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The Behaviour of Progestogens and Anabolic –Androgenic Steroids[AAS] : A Mini Review

Monika*[1], Arpit Raj[1], Dr. Anupama Diwan[1]
[1] School of Pharmaceutical Sciences, Apeejay Stya University,
Palwal-Sohan Road, Gurgaon-122103.

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ABSTRACT

Steroids are essential biological molecules that regulate a wide range of physiological processes, including metabolism, immune function, and reproductive health. They are classified into three main types: glucocorticoids, mineralocorticoids, and sex steroids, each with distinct roles in the body. Glucocorticoids, such as cortisol, play a crucial role in controlling inflammation and managing stress responses. Mineralocorticoids, like aldosterone, are responsible for maintaining electrolyte balance and regulating blood pressure. Sex steroids, including testosterone and estrogen, are critical for reproductive function and the development of secondary sexual characteristics. Deficiency in these steroids can result in severe health issues, such as Addison's disease (cortisol deficiency), hypoaldosteronism (aldosterone deficiency), and hypogonadism (sex steroid deficiency), leading to symptoms like chronic fatigue, low blood pressure, and infertility. Hormone replacement therapy (HRT) is a common treatment for such deficiencies, with synthetic glucocorticoids used to manage inflammation and sex steroid supplementation like prednisone employed in hypogonadism and menopause management. However, inappropriate or excessive steroid use can cause adverse effects, such as weakened bones, cardiovascular problems, and immune suppression. Recent advancements, such as selective steroid receptor modulators, aim to enhance therapeutic

outcomes while minimizing side effects. This review highlights the roles of steroids, their deficiencies, and therapeutic interventions.

1. Introduction

Steroids are intricate four-ringed organic molecules that play numerous vital roles and functions in multicellular organisms. They act as structural components of cell membranes, exemplified by the crucial dietary steroid cholesterol, and serve as functional regulatory agents in the form of modified cholesterol structures functioning as endogenous endocrine hormones.[1] Across all organisms, hormones in vivo play key regulatory roles in mediating communication and regulating essential functions and processes within and between cells, and across tissues, thereby connecting all organs of the body.[1] Endocrine hormones travel through the bloodstream, facilitating communication between cells and organs separated by considerable distances. Hydrophilic or water-soluble hormones primarily act at the cell surface by binding to protein receptors embedded in the plasma membrane.[2] Conversely, hydrophobic hormones primarily circulate bound to carrier plasma proteins and can diffuse freely across cell membranes to activate specific intracellular hormone receptors.[1,2]

1.1.Biosynthesis of steroids

All steroids in the body come from cholesterol through a well-regulated process that mostly takes place in specific endocrine organs like the adrenal gland, ovary, and testis. Further modifications of steroid structures and their functions can occur in various tissues and organs, including the liver, skin, brain, and prostate. In the steroidogenic cells of the adrenal gland, ovary, and testis, cholesterol is first converted to pregnenolone by the enzyme cholesterol side-chain cleavage enzyme (P450SSC), a cytochrome P450 enzyme. [3] This step is the key regulatory point for synthesizing most endogenous steroid hormones. The enzymes involved in this pathway are primarily cytochrome P450 enzymes or specialized hydroxysteroid dehydrogenases (HSD), which belong to the short-chain alcohol dehydrogenase reductase (SDR) enzyme superfamily. Adrenal steroid biosynthesis occurs in the outer cortex of the adrenal gland, which is divided into three layers where specific steroids are produced, depending on the expression of particular biosynthetic enzymes.[3] The outer zona glomerulosa expresses the Cyp11B2 P450 enzyme (aldosterone synthase) to produce the cardiovascular steroid aldosterone. The zona fasciculata, located centrally, expresses the Cyp11B1 P450 enzyme (11β-hydroxylase) to produce

glucocorticoid steroids like cortisol and corticosterone. [4] The inner zona reticularis produces adrenal androgens, such as dihydroepiandrosterone (DHEA), DHEA-sulphate (DHEAS), androstenedione, and testosterone, through the actions of enzymes like 3βHSD3, AKR1C3, and Sulfotransferase 2A1.[5] These adrenal androgens circulate in the bloodstream and serve as precursors for more biologically active reproductive steroids produced at higher levels in the gonads. There is evidence, mainly at the RNA level, that some of these steroid biosynthetic enzymes are also expressed in other tissues and organs such as the heart and brain, although it seems unlikely that these sites produce significant levels of bioactive steroids.[4,5]

