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1.

TESTOSTERONE

Testosterone is the primary male sex hormone. In male humans, testosterone plays a key role in the development of male reproductive tissues such as testes and prostate, as well as promoting secondary sexual characteristics such as increased muscle and bone mass, and the growth of body hair. In addition, testosterone is involved in health and well-being, and the prevention of osteoporosis.

Testosterone is a steroid from the androstane class containing a keto and hydroxyl groups. It is biosynthesized in several steps from cholesterol and is considered in the liver to inactive metabolites. It exerts its action through binding to and activation of the androgen receptor. Testosterone is secreted primarily by the testicles and, to a lesser extent the ovaries of females. On average, in adult male, levels of testosterone are about 7 to 8 times as great as in adult females. As the metabolism of testosterone in males is more pronounced, the daily production is about 20 times greater in men.

In addition to its role as a natural hormone, testosterone is used as a medication in the treatment of low testosterone levels in men, transgender hormone therapy for transgender men, and breast cancer in women. Since, testosterone levels decrease as men age, it is sometimes used in older men to counteract this deficiency. It is also used illicitly to enhance physique and performance, for instance in athletes.

BIOLOGICAL EFFECTS

In general, androgens such as testosterone promote protein synthesis and thus growth of tissues with androgen receptors. Testosterone can be described as having virilising and anabolic effects.

* Anabolic effects include growth of muscle mass and strength, increased bone density and strength, and stimulation of linear growth and bone maturation.
* Androgenic effects such as maturation of the sex organs, particularly the penis and the formation of the scrotum in the foetus, and after birth (usually at puberty), a deepening of the voice, growth of facial hair (such as the beard) and axillary (underarm) hair.

Testosterone effects can also be classified by the age of usual occurrence. For postnatal effects on both males and females, these are mostly dependent on the levels and duration of circulating free testosterone.

*BEFORE BIRTH*

Effects before birth are divided into two categories, in relation to the stages of development.

The first period occurs between 4 and 6 weeks of the gestation. Examples include genital virilisation such as midline fusion, phallic urethra, scrotal thinning and rogation, and phallic enlargement; although the role of testosterone is far smaller than that of dihydrotestosterone. There is also development of the prostate gland and seminal vesicles.

During the second trimester, androgen level is associated with sex formation. Specifically, testosterone, along with the anti-Mullerian hormone (AMH) prmote growth of the Wolffian ductand degeneration of the Mullerian duct respectively. This period affects the feminization or masculinization of the foetus and can be a better predictor of feminine or masculine behaviours such a sex typed behaviour than an adult’s own levels. Prenatal androgens apparently influence interests and engagement in gendered activities and have moderate effects on spatial abilities.

*EARLY INFANCY*

Early infancy androgen effects are the least understood. In the first weeks of life for male infants, testosterone levels rise. The levels remain in a pubertal range for a few months, but usually reach the barely detectable levels of childhood by 4–7 months of age. The function of this rise in humans is unknown. It has been theorized that brain masculinization is occurring since no significant changes have been identified in other parts of the body. The male brain is masculinized by the aromatization of testosterone into estrogen, which crosses the blood–brain barrier and enters the male brain, whereas female fetuses have α-fetoprotein, which binds the estrogen so that female brains are not affected.

*BEFORE PUBERTY*

Before puberty, effects of rising androgen levels occur both in boys and girls. These include adult-type body odour, increased oiliness of the skin and hair, acne appearance of pubic hair, axillary hair, growth spurt, accelerated bone maturation and facial hair.

