors⁵⁹ of a 2007 paper reported findings of a 6-week, randomized, double-blind, placebo-controlled investigation of 16 young, healthy males. Participants received 3.5 mg of testosterone enanthate or saline per kilogram of body weight each week for 6 weeks and performed a structured heavy-resistance training protocol throughout the trial. Living conditions were also controlled, and participants consumed standardized meals that included protein supplementation. The experimental group's 1RM bench press and total work values during a 10-second cycle sprint were improved at 3 and 6 weeks.⁵⁹

A well-designed project¹³² looked at the efficacy of AAS as related to endurance performance. Twenty-four young, active, healthy male volunteers participated. After initial testing to determine running velocity at individual anaerobic thresholds, they completed personalized 4-week training protocols designed to improve this measure and during that time ingested 300 mg/wk of 19-nor-4-androstenedione, 240 mg/wk of testosterone undecanoate, or 300 mg/wk of placebo. The study was randomized and double blinded. Anaerobic threshold velocities did not vary either before or after treatments. Although all groups improved their running velocity at anaerobic threshold after 4 weeks of AAS use and training, neither of the experimental groups (19-nor-4-androstenedione or testosterone) improved more than the placebo group.

Last, many recent scientific investigations involved the use of AAS for HRT among older adults or hypogonadal participants. Dozens of published papers examined the effects of HRT on a variety of variables, including muscular performance.^{6,62} The HRT findings were summarized in a meta-analysis⁶ and a systematic review.⁶² In their 2006 meta-analysis⁶ of 11 prospective studies, the authors suggested a "moderate increase in muscle strength" with HRT in elderly men. In a systematic review⁶² of various sarcopenia treatments, the authors concluded that male

HRT produced moderate strength increases, although not all protocols were successful.

Health Effects of AAS

Some⁵⁶ have suggested that AAS abuse risks are exaggerated, but others^{72,73,133,134} proposed that supra-physiologic AAS abuse negatively affects human health, even to the point of premature death. Therapeutic AAS use helps to resolve specific health maladies,^{6,7,13,18–20,24,26–33} but like all medicinal agents, AAS also have side effects (Table 5). Making definitive claims concerning the effects of long-term, high-dose, multidrug AAS abuse is difficult because researchers cannot ethically administer such protocols.^{11–13,51,52,55,56} These scenarios have never been and may never be duplicated in a controlled research setting.^{22,51,52,55,56} With AAS abuse, as with those seeking improved performances or physiques, the negative effects appear to increase, thus presenting health risks.

Effects of AAS Abuse on the Central Nervous System. Supraphysiologic doses of AAS are associated with a variety of neuropsychological effects. The best established of these effects are AAS-associated mood disorders, AAS dependence syndromes, and progression from AAS to other forms of substance abuse and dependence.

AAS-Associated Mood Disorders. A growing body of literature indicates that markedly supra-physiologic doses of AAS may cause hypomanic or manic syndromes in some individuals.^{21,54,67–78} These syndromes involve euphoric or irritable mood, exaggerated self-confidence (which may occasionally result in grandiose delusions), decreased need for sleep, hyperactivity (including sexual hyperactivity), and reckless or sometimes violent behavior.⁶⁷ However, these syndromes are idiosyncratic. Most AAS abusers, even when using high doses, do not display major mood disturbances, although a minority may exhibit dramatic changes, with behavior completely different from their baseline personalities.²¹ We have no clear way to predict who will and will not develop these syndromes. Hypomanic syndromes may occur in individuals without a history of mood disorder or personality disturbance.²¹

Some evidence suggests an association between AAS dose and the risk of manic or hypomanic episodes. For example, laboratory studies using only modestly supra-physiologic doses of testosterone or equivalent AAS (up to 300 mg/wk, representing 4 to 6 times normal male endogenous testosterone production) have rarely found clinically significant mood disorders among participants.²¹ Similarly, among illicit AAS abusers, mood disorders rarely appeared in individuals taking the equivalent of 300 mg/wk of testosterone¹³⁵ and are uncommon even in those taking the equivalent of 300 to 1000 mg/wk.^{21,68} However, these disorders occur at increased frequency among men taking doses of more than 1000 mg per week.⁶⁸ In women, AAS abuse is rare, and indeed, in only 1 study⁶⁹ in the past 15 years have investigators successfully recruited and interviewed female AAS abusers. Among the 25 AAS-abusing participants, none met the diagnostic criteria for hypomanic or manic syndrome, although some described feelings of euphoria, irritability, or increased libido.⁶⁹

The lay press commonly refers to AAS-induced mood syndromes by the popular term *roid rage*, implying that aggressive or violent behavior is a central feature. Although

