©Journal of Sports Science and Medicine (2006) **5,** 182-193
http://www.jssm.org

Review article

MEDICAL ISSUES ASSOCIATED WITH ANABOLIC STEROID USE: ARE THEY EXAGGERATED?

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Received: 10 February 2006 / Accepted: 09 March 2006 / Published (online): 01 June 2006

ABSTRACT

For the past 50 years anabolic steroids have been at the forefront of the controversy surrounding performance enhancing drugs. For almost half of this time no attempt was made by sports governing bodies to control its use, and only recently have all of the major sports governing bodies in North America agreed to ban from competition and punish athletes who test positive for anabolic steroids. These punitive measures were developed with the primary concern for promotion of fair play and eliminating potential health risks associated with androgenic-anabolic steroids. Yet, controversy exists whether these testing programs deter anabolic steroid use. Although the scope of this paper does not focus on the effectiveness of testing, or the issue of fair play, it is of interest to understand why many athletes underestimate the health risks associated from these drugs. What creates further curiosity is the seemingly well-publicized health hazards that the medical community has depicted concerning anabolic steroid abuse. Is there something that the athletes know, or are they simply naïve regarding the dangers? The focus of this review is to provide a brief history of anabolic steroid use in North America, the prevalence of its use in both athletic and recreational populations and its efficacy. Primary discussion will focus on health issues associated with anabolic steroid use with an examination of the contrasting views held between the medical community and the athletes that are using these ergogenic drugs. Existing data suggest that in certain circumstances the medical risk associated with anabolic steroid use may have been somewhat exaggerated, possibly to dissuade use in athletes.

KEY WORDS: Androgens, ergogenic aids, athletes, sport supplements, performance enhancing drugs.

INTRODUCTION

Anabolic-androgenic steroids (herein referred to as only anabolic steroids) are the man-made derivatives of the male sex hormone testosterone. Physiologically, elevations in testosterone concentrations stimulate protein synthesis resulting in improvements in muscle size, body mass and strength (Bhasin et al., 1996; 2001). In addition, testosterone and its synthetic derivatives are responsible for the development and maturation of male secondary sexual characteristics (i.e. increase in body hair, masculine voice, development of male pattern baldness, libido, sperm production and aggressiveness).

Testosterone was isolated in the early 20th century and its discovery led to studies demonstrating that this substance stimulated a strong positive nitrogen balance in castrated dogs and rats (Kochakian, 1950). Testosterone, because of its rapid degradation when given through either oral or parenteral administration, poses some limitations as an ergogenic aid. Although its potency is rapidly observed, the high frequency of administration needed becomes problematic. In addition, testosterone has a therapeutic index of 1 meaning there is similarity in the proportion between the anabolic and androgenic effects. As a result it becomes necessary to chemically modify testosterone to retard the degradation process and

reduce some of the negative side effects. This allows for maintenance of effective blood concentrations for longer periods of time, may increase its interaction with the androgen receptor, and achieves the desired anabolic and androgenic changes.

Boje (1939) was the first to suggest that exogenous testosterone administration may enhance athletic performance. By the late 1940's and 1950's testosterone compounds were experimented with by some west coast bodybuilders (Yesalis et al., 2000). The first dramatic reports of anabolic steroid use occurred following the 1954 world weightlifting championships (Yesalis et al., 2000). Use of these drugs spread quickly through the 1960's and became popular among athletes in a variety of Olympic sports (Dubin, 1990). Wide spread use has also been reported in power lifters (Wagman et al., 1995), National Football League players (Yesalis et al., 2000), collegiate athletes (Yesalis, 1992), and recent claims of wide spread use in many sports including Major League Baseball players has made anabolic steroids the number one sports story of 2005 in some markets (Quinn, 2006). The ergogenic effects associated with anabolic steroids are presented in Table 1.

Table 1. Ergogenic effects associated with anabolic steroid use.

