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Anabolic steroids and the athlete: a case study

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This paper examines the pharmacokinetic activities of anabolic steroids and their potential deleterious effects. A review of literature reveals the most significant pathological sequelae resulting from anabolic use to be peliosis hepatis and liver cell carcinoma. These ill effects have been more closely associated with those steroids whose chemical structures are specifically alkylated at the 17th carbon in the Alpha position as opposed to their Beta esterified counterparts. Testing of these drugs was attempted by way of a single case study. A 23 yr old male bodybuilder was subject to both oral and parenteral forms of steroid over a six week period of his training program. Serum, urinalysis and subjective parameters were monitored before during and after steroid administration. The results show elevated levels of urea, creatinine, bilirubin, CPK, AST, ALT and LDH. In this case study, the elevated parameters appear to be more a function of muscle breakdown induced by a combination of severe exercise and intramuscular injection than a measure of organ (liver) pathology. (JCCA 1989; 33(1): 27–33)

KEY WORDS: anabolic, androgenic, steroids, athletics.

Cette communication a pour objet d'examiner l'activité pharmacocinétique des stéroïdes anaboliques et de leurs effets délétères potentiels. Une étude de la documentation scientifique révèle que les séquelles pathologiques les plus significatives de l'usage des stéroïdes anaboliques sont la péliose hépatique et le carcinoma des cellules hépatiques. Ces effets néfastes sont plus étroitement associés aux stéroïdes dont les structures chimiques sont spécifiquement alcoylantes au 17e carbone de la position Alpha par opposition à leurs homologues Bêta éterfiés. L'essai de ces substances a été entrepris grâce à l'étude d'un seul sujet. Ainsi, un haltérophile masculin âgé de 23 ans a reçu des doses de stéroïdes par voie orale et parentérale sur un péroide de six semaines de son programme d'entraînement. Le sérum, les analyses d'urine et des paramètres d'ordre subjectif ont été contrôlés pendant et après l'administration des stéroïdes. Les résultats indiquent des teneurs élevées un urée, créatinine, bilirubine, CPK, AST, ALT et LDH. En conclusion, des études plus approfondies sur les effets des stéroïdes sur le rendement des athlètes doivent se poursuivre. (JCCA 1989; 33(1): 27–33)

MOTS CLEFS: anabolique, androgénique, stéroïdes, sports.

Introduction

Many health care practitioners are deeply committed to the well being of the contemporary athlete. Indeed, a number of practitioners have devoted their practices exclusively to the care of these individuals. Competitors are increasingly searching for what they consider to be the "winning edge". As a result, they have become the product of highly sophisticated training methods. Some of these methods incorporate the use of anabolic steroids as an integral part of their training regimen.

This article focuses on the potential deleterious effects of these drugs. Many athletes who elect to use anabolic steroids are unaware of their pharmacological properties, yet they utilize them in their relentless pursuit of victory. Hopefully the following revelations will better acquaint the practitioner and athlete with some relevant facts concerning their usage.

The use of anabolic steroids dates back to World War II when they were provided to German soldiers in order to enhance their aggressiveness. Since then, anabolic steroids have become increasingly popular amongst athletes, so much so, that steroid use in both professional and amateur athletics has reached epidemic proportions. In the late 1950's, Ciba Pharmaceuticals introduced what was to become the most popular anabolic drug for athletes, that being methandrostenolone *(Dianabal)*.[2,3] By this time, the era of the steroid athlete was well underway and world records were being shattered and reshattered with remarkable regularity.[2]

Anabolic steroids can generally be defined as that group of steroid compounds, chemically resembling the male sex hormone, one of whose principal functions is to stimulate the

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synthesis of cellular protein. The most potent steroid synthesized by the body and possessing anabolic properties is testosterone. Testosterone and various similar steroid hormones are termed androgens, from the Greek word meaning male-producing.[4,5] Testosterone is primarily produced by the interstitial cells of the testes. This and other less potent androgens are also synthesized by the ovaries and adrenal glands.[2] In males, testosterone is produced at a rate of approximately 4–10 mg/day and 0.1 mg/day in females. Some of the most dramatic and significant of the masculine characteristics, which develop as a male matures from childhood through puberty, can be attributed to a 20-fold increment in the testosterone titre.[6,7] Changes in skin, hair, voice, testes and accessory sexual organs are arbitrarily ascribed the "androgenic" effects of the hormone; while alterations in muscle, bone and blood are claimed to denote the "anabolic" effects.[8]

Mechanism of steroid action

At present there appear to be three methods by which testosterone and other hormones elicit their effects.[9]

1 by exerting direct effects on specific enzyme systems within cells, thus affecting reaction rates;
2 by changing the permeability of cell membranes, thus affecting the cellular supply of raw materials or nutrients; and
3 by stimulating or suppressing particular genes, thus increasing or decreasing production of certain substances.