Human plasma and tissue extracts can contain numerous different steroid compounds, many of which are biologically inactive. Active steroids are continually modified by other enzymes, particularly in the liver, to produce inactive metabolites. Most steroids are inactivated by hydroxylation or sulphation, which renders them unable to activate receptors or downstream intracellular signaling pathways and increases their solubility for renal excretion. Common hepatic modifications include 6β-hydroxylation, 5β-reduction of C21 and C19 steroids, and 4-hydroxylation of estrogens. Steroid sulfotransferases (SULT) are a large family of enzymes that transfer a sulphate moiety to the steroid ring from a donor molecule called 3'-phosphoadenine-5'-phosphosulphate. Important SULT enzymes include SULT2A1, which converts DHEA to DHEAS in the adrenal cortex, and SULT1E1, which sulphonates and inactivates estradiol for excretion. The hydroxysteroid dehydrogenases (HSDs) are also a significant enzyme family involved in steroid biosynthesis and inactivation.[6] Cortisol can be modified by two related HSDs, 11βHSD1 and 11βHSD2, which interconvert the keto/hydroxy side-chain on the 11th carbon of the cortisol steroid ring.[5,6] 11βHSD1 is a bidirectional enzyme but predominantly acts as a reductase, producing active cortisol in many metabolic tissues, such as the liver, brain, kidney, and white adipose tissue, thereby amplifying glucocorticoid-mediated signaling in target cells. 11βHSD2 is a unidirectional dehydrogenase that inactivates cortisol to cortisone in tissues, protecting the mineralocorticoid receptor (MR) from inappropriate activation by cortisol.[6,7] Loss of function mutations in the human 11βHSD2 gene cause the condition of 'apparent mineralocorticoid excess,' characterized by early-onset hypertension and cardiovascular complications in very young children. 11βHSD1 antagonists have been explored recently as potential treatments for metabolic syndrome by attenuating the metabolic effects of cortisol, although unwanted side-effect profiles have been a concern in early clinical trials.[7]

2. Types of steroids

2.1.Corticosteroids

Glucocorticosteroids (often called "steroids") are extensively used for their immunosuppressive properties. They are effective in managing cell-mediated immunity and inflammation in humans.[8] Despite their widespread use, many misconceptions persist regarding their mechanisms of action. Steroids are frequently prescribed to treat asthma and are also administered intranasally to alleviate allergic rhinitis. However, there is debate over which pathological mechanisms steroids impact in asthma—whether it is type I or type II. [9]The observed benefits of steroids in nasal allergies suggest that they likely reduce type I immunologic injury. It is important to note that decreasing histamine content takes several days.[8,9] The relationship between changes in intracellular cyclic AMP and clinical outcomes remains unclear. Steroids are also effective in mitigating type III hypersensitivity reactions, such as serum sickness or systemic lupus erythematosus.[10] It is noteworthy that in humans, steroids have minimal or no impact on antibody production or complement metabolism.[9,10]

2.2.Anabolic-Androgenic Steroids (AAS)

All anabolic steroids are derivatives of testosterone, possessing both androgenic and anabolic properties, as they promote growth and function of the male reproductive system. Individual drugs differ in their anabolic-to-androgenic activity ratio, but none of the currently available drugs are purely anabolic.[11] The anabolic steroids in use today are either derivatives of testosterone or structural modifications of testosterone that affect its pharmacokinetics, bioavailability, or the balance between androgenic and anabolic activity.[11] This category includes testosterone itself, all clinically used derivatives, and various plant products that claim to have anabolic effects. [12] Testosterone's actions encompass several activities. Firstly, it binds to the androgen receptor to exert its androgenic effects. Secondly, in some target tissues (such as the male urogenital tract, skin, liver, and sebaceous glands), it is reduced to dihydrotestosterone (DHT), which also acts on the androgen receptor.[12] Lastly, testosterone can be aromatized to estradiol, exerting estrogenic effects. The latter two actions are particularly undesirable in anabolic drugs; reduction to DHT decreases the ratio of anabolic to androgenic activity, while aromatization to estradiol can lead to feminizing side effects.[13]