*PUBERTAL*

Pubertal effects begin to occur when androgen has been higher than normal adult female levels for months or years. In males, these are usual late pubertal effects, and occur in women after prolonged periods of heightened levels of free testosterone in the blood. The effects include:

Growth of spermatogenic tissue in testicles, male fertility, penis or clitoris enlargement, increased libido and frequency of erection or clitoral engorgement occurs. Growth of jaw, brow, chin, and nose and remodeling of facial bone contours, in conjunction with human growth hormone occurs. Completion of bone maturation and termination of growth. This occurs indirectly via estradiol metabolites and hence more gradually in men than women. Increased muscle strength and mass, shoulders become broader and rib cage expands, deepening of voice, growth of the Adam's apple, enlargement of sebaceous glands. This might cause acne. Subcutaneous fat in face decreases. Pubic hair extends to thighs and up toward umbilicus, development of facial hair (sideburns, beard, moustache), loss of scalp hair (androgenetic alopecia), increase in chest hair, periareolar hair, perianal hair, leg hair, armpit hair.

*ADULT*

Testosterone is necessary for normal sperm development. It activates genes in Sertoli cells, which promote differentiation of spermatogonia. It regulates acute HPA (hypothalamic-pituitary-adrenal axis) response under dominance challenge. Testosterone regulates population of thromboxane A2 receptors on megakaryocytes and platelets and hence platelet aggregation in humans.

2.

MALE INFERTILTY

Male infertility refers to a male's inability to cause pregnancy in a fertile female. In humans, it accounts for 40–50% of infertility. It affects approximately 7% of all men. Male infertility is commonly due to deficiencies in the semen, and semen quality is used as a surrogate measure of male fecundity.

CAUSES

Factors relating to male infertility include:

*IMMUNE INFERTILITY*

Antisperm antibodies (ASA) have been considered as infertility cause in around 10–30% of infertile couples. ASA production are directed against surface antigens on sperm, which can interfere withTop of Form sperm motility and transport through the female reproductive tract, inhibiting capacitation and acrosome reaction, impaired fertilization, influence on the implantation process, and impaired growth and development of the embryo. Risk factors for the formation of antisperm antibodies in men include the breakdown of the blood-testis barrier, trauma and surgery, orchitis, varicocele, infections, prostatitis, testicular cancer, failure of immunosuppression and unprotected receptive anal or oral sex. Bottom of Form

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GENETICS

Chromosomal anomalies and genetic mutations account for nearly 10–15% of all male infertility cases.

*Klinefelter Syndrome*

One of the most commonly known causes of infertility is Klinefelter Syndrome, affecting 1 out of 500–1000 newborn males, Klinefelter Syndrome is a chromosomal defect that occurs during gamete formation due to a non-disjunction error during cell division. Resulting in males having smaller testes, reducing the amount of testosterone and sperm production. Males with this syndrome carry an extra X chromosome (XXY), meaning they have 47 chromosomes compared to the normal 46 in each cell. This extra chromosome directly affects sexual development before birth and during puberty (links to learning disabilities and speech development have also been shown to be affected). There are varieties in Klinefelter Syndrome, where some cases may have the extra X chromosome in some cells but not others, referred to as Mosaic Klinefelter Syndrome, or where individuals have the extra X chromosome in all cells. The reduction of testosterone in the male body normally results in an overall decrease in the production of viable sperm for these individuals thereby forcing them to turn to fertility treatments to father children.

*Y chromosome deletions*

Y chromosomal infertility is a direct cause of male infertility due to its effects on sperm production, occurring in 1 out of every 2000 males. Usually affected men show no signs or symptoms other than, at times, exhibiting smaller testes size. Men with this condition can exhibit azoospermia (no sperm production), oligozoospermia (small number of sperm production), or they will produce abnormally shaped sperm (teratozoospermia). This case of infertility occurs during the development of gametes in the male, where a normal healthy male will produce both X and a Y chromosome, affected males have genetic deletions in the Y chromosome. These deletions affect protein production that is vital for spermatogenesis. Studies have shown that this is an inherited trait; if a male is fathered by a man who also exhibited y chromosome deletions then this trait will be passed down. These individuals are thereby “Y-linked”, although daughters are not affected due to the lack of the Y chromosome.

