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ANABOLIC ANDROGENIC STEROIDS - RECENT PERSPECTIVES

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ABSTRACT

Anabolic Androgenic Steroids (AAS) are a wide class of chemicals that include naturally occurring androgens like testosterone and their synthetically created analogues. AAS usage is viewed as a public health issue because of the negative effects that AASs have on all organs, tissues and bodily processes, particularly long-term toxicity that affects the cardiovascular system and the reproductive system. A range of different performance and image enhancing drugs are frequently taken, while anabolic steroids are by far the most over used. Increased knowledge is required in this area among the general public and healthcare professionals for diagnostic, therapeutic, and preventative purposes. AAS side effects are caused by a number of processes that need to be better understood. Multiple organs and systems in both humans and animals are affected by AAS. In addition to anabolic steroids, some sportsmen also use other drugs such alcohol, opioids, cocaine, marijuana and gamma hydroxybutyrate, some of which might negatively interact with AASs. The sex, dosage and duration of administration of AAS all have an impact on its effects. Supraphysiologic doses of anabolic-androgenic drugs also have cardiovascular adverse effects. The often reported concomitant use of testosterone and cocaine increases the risk of thrombus, stroke and myocardial infarction. Steroids also cause hypertrophy of the myocardium, which also increases the likelihood of arrhythmias, sudden death, systolic and diastolic hypertension and myocardial infarction. AAS use is a decrease in tendon strength brought on by the dysplasia of collagen fibrils. In order to avoid AAS misuse, education and information are crucial.

KEYWORDS

Anabolic Androgenic Steroids, Drug abuse, AAS misuse and Supraphysiologic doses of AAS.

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INTRODUCTION

AASs, also referred to as anabolic steroids, are a wide class of chemicals that include naturally occurring androgens like testosterone and their synthetically created analogues[1]. The most often abused androgens are testosterone, nandrolone decanoate (ND), methandienone, and methenolol.

Due to its capacity to enhance muscle growth for aesthetic goals and athletes' performance while limiting androgenic effects, AAS are widely used[2]. While androgen use was once limited to professional bodybuilders, it has recently gained popularity among amateur athletes[3]. Indeed, androgens can increase both the size and strength of muscle fibers. The medicinal usage of AAS anabolic characteristics is common. In fact, AASs played a part in the treatment of disorders associated with a negative nitrogen balance, such as chronic kidney disease, osteoporosis in postmenopausal women, incurable breast cancer and chronic kidney disease[4].

The World Anti-Doping Agency (WADA) forbids the usage of AASs. The use of AAS is still relatively common, with estimates of its usage in the US ranging from 1-3%[5]. AAS usage is viewed as a public health issue because of the negative effects that AASs have on all organs, tissues, and bodily processes, particularly long-term toxicity that affects the cardiovascular system and the reproductive system[6]. A range of different performance and image enhancing drugs are frequently taken, while anabolic steroids are by far the most overused. Increased knowledge is required in this area among the general public and healthcare professionals for diagnostic, therapeutic and preventative purposes. These can be characterized as compounds that increase muscle mass, burn fat, act as pre-workout supplements, or prevent or treat negative effects. Additional negative health consequences may be anticipated depending on the composition and dosage of these drugs. The androgen receptor's (AR) role in signaling is connected to the anabolic androgenic effects. There are many androgen receptors in the tissues and organs of humans. There are three primary modes of action: (i) direct binding to the androgen receptor; (ii) through dihydrotestosterone (DHT) produced by the action of 5a-reductase; and (iii) through estrogen receptors via estradiol produced by CYP19 aromatase. Skeletal muscle and bone expand as a result of anabolic effects, which stimulate linear growth that eventually stops when the epiphysis closes. Androgens have a crucial part in the maintenance of skeletal muscle and bone, cognitive function and a sense of well-being in men. They are also crucial for the maintenance of reproductive function. The Androgens are essential for men's cognitive ability, sense of wellbeing, and preservation of skeletal muscle and bone. They are also essential for maintaining reproductive health. Examples of androgenic effects include larynx enlargement that deepens voice, terminal hair growth (in pubic, axillary and facial areas; in other regions, such growth depends on a number of factors), an increase in sebaceous gland activity that can cause acne and CNS effects (such as libido and increased aggression)[7]. Testosterone, an endogenous androgen, was isolated, described, and made accessible for external administration in 1935. The most major androgen secreted is testosterone.