- Testosterone is available in various forms, including injectables, transdermal patches, skin creams, and micronized oral preparations.[12]
- The 17-β esters of testosterone—such as testosterone cypionate, propionate, enanthate, and undecanoate—are more fat-soluble due to esterification at this site, which delays their absorption into the bloodstream. All these, except for undecanoate, must be administered via injection. Nandrolone also has 17-β esters.[12,13]
- The 17α-derivatives, including methyltestosterone, methandrostenolone, norethandrolone, fluoxymesterone, danazol, oxandrolone, and stanozol, resist liver metabolism, making them orally active. However, this modification is associated with significant liver toxicities.[13]
- Modifications of the A, B, or C rings—such as mesterolone, nortestosterone, methenolone, fluoxymesterone, methandrostenolone, northandrolone, danazol, nandrolone, and stanozol—achieve various objectives, including: a) slowing metabolism; b) enhancing affinity for the androgen receptor (e.g., 19-nortestosterone); c) resisting aromatization to estradiol (e.g., fluoxymesterone, 19-nortestosterone); and d) decreasing binding of metabolites to the androgen receptor (e.g., reduced metabolites of 19-nortestosterone, 19-nortestosterone). [14]

2.3.Progestogens

Progesterone is a naturally occurring steroid sex hormone produced by the ovaries. It interacts with specific receptors in the reproductive tract, mammary glands, and central nervous system.[15] Progesterone and its synthetic counterparts, progestins, have been used for decades in contraception, maintaining pregnancies at risk of miscarriage, treating postmenopausal symptoms, secondary amenorrhea, and abnormal uterine bleeding.[15] Over the past 30 years, progestins have been utilized to treat endometriosis and hormone-sensitive tumors, and in the last 15 years, they have been used in assisted reproductive technologies (ART) procedures. [16] Clinical studies have confirmed the efficacy of certain gestagens in managing cachexia and anorexia in cancer and AIDS patients.[17] The discovery of previously unknown targets for progestin action, such as membrane-associated progesterone receptors, xenobiotic transport proteins, mitochondrial pores, and checkpoint signaling pathway proteins, has opened up new clinical applications for progesterone and its synthetic analogues.[17]

The main pharmacological effects of progesterone, primarily mediated by nuclear receptors, include: endometrial secretory transformation, formation of thick and viscous cervical mucus, increased basal temperature, reduced activity of genital tract and uterine smooth muscles (tocolytic effect), activation of mammary gland secretory acini growth and induction of lactation, protein lipase stimulation, increased fat stores, increased basal and induced insulin levels and glucose utilization rate, liver glycogen accumulation, aldosterone production, hypoazotemia, and azoturia.[18] Progesterone also increases (in small doses) or suppresses (in large doses) gonadotropic hormone production in the pituitary gland. The pharmacological effects of progestins (progesterone analogues) are typically assessed through various preclinical studies in animals.[19] These studies include determining affinity for progesterone receptors, assessing gestagenic activity using endometrial transformation tests, pregnancy maintenance tests, and ovulation inhibition tests, evaluating androgenic activity by measuring the weight of the prostate or levator ani muscle, assessing anti-androgenic activity or feminizing activity in male rats, and analyzing glucocorticoid and antimineralocorticoid properties.[20]

