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SPERMATOGENESIS

Spermatogenesis is the process by which haploid spermatozoa develop from germ cells in the seminiferous tubules of the testis. The process starts with the mitotic division of the stem cells located close to the basement membrane of the tubules. These cells are called spermatogonial stem cells. Within the walls of the tubules, also, are many randomly scattered cells, called Sertoli cells, that function to support and nourish the immature sperm cells by giving them nutrients and blood products. As the young germ cells grow, the sertoli cells help to transport them from the outer surface of the seminiferous tubule to the central channel of the tubule. Sperm cells are continually being produced by the testes, but not all areas of the seminiferous tubules produce sperm cells at the same time. One immature germ cell takes as long as 74 days to reach final maturation, and during this growth process there are intermittent resting phases. The immature cells( called spermatogonia) are all derived from cells called stem cells in the outer wall of the seminiferous tubules. The stem cells are composed almost entirely of nuclear material. The nucleus of the cell is the portion containing the chromosomes. The stem cells begin their process by multiplying in the process of cell duplication known as mitosis. Hlf of the new cells from this initial crop go on to become the future sperm cells, and the other half remain as stem cells so that there is constant source of additional germ cells. Spermatogonia destined to develop into mature sperm cells are known as primary sperm cells. These move from the outer portion of the seminiferous tubule to a more central location and attach themselves around the sertoli cells. The primary cells then develop somewhat by increasing the amount of cytoplasm and structures called organelles within the cytoplasm. After a resting phase the primary cells divide into a form called a secondary sperm cell. During this cell division there is a splitting of the nuclear material.

The secondary sperm cell still must mature before it can fertilize an egg, maturation entails certain changes in the shape and form of the sperm cell. The nuclear material becomes more condensed and oval in shape, this area develops as the head of the sperm. The head is covered partially by a cap, called the acrosome, which is important in helping the sperm to gain entry into the egg. Attached to the opposite end of the head is the tailpiece. The tail is derived from the secondary sperm cells cytoplasm. In the mature sperm, it consists of a long, slender bundle of filaments that propel the sperm by their undulating movement. Once the sperm has matured, it is transported through the long seminiferous tubules and stored in the epididymis of the testes until it is ready to leave the male body. Because the type A1 spermatogonia are stem cells, spermatogenesis occur continuously. Each day, some 100 million sperm are made in each human testicle and each ejaculation releases 200 million sperm.

STAGES OF SPERMATOGENESIS

The entire process of spermatogenesis can be broken into several distinct stages, each corresponding to a particular type of cell in humans. The three stages are:

1. **Spermatocytogenesis:** Spermatocytogenesis is the male form of gametocytogenesis and results in the formation of spermatocytes possessing half the normal complement of genetic material. In spermatocytogenesis, a diploid spermatogonium, which resides in the basal compartment of the seminiferous tubules, divides mitotically, producing two diploid intermediate cells called primary spermatocytes. Each primary spermatocyte then moves into the adluminal compartment of the seminiferous tubules and duplicates its DNA and subsequently undergoes meiosis1 to produce two haploid secondary spermatocytes, which will later divide once more into haploid spermatids. This division implicates sources of genetic variation, such as random inclusion of either parental chromosomes or chromosomal crossover that increases the genetic variability of the gamete. The DNA damage response(DDR) machinery plays an important role in spermatogenesis. The protein FMRP binds to meiotic chromosomes and regulates the dynamics of the DDR machinery during spermatogenesis. FMRP appears to be necessary for the repair of DNA damage. Each cell division from a spermatogonium to a spermatid is complete, the cells remain connected to one another by bridges of cytoplasm to allow synchronous development. Not all spermatogonia divide to produce spermatocytes, otherwise, the supply of spermatogonia would run out.
2. **Spermatidogenesis:** Spermatidogenesis is the creation of spermatids from secondary spermatocytes. Secondary spermatocytes produced earlier rapidly enter meiosis II and divide to produce haploid spermatids.
3. **Spermiogenesis:** During spermiogenesis, the spermatids begin to form a tail by growing microtubules on one centrioles, which turns into a basal body. These microtubules form an axoneme. Later the centriole is modified in the process of centrosome reduction. The anterior part of the tail called midpiece thickens because mitochondria are arranged around the axoneme to ensure energy supply. Spermatid DNA also undergoes packaging, becoming highly condensed. The DNA is packaged firstly with specific nuclear basic proteins, which are subsequently replaced with protamines during spermatid elongation. The resultant tightly packed chromatin is transcriptionally inactive. The Golgi apparatus surrounds the now condensed nucleus, becoming the acrosome. Maturation then takes place under the influence of testosterone, which removes the remaining unnecessary cytoplasm and organelles. The excess cytoplasm, known as residual bodies, is phagocytosed by surrounding Sertoli cells in the testes. The resulting spermatozoa are now mature but lack motility. The mature spermatozoa are released from the protective Sertoli cells into the lumen of the seminiferous tubule in a process called spermiation. The non-motile spermatozoa are transported to the epididymis in testicular fluid secreted by the S ertoli cells with the aid of peristaltic contraction. While in the epididymis, the spermatozoa gains motility and becomes capable of fertilization. However, transport of the mature spermatozoa through the remainder of the male reproductive system is achieved via muscle contraction rather than spermatozoon’s recently acquired motility.