However, growth of human tissue is far more complicated than a protein synthesis apparatus merely influenced by testosterone. This complex growth process additionally involves the harmonious interaction of a number of the following diverse factors:

1 an individual's genetic capacity for growth;
2 the demand (both quality and quantity) placed on the body or muscle for additional growth;
3 the supply of nutrients (quality and quantity) available for use in the building of new tissues; and
4 the type and quantities of hormones present in the environment.[2,9]

As previously mentioned, athletes have attempted to capitalize and exploit the last of the above factors, namely the types and quantities of hormones present in the environment. Anabolic steroids were developed with the express intention of minimizing the androgenic effects, while maximizing their anabolic qualities. To date, however, it has been impossible to completely dissociate the two functions.[10,11] Therefore, individuals employing anabolic steroids and benefiting from their anabolic effects, must also incur the more undesirable side effects of these agents. What has ultimately resulted is the development of a large array of anabolic steroids, which are capable of inducing protein synthesis in both muscle and bone.

Anabolic steroid chemistry

Since anabolic steroids are derivatives of the testosterone molecule, they can be administered in either oral or parenteral

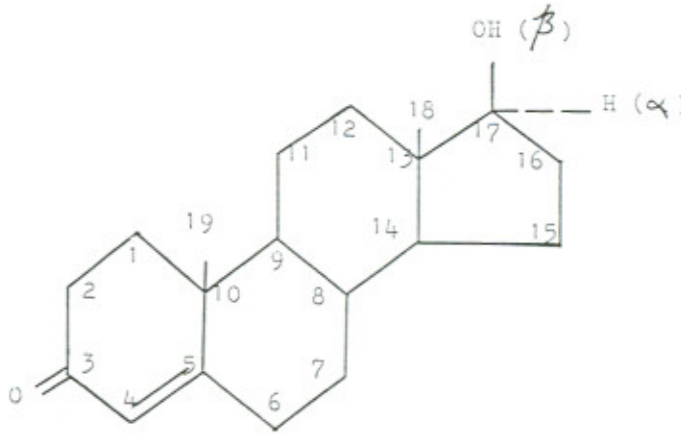

Figure 1 A representation of a model of testosterone molecule. The numbers refer to carbon atoms. β and α refer to the stereochemistry of the hydroxyl and hydrogen groups at Carbon 17.

forms. Alterations of the molecule at the 2, 9 and 4 positions enhance the anabolic activity of androgens, while adding side groups to the 17th (Alpha or Beta) positions affect the duration of its activity.[2,13] (Figure 1)

Esterification of the 17-Beta position of other steroid molecules with varying length carboxylic acids, increases the lypophilic nature of the drug and therefore, prolongs its activity. Thus, as the length of the carboxylic acid is increased, so is the duration of its activity. Table 1 illustrates the esters of testosterone and other androgens, presented in order from fastest acting, which are administered in one to three day intervals, to *Deconoate* and *Cypionate* esters which may retain their potency for 2 to 3 weeks or longer.[9] The most recent ester to undertake clinical trial is *Dimeric Testosterone*, in which a single injection in the rat was found to remain effective for as long as a 40 week period.[14]

Alkyl substitutions can also be made at the 17 alpha position by a methyl or ethyl group. Almost all of the orally active steroids are alkylated at this position with the exceptions of *Methenolone Acetate, Testosterone Undecanoate and Mesterolone*.[15,13,2,9] These oral compounds are potentially hazardous because they have been frequently associated with far more hepato-toxicity than their esterified counterparts.[16,17,18,19,2,9] Thus, the parenteral forms are preferred for both the medical and non-medical applications of these compounds.[20,21,22]

The most significant pathologic sequelae associated with the use of anabolic / androgenic steroids are peliosis hepatis and liver cell carcinoma. To date, no instances of peliosis hepatis have been reported in athletes or otherwise healthy individuals. Twenty-three cases have, however, occurred in patients being treated for various medical illnesses; twenty-two of these were associated with the use of orally active C-17 alpha derivatives of testosterone. The twenty-third patient was treated with a

parenteral anabolic steroid, *Testosterone Enanthate*, which is an androgenic/anabolic preparation rather than a true anabolic steroid.[13]

