Product Data Sheet

Product Name:	FSH
Cat. No.:	GP21245
Batch No.:	1

Product Data

Purity		Source	Urine of post-menopausal women.
Phycical Appearance	Sterile Filtered White lyophilized (freeze-dried) powder.	Shipping Condition	Shipped at Room temp.
Synonyms	Follitropin subunit beta; Follicle-stimulating hormone beta subunit; FSH-beta; FSH-B; Follitropin beta chain; FSH.		
Solubility	It is recommended to reconstitute the lyophilized Follicle Stimulating Hormone in sterile pyrogen free water at 100IU/0.1ml, which can then be further diluted to other aqueous solutions.		
Formulation	The FSH was lyophilized with no additives.		

Introduction

Follicle stimulating hormone (FSH) is a hormone synthesised and secreted by gonadotropes in the anterior pituitary gland. FSH and LH act synergistically in reproductionIn women, in the ovary FSH stimulates the growth of immature Graafian follicles to maturation. As the follicle grows it releases inhibin, which shuts off the FSH production. In men, FSH enhances the production of androgen-binding proteinby the Sertoli cells of the testes and is critical for spermatogenesis. In both males and females, FSH stimulates the maturation of germ cells. In females, FSH initiates follicular growth, specifically affecting granulosa cells. With the concomitant rise in inhibin B FSH levels then decline in the late follicular phase. This seems to be critical in selecting only the most advanced follicle to proceed to ovulation. At the end of the luteal phase, there is a slight rise in FSH that seems to be of importance to start the next ovulatory cycle.Like its partner, LH, FSH release at the pituitary gland is controlled by pulses of gonadotropin-releasing hormone(GnRH). Those pulses, in turn, are subject to the estrogen feed-back from the gonads.

Stability

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Lyophilized FSH although stable at room temperature for 3 weeks, should be stored desiccated below -18°C . Upon reconstitution FSH-beta should be stored at 4°C between 2-7 days and for future use below -18°C . For long term storage it is recommended to add a carrier protein (0.1% HSA or BSA).Please prevent freeze-thaw cycles.

Protocol

Cell experiment [1]:

Cell lines	Ovarian granulosa cells (GCs)
Preparation Method	GCs transfected with GFP-MAP1LC3B plasmid for 48 h were incubated with 200 μM H2O2 for 1 h and cultured for another 2 h in the presence or absence of FSH (7.5 IU/ml), pepstatin A and E64.
Reaction Conditions	FSH (7.5 IU/ml) for 2 h
Applications	FSH reduces oxidative injury in cultured GCs via inhibiting autophagic PCD.

Animal experiment [2]:

Animal models	Cebpb+/ 3xTg mutant mice
Preparation Method	I.p. injected 2.5- to 3-month-old compound mutant mice with FSH (5 IU per mouse) daily for 3 months
Dosage form	5 IU FSH for 3 months

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Applications	FSH-induced Cebpb, Lgmn, App and Mapt expression, AEP activation, APP and Tau cleavage, Aβ and pTau accumulation, dendritic spine deficits and cognition defects were all lower in Cebpb+/ 3xTg mice compared with 3xTg mice.

References:

[1].Shen M, Jiang Y, et,al.Protective mechanism of FSH against oxidative damage in mouse ovarian granulosa cells by repressing autophagy. Autophagy. 2017 Aug 3;13(8):1364-1385. doi: 10.1080/15548627.2017.1327941. Epub 2017 Jun 9. PMID: 28598230; PMCID: PMC5584866.
[2]. Xiong J, Kang SS, et,al. FSH blockade improves cognition in mice with Alzheimer's disease. Nature. 2022 Mar;603(7901):470-476. doi: 10.1038/s41586-022-04463-0. Epub 2022 Mar 2. PMID: 35236988.

Background

FSH, a major survival factor for antral follicles, has been suggested to improve GC resistance to oxidative stress during follicular atresia[2].

The decline in GCs viability caused by oxidant injury was remarkably reduced following FSH treatment, along with impaired macroautophagic/autophagic flux under conditions of oxidative stress both in vivo and in vitro. Blocking of autophagy displayed similar

levels of suppression in oxidant-induced cell death compared with FSH treatment, but FSH did not further improve survival of GCs pretreated with autophagy inhibitors. FSH inhibited the production of acetylated FOXO1 and its interaction with Atg proteins, followed by a decreased level of autophagic cell death upon oxidative stress[1]. FSH dampened stress-induced apoptosis and the expression of FoxO1 and pro-apoptosis genes in mouse granulosa cells (MGCs). The signaling cascades involved in regulating FoxO1 activity upon FSH treatment were identified using FSH signaling antagonists[3].

FSH acts directly on hippocampal and cortical neurons to accelerate amyloid-β and Tau deposition and impair cognition in mice displaying features of Alzheimer's disease. Blocking FSH action in these mice abrogates the Alzheimer's disease-like phenotype by inhibiting the neuronal C/EBPβ-δ-secretase pathway[4]. FSH plays an essential role in the pathogenesis of OA and acts as a crucial mediator[6]. When generated a mouse model of FSH elevation by intraperitoneally injecting exogenous FSH into ovariectomized (OVX) mice, in which a normal level of estrogen (E2) was maintained by exogenous supplementation. Consistently, the results indicate that FSH, independent of estrogen, increases the serum cholesterol level in this mouse model[5]. FSH can rescue impaired female fertility and ovarian function due to androgen insensitivity in female ARKO mice by maintaining follicle health and ovulation rates, and thereby optimal female fertility[7].

References:
[1]. Shen M, Jiang Y, et,al.. Protective mechanism of FSH against oxidative damage in mouse ovarian granulosa cells by repressing autophagy. Autophagy. 2017 Aug 3;13(8):1364-1385. doi: 10.1080/15548627.2017.1327941. Epub 2017 Jun 9. PMID: 28598230; PMCID: PMC5584866.
[2]. Peluso JJ, Steger RW. Role of FSH in regulating granulosa cell division and follicular atresia in rats. J Reprod Fertil. 1978 Nov;54(2):275-8. doi: 10.1530/jrf.0.0540275. PMID: 722676.
[3]. Shen M, Liu Z, et,al. Involvement of FoxO1 in the effects of follicle-stimulating hormone on inhibition of apoptosis in mouse granulosa cells. Cell Death Dis. 2014 Oct 16;5(10):e1475. doi: 10.1038/cddis.2014.400. PMID: 25321482; PMCID: PMC4237239.
[4]. Xiong J, Kang SS, et,al. FSH blockade improves cognition in mice with Alzheimer's disease. Nature. 2022 Mar;603(7901):470-476. doi: 10.1038/s41586-022-04463-0. Epub 2022 Mar 2. PMID: 35236988.

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[5]. Guo Y, Zhao M, et,al.Blocking FSH inhibits hepatic cholesterol biosynthesis and reduces serum cholesterol. Cell Res. 2019 Feb;29(2):151-166. doi: 10.1038/s41422-018-0123-6. Epub 2018 Dec 17. PMID: 30559440; PMCID: PMC6355920.
[6]. Zhang M, Wang Y, et,al. FSH modulated cartilage ECM metabolism by targeting the PKA/CREB/SOX9 pathway. J Bone Miner Metab. 2021 Sep;39(5):769-779. doi: 10.1007/s00774-021-01232-3. Epub 2021 May 14. PMID: 33988757.
[7]. Walters KA, Edwards MC, et,al. Subfertility in androgen-insensitive female mice is rescued by transgenic FSH. Reprod Fertil Dev. 2017 Jul;29(7):1426-1434. doi: 10.1071/RD16022. PMID: 27328025.

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