- Increase in lean body mass
- Increase in muscle cross-sectional area
- Decrease in body fat percent
- Increase muscle strength & power
- Enhance recovery between workouts
- Enhance recovery from injury
- Increase in protein synthesis
- Increase in muscle endurance
- Increase in erythropoiesis, hemoglobin, and hematocrit
- Increase in bone mineral density
- Increase in glycogen storage
- Increase in lipolysis
- Increase in neural transmission
- Reduced muscle damage
- Increase in pain tolerance
- Behavior modification (aggression)

Athletes typically use anabolic steroids in a "stacking" regimen, in which they administer several different drugs simultaneously. The rationale for stacking is to increase the potency of each drug. That is, the potency of one anabolic agent may be enhanced when consumed simultaneously with another anabolic agent. They will use both oral and parenteral compounds. Most users will take anabolic steroids in a cyclic pattern, meaning the athletes will use the drugs for several weeks or months and alternate these cycles with periods of discontinued use. Often the athletes will administer the drugs in a pyramid (step-up) pattern in which dosages are steadily increased over several weeks. Towards the end of the cycle the athlete will 'step-down' to reduce the likelihood of negative side effects. At this point, some athletes will discontinue drug use or perhaps initiate another cycle of different drugs (i.e., drugs that may increase endogenous testosterone production to prevent the undesirable drop in testosterone concentrations that follows the removal of the pharmaceutical agents). A recent study has shown that the typical steroid regimen involved 3.1 agents, with a typical cycle ranging from 5 – 10 weeks (Perry et al., 2005). The dose that the athlete administers was reported to vary between 5 - 29 times greater than physiological replacement doses (Perry et al., 2005). These higher pharmacological dosages appear necessary to elicit the gains that these athletes desire. In a classic study on the dose-response curve of anabolic steroids, Forbes (1985) demonstrated that the total dose of anabolic steroids have a logarithmic relationship to increases in lean body mass. These results exacerbate the athlete's philosophy that if a low dose is effective, then more must be better.

Adverse effects associated with anabolic steroid use are listed in Table 2. For years, the medical and scientific communities attempted to reduce anabolic steroid use by athletes by underscoring their efficacy and focusing on the unhealthy side effects (Biely, 1987; Darden, 1983; Fahey and Brown, 1973; Fowler et al., 1965; Golding et al., 1974). For the most part, this may have proved to be ineffective and caused athletes to lose trust in the physician's knowledge of anabolic steroids thereby forcing them to seek advice from friends, internet sites or drug suppliers (Pope et al., 2004). However, recent literature has suggested that the medical issues associated with anabolic steroids may be somewhat overstated (Berning et al., 2004; Sturmi and Diorio, 1998; Street et al., 1996) considering that many of the side effects associated with anabolic steroid abuse are reversible upon cessation. It is important to note that there are differences in the side effects associated with anabolic steroid use (i.e. under medical supervision) versus abuse (i.e. consumption of many drugs at high doses).

The clinical examination of anabolic steroid use is quite limited. Much of the problem in prospectively examining the effects of anabolic steroids on the athletic population is related to the unwillingness of institutional review boards to approve such studies in a non-clinical population. As a result, most of the investigations concerning

Table 2. Adverse effects associated with anabolic steroid use.

Cardiovascular ▪ Lipid profile changes ▪ Elevated blood pressure ▪ Decreased myocardial function	**Dermatological** ▪ Acne ▪ Male pattern baldness
Endocrine ▪ Gynecomastia ▪ Decreased sperm count ▪ Testicular atrophy ▪ Impotence and transient infertility	**Hepatic** ▪ Increased risk of liver tumors and liver damage
Genitourinary **Males** ▪ Reduced sperm counts ▪ Decreased testicular size **Females** ▪ Menstrual irregularities ▪ Clitoromegaly ▪ masculinization **Males and Females** ▪ Gynecomastia ▪ Libido changes	**Musculoskeletal** ▪ Premature epiphyseal plate closure ▪ Increased risk of tendon tears ▪ Intramuscular abscess **Psychological** ▪ Mania ▪ Depression ▪ Aggression ▪ Mood swings