TYPES OF ANABOLIC-ANDROGENIC STEROIDS

The active metabolites of testosterone the traditional AAS include dihydrotestosterone, androstanolone, estradiol and androstenedione[8]. Due to the hepatic isoenzymes of the cytochrome P450 family's fast metabolism, testosterone has a short half-life in free circulation[9]. Numerous synthetic AAS made from the chemical structure of testosterone have extended half-lives in circulation to counteract this fast metabolism. More than 1,000 testosterone derivatives have been created.

Three sorts of modifications apply to the AAS. Esterification of testosterone at the 17-betahydroxy site with various carboxylic acid groups constitutes class A modification. By lessening the polarity of the molecule, this change makes the AAS more lipophilic and hydrophobic, which boosts its androgenic characteristics and causes it to absorb more slowly when administered intramuscularly. The increased molecule's lipid solubility is a result of the esterification's lengthy carbon chains[10]. Unlike class A anabolic-androgenic steroid derivatives, which only need intramuscular dosing once every 2 to 12 weeks, depending on the carboxylic acid groups added, natural testosterone would need to be injected several times each week. Class A AAS is hydrolysed by the body after injection and share the same metabolic pathway as endogenous testosterone. After a class A injection, the levels of AAS reach a

peak and then progressively fall until the following injection, when they return to baseline levels. The exceptions are methenolone acetate and testosterone undecanoate class A AAS, which can be administered orally and either avoid the portal circulation or have a delayed liver metabolism. A testosterone derivative that can be administered orally with delayed hepatic degradation has undergone alkylation at the 17-beta hydroxy location in a class B derivative. As a whole, these chemicals are less potent than testosterone or class A AAS. They have also been demonstrated to stimulate the production of hepatic enzymes and to be harmful to the liver, particularly complement 1 inhibitor[11].

A, B, or C rings of the steroid backbone are modified through alkylation in Class C AAS compounds. Although hepatic metabolism has been reduced to zero, the alkylation of the steroid ring results in features that are similar to those of class B AAS (i.e., oral availability). The class C chemicals are eliminated either unaltered, as metabolites, or as conjugates in the urine or feces. Class C derivatives can un-esterify to form class AC analogs by undergoing a class A esterification. Also available as oral medications are these analogs[12].

Three factors are typically at play when novel testosterone derivatives are created. To increase potency is the first step. The second is to enhance the anabolic properties of the medication while minimizing its androgenic adverse effects. No "pure" or "clean" anabolic steroid has been discovered or produced as of yet. Making AAS that are difficult to detect by blood or urine testing is the third motive for some with each alteration, the drug's androgenic/anabolic profile and side effect profile are both altered[14].