With regard to hepatic tumors, thirty-six cases have been reported, one of which occurred in an athlete.[13] The balance arose in patients once again being treated for a variety of medical conditions. All but one of the reported cases were associated with the use of orally active C-17 alpha alkyl derivatives of testosterone. As was the case with peliosis hepatis, the other instance of liver tumor was related to the exclusive use of *Testosterone Enanthate*, an androgenic/anabolic steroid.[23]

The one reported case of a steroid induced hepatic tumor in an athlete resulted in his death.[24] This was a male bodybuilder who succumbed to hepatocellular carcinoma and hepatic cholangiocarcinoma. The athlete employed both oral and parenteral compounds for four years preceding his death including; *Methanorostenolone* (oral), *Oxandrolone* (oral), *Stanozol* (oral), *Methenolone* and *Nandrolone Decanoate* (administered intramuscularly). This individual utilized the 17-alpha alkyl derivatives of testosterone for a protracted period of time, as was the case with almost all of the other reported instances of hepatic tumors attributed to the use of anabolic steroids. It is significant to note that *Oxymetholone (Anadrol)* was the preparation most frequently associated with the incidences of both peliosis hepatis and hepatic carcinoma.[25]

Steroidal effect on strength

In theory, the use of anabolic steroids is meant to produce a direct relationship with athletic performance. In practice, many athletes who use steroids attest to the positive anabolic effects of these drugs. Yet the scientific community has produced much data, in which there appear to be many inconsistencies pursuant to testing the anabolic effects of synthetic steroids. In 1984, Haupt and Rovere[25] published an article in the American Journal of Sports Medicine, wherein they reviewed the scientific literature concerning this controversy. Their critical analysis revealed that the inconsistencies were the result of differences in study protocols. In addition, they concluded that those studies demonstrating significant increases in strength were consistent with the following characteristic protocols:

1 athletes who weightlifted prior to steroids being administered and continued to weight train during the anabolic steroid regimen;
2 they more frequently studied the effects of the orally active anabolic steroid, *Methandrostenolone* (dianabol), than the effects of all other steroids combined; and
3 they measured the athletes' strength by using the single repetition – maximal weight technique for the various weight-lifting exercises.

It is interesting to note, however, that the relationship between the anabolic steroid *Methandrostenolone* and significant strength increases, appears to be more a function of its popularity than its pharmacology.[25]

One of the main functions of anabolic steroids is to induce protein synthesis. This mechanism is achieved by steroid receptors which exist in the cytoplasm of most tissues within the body (muscle, prostate and other organs).[2] These receptors receive steroid hormones, that in turn initiate the protein synthesis of ribosomal and messenger RNA with a resultant accompanying increase in protein building enzymes at the ribosomal level.[15,3] This effect produces an increment in transcription and translation of genetic codes, which dictate protein synthesis.[15,19] It has recently been demonstrated, that anabolic steroids potentiate this effect throughout the duration of steroid therapy.[2]

Another anabolic aspect of steroid therapy is its apparent motivational effect. This factor is well documented, yet poorly understood.[15] Anabolic steroids often have a profound psychological effect on an athlete, thereby suggesting a placebo effect. Ariel and Saville demonstrated statistically significant improvements in the performances of athletes who thought they were administered anabolic steroids, compared to the same athletes' accomplishments prior to the placebo period.[28] Hence, using a placebo and thinking it was a steroid, created significant motivational changes with resultant increased output over what had been realistically expected in the absence of steroids. In addition, many athletes who used steroids claimed they experienced a sense of well-being or euphoria, inner strength and diminished fatigue which enhanced their training.[26,15,27,13] It has been suggested, that this phenomenon is directly attributable to the steroids' effect on the central nervous system and that this effect permits the athlete to conduct workouts at higher intensities for longer durations.[15]

Anabolic steroids, also have a significant influence on haemopoetic tissues. Primarily, the effects result in increases in hemoglobin concentration and overall blood volume. Thus, steroids have proven to be useful in the treatment of certain types of anemia. This positive effect on red blood cell volume is apparently elicited by an androgenic stimulation of erythropoetin production.[15,13,2] Further, Wright reports that changes in respiratory quotients upon steroid administration, reveal there may be a shift toward increased lipid metabolism.[9] The overall influence of steroid hormones on adipose tissue and fat metabolism is still, however, unclear.