medical issues associated with anabolic steroid administration have been performed on athletes self-administering the drugs. Anecdotally, it appears that a disproportionate magnitude of use and incidence of adverse effects are evident in bodybuilders (who are also known for consuming several other drugs that relieve some side effects but potentiate other risk factors as well, i.e. diuretics, thyroid hormones, insulin, anti-estrogens, etc.) compared to strength/power athletes. The mindset and motivation of these two types of athletes can be quite different. The strength/power athlete will typically use anabolic steroids to prepare themselves for a season of competition. They will generally cycle the drug to help them reach peak condition at a specific time of the training year. In contrast, bodybuilders use anabolic steroids to enhance muscle growth and definition. Their success is predicated on their aesthetic appearance. As a result many of these athletes may use anabolic steroids excessively for several years without cycling off or perhaps minimizing the length of "off cycles" depending on their competition schedule. Recent research has indicated that those athletes exhibit behavior that are consistent with substance dependence disorder (Perry et al., 2005). Although the medical issues associated with anabolic steroids may be quite different between these two types of athletes, the scientific literature generally does not differentiate between the two. The following sections will discuss adverse effects on specific physiological systems associated with anabolic-androgenic steroid use. It is important to note that many athletes consume multiple drugs in addition to anabolic steroids. Thus, the unhealthy side effects could be potentiated by the use of drugs such as human growth hormone or IGF-1.

CARDIOVASCULAR SYSTEM

In both the medical and lay literature one of the principal adverse effects generally associated with anabolic steroid use is the increased risk for myocardial infarction. This is primarily based upon several case reports published over the past 20 years describing the occurrence of myocardial infarctions in young and middle-aged body builders or weight lifters attributed to anabolic steroid use and/or abuse (Bowman, 1989; Ferenchick and Adelman, 1992; Gunes et al., 2004; Kennedy and Lawrence, 1993; Luke et al., 1990; McNutt et al., 1988). However, direct evidence showing cause and effect between anabolic steroid administration and myocardial infarction is limited. Many of the case studies reported normal coronary arterial function in anabolic steroid users that experienced an infarct (Kennedy and Lawrence, 1993; Luke et al., 1990), while others have shown occluded arteries with thrombus formation (Ferenchick and Adelman, 1992; Gunes et al., 2004; McNutt et al., 1988). Still, some of these studies have reported abnormal lipoprotein concentrations with serum cholesterol levels nearly approaching 600 $mg \cdot dl^{-1}$ (McNutt et al., 1988). Interestingly, in most case studies the effects of diet or genetic predisposition for cardiovascular disease were not disseminated and could not be excluded as contributing factors.

Alterations in serum lipids, elevations in blood pressure and an increased risk of thrombosis are additional cardiovascular changes often associated with anabolic steroid use (Cohen et al., 1986; Costill et al., 1984; Dhar et al., 2005; Kuipers et al., 1991; Laroche, 1990). The magnitude of these effects may differ depending upon the type, duration, and volume of anabolic steroids used. Interesting to note is that these effects appear to be reversible upon cessation of the drug (Dhar et al., 2005, Parssinen and Seppala, 2002). In instances where the athlete remains on anabolic steroids for prolonged periods of time (e.g "abuse"), the risk for developing cardiovascular disease may increase. Sader and colleagues (2001) noted that despite low HDL levels in bodybuilders, anabolic steroid use did not appear to cause significant vascular dysfunction. Interestingly, athletes participating in power sports appear to have a higher incidence of cardiovascular dysfunction than other athletes, regardless of androgen use (Tikkanen et al., 1991; 1998). Thus, a strength/power athlete with underlying cardiovascular abnormalities that begins using anabolic steroids is at a much higher risk for cardiovascular disease. However, anabolic steroid-induced changes in lipid profiles may not, per se, lead to significant cardiovascular dysfunction.