MECHANISM OF ACTION

Many different systems are thought to be involved in how anabolic steroids work. These processes include altering the topology of the androgen receptor, which in turn affects the androgen receptor's topology and, in turn, the interaction of the receptor with co-activators and transcriptional activity. Other mechanisms include an anti-catabolic effect by interfering with the expression of the glucocorticoid receptor and by Non genomic as well as by Genomic Pathways in the CNS Resulting in behavioural Changes[15]. Exploiting the basic difference may provide as a basis for raising the myotrophic to androgenic ratio. Between the levels of 5a reductase in both androgenic tissue and skeletal muscle. Giving a steroid with a higher binding affinity for the androgen receptor but a lower affinity when reduced to a 5a metabolite is one strategy to increase the anabolic androgenic dissociation. In contrast, the 5a-reduced form of 19-nortestosterone, 5a Dihydro-19Nortestosterone, has a smaller affinity for the androgen receptor than its parent steroid testosterone[16]. This is despite the fact that DHT has a higher affinity for the androgen receptor than its parent steroid testosterone. This myotrophic androgenic dissociation process having an additional double bond in the A-ring like chlorodehydromethyl testosterone and the favourable mytotrophic androgenic index of methandienone is[17]. However, the hypothesis proposed for the Differential action of a steroidal SARM called MENT (Non testosterone, Trestolone)[18]. Is that myotrophic androgenic dissociation may occur simply because the effect of the specific steroid cannot be amplified by 5a-reduction in androgenic target tissues. As an illustration, testosterone has a molecular weight of 288. Steroids are relatively tiny molecules that can passively move into cells. The hormone binds to the receptor ligand in target tissues, or the cells that contain steroid receptors.

The binding domain causes the receptor-Hsp90 complex to dissociate and the ensuring conformational (allosteric) shift makes the receptor active. The chaperone complex is found in the cytoplasm of the androgen (and glucocorticoid) receptor and after dissociation the activated receptor is transferred into the nucleus from the chaperone. When two receptors bind together cooperatively (with higher affinity and stability), they interact as homodimers with the steroid response element on the chromatin. The creation of a transcription complex a cluster of co regulators, also known as co modulators which fits around the receptors like "pieces in a jigsaw puzzle" is subsequently triggered by this connection to the DNA. Positive or negative

regulatory proteins, also known as co-activators or co repressors, can be co regulators[19]. The role of the receptor's transcriptional activation domains is to facilitate the binding of the receptor to co modulators. The receptor has an activation function-1 (AF-1) at the N-terminus and an activation function-2 (AF-2) at the other end. In the ligand-binding domain at the C-terminus. The mechanisms of AF-1 and AF-2 gene activation, with a focus on co-activator binding and AF-1 and AF-2 conformation[20]. Different affinities exist for anabolic steroids' interactions with the androgen receptor compared the relative binding affinity of the anabolic steroids tritiatedmethyltrienolone to that of ethylestrenol, fluoxymesterone, mesterolone, methandienone, methenolone, 17a-methyltestosterone, nandrolone, and oxymetholone (but with a 17a-alkyl substituent) to rat prostate gland, rat skeletal muscle, and rabbit skeletal muscle isolated androgen receptors. The comparison's respective binding affinities' order Nandrolone was more affine to methyltrienolone than methenolone, testosterone and meserolone[21].

The molecular chaperones heat shock protein, Hsp90, and p23, along with co-chaperones that use tetratricopeptide repeat (TPR) patterns, keep the steroid receptor inactive when hormone is not present. The Receptor-Hsp90 complex dissociates after hormone binding and the activated receptor is then translocated into the nucleus. A transcription complex, or a group of co regulators, is formed when activated receptors interact as homodimers with the steroid response element on the chromatin. This causes the gene to be activated, the gene to be transcribed, the protein to be translated and a change in the way a cell functions, grows, or differentiates. Although based in part on the figure in the paper by Weigel and Moore[22].

AAS USE AND ADVERSE EFFECT

AAS side effects are caused by a number of processes that need to be better understood. Multiple organs and systems in both humans and animals are affected by AAS. This may be a result of AR's pervasive presence in the body and the disruption of endogenous steroid production, transformation, and degradation[23]. By the time the receptors are saturated, AASs in supraphysiological levels may cause secondary effects[24]. AASs bind to a particular type of androgen receptor. But it's important to distinguish between adverse effects from AAS use (under medical supervision) and misuse (consumption of numerous medications at excessive doses; any non-medical use of these substances)[25]. In addition to anabolic steroids, some sportsmen also use other drugs such alcohol, opioids, cocaine, marijuana and gamma hydroxybutyrate, some of which might negatively interact with AASs. The sex, dosage and duration of administration of AAS all have an impact on its effects[26].