OTHERS

* Age
* Abnormal set of chromosomes
* Centriole
* Neoplasm, e.g. seminoma
* Idiopathic failure
* Cryptorchidism
* Trauma
* Hydrocele
* Hypopituitarism in adults, and hypopituitarism untreated in children (resulting in growth hormone deficiency and proportionate dwarfism.)
* Mumps
* Malaria
* Testicular cancer
* Acrosomal defects affecting egg penetration
* Idiopathic oligospermia- unexplained sperm deficiencies account for 30% of male infertility.

PRE-TESTICULAR CAUSES

Pre-testicular factors refer to conditions that impede adequate support of the testes and include situations of poor hormonal support and poor general health including:

VARICOCELE

Varicocele, is a condition of swollen testicle veins.

It is present in 15% of normal men and in about 40% of infertile men.

It is present in up to 35% of cases of primary infertility and 69–81% of secondary infertility.

* Hypogonadotropic hypogonadism due to various causes
	+ Obesity increases the risk of hypogonadotropic hypogonadism. Animal models indicate that obesity causes leptin insensitivity in the hypothalamus, leading to decreased Kiss1 expression, which, in turn, alters the release of gonadotropin-releasing hormone (GnRH).
* Undiagnosed and untreated coeliac disease (CD). Coeliac men may have reversible infertility. Nevertheless, CD can present with several non-gastrointestinal symptoms that can involve nearly any organ system, even in the absence of gastrointestinal symptoms. Thus, the diagnosis may be missed, leading to a risk of long-term complications. In men, CD can reduce semen quality and cause immature secondary sex characteristics, hypogonadism and hyperprolactinaemia, which causes impotence and loss of libido. The giving of gluten free diet and correction of deficient dietary elements can lead to a return of fertility. It is likely that an effective evaluation for infertility would best include assessment for underlying celiac disease, both in men and women.
* Drugs, alcohol
* Strenuous riding (bicycle riding, horseback riding)
* Medications, including those that affect spermatogenesis such as chemotherapy, anabolic steroids, cimetidine, spironolactone; those that decrease FSH levels such as phenytoin; those that decrease sperm motility such as sulfasalazine and nitrofurantoin
* Genetic abnormalities such as a Robertsonian translocation

TOBACCO SMOKING

There is increasing evidence that the harmful products of tobacco smoking may damage the testicles and kill sperm, but their effect on male fertility is not clear. Some governments require manufacturers to put warnings on packets. Smoking tobacco increases intake of cadmium, because the tobacco plant absorbs the metal. Cadmium, being chemically similar to zinc, may replace zinc in the DNA polymerase, which plays a critical role in sperm production. Zinc replaced by cadmium in DNA polymerase can be particularly damaging to the testes.

DNA DAMAGE

Common inherited variants in genes that encode enzymes employed in DNA mismatch repair are associated with increased risk of sperm DNA damage and male infertility. As men age there is a consistent decline in semen quality, and this decline appears to be due to DNA damage. The damage manifests by DNA fragmentation and by the increased susceptibility to denaturation upon exposure to heat or acid, the features characteristic of apoptosis of somatic cells. These findings suggest that DNA damage is an important factor in male infertility.

EPIGENETIC

An increasing amount of recent evidence has been recorded documenting abnormal sperm DNA methylation in association with abnormal semen parameters and male infertility. Until recently, scientists have thought that epigenetic markers only affect the individual and are not passed down due to not changing the DNA. New studies suggest that environmental factors that changed an individual's epigenetic markers can be seen in their grandchildren, one such study demonstrating this through rats and fertility disruptors. Another study bred rats exposed to an endocrine disruptor, observing effects up to generation F5 including decreased sperm motility and decreased sperm count. These studies suggest that environmental factors that influence fertility can be felt for generations even without changing the DNA.

POST-TESTICULAR CAUSES

Post-testicular factors decrease male fertility due to conditions that affect the male genital system after testicular sperm production and include defects of the genital tract as well as problems in ejaculation:

* Vas deferens obstruction
* Lack of vas deferens, often related to related to genetic markers for cystic fibrosis
* Infection, e.g. prostatitis
* Retrograde ejaculation
* Ejaculatory duct obstruction
* Hypospadias
* Impotence