2.4.Estrogens

Estradiol is commonly known as an endocrine product of the ovary, but many tissues have the ability to synthesize estrogens from androgens and use them in a paracrine or intracrine manner. [21] Additionally, organs like adipose tissue significantly contribute to the circulating pool of estrogens. Evidence suggests that extraglandular production of C18 steroids from C19 precursors plays a crucial role in both normal physiology and pathological states in both men and women.[22] The enzyme aromatase, which catalyzes the conversion of C19 steroids to estrogens, is present in various human tissues and cells, including ovarian granulosa cells, placental syncytiotrophoblasts, adipose and skin fibroblasts, bone, and the brain. Aromatase expression in adipose tissue and potentially the skin primarily accounts for the peripheral formation of estrogen and increases with body weight and age.[22] Sufficient circulating levels of the biologically active estrogen estradiol can be produced from the extraglandular aromatization of androstenedione to estrone, which is then reduced to estradiol in peripheral tissues, leading to uterine bleeding, endometrial hyperplasia, and cancer in obese anovulatory or postmenopausal women. [23]Extraglandular aromatase expression in adipose tissue and skin (by increasing circulating levels of estradiol) and bone (by increasing local estrogen concentrations) is vital in slowing the rate of postmenopausal bone loss. Moreover, excessive or inappropriate aromatase

expression has been observed in adipose fibroblasts surrounding a breast carcinoma, endometriosis-derived stromal cells, and stromal cells in endometrial cancer, resulting in increased local estrogen concentrations in these tissues. Elevated estrogen levels, whether systemically delivered or locally produced, will promote the growth of these steroid-responsive tissues. [24] Additionally, local estrogen biosynthesis by aromatase activity in the brain may play a role in regulating various cognitive and hypothalamic functions. The regulation of aromatase expression in human cells involves multiple promoters, which can be activated or inhibited by various hormones, adding complexity to estrogen biosynthesis in the body. [24]Aromatase expression is controlled by the classically located proximal promoter II in the ovary and a distal promoter I.1 (40 kilobases upstream of the translation initiation site) in the placenta. In the skin, the promoter is I.4, while in adipose tissue, two other promoters (I.4 and I.3) located between I.1 and II are also used in addition to the ovarian-type promoter II. [25] Promoter use in adipose fibroblasts shifts between promoters II/I.3 and I.4 upon treatment with PGE2 or glucocorticoids plus cytokines. Furthermore, the presence of a carcinoma in breast adipose tissue also causes a switch in promoter use from I.4 to II/I.3. Thus, there are complex mechanisms that regulate extraglandular estrogen production in a tissue-specific and state-specific manner.[25]

2.5.Phytosteroids

Plant steroids are a unique class of chemical compounds found throughout the animal and plant kingdoms. Glucocorticoids are steroidal agents used to treat inflammatory disorders; however, long-term use can lead to severe side effects. [26] To mitigate these undesirable consequences, research efforts aim to identify novel bioactive phytochemicals with therapeutic potential that have minimal or significantly reduced side effects. These plant steroids are classified into different categories based on their chemical structure, pharmacological activities, and the sources from which they are isolated. Modern clinical and preclinical studies have demonstrated the anti-inflammatory activity of plant-derived steroids, such as chemical constituents from Trigonella foenum graecum L. (Family: Fabaceae), Solanum xanthocarpum L. (Family: Solanaceae), Boswellia serrata Roxb. (Family: Burseraceae), Glycyrrhiza glabra L. (Family: Fabaceae), Commiphora mukul (Family: Burseraceae), and Withania somnifera (Family: Solanaceae). [27]There is significant potential for investigating the anti-inflammatory activity of plant steroids that are structurally similar to glucocorticoids in various inflammatory conditions. Further research is needed to explore more potent lead compounds with fewer side effects.[26,27]

2.6. Neurosteroids

Neurosteroids, such as 3α,5α-THPROG, are now recognized as highly selective and potent modulators of GABAA receptor-mediated neurotransmission. Their interaction with neuronal GABAA receptors is highly specific and is influenced by the receptor's subunit composition, local metabolism, and phosphorylation. [28] However, more research is needed to understand the relative impact of these mechanisms on inhibition within specific neuronal circuits. [28] The use of drugs that affect steroid synthesis and metabolism in behavioral and electrophysiological experiments has provided strong evidence that neurosteroids can act as paracrine messengers, locally influencing neuronal activity. Recent experiments have even shown that endogenous neurosteroids can affect GABA-mediated inhibition in well-perfused brain slice preparations. [29] Nevertheless, the spatial relationship between neurosteroid production and GABAergic synapses in regions associated with their neurobiological effects, as well as the gender specificity of this relationship, remain challenging areas for future research.[28,29]

3. Marketed formulation:

There are some steroid containing marketed formulation approved by FDA are listed below in table 1,

Table 1: Steroid containing Marketed Formulation approved by FDA.

