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Commentary

The Imapct of Internal Structures on the Male Reproductive System

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DESCRIPTION

The male reproductive system consists of internal structures: Testicles, epididymis, and vas deferens, prostate and external structures: Scrotum and penis. These structures line the blood vessels well with many glands and vessels to promote sperm formation, storage and ejaculation for fertilization and to produce androgens important for male development. The main male androgen is testosterone, which is produced by the laryngeal cells in the testicles. Testosterone can be converted to a more active form by dihydrotestosterone or aromatase by 5-alpha-reductase. Other vital hormones include inhibitory B and the Mullerian Inhibitory Hormone (MIH), both produced by testosterone cells. The important hormones that modulate these are Follicle-Stimulating Hormone (FSH) and Luteinizing Hormone (LH), which are released from the anterior pituitary gland and regulated by Gonadotropin-Releasing Hormone (GnRH) produced by the hypothalamus. Together, these hormones form the hypothalamic-pituitary-gonadal axis, which promotes and maintains sexual development and function in men.

Testosterone plays an important role in stimulating the development of Wolffian vessels in the male fetus, turning it into testes, epididymis, vas deferens and seminal vesicles. Testosterone is also responsible for atherosclerosis, puberty, increased bone density, closure of epiphyseal plates, deepening of the voice, increase in muscle mass, and anatomy and libido. In addition, testosterone is converted to Dihydrotestosterone (DHT) by 5-alpha-reductase, an enzyme produced by the prostate gland. Both DHT and testosterone bind to the same androgen receptors intracellularly, but have a high affinity for DHT. DHT aids in the growth of the prostate, scrotum, and penis. Male hair pattern (facial, axillary, and pubic hair), as well as the pathology of male pattern baldness, increased sebaceous gland secretion, and acne, are all caused by DHT. These hormones work together to promote puberty and the maintenance of the male reproductive system.

The anterior pituitary responds by secreting LH and FSH into the bloodstream. These gonadotropic hormones bind to membrane receptors in the Leydig and Sertoli cells of the testis. Both hormones come from the same glycoprotein family and have the same alpha subunits, but their different beta-subunits distinguish their function. Both exert their physiological effects by binding and activating the G protein receptor that activates adenocyl cyclase and stimulates cellular cAMP levels to stimulate serotonin and Leydig cells. LH stimulates Ledig cells in the testicular interstitium to produce testosterone from cholesterol. LH promotes desmolase, the initial rate-limiting enzyme that converts cholesterol into pregnancynolone. It produces two key weak androgen mediators: Dehydroepiandrosterone (DHEA) and androstenedione. The enzyme 17-beta-hydroxysteroid dehydrogenase converts androstenedione to testosterone. In the hypothalamus and anterior pituitary, testosterone lowers LH and FSH secretion by negative feedback. Serotonin cells, which are found at the edge of the testicular seminiferous tubules, are similarly affected by testosterone. FSH and testosterone can stimulate serotonin cells to release Androgen-Binding Protein (ABP), which delivers testosterone to germ cells during spermatogenesis. FSH stimulates Sertoli cells to stimulate sperm production and release inhibin B and MIS. Inhibin acts as a negative feedback regulator of serotonin cells on the hypothalamic-pituitary system to reduce FSH release.

Although most of the testosterone production in men comes from the leydig cells in the testicles, the adrenal cortex contributes to some androgen production. Like the hypothalamic-pituitary-gonadal axis, the adrenal glands are regulated by the hypothalamus and anterior pituitary to form the hypothalamic-pituitary-adrenal axis. The hypothalamus secretes Corticotropin-Releasing Hormone (CRH), which causes the anterior pituitary to secrete Adrenocorticotropic Hormone (ACTH). The ACTH enzyme desmolase induces the conversion of cholesterol in the adrenals into pregnancy, similar to testosterone synthesis in the testes. The adrenal medulla's zona reticularis is in charge of creating the weak androgens DHEA and androstenedione, which are then converted to testosterone or estradiol. Defects of the male reproductive system are usually caused by low testosterone levels or sensitivity to testosterone, which can lead to low libido, failure to ejaculate, bone density, muscle loss, infertility, premature puberty or premature puberty. Other less specific symptoms of low testosterone are low energy/depressed mood, anemia or increased body fat. Metabolic

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Received: 01-Apr-2022, Manuscript No. ANO-22-22468; **Editor assigned:** 04-Apr-2022, PreQC No. ANO-22-22468(PQ); **Reviewed:** 18-Apr-2022, QC No. ANO-22-22468; **Revised:** 25-Apr-2022, Manuscript No. ANO-22-22468(R); **Published:** 02-May-2022, DOI:10.35248/2167-0250.22.11.281.

Citation: Yang C (2022) The Imapct of Internal Structures on the Male Reproductive System. Andrology.11:281.

syndrome, insulin resistance, and atherosclerosis are some of the other comorbidities linked to low testosterone.