Table 5. Possible Anabolic-Androgenic Steroid Abuse Health Effects

System	Possible Adverse Effects
Cardiovascular	Cardiac dysfunction: ↓ left ventricle peak ejection fraction, systolic strain, early diastolic peak velocities, E/A ratio, right ventricle function
Central nervous	Major mood disorders, dependence, progression to other forms of substance dependence
Hematologic	↑ total cholesterol, low-density lipoproteins; ↓ high-density lipoproteins
Hepatic	Cholestatic hepatitis, cholestatic jaundice, peliosis hepatis, adenomas, carcinomas
Immune	General immunosuppression: ↓ antibody synthesis, B- and T lymphocyte maturation, natural killer cell activity; systemic infection (from needle, syringe misuse)
Integumentary	Sebaceous gland hypertrophy, ↑ sebum and skin lipid excretion, ↑ acne, seborrhea, striae distensae, hypertrichosis, hirsutism, rosacea, folliculitis, keloids, furunculosis
Neuroendocrine	
Male	Hypothalamic-pituitary-testicular axis suppression, hypogonadism, testicular atrophy, sexual dysfunction, infertility, gynecomastia, male-pattern baldness, libido changes, prostate hypertrophy, cancer
Female	General virilization, hirsutism, voice deepening, male-pattern baldness, clitoral hypertrophy, breast reduction, menstrual dysfunction, libido changes
Musculoskeletal	Premature epiphyseal closure, tendinopathies
Nephritic	Kidney stones, cholestasis, tubular necrosis, hyperbilirubinemia, proteinuria, focal segmental glomerulosclerosis, renal cell cancer, Wilms tumor
Oral	Gingival hypertrophy, ↑ temporomandibular dysfunction

Abbreviations: ↑, increase; ↓, decrease; E/A ratio, the ratio of passive ventricle filling (early [E] wave) and active filling with atrial systole (atrial [A] wave).

a colorful term, *roid rage* has no agreed scientific meaning. Nevertheless, AAS-induced hypomanic and manic syndromes often are associated with irritable or aggressive behavior.²¹ In some, this behavior may rise to the level of outright violence, and the victims are often women.⁷⁰ Several case reports²¹ have described individuals who committed violent crimes while using AAS. Some of them had no history of violence, no criminal records, and no evidence of psychiatric disorder before AAS exposure, suggesting that the observed behavior was attributable to a biological AAS effect. The authors⁷¹ of a literature review on AAS-induced aggression suggested that this phenomenon is sufficiently well documented to meet the so-called Daubert criteria, as a scientifically accepted event admissible in court testimony.

Various forensic studies suggest an association between AAS abuse and violent or criminal behavior.^{72–76} These include case series, describing unusual violence among young AAS abusers⁷⁴; epidemiologic data suggesting both a possible link between AAS abuse and death by homicide or suicide^{72,73} and a relationship between AAS abuse and conviction for weapons offenses.⁷⁶ It is important to note the direction of causality cannot be determined in such studies; it may be that AAS abuse predisposes individuals to violent behavior, that violent individuals are more predisposed to AAS abuse, or both.

This issue of causality raises the more general question of whether AAS-associated manic syndromes are attributable to a biological effect of AAS themselves, as opposed to underlying personality attributes of the abuser or psychosocial factors surrounding AAS abuse.^{21,71,136} One might argue that individuals who abuse AAS are inherently more prone to aggression or, alternatively, that expectations may prompt individuals to behave aggressively, even in the absence of an actual biological effect. However, in several prospective, randomized, double-blind, placebo-controlled studies,^{54,77,78} investigators administered supraphysiologic doses of AAS to normal volunteers and demonstrated hypomanic effects in some participants, suggesting that these effects cannot be entirely explained by psychosocial factors. Specifically, among 109 men in 4 studies who

received doses of at least 500 mg/wk of AAS under blinded conditions, 5 (4.6%) developed hypomanic or manic syndromes on AAS, but none developed these syndromes on placebo.⁵⁴ This figure is likely an underestimate of the true prevalence of hypomanic or manic episodes among AAS abusers, who frequently receive much larger doses.²¹ Abusers of AAS typically use several AAS simultaneously,^{1,4,8,9,21,41,45} thus possibly further increasing their psychiatric vulnerability. Finally, these studies generally excluded participants with preexisting psychopathology, whereas AAS abusers do not similarly screen themselves.

Other lines of evidence also suggest that AAS-associated mood syndromes cannot be entirely explained as the result of premorbid personality factors or expectations. For example, 3 reports collectively described 4 monozygotic twin pairs in which 1 twin abused AAS and the other did not.^{137–139} In each case, the AAS-abusing twin exhibited prominent AAS-associated symptoms, including suicide in 1 case¹³⁹ and extreme violence in another.¹³⁸ None of the non-AAS-abusing twins exhibited any significant psychopathology.^{137–139} The AAS also induce aggression in rodent models,^{50,140} again suggesting a biological component in these effects. However, the reasons for individual variability (in both humans and animals) remain unclear.