Active Ingredient	Dosage Form	Concentration/Strength	Brand Name(s)
Hydrocortisone	Oral tablets	5 mg, 10 mg, 20 mg	Cortef
	Topical cream/ointment	0.5%, 1%, 2.5%	Cortizone
	Injectable solution	100 mg/2 mL	Solu-Cortef
Prednisone	Oral tablets	1 mg, 2.5 mg, 5 mg, 10 mg, 20 mg, 50 mg	Deltasone
	Oral solution	5 mg/5 mL	Prednisone

Active Ingredient	Dosage Form	Concentration/Strength	Brand Name(s)
			Intensol
Methylprednisolone	Oral tablets	2 mg, 4 mg, 8 mg, 16 mg, 32 mg	Medrol
	Injectable suspension	40 mg/mL, 80 mg/mL	Depo-Medrol
Dexamethasone	Oral tablets	0.5 mg, 0.75 mg, 1.5 mg, 4 mg, 6 mg	Decadron
	Injectable solution	4 mg/mL, 10 mg/mL	Decadron Phosphate
Betamethasone	Injectable suspension	6 mg/mL	Celestone
	Topical cream/ointment	0.05%	Diprolene

4. The behavior of Progestogens and Anabolic-Androgenic Steroids (AAS)

4.1Progestogens

George W. Corner and Allen M. Willard first isolated and characterized progesterone (Pregn-4-ene-3, 20-dione), while Ludwig Fraenkel later provided experimental proof of the endocrine function of the corpus luteum.[30,31,32] Progesterone, derived from the terms "progestin" (pro, meaning "for") and "gest" (pregnancy), is the most abundant hormone produced by the gonads. It is synthesized primarily by the corpus luteum, adrenal cortex, and placenta during pregnancy, while in men, it is produced by the testes and adrenal cortex.[33-40] Progesterone(Figure 1) has the chemical formula $C_{21}H_{30}O_2$ and a molecular weight of 314.469 g/mol. It appears as a white crystalline powder with a melting point of 126°C and is in a solid state. It is slightly soluble in water but soluble in ethanol, ether, and chloroform. Progesterone is optically active and exists as a mixture of stereoisomers. It is classified under BCS Class II, indicating low solubility and high

permeability. The volume of distribution ranges from 8 to 10 L/kg, and it has a protein binding rate of approximately 98%, primarily to albumin. The half-life of progesterone varies depending on the route of administration: 16-18 hours for oral, 22-26 hours for intramuscular, and 13-18 hours for subcutaneous. Food can influence the absorption of oral progesterone, which is why it is often recommended to take it on an empty stomach. [41-47]