The above cited influences of anabolic steroids on athletes may be viewed in a rather favourable manner. Yet not all the effects of these drugs can be regarded as positive. Scientific literature is replete with data, which describes the significant health hazards associated with these drugs. The major organ systems whose functions may be altered by steroid use include those which are involved in the transport, metabolism, detoxification and excretion of these drugs and their metabolic by-products.[28,25,14] Therefore, the organ systems primarily involved are the hepatic, renal, skin, reproductive organs (i.e. testes and ovaries) cardiovascular and neuroendocrine systems.[25,15] As previously indicated, the liver is particularly vulnerable. The liver is at high risk with exposure to steroids and

when extensively affected may present the gravest of health consequences.

To assess liver function, blood samples are secured and specific serum enzyme levels are monitored. There are essentially two categories of liver function tests (LFT's). The first one is the non-specific LFT's which include aspartate alanine transferase (ALT), aspartate oxaloacetate transaminase (AST) and lactate dehydrogenase (LDH). These indicators are non specific for this organ, since they exist in tissues other than the liver.[13,25] AST not only occurs in significant concentrations in the liver but is also produced by the heart, skeletal muscle, kidney and pancreas. ALT is found in the liver, kidney, heart and skeletal muscles. LDH is present in high concentrations in the liver, heart, skeletal muscles and erythrocytes.[25,29,13] Therefore, a disturbance in the function in any one of or a combination of these organs other than the liver, may result in a false positive impression of hepatic dysfunction. The second category of LFT's are the specific LFT's which consist of the liver isoenzymes of LDH and alkaline phosphatase (ALP).[29] LDH can further be separated into liver, brain and cardiac isoenzymes.[29] Separation is performed by various electrophoretic, chromatographic and immunologic characteristic techniques. ALP concentrations circulating in serum are principally derived from liver and bone tissue in adults. Although isoenzymes can be separated to represent either of these tissues, Haupt and Rovere suggest that this is unnecessary since anabolic steroids do not significantly stimulate osteoblastic activity which would elevate serum ALP from bone.[25] Thus, ALP and the liver isoenzyme LDH are regarded as specific LFT's. Studies which demonstrated elevations in LFT's revealed abnormally high levels of non-specific and specific LFT's, yet alterations in any one specific test were not significantly predictable. At any rate, abnormal LFT's have been associated with severe liver dysfunction, namely progressive cholestasis with resultant jaundice. Furthermore, the extremely serious conditions of peliosis hepatis and subsequent tumor formation have resulted in death.[30]

Peliosis hepatis has been increasingly documented in various groups of patients receiving oral steroids.[31] This liver condition results in the development of dilated sanguinous cysts throughout the liver, which may ultimately hemorrhage or result in liver failure.[24,31] In addition, the reported cases of hepatic tumors were associated with longer durations of steroidal administration than were the instances of peliosis hepatis.[31] Paradinas[31] recognized this fact and suggested that peliosis hepatis may be a pretumorous state, which may predispose to malignancy by the administration of oral anabolic steroids. More specifically, the parenchymal changes observed in peliosis may potentiate nodular hepatocyte development and eventual neoplasia. As with other hepatotoxic and systemic sequellae, these ill-effects have been associated with those testosterone analogues which are specifically alkylated at the 17th carbon in the Alpha position.[25,15,13,2,9] The hepatotoxic types of synthetic androgenic anabolic steroids are almost always administered in their oral form.

The reproductive organs may also be affected by the use of anabolic steroids. Changes in reproductive behavior and physiology are well documented. These alterations include changes in plasma testosterone, secretion of gonadotrophins, spermatogenesis, testicular size and libido.[32,30] Testosterone focuses primarily on the testosterone-binding globulin, resulting in decreases in this protein-bound fraction of plasma testosterone. A proposed mechanism responsible for this phenomenon is the displacement of testosterone by the synthetic steroid from the protein globulin, through competitive inhibition.[30] Alterations in gonadotrophins,interstitial-cell-stimulating hormone (ICSH) and follicle stimulating hormone (FSH) have also been reported.[32] It is postulated that high serum levels of anabolic steroid act to replace testosterone causing the pituitary to shut down its secretion of these gonadotrophins. This reduced state of ICSH and FSH appears to be associated with alterations in spermatogenesis. Examination of sperm from athletes using steroids reveals pathologic changes as related to the fertility index.[33] Changes in all indices including sperm density, motility and morphology were noted. These effects however, were reversible upon discontinuance of the drug.[25]

Case presentation

This case studies a 23 year old male bodybuilder who, with a healthy history, incorporated the use of anabolic steroids into his training program for an upcoming competition. The subject administered both oral and parenteral forms of steroid over a 6 week period. Serum and urinalysis were conducted before, during and after a six week interval of steroid administration. In addition, the subject recorded his subjective side effects during this period.