Evidence from naturalistic studies also suggests that episodes of major depression may arise in AAS abusers, especially during withdrawal.⁷⁹ These depressive symptoms may be partially attributable to suppression of the hypothalamic-pituitary-testicular axis by AAS.^{7,11–14,22,51} As in hypomanic syndromes, depressive episodes appear to be highly idiosyncratic, with many individuals exhibiting virtually no depressive symptoms during withdrawal and occasional individuals becoming profoundly depressed and even suicidal.⁸⁰ Again, the reasons for this variability are unknown.

For largely ethical reasons,^{11–13,23,52,55} no laboratory studies have been conducted on the withdrawal effects from supraphysiologic AAS doses. It is, of course, unethical to administer massive, prolonged AAS doses to normal volunteers and then deliberately precipitate withdrawal. Thus, evidence regarding these depressive effects

necessarily stems almost entirely from naturalistic investigations and is subject to all related limitations, such as selection bias, recall bias, and the presence of confounding variables.⁸⁴ Yet laboratory investigations of normal volunteers suggest that pharmacologically induced hypogonadism may indeed be associated with depressive reactions that occur idiosyncratically in some individuals.⁸¹

AAS Dependence. Increasingly acknowledged is that some AAS abusers develop a syndrome of AAS dependence.^{82,83} To date, 8 field studies conducted in the United States, Great Britain, and Australia have collectively noted AAS dependence in 197 (30.2%) of 653 AAS abusers.⁸² Even if one was to assume an estimate of much less than this 30% figure, it would follow that there are still hundreds of thousands of AAS-dependent Americans and millions more worldwide who have consumed AAS for prolonged periods, often despite adverse medical, psychiatric, and social consequences. Dependence on AAS may arise via any or all of 3 possible mechanisms: anabolic, androgenic, or hedonic.⁸⁴ The anabolic mechanism refers to the muscle-building effects of AAS, which may be particularly appealing to men (and, in rare cases, women) with body-image disorders who wish to look leaner and more muscular.⁸⁴ Some individuals experience a syndrome termed *muscle dysmorphia*, or *reverse anorexia nervosa*, a form of body dysmorphic disorder in which individuals become preoccupied with their perceived lack of muscularity.¹⁴¹ People with this form of body dysmorphic disorder may be particularly at risk for repeated AAS abuse because they constantly strive to be bigger.^{85,86} Paradoxically, however, some individuals develop worsening symptoms of muscle dysmorphia with AAS abuse, perhaps because they become increasingly preoccupied with their muscularity.⁸⁴ Thus, they may continue to repeat AAS cycles, afraid of the possibility of losing even a little muscle size if they discontinue the drugs. Published descriptions of such individuals suggest this phenomenon is quite similar (albeit in reverse) to classical anorexia nervosa, in which the patient experiences severe anxiety about the prospect of gaining even a pound.¹⁴² Additionally, athletes may be further disposed to muscle dysmorphia and related body-image concerns due to pressures to be and appear athletically competent. This may encourage many to train with the dual intents of improving both performance and appearance. As such, some may decide to abuse AAS to achieve not only their athletic demands but also their psychological expectations.¹⁴³

In susceptible individuals, AAS dependence may be further potentiated by the androgenic effects whereby these drugs affect the abuser's own endocrine function. Specifically, men who abuse AAS for prolonged periods may profoundly suppress their hypothalamic-pituitary-testicular functioning, to the point that protracted AAS-withdrawal hypogonadism develops every time they stop AAS.^{84,87} This hypogonadism, as mentioned earlier, may be associated with depression or with other symptoms, such as loss of sex drive and fatigue.¹⁴⁴ Therefore, these individuals may soon be tempted to resume AAS to abort these dysphoric effects and may eventually develop a pattern of continuous AAS abuse.

It now appears that AAS dependence may evolve by a third pathway in a mechanism similar to that of classical drug addiction.^{84,88} Documentation of this hedonic pathway

has occurred in laboratory animals. For instance, rodents will self-administer testosterone, even to the point of death¹⁴²; some evidence suggests that this self-administration process may be at least partially mediated by opioidergic mechanisms.⁸⁹ A similar process likely occurs in humans. It is notable that AAS-dependent humans often report other forms of classical substance dependence, indicating that certain individuals may have an innate susceptibility to substance-dependence syndromes in general, including dependence upon AAS.⁸² Clinical observations suggest that AAS dependence, as it evolves, may increasingly resemble classical substance dependence in its behavioral manifestations. In particular, AAS abuse and the surrounding lifestyle of the "gym culture" may become the central focus of an individual's life.¹⁴⁵

Progression to Other Forms of Substance Dependence. Results from several studies suggest that AAS abusers may be prone to develop other forms of drug dependence, especially opioid dependence.^{83,90–93} For example, 40 (64%) of 62 male AAS abusers recruited in the field reported a lifetime history of some form of illicit drug dependence other than AAS.⁸³ In another investigation, 21 (9%) of 227 sequentially admitted male heroin addicts appeared to have been first introduced to opioids via AAS abuse.⁹¹ Similarly, among 88 men with a primary diagnosis of opioid dependence, 22 (25%) reported prior AAS abuse, as compared with only 7 of 135 (5%) men with other forms of substance dependence ($P < .001$).⁹⁰ Notably, recent observations indicate that AAS abusers may develop other forms of substance dependence before, during, or after their AAS abuse^{83,92}; AAS abuse may not necessarily predispose an individual to other forms of substance dependence, but these various forms of drug abuse may arise from a common underlying susceptibility, as mentioned earlier.