CARDIOVASUCLAR SYSTEM

AAS use most usually results in heart issues. Heart impairment results from long-term high AAS dosage treatment. AAS therapy negatively affected the autonomic control of cardiac muscle and prevented parasympathetic cardiac modulation in mice, according to an animal experiment[27]. Additionally, animals receiving AAS stop adapting to exercise-induced antioxidant repair. In addition to causing hypertension, left ventricular hypertrophy and an increase in the synthesis of cardiac muscle, high dosages of AAS that activate androgen receptor signalling also cause these side effects. Long-term exercise and the use of AAS reduce the left ventricle's capacity to relax. In this instance, taking AAS negates the benefits of exercise on left ventricular function. AAS users have thicker left ventricular walls, greater left ventricular masses, and mechanical dysfunction in their myocardium than AAS nonusers, according to various studies. Due to its pro-arrhythmic activities, creation of myocardial ischemia, and poor repolarization, the use of AAS also perhaps most importantly increases the risk of sudden death and life-threatening arrhythmias[28].

MUSCULOSKELETAL SYSTEM

AAS is frequently used to increase muscle mass. Actually, AAS increases IGF-1 production and androgen receptor signalling. This is related to an increase in protein synthesis in addition to enhancing muscular mass and strength[29]. High doses of AAS

use can adversely damage skeletal muscles. Vascular endothelial growth factor (VEGF), which is involved in this mechanism, mediates exercise adaption. The expression of VEGF will drop as a result of using AAS. Due to inadequate vascularization and remodelling brought on by the use of AAS in this case, lowering VEGF will result in bone loss. One study found a connection between AAS and tendon damage. The use of AAS can increase the risk of tendon rupture due to increased muscle mass and muscle strength during exercise[30].

REPRODUCTIVE SYSTEM

Testosterone is naturally produced by the male reproductive system. This hormone has a big impact on the physical and sexual growth of males. AAS is misused by athletes to increase their strength and muscle mass because it operates similarly to the hormone testosterone[31]. Infertility is brought on by the use of AAS because it alters the release of follicle-stimulating hormone (FSH) and luteinizing hormone (LH), which has a detrimental effect on the hypothalamic-pituitary axis. AAS administration lowers natural testosterone levels through interfering with the enzymes 17a-hydroxylase (CYP17A1) and the steroidogenic acute regulatory protein (StAR), according to a study using leydig cell culture. In another study, long-term high dose AAS use decreased sexual dysfunction after AAS cessation. A study on experimental animals found that AAS use reduced the number of sperm cells in male rats, which in turn affected faulty spermatogenesis. The rat seminiferous tubules' breadth and thickness also changed after receiving AAS.Long-term AAS use reduced the amount of sperm in the testicles, according to findings from another study on bodybuilders. Therefore, sperm production will increase again[32].

LIVER

One of the most common negative effects of AAS abuse is hepatotoxicity .Peliosis and cholestasis are two of the most frequent liver side effects that follow supraphysiological dosages of AASs. Peliosis is characterized by the presence of numerous tiny, blood-filled cystic spaces that are dispersed throughout the liver parenchyma. Antioxidant compounds offer protection from hepatotoxicity caused by AASs. Additionally, it has been shown that metabolic resistance and androgenic potency are positively correlated with the severity of liver damage. In AAS-induced hepatotoxicity, inflammation and necrosis may result in a regenerative signal[33].

CANCER

AASs have a biochemical process that is comparable to testosterone. AASs bind to DNA sequences and cause changes in gene expression. Combinations of genetic and epigenetic factors are the source of the toxicity, mutagenicity, genotoxicity, and carcinogenicity of sexual hormones, according to a recent review on the effects of androgens on cell functions.In adipose, cerebral, and testicular tissues, testosterone synthetic derivatives can be converted to 17-estradiol, a steroid known to be potentially mutagenic[34].