The risk of sudden death from cardiovascular complications in the athlete consuming anabolic steroids can occur in the absence of atherosclerosis. Thrombus formation has been reported in several case studies of bodybuilders self-administering anabolic steroids (Ferenchick, 1991; Fineschi et al., 2001; McCarthy et al., 2000; Sahraian et al., 2004). Melchert and Welder (1995) have suggested that the use of 17α-alkylated steroids (primarily from oral ingestion) likely present the highest risk for thrombus formation. They hypothesized that anabolic steroid consumption can elevate platelet aggregation, possibly through an increase in platelet production of thromboxane A_2 and/or decreasing platelet production of prostaglandin PgI_2, resulting in a hypercoagulable state.

Left ventricular function and anabolic steroid use/abuse has been examined. Climstein and colleagues (2003) demonstrated that highly strength-trained athletes, with no history of anabolic steroid use exhibited a higher incidence of wave form abnormalities relative to recreationally-trained or sedentary individuals. However, when these athletes self-administered anabolic steroids, a higher percentage of wave form abnormalities were exhibited. Further evidence suggestive of left ventricular dysfunction has been reported in rodent models. A study on rats has shown that 8 weeks of testosterone administration increased left ventricle stiffness and caused a reduction in stroke volume and cardiac performance (LeGros et al., 2000). It was hypothesized that the increased stiffness may have been related to formation of crosslinks between adjacent collagen molecules within the heart. Others have suggested that anabolic steroid use may suppress the increases normally shown in myocardial capillary density following prolonged endurance training (Tagarakis et al., 2000). However, there are a number of interpretational issues with this study. The changes reported were not statistically significant. In addition, the exercise stimulus employed (prolonged endurance training) is not the primary mode of exercise frequently used by anabolic steroid users. Resistance training, independent of anabolic steroid administration, has been shown to increase left ventricular wall and septal thickness due to the high magnitude of pressure overload (Fleck et al., 1993; Fleck, 2003; Hoffman, 2002). This is known as concentric hypertrophy and does not occur at the expense of left ventricular diameter. In general, cardiac hypertrophy (resulting from a pressure overload, i.e. hypertension) may not be accompanied by a proportional increase in capillary density (Tomanek, 1986). Therefore, the potential for a reduction in coronary vasculature density exists for the resistance-trained athlete. However, it does not appear to pose a significant cardiac risk for these athletes. Recent observations have shown a dose-dependent increase in left ventricular hypertrophy (LVH) in anabolic steroid users (Parssinen and Seppala, 2002). This may have the potential to exacerbate the reduction in coronary vasculature density. However, the authors have acknowledged that their results may have been potentiated by a concomitant use of human growth hormone by their subjects. Other studies have failed to show additive effects of anabolic steroid administration and LVH in resistance-trained athletes (Palatini et al., 1996; Dickerman et al., 1998).

HEPATIC SYSTEM

An elevated risk for liver tumors, damage, hepatocellular adenomas, and peliosis hepatitis are often associated with anabolic steroid use or abuse. This is likely due to the liver being the primary site of steroid clearance. In addition, hepatic cancers have been shown to generally occur with higher frequency in males compared to females (El-Serag, 2004). It is thought that high endogenous concentrations of testosterone and low estrogen concentrations increase the risk of hepatic carcinomas (Tanaka et al., 2000). However, this appears to be prevalent for men with pre-existing

liver disease. In normal, healthy men the relationship between testosterone concentrations and liver cancer has not been firmly established. Additional reports of liver cancer and anabolic steroids have been reported in non-athletic populations being treated with testosterone for aplastic anemia (Nakao et al., 2000). In regards to liver cancer and disease in athletes consuming anabolic steroids, many concerns have been raised based primarily on several case studies that have documented liver disease in bodybuilders using anabolic steroids (Cabasso, 1994; Socas et al., 2005; Soe et al., 1992).

