Research Article

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Sex Steroid Hormones Regulate the Function of the Sexual Organs

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Abstract

A hormone is a type of chemical that cells release into bodily fluids to influence the actions of other cells. Endocrine glands primarily secrete various cystic hormones. The endocrine system works alongside the nervous system to help the body function smoothly and in a coordinated manner. Hormones travel through the bloodstream, reaching various organs and tissues to perform their functions. They manage essential body activities like growth, reproduction, tissue renewal, metabolism, stress adjustment, and bone strength, among many others. Steroid hormones share a chemical structure with cholesterol and are typically derived from it. They are produced by glands like the adrenal cortex, ovaries, testes, and placenta.

Keywords: Hormone, Steroid, Sexual Function, Puberty, Health

Introduction

It's widely recognized that the main function of sex steroid hormones is their contraceptive ability [1]. Nevertheless, they can also serve therapeutic roles in various clinical situations, providing significant benefits beyond birth control. Therapeutic advantages can include suppressing ovulation, and exhibiting antiestrogenic, antiandrogenic, and progestin actions. The positive effects of progestins can be amplified by the presence of estrogen; for instance, higher doses of EE, such as 30 µg, can enhance the antiandrogenic effects of certain progestins. Reducing the estrogen dosage has made estrogen-progestin treatments easier for patients to tolerate by lessening side effects along with their influence on liver metabolism and the risk of blood clots. Hormonal therapy that uses estrogen and progestin is one of the most crucial and adaptable treatments available, providing immediate relief for issues like seborrhea, dysmenorrhea, acne, and hirsutism in women. The long-term protection against several cancers, especially ovarian cancer, is also significant. Conditions related to menstrual cycles, such as dysmenorrhea, endometriosis, lack of ovulation, PCOS, and abnormal uterine bleeding, often improve with estrogen-progestin therapy. After a thorough medical evaluation, it has long been established that the prescription of these treatments carries fewer risks for women compared to their benefits.

Role

In many species, the functions of sex steroid hormones are well established—estrogen encourages and progesterone hinders the processes that lead to parturition [2]. The withdrawal of progesterone, meaning the removal of this hormone, directly signals the onset of parturition. Additionally, administering progesterone in certain species can postpone parturition due to reduced myometrial activity and sustained cervical competency. However, in humans, it appears that both estrogen and progesterone are part of a larger molecular system that keeps the uterus calm. During a normal pregnancy, the levels of estrogen and progesterone in the blood are extremely high and far exceed the affinity constants for their receptors. Therefore, it is challenging to understand how minor adjustments in their concentration ratios could influence physiological functions during pregnancy. Nonetheless, the evidence suggesting that a higher progesterone-to-estrogen ratio is crucial for sustaining pregnancy and that a decrease in this ratio facilitates parturition is compelling. Across all species examined, including humans, the administration of progesterone-receptor antagonists such as mifepristone (RU-486) or onapristone encourages various aspects of parturition, such as cervical ripening, increased cervical distensibility, and heightened uterine sensitivity to uterotonins.

The specific role of estrogen in regulating uterine calmness and cervical competency in humans is not entirely clear. Nevertheless, estrogen can enhance the responsiveness to progesterone, thus promoting uterine calm. Towards the end of pregnancy, estrogen supports the processes that facilitate uterine activation and cervical ripening.

Both progesterone and estrogen attach to nuclear receptors that control gene transcription in a way that is specific to each cell type and situation. Estrogen has two nuclear receptors: estrogen receptor α (ERα) and estrogen receptor β (ERβ). Different transcripts derived from a single gene encode the nuclear receptor isoforms for progesterone (PR-A and PR-B).

The Uterine Endometrium

The uterine endometrium serves as a target for sex steroids and undergoes a coordinated sequence of exposure to circulating estrogen, progesterone, and progesterone withdrawal, which occurs due to the regression of the corpus luteum when there is no pregnancy [3]. The staggered exposure to estrogen and progesterone from the cyclical activity of the ovaries leads to repeated cycles of cell growth and differentiation, accompanied by regular menstrual bleeding. The proliferative phase of the endometrial cycle aligns with the ovarian follicular phase. The secretory phase of endometrial differentiation is governed by progesterone and coincides with the ovarian luteal phase. During the follicular phase, estrogen released from the dominant follicle stimulates the regeneration and growth of the endometrium while increasing the levels of both estrogen and progesterone receptors. The mid-secretory phase represents the peak exposure to circulating progesterone and is commonly known as the "implantation window."