GYNAECOMASTIA

The benign expansion of the glandular tissue of the breast is known as gynecomastia. Contrary to pseudogynecomastia, also known as lipomastia or adipomastia, in which excessive adipose tissue provides the appearance of gynecomastia, the disorder is not to be confused with gynecomastia frequently manifests throughout puberty, with a peak frequency of 65% in males . One study found that 40.5% of healthy young men had gynecomastia and another found that 36% of healthy adult men had detectable palpable breast tissue.Among AAS users there is the belief that AAS might cause gynecomastia through alternative pathways, such as increased progestin action at the mammary glands or increased prolactin levels. While gynecomastia can develop in patients with hyperprolactinemia, the condition arises secondary to the gonadotropin suppression prolactin can cause. Importantly, prolactin levels are suppressed by androgens while not approved for treatment of gynecomastia, anti-estrogenic drugs such as aromatase inhibitors and SERMs are prescribed off-label and used in clinical trials to treat gynecomastia[35]. AAS users also self-

medicate with these drugs to either prevent gynecomastia from developing or to reduce the size of existing gynecomastia.

HEALTH EFFECT OF SUPERPHYSIOLOGIC DOSES OF AAS

Supraphysiologic doses of anabolic-androgenic drugs also have cardiovascular adverse effects. The HDL is lowered by approximately 33% by alkylating drugs like stanozolol, especially HDL2, which is reduced to 80% with only a 9% fall in HDL, testosterone's effect is far less pronounced. While testosterone has been decrease low-density lipoprotein by 16%, the alkylating AAS have been demonstrated to increase hepatic triglyceride lipase activity by 21 to 123% and low density lipoprotein by as much as 29%.

This increased risk for myocardial infarction and stroke is attributed to the increased platelet count and platelet aggregation that occurs in people who abuse AAS. Testosterone, even at concentrations in which it does not affect thrombus risk, can potentiate cocaine's effects on both the endothelium and platelet function. Therefore, the often reported concomitant use of testosterone and cocaine increases the risk of thrombus, stroke and myocardial infarction. Steroids also cause hypertrophy of the myocardium, which also increases the likelihood of arrhythmias, sudden death, systolic and diastolic hypertension and myocardial infarction[36]. AAS boosts bone and muscular strength, but an intriguing and seemingly counterintuitive side effect of high-dose AAS use is a decrease in tendon strength brought on by the dysplasia of collagen fibrils. As a result, those who take steroids to bulk up are more likely to experience either short-term or long-term crippling tendon ruptures. AAS abusers had a higher risk of experiencing rare triceps, biceps, and bilateral quadriceps ruptures, according to several studies.

AAS undergoes aromatization when levels are high, turning into estrogens in peripheral tissue through the action of fat and other cells. Gynecomastia in males may result from this increase in estrogen levels, either reversibly or irreversibly. In females, increased AAS levels cause breast atrophy and irregular menstruation. Additionally, there may be long-lasting masculinizing effects such clitoromegaly, hirsutism, a deeper voice and male-pattern baldness[37].

Those who use anabolic steroids in excess (more than 1,000mg/wk.) or who combine them with other medicines experience clear psychiatric side effects. The most prominent mental health symptoms were manic-like manifestations, which were characterized by agitation, aggression, euphoria, grandiose beliefs, hyperactivity and careless or dangerous behavior.Acute confusional state development, worsening optics, and the emergence of acute psychoses have all been reported as additional presentations. Users of steroids for extended periods of time may have steroid withdrawal symptoms such as suicidal thoughts and behavior, anhedonia, exhaustion, and weariness. It has been noticed that these withdrawal symptoms could exacerbate the dependent syndrome[38].