Effects of AAS Abuse on the Cardiovascular System. A growing literature, including anecdotal reports, animal trials, clinical studies, and comprehensive reviews, describes possible adverse cardiovascular effects of AAS abuse.^{67,94–97} The mechanisms of AAS-induced cardiovascular toxicity remain uncertain, although one group⁹⁸ proposed that AAS may be directly toxic to cardiac tissue, resulting in a cardiomyopathy characterized by decreased myocardial compliance.

Myocardial function studies in AAS abusers, using sensitive tissue Doppler imaging and strain imaging, documented marked cardiac deficits in AAS abusers.^{96,99,100} For instance, AAS abusers had lower early and late diastolic tissue velocities and reduced peak systolic strain versus nonusers.⁹⁶ Other authors¹⁰⁰ reported similar impairment of diastolic function in 6 AAS abusers, and tissue Doppler imaging showed impaired right ventricular function in AAS-using weight lifters.⁹⁹ A recent US study compared 12 long-term AAS abusers and 7 non-AAS-using weight lifters matched for age and exercise history and, alarmingly, found cardiac deficits in the former group.¹⁰¹ In particular, AAS abusers showed strikingly lower left ventricular ejection fractions and strain measures, together with evidence of diastolic impairment, as illustrated by markedly lower early versus late diastolic transmitral blood-flow velocities (ie, a reduced ratio of early to late ventricular filling velocity).¹⁰¹ As such, cardiotoxicity from

long-term AAS abuse may be more serious than previously recognized and may increase the risk for heart failure.¹⁰¹

The cardiovascular effects of AAS are not limited to the myocardium, as evidenced by a number of studies and summarized in 3 systematic reviews or meta-analyses.^{25,94,95} The evidence indicates that AAS abuse increases low-density lipoprotein cholesterol and decreases high-density lipoprotein cholesterol: a lipid profile recognized as a major risk factor for coronary heart disease.^{25,95,102} However, despite the well-documented changes in circulating lipids caused by AAS, direct evidence that AAS causes atherosclerosis remains limited, albeit suggestive.^{55,103} These effects are a concern because widespread AAS abuse is a relatively recent phenomenon.^{3,8,9,34-47,49,128} Thus, most long-term illicit AAS abusers are not yet 50 years old and may soon display increasing rates of cardiac problems as they age.⁶⁷

Effects of AAS Abuse on the Hepatic System. The abuse of AAS may occasionally be associated with liver abnormalities, such as cholestatic hepatitis, cholestatic jaundice, peliosis hepatis, and liver adenomas and carcinomas.^{7,11-14,22,24,51,55,104} These effects are rare and occur almost entirely from the abuse of oral C17 α -alkylated AAS (ie, methyltestosterone, oxymetholone, fluoxymesterone, norethandrolone, methandienone).^{7,12,24,55,102,104} The frequency of AAS-induced hepatic abnormalities may be overestimated because many liver function tests involve enzymes present in muscle, which may be elevated from muscular trauma after high-intensity training.⁵¹ Consequently, to test for genuine liver abnormalities possibly attributable to AAS abuse, it is critical to assess γ -glutamyl transpeptidase because this enzyme is present in liver but not in muscle tissue.⁵¹ Although it may take an extended time, many AAS-induced liver conditions appear to resolve after AAS disuse.^{12,22,24,51}

Effects of AAS Abuse on the Neuroendocrine System. Abuse of AAS suppresses the hypothalamic-pituitary-testicular axis in males.^{7,11-14,22,51} The resulting hypogonadism is thought to relate to AAS dosage, the specific agent abused, and the duration of abuse.¹¹ Hypogonadism can lead to testicular atrophy,^{7,11-14,22,51,55} sexual dysfunction,^{11,12,51,55} and infertility.^{7,11-13,22,51,55} Many of these conditions appear to be reversible upon cessation, although complete resolution may take more than 1 year.^{7,11-13,22,51,55}

Other possible male endocrine effects of AAS abuse include gynecomastia due to AAS conversion to estrogens, which typically requires surgical intervention because cessation does not reverse this change.^{11-14,22,51,55} Irreversible male-pattern baldness is another potential side effect as testosterone is converted to dihydrotestosterone.^{11-14,22,51,55} Effects on the prostate gland include hyperplasia, hypertrophy, and possibly cancer.^{11,12,55} As shown in a meta-analysis,¹⁷ therapeutic AAS use in middle-aged to older men led to an increased number of prostate problems.