Endometrial expressions of sex steroid receptors enable the endometrium to react to ovarian oestradiol and progesterone. When the endometrium encounters these sex steroids, which act through their specific receptors (ERs and PRs), it triggers a series of gene expressions necessary for getting the endometrium ready for the possibility of successful implantation. Without pregnancy, the corpus luteum shrinks, leading to reduced progesterone levels, and this progesterone withdrawal starts the chain of events that results in the shedding of the upper layer of the endometrium.

Menstruation is a key reproductive function in women and has all the characteristics of an inflammatory response. Regeneration of the endometrium, which is as crucial as its shedding, begins 36 hours after bleeding starts and usually wraps up by days 5 to 6 of the cycle. The exact processes involved in endometrial repair are not yet fully understood, but they likely resemble wound-healing mechanisms. Therefore, the repair of the endometrium will include overlapping stages of inflammation, its resolution, tissue formation, remodeling, and angiogenesis. The ability of the human endometrium to regenerate efficiently every month suggests that adult progenitor cells are present in the local endometrial environment.

Sexual Function

Normal male sexual functioning encompasses four key elements: (1) having a healthy libido; (2) the ability to achieve and sustain an erection; (3) ejaculation; and (4) the process of detumescence [4]. Libido, defined as sexual desire, is influenced by several factors including visual, olfactory, tactile, auditory, imaginative, and hormonal signals. Hormones like testosterone play a crucial role in boosting libido, but it can be negatively affected by hormonal imbalances, mental health issues, or certain medications.

The process of achieving an erection involves increased blood flow into the lacunar network, resulting from the complete relaxation of the arteries and smooth muscle in the corpora. The corpora structure consists of smooth muscle (trabecula) interwoven with a network of vessels (lacunar spaces) lined with endothelium. As the trabecular smooth muscle compresses against the tunica albuginea, it leads to the closure of the emissary veins, trapping blood within the corpora. When a full erection occurs and the valvular mechanism is functioning properly, the corpora transform into noncompressible cylinders, preventing blood from escaping.

The central nervous system plays a significant role by either enhancing or inhibiting the spinal pathways responsible for erectile function and ejaculation. The erectile response is a result of both central (psychogenic) and peripheral (reflexogenic) nervous system activity.

Sensory nerves from the receptors located in the penile skin and glans merge to create the dorsal nerve of the penis, which connects to the S2-S4 dorsal root ganglia through the pudendal nerve. Parasympathetic fibers that target the penis originate from neurons in the intermediolateral columns of the S2-S4 sacral spinal segments. In contrast, sympathetic nerves come from the spinal segments T-11 to L-2, descending via the hypogastric plexus.

The role of neural input in modulating the tone of smooth muscle is vital for both initiating and maintaining an erection. Additionally, a complex interaction occurs between the corporal smooth muscle cells and their associated endothelial lining. Nitric oxide, which promotes blood vessel relaxation, is essential for achieving an erection and is balanced by endothelin-1 (ET-1), which causes vessel contraction. Nitric oxide is generated from L-arginine by nitric oxide synthase and is released by the nonadrenergic, noncholinergic (NANC) autonomic nerve fibers to act on smooth muscle cells after the junction. This compound helps increase levels of cyclic 3',5'-guanosine monophosphate (cyclic GMP), leading to the relaxation of smooth muscle. Phosphodiesterase type 5 (PDE-5) gradually breaks down cyclic GMP. Oral medications like sildenafil inhibit PDE-5, helping to sustain erections by limiting the breakdown of cyclic GMP. However, if nitric oxide is not produced at all, PDE-5 inhibitors will not work effectively because these drugs facilitate rather than initiate the enzymatic process. Besides nitric oxide, vasoactive prostaglandins are also

produced within the cavernosal tissue, which elevate cyclic AMP levels and contribute to the relaxation of the cavernosal smooth muscle.

Cancer Risk

Research has shown that hormonal contraception can reduce cancer rates overall [1]. Specifically, prolonged use of hormonal contraceptives has been associated with a decrease in the incidence of certain cancers, including colorectal, endometrial, and ovarian, among others. Additionally, the drop in the rate of endometrial, ovarian, and colorectal cancer lasts for a minimum of 30 years following the stop of COC use. For endometrial cancer, the protective effects brought on by estrogen-progestin, progestin-only, or intrauterine methods seem to depend on the duration of therapy and remain after treatment ends. This positive impact stems from the antiproliferative effects on the endometrium, lowering the chances of both endometrial hyperplasia and cancer. Furthermore, the risk reduction of malignant and borderline ovarian epithelial carcinoma, aside from mucinous epithelial carcinoma, correlates with how long hormonal contraceptives are used and continues for over 30 years. Oral estrogen-progestins create the most substantial effect by inhibiting ovulation, suppressing the secretion of gonadotropins, and decreasing retrograde menstruation. Prolonged use of LNG-IUD has also been shown to protect against this cancer type.

