

Processes of spermatogenesis



The testes that is responsible for producing the male gametes and carries the sequence of processes in the seminiferous tubulus is known as Spermatogenesis. Such process commence during puberty, at and around of 14 years of age in males and stage a continuous process throughout a person's life. The production of sperm in a healthy male is found in researches to be 400 million sperms. Spermatogenesis takes 64 days for development from a spermatogonium to a mature sperm. At any given time, different seminiferous tubules are in different stages, so that up to several hundred million sperm may reach maturity daily. Spermatogenesis encompasses three major stages; mitotic proliferation, meiosis and packaging.

Two gonadotropic hormones are controlled by the testes in a male which is secreted by the anterior pituitary, luteinizing hormone (LH) and follicle-stimulating hormone (FSH), the same cell type is responsible for the production which is gonadotrope. The luteinizing hormone and follicle-stimulating hormone tend to act as separate elements of the testes. The luteinizing hormone displays on the Leydig cells to generate testosterone secretion. While on the other hand, follicle-stimulating hormone displays on the sertoli cells to improve the vitality of the spermatogenesis. Secretion of the luteinizing hormone and follicle-stimulating hormone from the anterior pituitary is stimulated in turn by a single hypothalamic hormone, gonadotropin-releasing hormone (GnRH). However, FSH and LH are segregated to a large extent into separate secretory vesicles in the gonadotrope and are not secreted equal amounts because other regulatory factors influence how much of each gonatropin is secreted. Testosterone, the

creation and triggering of luteinizing hormone of the Leydig cells, behave in a negative manner to interfere with the secretion of the luteinizing hormone in dual ways. The most important negative effect of testosterone is to reduce GnRH to be released by an act on the hypothalamus. This will result in an indirect reduction in both luteinizing hormone and follicle-stimulating hormone to be released by the anterior pituitary to selectively reduce LH secretion. The latter action explains why the exertion from the testosterone has greater impact on luteinizing hormone rather than the follicle-stimulating hormone.

The hypothalamus releases GnRH, which dominates the letting off of the anterior pituitary gonadotropins, the follicle-stimulating hormone and luteinizing hormone. The GnRH reaches the anterior pituitary cells via the blood of the hypophyseal portal system. Binding of GnRH to pituitary cells (gonadotrophs) prompts both to secrete follicle-stimulation hormone and luteinizing into the bloodstream. The follicle-stimulation hormone triggers spermatogenesis consequentially by triggering the sustentacular cells to let off androgen-binding protein (ABP). ABP stimulates the spermatogenic cells to hold together and focus on testosterone, which will then trigger spermatogenesis. The reaction of follicle-stimulating hormone then makes the cells responsive to testosterone's trigger the effects. The luteinizing hormone holds together the interstitial cells and triggers them to release testosterone. LH is therefore sometimes known as interstitial cell-stimulating hormone (ICSH) in males. Locally testosterone benefits as the end stimulant for spermatogenesis. Testosterone flows into the blood stream that exerts a number of effects at other body sites. Both hypothalamus and

the anterior pituitary are subject to feedback inhibition to blood borne hormones. Testosterone slow down the process of hypothalamic release of GnRH thus acts instantly on the anterior pituitary to slow down gonadotropin release. A protein hormone called Inhibin, which is made by the sustentacular cells, carry out as a barometer of the normalcy of spermatogenesis. Inhibin release will increase tremendously when the sperm count is high, and it slow down anterior pituitary secretion of Follicle-stimulating hormone and GnRH secretion by the hypothalamus. When the sperm rate falls 20 million/ml and below, the inhibin releases declines steeply.

Mass production of testosterone and sperm by the testes shows a balance reaction of the three hormones which are GnRH, gonadotropins and inhibin. GnRH, triggers the testes through the follicle-stimulating hormone and the interstitial cell-stimulating hormone. Secondly, the gonadotropins, which instantly stimulates the testes while the testicular hormones which is (testosterone and inhibin), shows opposing command on the hypothalamus and anterior pituitary.

In the absence of GnRH and gonadotropins, the testes atrophy, and for all practical purposes, sperm and testosterone production ceases. As more GnRH is released, more testosterone is secreted by the testes, but the threshold for hypothalamic inhibition keeps rising until the adult pattern of hormone interaction is achieved. Maturation of the brain-testicular axis takes about three years, and once established, the balance between the interacting hormones remains relatively constant.

The testicular inhibitory signal specifically directed at controlling FSH secretion is the peptide hormone inhibin, which is secreted by the Sertoli cells. Inhibin behaves instantly on the anterior pituitary and cautiously inhibits follicle-stimulating hormone release. Thus the understanding that inhibition of the follicle-stimulating hormone by a Sertoli cell product is appropriate, because FSH stimulates spermatogenesis by acting on the Sertoli cells.

Testosterone, secreted by Leydig cells under LH stimulation, acts as a paracrine regulator by stimulating spermatogenesis in the adult human testis. FSH is not absolutely required for spermatogenesis, as demonstrated by men who have mutated and nonfunctional FSH receptors. LH-stimulated testosterone secretion promotes spermatogenesis, whereas FSH only enhances this effect. The FSH receptors are located in the Sertoli cells, and the follicle-stimulating hormone triggers Sertoli cells which produce androgen-binding protein and inhibin. Newborn males have only about 10% of their adult number of Sertoli cells, and this increases to the adult number as the boy enters puberty. It appears that FSH, acting together with testosterone, promotes this proliferation of Sertoli cells. Without FSH, spermatogenesis would still occur but would begin later in puberty. FSH is required for maximal sperm production, and so it may be required for optimal fertility.

Follicle-stimulating hormone and testosterone performed a very important task in managing spermatogenesis, both play their own effect by acting on the Sertoli cells. The germ cells of mitosis and meiosis are very important for Testosterone, while follicle-stimulating hormone is required for spermatid remaking. The Leydig cells which is produced locally is withheld in the intra

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tubular fluid complex added with androgen-binding protein which will be secreted by the Sertoli cells, created a much higher testosterone concentration in the testes than in the blood stream. The sperm production is sustainable through high concentration of testicular testosterone.