A few studies have recently questioned the risk to hepatic dysfunction from anabolic steroid use (Dickerman et al., 1999). A recent study examining the blood chemistry of bodybuilders self-administering anabolic steroids reported elevations in aspartate aminotransferase (AST), alanine aminotransferase (ALT) and creatine kinase (CK), but no change in the often-regarded more sensitive gamma-glutamyltranspeptidase (GGT) concentration (Dickerman et al., 1999). Thus, some experts have questioned these criteria tools because of the difficulty in dissociating the effects of muscle damage resulting from training from potential liver dysfunction. This has prompted some researchers to suggest that steroid-induced hepatotoxicity may be overstated. Another study involved a survey sent to physicians asking them to provide a diagnosis for a 28-year-old anabolic steroid using bodybuilder with abnormal serum chemistry profile (elevations in AST, ALT, CK, but with a normal GGT) (Pertusi et al., 2001). The majority of physicians (63%) indicated liver disease as the primary diagnosis as 56% of physicians failed to acknowledge the potential role of muscle damage or disease thereby increasing the likelihood of overemphasized anabolic steroid-induced hepatotoxicity diagnoses. Many case reports involving anabolic steroid administration and hepatic cancers examined individuals who were treated with oral steroids (17α-alkylated) for many years. No cysts or tumors have been reported in athletes using 17β-alkylated steroids. Thus, evidence appears to indicate that the risk for hepatic disease from anabolic steroid use may not be as high as the medical community had originally thought although a risk does exist especially with oral anabolic steroid use or abuse.

BONE AND CONNECTIVE TISSUE

The issue of anabolic steroids and bone growth has been examined in both young and adult populations. In both populations, androgens have been successfully used as part of the treatment for growth delay (Albanese et al., 1994; Bagatell and Bremner, 1996; Doeker et al., 1998), and for osteoporosis in women (Geusens et al., 1986). Androgens are bi-phasic in that they stimulate endochondral bone formation and induce growth plate closure at the end of puberty. The actions of androgens on the growth plate are mediated to a large extent by aromatization to estrogens (Vanderschueren et al., 2004; Hoffman, 2002). Anabolic steroid use results in significant elevations in estrogens thought to impact premature closure of the growth plate. The acceleration of growth in adolescents treated with testosterone has raised concern for the premature closure of the epiphyseal plate (NIDA, 1996; Sturmi and Diorio, 1998). However, there does not appear to be any reports documenting the occurrence of premature stunted growth in adolescents taking anabolic steroids. Interesting, anabolic steroid administration in colts has been reported to delay epiphyseal plate closure (Koskinen and Katila, 1997). Although comparisons between humans and animals are difficult to make, suprapharmacological dosages that most athletes use may pose a greater risk than the doses studied to date. Thus, for the adolescent athlete using anabolic steroids the risk of premature epiphyseal plate closure may exist.

Anabolic steroids have been suggested to increase the risk of tendon tears in athletes (David et al., 1994; Stannard and Bucknell, 1993). Studies in mice have suggested that anabolic steroids may lead to degeneration of collagen (proportional to duration of steroid administration) and potentially lead to a decrease in tensile strength (Michna, 1986). In addition, a decrease in collagen synthesis has been reported from anabolic steroid administration in rats (Karpakka et al., 1992). The response in humans has been less clear. Mechanical failure has been suggested as a mechanism in anabolic steroid-using athletes. Skeletal muscle adaptations (i.e. hypertrophy and strength increases) take place rather rapidly in comparison to connective tissue. Therefore, tendon injuries in athletes are thought to occur from a rapid increase in training intensity and volume where connective tissue fails to withstand the overload. However, case reports of spontaneous tendon ruptures of weightlifters and athletes are limited. Although experimental data from animal models suggest that anabolic steroids may alter biomechanical properties of tendons, ultrastructural evidence supporting this claim is lacking. One study has shown that high doses of anabolic steroids decrease the degradation and increase the synthesis of type I collagen (Parssinen et al., 2000). Evans and colleagues (1998) performed an ultrastructural analysis on ruptured tendons from anabolic steroid users. They concluded that anabolic steroids did not induce any ultrastructural collagen changes that