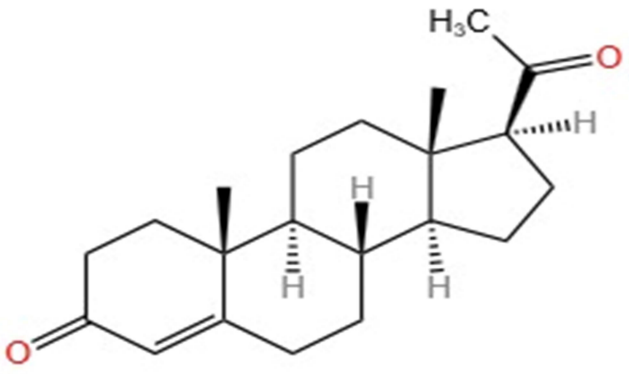

Figure 1. Progesterone

4.1.1 Mechanism of action of Progesterone

Progesterone produced by the gonads is primarily carried in the blood to perform its biological functions, while adrenal progesterone is largely converted into glucocorticoids and androgens. [48] It circulates in the bloodstream bound to cortisol-binding globulin and serum albumin, and has a short half-life of only five minutes. The liver metabolizes progesterone into sulfates and glucuronides, which are excreted in the urine. [49] Circulating progesterone can be converted to desoxycorticosterone (DOC) by renal 21-hydroxylation, especially during the luteal phase, pregnancy, or with exogenous progesterone administration, which may lead to unwanted side effects.[50] As a lipophilic molecule, progesterone easily crosses cell membranes and interacts with specific nuclear receptors (PR-B and PR-A), activating about 300 co-regulators that influence ribosomal RNA and protein production, making it a key regulator of female reproduction.[51] Genomic receptors PR-B and PR-A share parts of the DNA binding and ligand binding domains but differ in amino acid sequence, with PR-A being shorter. [52] Cells

expressing PR-B and PR-A are equally represented under physiological conditions, but PR-C is abundant in myometrial tissue as shown in figure 2. Progesterone is essential not only for reproductive health but also shows potential in managing conditions such as Alzheimer's disease, cerebral edema, osteoporosis, and diabetic neuropathy. [53]

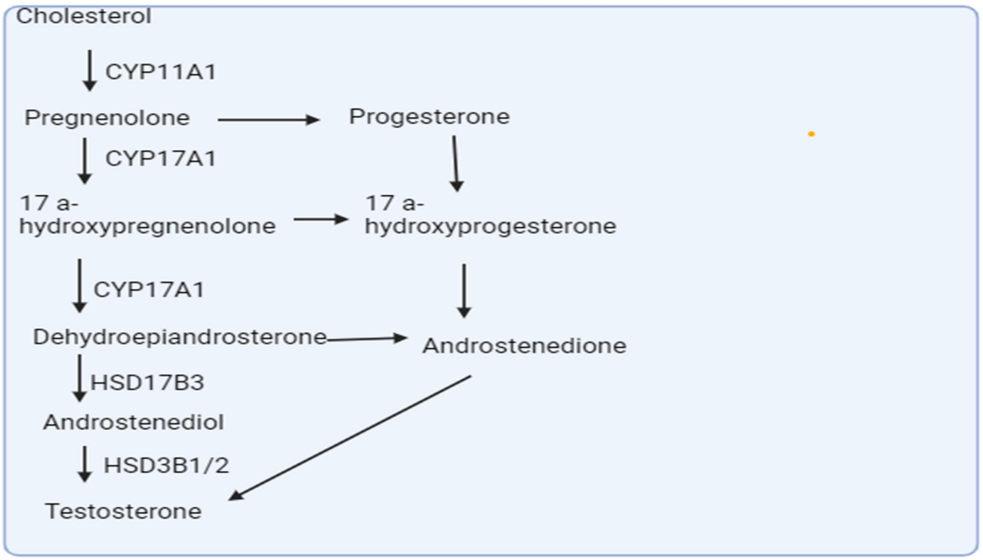

Figure 2. MOA of Progesterone

4.2. Anabolic-Androgenic Steroids (AAS)

An androgenic steroid hormone named Testosterone play a vital role in regulating the metabolic process of human individuals. It play a numerous of primary function particularly in male individual like muscular development, adipose tissue distribution,Sexual drives, Spermatogenesis, Erythrogenesis.[54]

Testosterone (Figure 3), with the chemical formula $C_{19}H_{28}O_2$ and a molecular weight of 288.4244, appears as a white crystalline substance. Its melting point ranges between 152–155°C. Testosterone is freely soluble in ethanol but practically insoluble in water and fatty oils. It has a specific optical rotation of +107.3 and is classified as a Class II compound under the Biopharmaceutics Classification System (BCS). The volume of distribution of testosterone in elderly men is approximately 80.36 ± 24.51 L. About 40% of testosterone is bound to sex hormone-binding globulin (SHBG), 2% remains unbound, and the remainder binds to albumin and other proteins. The half-life of testosterone is highly variable, ranging from 10 to 100

minutes. Food has no significant interaction with testosterone. It‘s chemical structure is shown below.