Well-designed research involving female use of AAS is limited.⁵¹ Abuse of AAS in females apparently leads to increased masculinization, including voice deepening, hirsutism, male-pattern baldness, clitoral hypertrophy, and breast reduction.^{11-14,22,51} Related changes often seen in females include acne and changes in libido and menstruation.^{11,12,14,22,51} Some changes in females appear to be

permanent.^{12,14,22} One review¹² suggested that AAS abuse may lead to birth defects.

Limited evidence^{11,12,22} indicates that AAS abuse may influence thyroid function, with possible decreased production of triiodothyronine (T₃), thyroxine (T₄), and thyroid-binding globulin and increased production of thyrotropin and free T₄. The clinical relevance of possible thyroid changes is presently unclear.¹² Moreover, AAS abuse may decrease glucose tolerance and increase insulin resistance in humans.^{12,22,105}

Effects of AAS Abuse on the Musculoskeletal System. Skeletally immature individuals who abuse AAS risk decreased adult height due to premature epiphyseal closure.^{11,12,14,22,51} Further evidence suggests a potential link between AAS abuse and assorted musculoskeletal conditions, including various tendinopathies and tendon ruptures.^{7,11-13,22,51} Possible causes include relative tendon versus muscle weakness and actual collagen fiber dysplasia.^{7,11-13,22,51} The use of AAS for sport injury recovery has limited support in the literature. Most wound-healing studies were of burn patients,⁷ but one group¹⁰⁶ did report improved recovery time with therapeutic doses of AAS after induced muscle contusions in rats. Further study is needed.

Effects of AAS Abuse on the Other Biologic Systems and Areas. Additional health effects on other human biologic systems or regions may result from AAS abuse.

Immune System. Literature reviews of both animal and human studies suggest that immune system changes depend on AAS dose and type, but AAS generally act as immunosuppressants.¹⁰⁷ Specifically, AAS appear to decrease antibody synthesis, limit T- and B-lymphocyte maturation, and diminish natural killer cell activity.^{11,22,107} Additionally, unhygienic needle or syringe use may lead to local abscesses or systemic infections, including human immunodeficiency and the hepatitis viruses.^{11,12,14,22}

Integumentary System. Nontherapeutic abuse of AAS affects various aspects of human skin.^{11,12,14,22,108,109} Two reviews^{108,109} summarized the possible deleterious effects of high-dose AAS abuse related to the integumentary system. High doses of AAS promote the hypertrophy of the sebaceous glands and production of sebum and skin surface lipids and increase the concentration of *Propionibacterium acnes*. These changes increase the risk for conditions such as acne (conglobata, vulgaris, or fulminans), seborrhea, striae distensae, hypertrichosis, hirsutism, rosacea, folliculitis, keloids, and furunculosis.^{108,109} Furthermore, preexisting dermatologic conditions may be exacerbated by AAS abuse, including psoriasis, rosacea, angioliomatosis, and acne vulgaris.¹⁰⁸

Nephritic System. Although less studied and reported, renal side effects may occur with AAS abuse.^{11,22,51,55,110,111} Possible nephrologic conditions include kidney stones, cholestasis, tubular necrosis, hyperbilirubinemia, proteinuria, focal segmental glomerulosclerosis, renal cell cancer, and Wilms tumor.^{11,51,55,110,111} The pathophysiology of AAS-induced kidney conditions is unclear.¹¹⁰ Complicating factors in athletic and active individuals may include the frequent use of nonsteroidal anti-inflammatory agents, nutritional supplements, high-protein diets, and dehydration (purposeful or otherwise).^{110,111}

Oral Region. Limited research suggests a possible association between AAS abuse and maladies related to

the gingival tissues⁵² and masticatory system.¹¹² A preliminary paper⁵² indicated that prolonged AAS abuse led to gingival tissue enlargement, including significantly thicker gingival tissue, gingival encroachment, and total gingival enlargement scores compared with AAS nonusers. These changes sometimes require surgical intervention.⁵² Another study¹¹² indicated that AAS abuse may play a role in altering the masticatory structures, thus increasing the incidence of temporomandibular dysfunction.