would increase the risk of tendon ruptures. Although the incidences of tendon rupture in anabolic steroid users should not be discounted, it is important to consider it in relation to the mechanical stress encountered from the rapid increases in muscular performance. Prospective research on anabolic steroid use and connective tissue injury is warranted.

PSYCHOLOGICAL AND BEHAVIORAL

An issue that is often raised with anabolic steroid use is the psychological and behavioral effects. Increases in aggressiveness, arousal and irritability have been associated with anabolic steroid use. This has potentially beneficial and harmful implications. Elevations in arousal and self-esteem may be a positive side effect for the athlete. The increase in aggressiveness is a benefit that athletes participating in a contact sport may possess. However, increased aggressiveness may occur outside of the athletic arena thereby posing significant risks for anabolic steroid users and those they come in contact with. Anabolic steroids are associated with mood swings and increases in psychotic episodes. Studies have shown that nearly 60% of anabolic steroid users experience increases in irritability and aggressiveness (Pope and Katz, 1994; Silvester, 1995). A recent study by Pope and colleagues (2000) reported that significant elevations in aggressiveness and manic scores were observed following 12 weeks of testosterone cypionate injections in a controlled double-blind cross-over study. Interestingly, the results of this study were not uniform across the subjects. Most subjects showed little psychological effect and few developed prominent effects. A cause and effect relationship has yet to be identified in anabolic steroid users and it does appear that individuals who experience psychological or behavioral changes do recover when steroid use is discontinued (Fudula et al., 2003).

ADDITIONAL ADVERSE EFFECTS ASSOCIATED WITH ANABOLIC STEROID USE

Other adverse events generally associated with anabolic steroid use include acne, male pattern baldness, gynecomastia, decreased sperm count, testicular atrophy, impotence, and transient infertility. Acne is one of the more common side effects associated with anabolic steroid administration. One study reported that 43% of users experienced acne as a consequence from androgen use (O'Sullivan et al., 2000). Few other investigations have been able to prospectively determine the occurrence of side effects associated with androgen administration. Increases in acne are thought to be related to a stimulation of sebaceous glands to produce more oil. The most common sites of acne development are on the face and back. Acne appears to disappear upon cessation of androgen administration.

Male pattern baldness does not appear to be a common adverse effect, but is often discussed as a potential side effect associated with androgen use. This is likely related to the role that androgens have in regulating hair growth (Lee et al., 2005). An abnormal expression of a specific cutaneous androgen receptor increases the likelihood of androgenic alopecia (Kaufman and Dawber, 1999; Lee et al., 2005). Thus, it is likely that androgenic alopecia observed as a result of exogenous androgen use is more prevalent in individuals that have a genetic predisposition to balding.

Gynecomastia is a common adverse effect associated with anabolic steroid use. Research has demonstrated a prevalence rate of 37% in anabolic steroid users (O'Sullivan et al., 2000). Gynecomastia is a benign enlargement of the male breast resulting from an altered estrogen-androgen balance, or increased breast sensitivity to a circulating estrogen level. Increases in estrogen production in men are seen primarily through the aromatization of circulating testosterone. Many anabolic steroid users will use anti-estrogens (selective estrogen receptor modulators) such as tamoxifen and clomiphene or anastrozole which is a nonsteroidal aromatase inhibitor to minimize side effects of estrogen and stimulate testosterone production. Once gynecomastia is diagnosed cosmetic surgery is often needed to correct the problem.