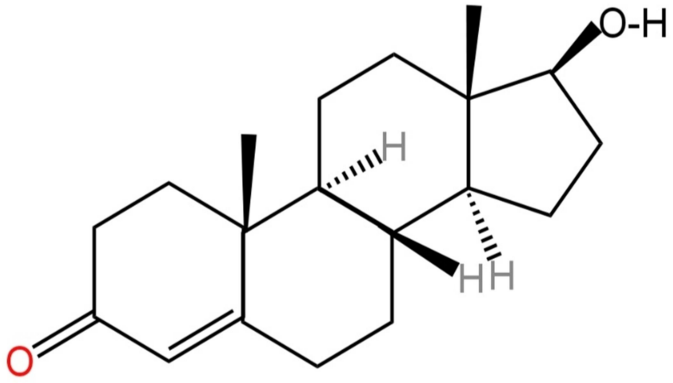

Figure 3 : Structure of Testosterone

4.2.1 Mechanism of action of testosterone:

Biologically this vary androgenic hormone is produced by chemical co-ordination in the body. These are often controlled by the active feedback regulation process by the special hormone named luteinizing hormone (LH) by the anterior pituitary gland. As per the normal human physiology the age after preadolescent age this next puberty age groups starts which is also considered as initial stages of spermatogenesis.[55] At this stages the peak level of hormonal changes is reported which is often regulated by the two specific hormones named LH(Luteinizing Hormone) and FSH(Follicular Stimulating Hormone) regarding Spermatogenesis(as shown in figure 4). As per the medical steroidal deficiency survey report it was well evaluated, The well named condition is called as hypogonadism (means the low testosterone level inside the body), is well categorized according to its origination i.e if this vary condition is due to inefficient chemical co-ordination (origin through anterior pituitary gland) it is named as secondary problem, otherwise if problem arise through the male sex organ i.e testis this is consider as primary problems.[56] Apart from this there are several comorbidities problems also which is aroused due to low deficiency testosterone like Gene based disorder such as turner’s disorder, Autoimmunological conditions such as Hypoparathyroidism, Apart from these many physical stimuli also results in the steroidal deficiency like over exposure to chemical like heavy metals , heat radiation, Chemotherapy and Tumours.[57] As men age, testosterone

levels gradually decline, accompanied by a disruption in the normal diurnal rhythm of testosterone production, which can also lead to clinical manifestation.[58]