Table No.1: Commonly used drugs[13]

S.No	Administered orally	Administered intramuscularly	Administered trans dermally
1	Fluoxymesterone	Boldenoneundecylenate Equipoise	Testosterone
2	Halotestin Android-F, Ultandren	Methenoloneenanthate Primobolan depot	Androderm, Androgen, Testim, Testoderm
3	II Mesterolone	Nandrolone decanoate Deca-Durabolin	-
4	Mestoranum, proviron	Nandrolone phenpropionate	-
5	Methandienone, methandrostenolone	Durabolin	-
6	Dianabal	Nandrolone undecanoate Dynabolan	-
7	Methyltestosterone	Stanozolol Winstrol-V	-
8	Android, Testred, Virilon	Testosterone cypionate Depo-Testosterone, Virilon IM	-
9	Mibolerone	Testosterone enanthate Delatestryl	-
10	Cheque Drope	Testosterone esters blends Sustanon,Sten	-
11	Oxandrolone	Testosterone propionate Testoviron, Androlan	-
12	Anavar, Oxandrin	Testosterone undecanoate Andriol, Restandol	-
13	Oxymetholone	Trenbolone acetate Finajet, Finaplix	-
14	Anadrol-50, Hemogenin	Trenbolonehexahydrobencylcarbonate	-
15	Stanozolol	Parabolan	-
16	Winstrol	-	-

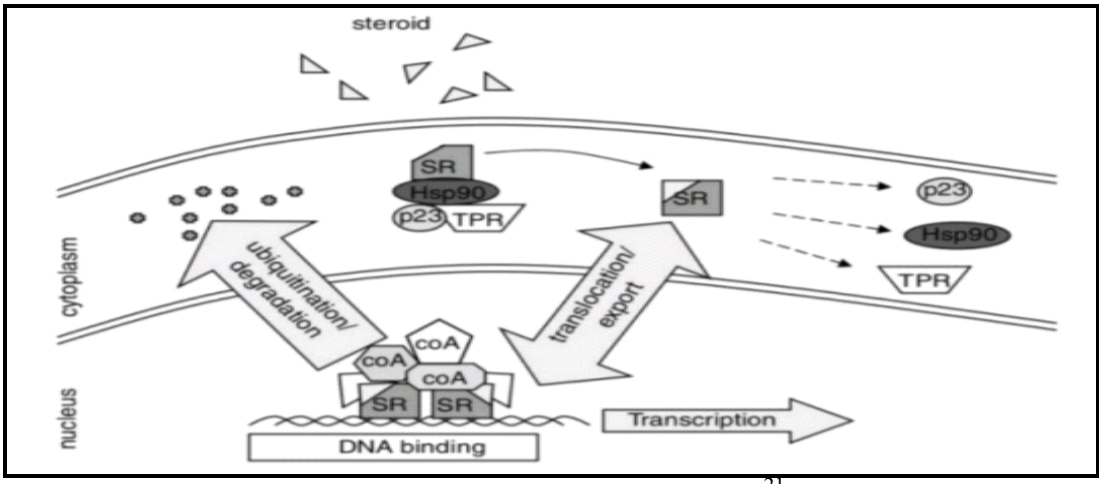

Figure No.1: Mechanism of AAS[21]

CONCLUSION

Organ health is negatively impacted by AAS. When AAS is utilized, harm mechanisms include apoptosis and oxidative stress. The use of high doses of AAS for an extended period of time has been shown to have serious adverse effects on various organs, including heart dysfunction, damage to bones and tendons, decreased sperm production, decreased sexual dysfunction, kidney damage, brain cell apoptosis, depression, anxiety, aggressiveness, addiction, and dependence. The long-term administration of high doses of AASs may lead to serious consequences, such as hypogonadism, cardiac impairment, neurodegeneration, coronary artery disease and sudden cardiac death. The long-term side effects affect the cardiovascular system, such as cardiomyopathy and atherosclerotic disease. Hypogonadism is a frequent finding in AAS abusers and need to be taken into consideration when AAS use is suspected in order to undertake aggressive treatment. The AAS misuse and abuse lead to adverse effects in all body tissues and organs. In order to avoid AAS misuse, education and information are crucial. By doing this, it is believed that the prevalence of AAS use would decline in the future.

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CONFLICT OF INTEREST

We declare that we have no conflict of interest.

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