Changes in libido appear to be the most common adverse event (approximately 61% of users) reported in a small sample of anabolic steroid users (O'Sullivan et al., 2000). Although testosterone is often used in hypogonadal men to restore normal sexual function, increasing testosterone above the normal physiological range does not appear to increase sexual interest or frequency of sexual behavior in healthy men administered anabolic steroids in supraphysiological dosages (up to 500 $mg \cdot wk^{-1}$) for 14 weeks (Yates et al., 1999). Other studies confirm unchanged libido following 10 weeks of anabolic steroid administration in dosages ranging up to 200 $mg \cdot wk^{-1}$ (Schurmeyer, et al., 1984). However, reports do indicate that towards the end of an androgen cycle some men may experience loss of libido (O'Sullivan et al., 2000). It was thought that the decreased libido was related to the transient hypogonadism which

typically occurs during exogenous androgen administration. Decreases in libido as a result of hypogonadism appear to be a function of high baseline levels of sexual functioning and desire (Schmidt et al., 2004). This may explain the conflicting reports seen in the literature. Regardless, changes in libido do appear to normalize once baseline endogenous testosterone concentrations return (Schmidt et al., 2004).

Another frequent adverse event relating to sexual function in males administering anabolic steroids is reversible azoospermia and oligospermia (Alen and Suominen, 1984; Schurmeyer et al., 1984). As exogenous androgen use increases, endogenous testosterone production is reduced. As a result, testicular size is reduced within three months of androgen administration (Alen and Suominen, 1984). In addition, sperm concentration and the number of spermatozoa in ejaculate may be reduced or eliminated by 7 weeks of administration (Schurmeyer et al., 1984). During this time risk for infertility is elevated. However, the changes seen in testicular volume, sperm count and concentration are reversible. Anabolic steroid-induced hypogonadism returns to baseline levels within 4 months following discontinuation of androgen use (Jarow and Lipshultz, 1990), and sperm counts and concentration return to normal during this time frame (Alen and Suominen, 1984; Schurmeyer et al., 1984).

MEDICAL ISSUES ASSOCIATED WITH FEMALE STEROID USE

In female anabolic steroid users the medical issues are quite different than that shown in men. Deepening of the voice, enlargement of the clitoris, decreased breast size, altered menstruation, hirsutism and male pattern baldness are all clinical features common to hyperandrogenism in females (Derman, 1995). Androgen excess may occur as the result of polycystic ovary syndrome, congenital adrenal hyperplasia and possibly Cushing's syndrome (Derman, 1995; Redmond, 1995). However, these clinical symptoms are seen in young, female athletes that are self-administering anabolic steroids. In contrast to men, many of these adverse events in the female anabolic steroid user may not be transient (Pavlatos et al., 2001).

LONG TERM HEALTH ISSUES ASSOCIATED WITH ANABOLIC STEROID ADMINISTRATION

The acute health issues associated with anabolic steroid use appear to be transient and more prevalent in individuals with genetic predisposition (e.g. hair loss, heart disease). It is the long-term effects that become a larger issue. However, limited data are available. In one study in mice, anabolic steroids were administered in relative dosages typically used by bodybuilders. However, the duration of the study was 1/5 the life span of the mouse which is relatively greater than that experienced by most athletes self-administering androgens. The results demonstrated a shortened life span of the mice with evidence of liver, kidney and heart pathology (Bronson and Matherne, 1997). In a study on Finnish power lifters, investigators examined 62 athletes who finished in the top 5 in various weight classes between the years 1977 and 1982 (Parssinen et al., 2000). These investigators reported that during a 12-year follow-up, the mortality rate for the power lifters was 12.1% compared to 3.1% in a control population. They concluded that their study depicted the detrimental long-term health effects from anabolic steroid use. Others have suggested that prolonged anabolic steroid use may increase the risk for premature death, but this may be more relevant in subjects with substance abuse or underlying psychiatric disease (Petersson et al., 2006).