Fibroids

More insights exist regarding managing the growth of uterine fibroids than about the causes of these non-cancerous tumors [5]. Growth factors play a crucial role in regulating fibroid growth and their makeup. Increased levels of the angiogenic fibroblast growth factor have been detected in fibroids when compared with the nearby myometrium. Moreover, the roles of transforming growth factor β, granulocyte-macrophage colony-stimulating factor, and epidermal growth factor (EGF), among others, have shown distinct differences between fibroid tissue and normal myometrium.

Given that fibroids have not been found in prepubescent girls and tend to shrink during menopause, it has commonly been believed that their presence relies on the sex steroids estrogen and progesterone. This belief has led much fibroid research to focus on these hormones in an effort to create new medical treatments for these tumors.

Sex steroids function through receptors. The steroid attaches to the receptor, which then moves into the cell's nucleus. Research has indicated that the concentration of steroid receptors is higher in fibroids compared to surrounding myometrium and that the availability of these receptors is affected significantly by agents that change circulating estrogen levels. Further studies have explored the connection between steroid hormones and growth factors, such as EGF and insulin-like growth factor, which may serve as mediators for estrogen's effects. In contrast, the role of progesterone remains less defined. There are more progesterone receptors in fibroids than in the nearby myometrium. Similar to estrogen, progesterone also influences EGF receptor levels and inhibits apoptosis. Research utilizing antiprogestins, progesterone receptor modulators, and administering progestogens to women with low estrogen suggests that progesterone could encourage fibroid growth. Nonetheless, the specific roles of estrogen and progesterone in such growth are still not fully understood.

POI

Patients diagnosed with primary ovarian insufficiency, or POI, receive hormone replacement therapy that aims to imitate the natural hormone levels typical for their age group [6]. Transdermal estrogen is a commonly recommended option for hormone replacement therapy because it delivers serum estradiol levels that are closest to what is found in a healthy physiology, and it is considered safer than oral options since it skips liver metabolism. This approach reduces the chances of altering clotting factors.

The objectives of long-term hormone replacement are to achieve normal adult levels of sex steroids, which help maintain bone density and prevent endothelial dysfunction, both of which are important for reducing cardiovascular disease risk. For patients experiencing delayed puberty or primary amenorrhea, there are additional immediate goals, such as developing secondary sexual characteristics and promoting a growth spurt to reach their potential height.

Patients dealing with the emotional and psychological effects of POI can face considerable challenges. This aspect becomes particularly critical when they first learn about their diagnosis. Menstrual irregularities can lead to heightened risks of anxiety and depression, and the loss of natural fertility can be particularly hard on young women, potentially worsening mood disorders.

Another key point in managing POI, especially for teens and young adults, is the importance of contraception. Since ovarian function is diminished but not completely absent, women with POI may still have unexpected pregnancies. Ironically, hormone replacement therapy could enhance fertility for unpredictable periods. Therefore, those who think they are not at risk for getting pregnant might end up with unplanned pregnancies.

Unfortunately, hormonal contraceptives like combined oral contraceptives may not be very effective, as these work by suppressing the hypothalamic-pituitary-ovarian axis. Thus, there should be a strong recommendation for consistent use of barrier methods like condoms and consideration of long-acting reversible contraceptives such as intrauterine devices (IUD), including both the levonorgestrel and copper types. The levonorgestrel IUD not only provides contraception but also protects the endometrium by offering an intrauterine source of progestin.

Conversely, some patients may express a desire for fertility preservation options. It is important to understand that most patients cannot access oocyte cryopreservation due to

very high follicle-stimulating hormone (FSH) levels. Freezing oocytes necessitates the stimulation of ovaries to produce follicles, something that these menopausal FSH levels generally prevent. However, given that ovarian function in individuals with POI can be variable and sporadic, fertility preservation may still be a possibility for some. Patients interested in this should be referred to reproductive endocrinology and infertility specialists if they wish to explore their options.

Puberty

Precocious puberty is when the body starts developing earlier than the usual age for puberty [7]. For girls, this occurs if they begin to show secondary sexual traits before age 8 in Caucasian girls and before age 7 in African-American and Hispanic girls. Girls are more frequently affected by precocious puberty compared to boys. Many girls who display signs of puberty between 6 and 8 years old have a mild form that does not need any treatment. Being overweight can lead to earlier puberty.