At 7 weeks precompetition, the athlete initiated an anabolic steroid program for a 6 week period. He discontinued his drug use 1 week prior to competition. During the initial 3 week period, the subject administered parenteral agents, *Nandrolone Deconoate* 300 mg/wk, *Bolasterone* 30 mg/wk, and *Mesterolone*, which is an oral compound but is altered at the 17-beta position so that it possesses pharmacological characteristics similar to an injectable steroid (25 mg/day). During the next 3 weeks of therapy, the steroid types used were ones which were associated with less water retention and therefore, minimized the "bloated" appearance which is an undesirable state for competition. These included most of the oral preparations which have short side chains at the 17 alpha position of the steroid molecule. The drugs in this phase included *Mesterolone* 50 mg/day, *Oxandrolone* 35 mg/day, *Boldenolone Undecylenate* 150 mg/wk, and *Methenolone Actate* 60 mg/wk.

Serological assessments were completed prior to initiation of anabolic therapy, twice during the 6 week drug session and at five and 13 weeks following discontinuance of drug use. The results of these tests are recorded in Table 2. Also recorded were any subjective side effects noted by the athlete. (Table 3)

Table 1

Testosterone – Androgen Ester	Side Group Structure
Acetate	R = —$COCH_3$
Proprionate	—$COCH_2CH_3$
Phenylocetate	—$COCH_2$
Phenylproprionate	—$COCH_2CH_2$
Enanthate	—$CO(CH_2)5CH_3$
Deconate	—$CO(CH_2)8\ H_3$
Cypionate	—$COCH_2CH_2$

Table 2 Objective parameters measured before, during and after steroid administration. (Normal values in brackets)

STEM Parameters Measured	Pre drug use	3 wks drug use	6 wks drug use	Post 5 wks drug use	Post 13 wks drug use
Urea (3.0–7.0 Mol/L)	7.8	7.9	8.0	7.5	6.9
Creatinine (50–129 mol/L)	122	138	142	135	100
Bilinubin (0–20 mol/L)	7	7	10	7	17
Total Protein (60–80 g/L)	69	70	71	70	69
Albumin (35–50 g/L)	47	49	46	49	42
CPK (0–100 IU/L)	1532	1649	1833	240	184
AST (0–30 IU/L)	54	57	98	41	43
ALT (0–30 IU/L)	53	58	137	109	93
ALP (30–90 IU/L)	48	52	42	54	40
LDH (105–193 IU/L)	205	209	251	203	200
Cholesterol (<7.0 MMol/L)	3.4	3.35	2.91	3.1	3.7
T.G (<1.8 MMol/L)	1.1	1.32	0.62	0.81	0.9

Table 3 Subjective side effects reported by the athlete throughout case study

1 Increased libido
2 Increased frequency of erection
3 Priapism
4 Testicular atrophy
5 Muscle spasm
6 Increased acne
7 Water retention
8 Increased urinary output
9 Increased sensitivity of aereola
10 Increased frequency of headaches upon drug discontinuance
11 Increased muscle mass, irrespective of an increase in training intensity

Discussion

Urea values were elevated throughout the entire study period. This was likely due to the high protein intake of the athlete and not necessarily attributed to any degree of renal dysfunction.[34] Creatinine, also a kidney function test, was elevated throughout most of the study with the exception of 13 weeks post drug use. Creatinine excretion was consistent in a given time period and was proportional to the increased amount of muscle mass acquired by the subject.[35,34] Table 2 reveals a steady increase in creatinine excretion from the non-drug use state to the high point of drug consumption and then steadily diminished thereafter. This seemed logical since the athlete acquired muscle mass in proportion to the creatinine increase. The subject trained little from the day of competition to the 13 week post period and hence lost approximately 10 pounds of muscle mass which was reflected by diminishing creatinine levels.

Bilirubin remained normal throughout the study suggesting undisturbed liver bilirubin metabolism. Total protein and albumin changes, in this study, were also unremarkable.