AAS Abuse Prevention

Individuals typically abuse AAS to gain performance advantages and enhance their physique or body image (or both).^{1,2,4,5} The abuse of AAS often begins during adolescence,^{35,36,41-43,46,47} and thus prevention strategies should also begin at a young age. Prevention techniques generally include educational or screening paradigms (or both). Although relatively few scholarly publications specifically address AAS abuse prevention, general drug abuse prevention requires detailed understanding, including resistance skill development, learning peer norms, and being involved in a multisubstance abuse prevention program that does not focus solely on one substance of abuse. Using related theory in prevention efforts may be clinically helpful. For example, the transtheoretical model helped to explain and address smoking behaviors in specific populations and may also apply to AAS abuse.^{125,126} Understanding the reasons for AAS abuse or intent to abuse, as well as abuse progressions, may be an effective AAS prevention strategy in conjunction with other prevention initiatives.^{125,126}

Drug Testing for AAS Abuse Prevention. Over the past half century, screening for general drug abuse evolved as part of prevention programs. Drug-tested groups are many and varied: for example, military personnel, civilian and governmental employees, and amateur and professional athletes. For some testing agencies, AAS are not drugs of interest, but the more common screening is for illicit abuse of recreational and other prescription drugs. Athlete AAS testing began more than 30 years ago.^{1,4,5} Testing for AAS typically involves only organized, competitive sport participants and thus is generally not targeted at the noncompetitive fitness or bodybuilding population.¹⁴⁶ Screens for AAS initially focused on elite athletes but have since evolved to include collegiate and some high school student-athletes.¹¹⁴ Numerous international, national, and regional bodies oversee sport AAS testing.¹¹³ The World Anti-Doping Agency and related national organizations have strict, year-round, no-notice, accredited-laboratory drug testing, whereas other programs are less stringent and sometimes limited by collective bargaining agreements.^{4,113} Although these programs are varied, expensive, and time intensive, their effectiveness as preventive measures remains unclear.¹¹⁴ The many inherent testing variables make conducting related studies and drawing appropriate conclusions challenging.

The authors of one early study¹¹⁵ reported that more frequent drug testing resulted in a gradual decline in the number of positive drug tests for Norwegian athletes from 1977 to 1995. Only one group¹¹⁴ conducted a prospective randomized controlled study to investigate the deterrent efficacy of AAS testing for adolescent athletes. Student-

athletes (N = 1396) from 11 high schools (5 drug testing, 6 controls) completed the 2-year project. Using a method similar to that used for out-of-competition Olympic testing, the investigators found that AAS abuse remained unaffected over a 2-year period. In addition, those high school student-athletes randomized to testing had increased risk factors for future drug abuse. No known prospective randomized trials exist to demonstrate the deterrent efficacy or inefficacy of AAS abuse testing in other populations such as collegiate, professional, or Olympic athletes. With only 1 well-designed study investigating AAS screening in a very specific population,¹¹⁴ the overall deterrent efficacy of such testing remains understudied and unclear. Additional research on the deterrent effect of AAS testing is greatly needed.

Education for AAS Abuse Prevention. When implemented as a stand-alone intervention, AAS testing focuses on identifying the abuser, but health care professionals should also be concerned about identifying the *potential* AAS abuser. This paradigm involves education of not only AAS abusers but also potential abusers, educators, and medical professionals.

Numerous authors investigated educational programs as a preventive measure for AAS abuse.^{114,116-120,147} One 2004 European group¹²⁰ reported positive results after the implementation of a health promotion program to prevent AAS misuse among adolescents. The 2-year teaching session and group-discussion intervention program focused on appearance and self-confidence, rather than AAS abuse. Group discussions included both sexes. Results indicated that AAS abuse in adolescent boys, ages 16 through 17 years, might decrease after an educational program focused on appearance and self-confidence, although the authors recommended further study. Another project by this group¹²⁰ emphasized the varied attitudes between AAS abusers and AAS nonusers and the need to understand these differences in designing an effective prevention program. For example, understanding is limited about the many reasons for AAS abuse, such as body-image enhancement, improved performance, and others. Gaining insight into the phenomenologic patterns of AAS abuse warrants further study. Similar to eating disorder prevention and screening protocols, simple body-image screening instruments may need to be included during the preparticipation physical examination process or during neuropsychological testing to help identify those more at risk for AAS abuse.

One US-based research group^{147,148} developed and tested a number of AAS prevention protocols for adolescents. An early study¹⁴⁷ suggested that educational interventions stressing solely the negative effects of AAS were not only ineffective but increased adolescents' desire to abuse androgens. Programs focusing on knowledge of risks and benefits of AAS improved knowledge but did not change attitudes toward abuse; thus, they were deemed ineffective prevention approaches.¹⁴⁸ Since then, the authors have developed team-centered, sex-specific educational programs designed to deter AAS abuse among adolescent boys.