HORMONAL CONTROL

Hormonal control of spermatogenesis varies among species. In humans the mechanism is not completely understood, however it is known that initiation of spermatogenesis occurs at puberty due to the interaction of the hypothalamus and pituitary glands. If the pituitary gland is removed, spermatogenesis can still be initiated by follicle stimulating hormone (FSH) and testosterone. In contrast to FSH, lutenizing hormone (LH) appears to have little role in spermatogenesis outside inducing gonad testosterone production. FSH stimulates the production of androgen binding protein by Sertoli cells, and the formation of the blood testes barrier. Androgen binding protein is essential to concentrating testosterone in levels high enough to initiate and maintain spermatogenesis.

TESTOSTERONE

Testosterone is the primary male sex hormone and anabolic steroid. In male humans, testosterone plays a key role in the development of male reproductive tissues such as testes and prostrate, as well as promoting secondary sexual characteristics such as increased muscle and bone mass, and the growth of body hair. Testosterone is the hormone responsible for the development of male sexual characteristics. Hormones are chemical messengers that trigger necessary changes in the body. Females also produce testosterone but in smaller amounts. It is a type of androgen produced primarily by the testicles in cells called the Leydig cells. In men, include:

* Sex drive
* Bone mass
* Fat distribution
* Muscle size and strength
* Red blood cell production

Without adequate amounts of testosterone, men become infertile. This is because testosterone assists in the development of mature sperm. Despite being a male sex hormone, testosterone also contributes to sex drive, bone density and muscle strength in women. However, an excess of testosterone can also cause women to experience male pattern baldness and infertility. The brain and pituitary gland control testosterone levels. Once produced, the hormone moves through the blood to carry out its various important functions. High or low levels of testosterone can lead to dysfunction in the parts of the body normally regulated by the hormone. When a man has low testosterone, pr hypogonadism, he may experience:

* Reduced sex drive
* Erectile dysfunction
* Low sperm count
* Enlarged or swollen breast tissue
* Loss of body hair
* Loss of muscle bulk
* Loss of strength
* Increased body fat

Chronic or ongoing low testosterone may lead to osteoporosis (a medical condition in which the bones become brittle and fragile from loss of tissue, typically as a result of hormonal changes, or deficiency of calcium or vitamin D), mood swings, reduced energy, and testicular shrinkage.

CAUSES

* Testicular injury, such as castration
* Infection of the testicles
* Medications, such as opiate analgesiscs
* Disorders that affect the hormones, such as pituitary tumors or high prolactin levels
* Chronic diseases, including type 2 diabetes, kidney disease, liver disease, obesity and HIV/AIDS
* Genetic diseases such as Klinfelter syndrome, Prader-Willi syndrome, hemochromatosis, Kallman syndrome, and myotonic dystrophy

Too much testosterone, on the other hand, can lead to the triggering of puberty before the age of 9 years. This condition would mainly affect younger man and is much rarer. In women, however, high testosterone levels can lead to male pattern baldness, a deep voice and menstrual irregularities as well as: Growth and swelling of the clitoris, changes in body shape, reduction in breast size, oily skin, acne, facial hair growth around the body, lips and chin. High testosterone levels in women can cause uterine fibroids.

TESTOSTERONE LEVELS AND AGING

Testosterone levels naturally decrease as a man ages. The effects of gradually lowering testosterone levels in men age have received increasing attention in recent years. It is known as late onset hypogonadism. After the age of 40, the concentration of circulating testosterone falls by about 1.6 percent every year for most men. By the age of 60, the low levels of testosterone would lead to a diagnosis of hypogonadism in younger men. About 4 in 10 men have hypogonadism by the time they reach 45 years of age. The number of cases in which older men have been diagnosed as having low testosterone increased by 170 percent. Late testosterone has been associated with increased mortality in male veterans. Late onset hypogonadism has become a recognized medical condition, although many of the symptoms are associated with normal aging. The following are symptoms of late onset hypogonadism:

* Diminished erectile quality, particularly at night
* Decreased libido
* Mood changes
* Reduced cognitive function
* Fatigue, depression and anger
* A decrease in muscle mass and strength
* Loss of body hair (pubic, axilliary and facial)
* Skin changes
* Decreased bone mass and bone mineral density
* Increase in abdominal fat mass
* Low sperm count
* Lethargy
* Sleep disturbances
* Breast discomfort and enlargement
* Hot flashes
* Sweating

In primary hypogonadism, the testicles do not respond to hormone stimulation. This can be due to congenital disorder such as Klinfelter’s syndrome, or acquired as a result of radiation treatment, chemotherapy, mumps, tumors or trauma to the testes.

In secondary hypogonadism, a disease state interferes with either the hypothalamus or pituitary gland, the main glands that release hormones to stimulate the testes to produce testosterone. Situations that can cause secondary hypogonadism include:

* Malnutrition
* Systemic illness
* Stress
* Medication side effects
* Liver cirrhosis
* Toxins (alcohol and heavy metals)
* Morbid obesity

As well as sexual dusfunction, late onset hypogonadism has also been associated with metabolic disease and cardiovascular disease.