Creatinine phosphakinase (CPK) determinations were highly elevated throughout the drug use period but decreased markedly in both the non-training and non-drug use periods. The elevated titres of CPK were attributed to muscle damage induced by severe exercise[35,34] (4–5 hours daily) and to the intramuscular injections administered by the subject. During the pre-steroid phase, CPK titres were lower than both the 3 week and 5 week drug stages. An elevation of 301 IU/L of CPK from the pre-steroid to the 5 week steroid stage was likely the result of the intramuscular injections since the intensity of training did not appreciably alter during these stages of the study.

The non specific liver function tests, AST and ALT, maintained higher than normal values throughout the entire study. Note, these values were elevated prior to anabolic therapy initiation. This was not surprising since both AST and ALT are found in skeletal muscle and are released into the serum when muscle damage is increased.[35,34,29] This is explained in a manner similar to that of CPK elevation. Strauss et al studied 32 weightlifters of which 20 were using oral anabolics while the remaining 12 were not.[36] The steroid subjects demonstrated ALT levels greater than normal and AST values on the high side of normal. The 12 individuals not using steroids, demonstrated ALT and AST levels virtually identical to those using the steroids. Another study conducted by Hogerman[14] of 5 weightlifters, 3 of which were taking steroids and 2 who were not, determined that all five lifters displayed elevated AST and LDH levels while ALT and ALP were normal. The studies suggest that intense weight training alone, can elevate non-specific LFT's.

At the peak of drug use, AST and ALT levels reached 98 and 137 IU/L, respectively. This could arise secondarily to the increased drug consumption at this stage of the study and/or to the change to the oral forms of steroids, namely *Oxandrolone*. This specific drug, of course, has been associated with higher incidences of hepatotoxicity than the parenteral forms.[15,28,31]

Another hypothesis suggests that the elevation of LFT's is due to skeletal muscle breakdown rather than any hepatic dysfunction.[28] There are two aspects which reinforce this conjecture. One proposes that these elevations parallel those of CPK which reflects muscle damage due to exercise and intramuscular injections. Secondly, the specific LFT, especially alkaline phosphatase, remained within normal parameters throughout the entire study, even on the lower side of normal. It appears likely that a combination of all the above exists and that the latter factor, muscle breakdown, plays the largest role in the elevation of non-specific LFT's.

Total LDH levels were monitored and since this enzyme is found in skeletal muscle as well, it is not surprising that these changes parallel those of CPK. Haupt and Rovere suggest that measurement of the hepatic fractionation of LDH is a useful specific LFT.[25] Biochemical analysis during this study did not include LDH fractionation, as such LDH was considered as a non-specific liver function test. Both cholesterol and triglycerides (TG) levels were unremarkable throughout the study. Urinalysis was similarly unremarkable.

Conclusion

In view of the measured results in this isolated case study, it was considered that the only noteworthy alterations in blood chemistry were CPK, AST, ALT, and LDH. It was also felt that no pathophysiological processes were occurring with respect to liver dysfunction and that elevated non-specific LFT's were more a product of muscle breakdown induced by severe exercise and by intramuscular injection. This can be substantiated by the elevations and reductions of LDH, AST, and ALT, all of which parallel CPK levels with the exception observed 5 weeks post-drug therapy, where ALT levels assumed a value of 109 IU/L. This might be explained by the fact that ALT is cleared from the bloodstream more slowly than AST and hence its levels remain higher for a longer period of time once stimulatory factors are removed.[35,34]

In subsequent studies, it will be important to measure HDL/LDL concentrations, as it has been shown that dramatic decreases in HDL result with anabolic use.[34] In Webb's study, the subject used a dosage range of 550–1100 mg/week of anabolic steroid for an average period of two months.[39] This study's subject averaged a weekly dosage of 600 mg for a period of six weeks, therefore some serological alterations could realistically be expected. It is interesting to note undocumented statements, that support some competitors consuming up to 2000 mg of steroids per day.[15] The serum chemistry profiles of these individuals would certainly be of interest.

What is required at this time are continued studies which regard the effects of the ever increasing dosages used by today's athletes. Almost all the information documented regarding the iatrogenic effects of anabolic steroids are provided by patients who required these preparations for medical purposes (ie., aplastic anemia, Fanconi's anemia, hypogonadism, neoplasm, pancytopenia, osteoporosis and others). These dosages administered to medical patients, however, were generally quite small.[25]

As abuse by athletes escalates, new investigations into the potential health risks arising in otherwise healthy individuals, using massive sustained exogenous doses of steroids, should be conducted. Their use cannot be condoned until these factors are known.

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