Three distinct cohorts implemented Adolescents Training and Learning to Avoid Steroids (ATLAS), which successfully inhibited AAS abuse among adolescent boys involved in team sports.^{117,118} Results from the 1996 randomized, prospective study,¹¹⁷ involving more than 1500 male youth from 31 high schools, suggested the ATLAS intervention

Table 6. Possible Indicators of Anabolic-Androgenic Steroid Abuse

Behaviors	Secretive actions, excessive dietary supplement use, mood swings, aggressiveness, excessive exercise, periods of abnormal healthy patterns, ^a physique and/or diet obsession, high-risk behaviors, rapid performance increases
Dermatologic	Abnormal, excessive, or unexpected acne; jaundice, needle marks, abscesses
Cardiovascular	Changes toward hypertension, dyslipidemia
Neuroendocrine	Male-pattern baldness, gynecomastia, testicular atrophy, female virilization (clitoral hypertrophy, hirsutism, amenorrhea, breast reduction, voice changes)
Musculoskeletal	Extreme musculature, rapid growth, extreme leanness, unexpected or unexplainable tendinopathies, premature growth-plate closure

^a During an anabolic-androgenic steroid cycle (time period of abuse), individuals may simultaneously focus on healthy behaviors such as a healthful diet, sufficient sleep, and regimented workouts.

decreased factors that encourage AAS misuse and lowered the intent to abuse AAS while enhancing nutrition behaviors. A follow-up 2000 ATLAS study¹¹⁸ reconfirmed the earlier results with 31 high school football teams and 3207 student-athletes. Results suggested a significant reduction in the intent to abuse AAS as well as the actual AAS abuse within the ATLAS intervention group. Other abuse patterns also were reduced, including ingestion of sport supplements, alcohol, and illicit drugs and illicit drinking and driving behaviors.¹¹⁸

Athletes Targeting Healthy Exercise and Nutrition Alternatives (ATHENA) is a scripted, athletic team-centered, peer-taught, and coach-facilitated program similar to ATLAS that focuses on adolescent female risk factors. More than 925 students from 18 high schools in 2 states participated. Compared with the control group, the ATHENA group used fewer athletic-enhancing substances, including AAS, amphetamines, supplements, and diet pills. In addition, ATHENA participants improved their diets and displayed fewer risky behaviors.¹¹⁶ Long-term follow-up (1 to 3 years after high school graduation) indicated persistently lower rates of substance abuse.¹²² Both ATLAS and ATHENA have undergone mediation analysis to assess the reasons for the programs' effectiveness.^{123,124} A systematic review of educational programs commissioned by the World Anti-Doping Agency showed that ATLAS and ATHENA provided the only high-quality evidence available as to the best way to educate adolescents about doping and are the only programs that have been monitored regularly over an extended follow-up period.¹²¹ Sex-specific, sport-centered, and coach-facilitated educational programs that promote performance-enhancing alternatives (eg, sport nutrition and strength training) appear to be effective AAS abuse deterrents for adolescent student-athletes.^{116–120} Presently, other than adolescent educational AAS abuse-prevention programs, no additional studies involving other populations are found in the scientific literature.

AAS Abuse Identification and Intervention

Health care professionals and educators have a responsibility to assist individuals in making informed, positive health decisions.²² The Board of Certification details the following areas of professional athletic training practice related to AAS abuse: (1) injury/illness prevention and wellness protection, (2) treatment and rehabilitation, and (3) organizational and professional health and well-being.¹⁴⁹ Recognizing AAS abuse or the intent to abuse (one aspect of prevention); knowing when and how to intervene (which guides referral options); and knowing how

to implement health care administration for those in need all are responsibilities of athletic trainers.

AAS Abuse Identification. Health care professionals must understand the epidemiology of AAS abuse in strength and power athletes, as well as other, sometimes less obvious groups and individuals.^{3,8,9,34–49} A vigilant approach by athletic trainers is critical due to their extended time spent with these populations. The health care professional should be aware of related conversations, as well as changes in relationships,^{90,93} behaviors,^{54,67,68,82–85,128,145} and general health,^{11,13,22,51,55,127} all of which can provide valuable clues to AAS abuse. Although every situation varies, a number of behavioral changes and other signs and symptoms may indicate AAS abuse (Table 6).^{*} If AAS abuse is suspected, consider comparing the patient's current medical records with his or her baseline.

AAS Abuse Intervention. To date, no validated management strategies are available for AAS abuse⁸⁴; thus, much of the following stems from anecdotal evidence or experiences. Referral and counseling are paramount to avoiding possible consequences of AAS abuse, such as negative health effects and psychological dependence.¹⁴⁹ However, not all health care professionals or educators understand AAS pharmacology, the mentality of athletes and AAS abusers, or common AAS abuse patterns.^{67,82,84} Few AAS abusers seek treatment,⁸⁴ and they are historically skeptical of the medical-scientific establishment.⁵ As such, health care professionals should develop a referral network with other well-qualified colleagues, possibly including specialists such as endocrinologists and psychiatrists. Considering the various AAS abuse (or intent to abuse) stages may assist health care professionals in offering specific support, as described in the following examples.

- Keep athletes' self-esteem high with alternatives to AAS, such as evidence-based nutrition and weight-training principles, along with collaboration from conditioning experts and sport nutritionists, among others.
- Build trust with patients, clients, athletes, and coaches through consistent, year-round, evidence-based educational meetings and in-service sessions.
- Avoid unfounded scare tactics and hype concerning the negative consequences of AAS.
- Be alert for excessive discussion about and focus on supplements and nutrition practices or self-education about AAS (eg, types, stacks, and cycles).

