

ETIOLOGY OF TESTOSTERONE DEFICIENCY IN MEN

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Under physiologic conditions in men, the hypothalamus produces gonadotropin-releasing hormone (GnRH), which induces the anterior pituitary gland to secrete 2 gonadotropins: luteinizing hormone (LH) and follicle-stimulating hormone (FSH). In turn, these gonadotropins respectively stimulate Leydig cells to produce testosterone (T) and induce Sertoli cells to nurture spermatogenesis. Sperm and T downregulate their own production through a feedback loop that reduces the secretion of hormones by the hypothalamus and pituitary.

In the blood, T circulates principally in bound form, mainly to sex hormone-binding globulin (SHBG) and albumin. It tightly binds to SHBG and is not biologically available, whereas the T fraction associated with albumin is weakly bound and can dissociate to free, active T. In young adult men, only about 2% of T is in the free form, 30% is bound tightly to SHBG, and 68% is weakly bound to albumin.

Testosterone deficiency (TD) - is a clinical and biochemical syndrome frequently associated with age and comorbidities, and characterized by a deficiency in T and relevant symptoms: incomplete or delayed sexual development, secondary sexual characteristics, sexual disorders and infertility.

Etiology TD represented classification, which reflects the basic types.

1. The primary TD (disorders at the testicular level): anorchia, cryptorchidism, varicocele, orchitis, Klinefelter syndrome, XX syndrome men, XYY syndrome, testicular tumors, chronic diseases, injury testicles, radiation treatment or chemotherapy.

2. Secondary TD (diseases of the hypothalamus and the pituitary gland): Kallmann syndrome, Prader-Willi syndrome, chronic systemic illness (chronic organ failure, diabetes mellitus, malignancy, rheumatic disease, HIV infection, inherited metabolic storage diseases), congenital adrenal hypoplasia, constitutional delay of development, secondary GnRH deficiency, fractured skull, central ischemia, radiation effects in connection with treatment brain tumors, hypopituitarism, isolated LH or FSH deficiency, hyperprolactinemia, mutation receptor GnRH hormone.

3. Mixed primary and secondary TD: late-onset TD.

4. Tertiary TD (disorders of androgen target organs): testosterone insensitivity. TD in men has a multifactorial etiology and results from testicular failure, or is due to the disruption of one or several levels of the hypothalamic-pituitary-gonadal axis.