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Perspective

A Brief Note on Follicle Stimulating Hormone

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DESCRIPTION

Follicle Stimulating Hormone (FSH) is a glycoprotein polypeptide hormone and a gonadotropin. The anterior pituitary gland's gonadotropic cells synthesis and secrete FSH, which regulates the body's development, growth, pubertal maturation, and reproductive activities. In the reproductive system, FSH and Luteinizing Hormone (LH) operate together. The pituitary gland's ability to control FSH secretion is uncertain. Low-frequency Gonadotropin-Releasing Hormone (GnRH) pulses raise FSH mRNA levels in rats; however this is not linked to an increase in circulating FSH.

GnRH has been found to play a key role in FSH secretion, with hypothalamic-pituitary disconnection resulting in FSH cessation. FSH secretion returns after GnRH treatment. The gonads provide oestrogen feedback to FSH *via* the pituitary.

Effects in females

In the ovary, FSH promotes the growth and recruitment of immature ovarian follicles. FSH is the key survival factor in early (small) antral follicles, rescuing the small antral follicles (2-5 mm in diameter for humans) from apoptosis (programmed death of the somatic cells of the follicle and oocyte). Because serum levels of progesterone and oestrogen (mainly estradiol) fall throughout the luteal-follicle phase transition period and no longer restrict FSH release, FSH peaks around day three. The tiny antral follicle cohort is generally large enough to produce enough Inhibin B to reduce FSH serum levels.

Furthermore, evidence suggests that gonadotropin surge attenuating factor produced by small follicles during the first half of the follicle phase has a negative feedback on pulsatile Luteinizing Hormone (LH) secretion amplitude, allowing for a more favourable environment for follicle growth and preventing premature luteinisation.

The number of tiny antral follicles recruited in each cycle decreases as a woman approaches perimenopause, and as a result, insufficient Inhibin B is produced to entirely suppress FSH, and the serum level of FSH begins to rise. FSH levels eventually rise to the point where FSH receptors are downregulate, and any residual tiny secondary follicles do not contain FSH or LH receptors by postmenopause.

When the follicle grows and reaches a diameter of 8-10 mm, it begins to secrete large levels of estradiol. In humans, only one follicle becomes dominant and survives to grow to a size of 18-30 mm and ovulate, while the rest of the follicles in the cohort undergo atresia. The quick increase in estradiol synthesis by the dominant follicle has a favourable effect on the brain and pituitary, resulting in rapid GnRH pulses and an LH surge. By blocking GnRH production in the hypothalamus, an increase in serum estradiol levels causes a decrease in FSH output.

Because the current cohort's smaller follicles lack sufficient sensitivity to FSH, they suffer from atresia as a result of the fall in serum FSH levels. By coincidence, two follicles may reach the 10 mm stage at the same time, and because both are equally sensitive to FSH, both survive and mature in the low FSH environment, resulting in two ovulations in one cycle, potentially resulting in non-identical (dizygotic) twins.

Effects in males

FSH causes primary spermatocytes to divide for the first time, resulting in secondary spermatocytes. FSH stimulates the creation of androgen-binding protein in the sertoli cells of the testes by binding to FSH receptors on their basolateral membranes, which is necessary for spermatogenesis to begin.

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