

## Male reproductive endocrinology

James Adams

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Adams J. Male reproductive endocrinology. *J Endocr Disord Surg.* 2023;7(1):9-10.

### ABSTRACT

The testes produce sperm, which is necessary for male fertility, and testosterone, which is essential for the growth and maintenance of many

physiological processes. Endocrine hormones made locally in the testis as well as centrally in the brain and pituitary control the synthesis of both products.

**Key Words:** *Testosterone; Endocrine; Hormones*

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### INTRODUCTION

While testosterone is necessary for the production of sperm, Follicle-Stimulating Hormone (FSH) is also required for the best possible testicular growth and maximum sperm production. Spermatogenesis, an incredibly intricate and dynamic process that involves cooperation between various testicular cell types, produces sperm. Although the regulation of spermatogenesis by testosterone and FSH has long been understood, years of study have revealed many of the complex mechanisms by which spermatogonial stem cells transform into highly specialized, motile spermatozoa. As well as numerous paracrine and growth factors, tightly regulated gene and protein expression programs, epigenetic genome modifications, and various non-coding RNA species, spermatogenesis includes coordinated interactions of endocrine hormones.

The testis seminiferous tubules are where spermatogenesis occurs. These tubules connect to a network of anastomosing tubules known as the rete testis by passing into the mediastinum of the testis and forming lengthy, convoluted loops. Spermatozoa leave the testes through the rete and move into the efferent ductules before passing through the epididymis and reaching their full maturity there. The somatic Sertoli cells and the developing male germ cells at different stages of development make up the seminiferous tubules' seminiferous epithelium. Basement membrane and layers of peritubular myoid cells, modified myofibroblastic cells, surround the seminiferous epithelium. The interstitial area between the tubules is home to steroidogenic Leydig cells, lymphatic and blood arteries, and immune cells such as macrophages and lymphocytes.

Recently, gonadotropin receptor mutations in human cases were found. These results have clarified the molecular pathogenesis of some disturbances in the reproductive function, despite the fact that they are extremely uncommon. Male-limited, early-onset, gonadotropin-independent premature puberty (testotoxioicosis), which is caused by an activating mutation of the LH receptor (R) gene, appears to be unaffected in women who have the same

mutation. The patient displayed testicular Leydig cell adenomas if the activating LHR mutation activates the inositol trisphosphate cycle, as was discovered with one specific mutation. By highlighting the potential role of gonadotropins as tumor promoters, this result lends support to a theory that has been advanced in light of some clinical findings regarding gonadal malignancies. The involvement of gonadotropins in the development of gonadal tumors in gene-modified mice, such as inhibin-KO mice with high FSH levels, LH overexpressing mice, and those expressing the viral oncogene SV40 T-antigen, has also been studied. Such models provide excellent opportunities for further investigation of this possibly significant but poorly understood gonadotropin action.

Human LHR inactivating mutations impair male sex differentiation, which can vary from mild under virilization to complete lack of genital masculinization, depending on the severity of the receptor inactivation. Females have a weaker phenotype that is only associated with anovulatory infertility. In contrast to the corresponding receptor mutation, the phenotype of a single man with an inactivating mutation of the LH gene differs in that the subject was normally masculinized at birth but did not experience sexual maturation.

The LHR mouse model, which was just recently created, provides for an intriguing comparison between the effects of LH and hCG on male and mouse sexual differentiation. The male LuRKO mice were typically masculinized at birth, just like the male patient with inactivated LH, but they did not exhibit postnatal sexual maturation. Therefore, unlike in humans, a functional LHR is not required for the rodent fetus to produce enough testicular androgen to cause masculinization. In the lack of LH action, it appears that other blood-borne or paracrine factors can sustain adequate androgen production. It is interesting that there is such a significant hormonal variation between rodents and humans in how masculinization is regulated.

Studies on the impacts of lifestyle and environmental variables on the male reproductive potential have been sparked by reports of the rising

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*Editorial Office, Department of Endocrine Disorder and Surgery, United Kingdom*

*Correspondence: James Adams, Editorial Office, Department of Endocrine Disorder and Surgery, United Kingdom; E-mail: jamesadam93050@gmail.com*

*Received: 05-Jan-2023, Manuscript No. puljeds-23-6258; Editor assigned: 12-Jan-2023, Pre-QC No. puljeds-23-6258 (PQ); Reviewed: 17-Jan-2023, QC No. puljeds-23-6258 (Q); Revised: 20-Jan-2023, Manuscript No. puljeds-23-6258 (R); Published: 01-Feb-2023, DOI: 10.37532/puljeds.2023.7 (1).09-10.*

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## Adams

incidence of male infertility coupled with decreasing semen quality. Numerous endogenous and exogenous factors have the ability to increase the creation of Reactive Oxygen Species (ROS) in excess of what the body's antioxidant defences can handle, leading to oxidative stress. The Hypothalamus-Pituitary-Gonadal (HPG) axis is adversely impacted by oxidative stress, and this can lead to infertility either directly or indirectly by interfering with the HPG axis' ability to communicate with other hormonal axes.

Through enzymatic (superoxide dismutase, catalases, and peroxidases) and non-enzymatic (vitamins, steroids, etc.) processes, antioxidants protect against excessive ROS levels. Oxidative Stress (OS), which places the body's cells under stress, happens when the balance between oxidants and antioxidants leans toward the oxidants. As a consequence, too much ROS can cause lipid peroxidation and interfere with the functions of DNA, RNA, and proteins in spermatozoa and other testicular cells. High ROS levels can increase the likelihood of infertility both directly by causing OS and implicitly by influencing the hypothalamic axes that control the release of hormones. Infertility is brought on by ROS, which lower male sex hormone levels and disturb the hormonal equilibrium that controls male

reproductive function. These "endocrine disruptors" obstruct cross-talk between the Hypothalamic-Pituitary-Gonadal (HPG) axis and other hypothalamic hormonal axes as well as contact between the testis and the hypothalamic-pituitary unit.

Reactive Oxygen Species (ROS), which have at least one oxygen atom and are short-lived, unstable, and extremely reactive, can steal electrons from other molecules to become an electronically stable state. The other molecule drops an electron during this process, which results in the formation of a new radical. This radical then interacts with a nearby molecule, passing on its radical status through a process known as a "radical-chain reaction," up until two radicals interact with one another to create a stable bond. The degree of changes in the cellular structures is amplified by these processes.

One of the more prevalent conditions affecting the pituitary system is pituitary tumors. Although almost all of these tumors are benign (noncancerous), they can still result in hormonal abnormalities that affect general health by increasing or decreasing hormone production. Not all tumors will exhibit signs, but once they are identified it is crucial that the patient go through a thorough examination by a qualified team to stop the condition from getting worse.