The use of anabolic steroids in strength/power athletes has been reported for more than 50 years in North America. As discussed in the beginning of this review, during the 1970's and 1980's anecdotal reports on the rampant use of anabolic steroids in professional athletes were prevalent. However, little information is available concerning steroid-related diseases or associated deaths in these former strength/power athletes who are now well into middle age. Regardless, research should focus on these former athletes to ascertain possible long-term effects from androgen use.

IS THERE A CLINICAL ROLE OF ANDROGENIC ANABOLIC STEROIDS?

The efficacy of anabolic steroids in enhancing muscle strength and lean tissue accruement is no longer an issue for debate. While the issue of medical risks in individuals self-administering anabolic steroids is still being hotly debated, the medical community is no longer denying the potential clinical use of these androgens (Dobs, 1999). In recent years clinical treatment with anabolic steroids has increased lean tissue and improved daily functional performance in AIDS patients (Strawford et al., 1999) patients receiving dialysis (Johansen et al., 1999), patients with chronic obstructive pulmonary disease (Ferreira et al., 1998), and patients recovering from a myocardial infarction (Nahrendorf et al., 2003). In addition, research has

demonstrated a positive effect on healing from muscle contusion injuries (Beiner et al., 1999). Although the medical community has generally taken a conservative approach to promoting anabolic steroids as part of a treatment plan in combating diseases involving muscle wasting, the body of knowledge that has developed indicates the potential positive effects of androgen therapy for certain diseased populations.

CONCLUSIONS

For many years the scientific and medical communities depicted a lack of efficacy and serious adverse effects from anabolic steroid use. However, competitive athletes continued to experiment with, use, and abuse anabolic steroids on a regular basis to enhance athletic performance despite the potential harmful side effects. The empirical evidence that the athletes viewed may have led to the development of distrust between the athletic and medical communities. Science has been lagging several years behind the experimental practices of athletes. In fact, most athletes consume anabolic steroids on a trial and error approach based on information gained from other athletes, coaches, websites, or gym "gurus." Science has lacked in its approach to study anabolic steroids because only few studies have examined long-term cyclical patterns, high doses, and the effects of stacking different brands of steroids. These practices are common to the athletic community and not for the medicinal purposes of anabolic steroid therapy. In addition, some athletes (especially bodybuilders) have experimented with drugs unbeknown to the medical community, i.e. insulin, thyroid hormones, and site-specific enhancers such as Synthol and Esiclene to name a few.

When examining the potential medical issues associated with anabolic steroid use, evidence indicates that most known side effects are transient. More so, few studies have been able to directly link anabolic steroids to many of the serious adverse effects listed. Although clinical case studies continue to link anabolic steroid administration with myocardial infarct, suicide, and cancer, the evidence to support a cause and effect relationship is lacking and it may be other contributing factors (i.e. genetic predisposition, diet, etc.) play a substantial role and potentiate the harmful effects from anabolic steroids. Consistent physician monitoring is critical to the athlete who consumes anabolic steroids. However, many athletes may not undergo extensive medical exams prior to androgen administration and few physicians may be willing to provide such monitoring.

The purpose of this review was not to support or condone anabolic steroid use. Rather, the aim was to discuss pertinent medical issues and provide another perspective in light of the fact that many anabolic steroids users do not appear to prioritize the health/safety hazards or potential adverse medical events. In order to maintain credibility with the athlete, it is important to provide accurate information to the athlete in regards to these performance enhancing drugs, and provide education about alternative means and potential risks. Finally, anabolic steroids have been used legitimately for several clinical purposes such as muscle wasting or hypogonadal related diseases.

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KEY POINTS

- For many years the scientific and medical communities depicted a lack of efficacy and serious adverse effects from anabolic steroid use.
- Clinical case studies continue to link anabolic steroid administration with myocardial infarct, suicide, and cancer, evidence to support a cause and effect relationship is lacking.
- It may be other contributing factors (i.e. genetic predisposition, diet, etc.) that play a substantial role and potentiate the harmful effects from anabolic steroids.

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