Central precocious puberty, which depends on gonadotropin-releasing hormone (GnRH), happens when the GnRH pulse generator in the hypothalamus activates, leading to increased gonadotropin levels and, as a result, more sex hormones. The hormonal changes and physical developments in central precocious puberty mirror those seen in typical puberty. In most cases, the cause of central precocious puberty in girls is unknown, but it can stem from central nervous system (CNS) issues that interfere with the normal control of the GnRH pulse generator. These CNS problems include hypothalamic hamartomas, brain tumors, cranial radiation, hydrocephalus, and injuries. On the other hand, peripheral precocious puberty occurs regardless of gonadotropin secretion. In girls, this can result from ovarian or adrenal tumors, ovarian cysts, late-onset congenital adrenal hyperplasia, McCune-Albright syndrome, or exposure to outside estrogen. McCune-Albright syndrome consists of a combination of café au lait spots, fibrous dysplasia in multiple bones, and GnRH-independent precocious puberty. It is caused by a mutation that activates the gene for the α-subunit of Gs, a G protein that triggers adenyl cyclase. Endocrine cells with this mutation produce hormones excessively due to autonomous hyperfunction.

Delayed Puberty

Delayed puberty occurs when early signs of development do not appear within the expected age range for the population, typically 12 to 13 years for girls [8]. The main reason for delayed puberty is a slowdown in GnRH secretion, which subsequently affects the release of FSH and LH, leading to a delay in sex steroid production, a condition often referred to as constitutional delay of growth and puberty. This situation is generally temporary. If GnRH secretion does not return after several years, it may result in secondary hypogonadism. Another potential cause could be gonadal atresia or atrophy, which relates to primary hypogonadism, characterized by low levels of sex steroids alongside elevated FSH and LH levels. Various gonadal issues, such as Turner syndrome, Klinefelter syndrome, or damage from treatments like chemotherapy and radiotherapy, can lead to this condition.

For delayed puberty cases where an underlying issue cannot be resolved, short-term hormone treatment is recommended. In girls, the preferred method is administering low-dose estradiol either orally or through the skin. Typically, very small amounts of 17-beta estradiol are used, well below the doses for hormone replacement therapy. The usual starting dose is 6.25 mcg of 17-beta estradiol daily (if only higher-dose patches are available, they can be cut to achieve a lower dose). After six months, if the treatment is tolerated well, the dosage can be raised to 12.5 mcg per day. By the 18-month mark, this can be increased to 25 mcg daily, and after two years of treatment (or once there is intermenstrual bleeding with estradiol alone), 200 mg of progesterone can be added alongside the current dose of estradiol from days 1 to 12 of each cycle. It's vital to avoid adding progesterone if breast development hasn't started yet since early initiation may affect final breast growth. Patients over 18 who have not had periods likely have a congenital GnRH deficiency, and hormone replacement involving estrogen and progesterone should be initiated, similar to women with primary ovarian failure.

Activation

The puberty process begins with the activation of the hypothalamic GnRH pulse generator, leading to the pulsatile release of GnRH [9]. This action triggers an increase in the levels of LH and FSH in the plasma, along with sex steroids, which include testosterone for boys and estradiol for girls.

In the early phases of puberty, there is a noticeable increase in serum LH levels during nocturnal sleep. As puberty progresses, LH levels during the day also rise, eventually establishing the adult pattern of pulsatile release every 90 minutes throughout both day and night.

Various neuronal and hormonal signals that are not entirely understood likely influence the activation of the GnRH pulse generator and the beginning of puberty.

It has been suggested that reaching a certain body weight or body composition is essential for puberty to begin. Leptin, a hormone mainly produced by fat cells, may serve as an indicator of the metabolic energy needed for the onset of puberty.

Kisspeptin is a neuropeptide found in the hypothalamus that interacts with the GPR54 receptor. The signalling pathway involving Kisspeptin and GPR54 has been identified as critical for the onset of puberty. When there are mutations in GPR54, this can lead to a condition in humans called autosomal recessive hypogonadotrophic hypogonadism.

Additionally, about 18 months prior to the start of central puberty, the zona reticulosa of the adrenal gland enlarges. This is followed by the release of adrenal androgens. The

clinical implications of adrenarche are still not completely clear.

Conclusion

Steroid hormones, which include estrone, progesterone, and testosterone, are also referred to as sex hormones. They play a key role in regulating the functions of both primary and secondary sexual organs. While testosterone is important for sperm production, estrogen and progesterone are crucial in women for managing the menstrual cycle and supporting fertilization and pregnancy. Although hormones constitute a small portion of our bodies, their impact is significant.

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