^{*}References 11, 14, 22, 51, 54, 67, 68, 82–85, 90, 93, 127, 128, 133, 141, 145.

- Do not threaten suspected AAS abusers but instead cautiously state your suspicions (eg, “I have noticed a change in your behavior, supplement use, or interest in AAS. . .”) and offer evidence-based information and assistance. Be an accessible agent of positive behavioral changes.
- Acknowledge the minimal, short-term (physical) consequences of AAS abuse but stress education from health, moral and ethical, and legal perspectives.
- Comment on the negative aspects of AAS abuse, such as increased acne, stunted growth, or jaundice (ie, the issues younger adolescents and adults will likely be concerned about). Be sure not to compliment their physical appearance (eg, muscularity) or strength gains because this may reinforce or encourage AAS abuse.
- Discuss personal sacrifices that accompany AAS abuse, such as money, time, social experiences, risk of disease and injury, and counterfeit or tainted products, among others.
- Remain attentive because AAS abusers typically are secretive and good at not getting caught. A passed drug screen does not prove AAS nonuse.

Consider using available tools, such as validated body-image screening instruments: for example, the male version of the eating disorder inventory⁹³ or the muscle dysmorphia version¹⁴⁵ of the Body Dysmorphic Disorder Modification of the Yale Brown Obsessive-Compulsive Scale. Objective measures, such as the fat-free mass index, use basic anthropometric data to determine the upper physiologic limits of muscularity. Involve colleagues and other professionals as part of a holistic approach. For instance, integrate evidence-based strength and nutrition principles into all activities and programs. Cooperatively implementing these techniques and principles may allow us to better fulfill our comprehensive responsibility to athletes and the physically active. That is, health care professionals should constantly seek high-quality new resources and readily call on available professionals, while coordinating the process as with any other health care issue. Referral to an AAS-knowledgeable specialist for counseling and possible pharmacologic intervention may be beneficial.⁸⁴

Health care professionals, including athletic trainers and others, manage many illnesses and injuries in their clinical practices.¹⁴⁹ Each condition has unique characteristics and clinical features, just as each AAS abuser is different. Health care professionals may have a close relationship with those in their care. Such relationships provide excellent, critical opportunities to prevent AAS abuse through an active approach that includes being alert to abuse patterns, forthright in terms of approaching the issue, and effective in offering counseling and referral services. Health care professionals and educators serve as advocates and resources for all those with health-related issues, including AAS abuse.

CONCLUSIONS

Anabolic-androgenic steroids are powerful Schedule III pharmaceuticals related to human androgens, primarily testosterone. They act as both anabolic and androgenic agents. Although these drugs have therapeutic roles, athletes and nonathletes often abuse AAS to improve their physical performance, physique, and body image. Abusers of AAS most frequently use a cyclic, high-dose, multidrug regimen,

the consequences of which are poorly understood. Rarely do they obtain their information or their drugs from valid and reliable sources. Anabolic-androgenic steroids carry a dose-related potential for increased lean body mass, and these agents generally improve strength- and power-related performances. Conversely, when abused, AAS may lead to negative health effects on the central nervous, cardiovascular, hepatic, reproductive, musculoskeletal, immune, integumentary, and nephritic systems and possibly others. Investigations into useful techniques for preventing AAS abuse are limited. Testing for AAS, by itself or in combination with other interventions, is not well studied, and its deterrent efficacy remains uncertain. Well-designed and properly implemented education appears to be useful for adolescent athletes, but other populations remain understudied. Health care professionals and educators must manage the AAS abuser and potential abuser based on the best available evidence, including studies detailing prevention and intervention strategies. Athletic trainers are health care professionals who are often in a unique position to assess and assist AAS abusers and those who may become AAS abusers.

ACKNOWLEDGMENTS

We gratefully acknowledge the efforts of Don Hooton; Cindy Thomas, MS, ATC; Teresa Seefeldt, PhD; and Andrew P. Winterstein, PhD, ATC in the review of this position statement.

DISCLAIMER

The NATA and NATA Foundation publish their position statements as a service to promote the awareness of certain issues to its members. The information contained in the position statement is neither exhaustive nor exclusive to all circumstances or individuals. Variables such as institutional human resource guidelines, state or federal statutes, rules, or regulations, as well as regional environmental conditions, may impact the relevance and implementation of these recommendations. The NATA and NATA Foundation advise members and others to carefully and independently consider each of the recommendations (including the applicability of same to any particular circumstance or individual). The position statement should not be relied upon as an independent basis for care but rather as a resource available to NATA members or others. Moreover, no opinion is expressed herein regarding the quality of care that adheres to or differs from the NATA and NATA Foundation position statements. The NATA and NATA Foundation reserve the right to rescind or modify position statements at any time.

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