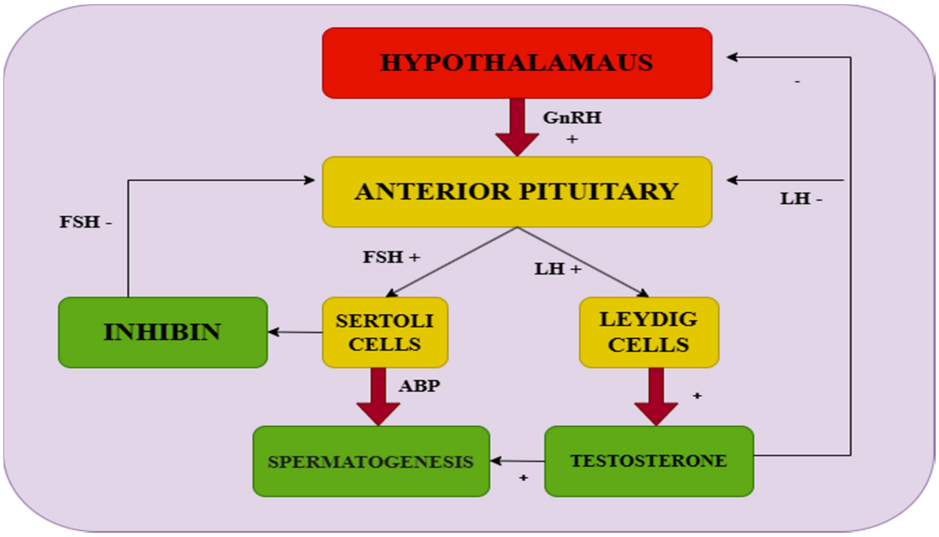

Figure 4: MOA of Testosterone

5. Market formulations of progesterone and testosterone

Table 2. Market formulations of Progesterone

Form	**Brand Name**	**Dosage/Composition**
Oral Capsules	Prometrium	100 mg
	Utrogestan	100/200 mg
Vaginal Inserts and Gels	Crinone (Gel)	8%
	Endometrin (Vaginal Insert)	100 mg
	Prochieve (Gel)	4% and 8%
Injectable Forms	Progesterone in Oil	500 mg/10 mL
	Gestone	Capsule: 400 mg, Injection: 100 mg
Intrauterine Devices (IUDs)	Mirena (Levonorgestrel, synthetic progestogen)	52 mg
	Kyleena (Levonorgestrel)	19.5 mg
Combination Packs	Estrogel Propak	Micronized Progesterone capsules and

Form	Brand Name	Dosage/Composition
		estradiol gel
Other Forms	Provera (Medroxyprogesterone Acetate)	10 mg tablet
	Duphaston (Dydrogesterone)	10 mg tablet
Vaginal Suppositories	Cyclogest	Also available as an oral tablet
	Lutinus	-
Injectable Forms	Proluton Depot	250 mg
	Depo-Provera (Medroxyprogesterone Acetate)	150 mg/mL
Combination Hormone Therapies	Femoston	Dydrogesterone and Estradiol
	Angeliq	Drospirenone and Estradiol

Table 3: Marketed Formulation of Testosterone

Dosage Form	Brand Name(s)	Dosage/Composition
Buccal tablet	Striant	30 mg
Oral capsule	Jatenzo, Kyzatrex, Tlando	40 mg
Nasal gel	Natesto	5.5 mg/actuation
Topical gel	AndroGel, Testim, Fortesta, Vogelxo	1% & 1.62%
Transdermal patches	Androderm	2–4 mg/patch
Subcutaneous	Testopel	75 mg/pellet

Dosage Form	Brand Name(s)	Dosage/Composition
implant		
Injectable solution	Delatestryl, Xyosted	200 mg/mL
	Depo-Testosterone	100–200 mg/mL
	Nebido, Aveed	250 mg/mL
	Sustanon 250	250 mg/mL

6. Future perspectives

Steroid hormones, including androgens, estrogens, and corticosteroids, play essential roles in regulating numerous physiological processes in the body, such as metabolism, immune function, reproduction, and stress response. These hormones are synthesized from cholesterol and act on various tissues through specific receptors, influencing processes from bone health to cardiovascular function.[59] The regulation and maintenance of normal steroid hormone levels are critical for overall health, and any imbalance can lead to a range of medical conditions such as osteoporosis, infertility, diabetes, and autoimmune diseases. In this context, the role of steroid hormone therapies (SHTs) becomes increasingly important in both clinical practice and emerging treatment paradigms.In the future, the approach to steroid hormone therapies is likely to evolve significantly due to advances in biotechnology, genomics, and personalized medicine. One major trend is the growing focus on individualized hormone replacement therapies (HRTs).[60] Currently, many steroid hormone therapies, such as those used for menopause or testosterone replacement in men with low levels, are standardized, often leading to suboptimal results for some patients. Personalized medicine, based on genetic and phenotypic profiling, promises to create tailored treatments that take into account individual variations in hormone receptors, metabolism, and responses to medications.

7. Conclusion

Steroid hormones are integral to human health, and maintaining their balance is crucial for normal physiological functioning. With advancements in medical science, the future of steroid hormone therapy holds tremendous promise. Personalized and precision medicine will ensure more effective and safer treatments, tailored to individual genetic makeup. Additionally, new therapeutic approaches, including gene therapy and synthetic analogs, will allow for more targeted interventions, reducing side effects and offering longer-term solutions for those with chronic hormone deficiencies or imbalances.Despite these advancements, challenges remain, such as addressing the complexities of hormone regulation and ensuring accessibility to these innovative therapies. The development of safer, more effective steroid hormone therapies will continue to evolve, offering a brighter future for individuals affected by hormonal disorders and helping maintain optimal health for the broader population.

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