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Role of Follicle-stimulating hormone (FSH) and Testosterone in abnormal sperm parameters – A review article

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ABSTRACT

Follicle-stimulating hormone (FSH) and testosterone are essential for the spermatogenesis. FSH acts on FSH receptor (FSHR) present in Sertoli cells to support spermatogenesis. Luteinizing hormone (LH) act on Leydig cells which stimulates testosterone production which completes the spermatogenesis. Their levels are regulated by a negative feedback loop to balance the hormone production. The balanced levels of these hormones are essential for maintaining the sperm parameters. In case of excessive or deficiency of FSH and testosterone levels, sperm quantity and quality got affected and lead to male infertility. In male factor infertility with low to normal FSH and testosterone levels, FSH supplements with or without LH or Human chorionic gonadotropins (HCG) supplements can maintain spermatogenesis even in presence of low levels of testosterone. Only testosterone replacement therapy can suppress of FSH and LH that can lead to decrease in semen parameters and male infertility. Also, high level of testosterone can damage the sperm and can lead to male infertility. In this article, role of FSH and testosterone in abnormal semen parameters had been explained and type of hormonal therapy which can be beneficial in these cases.

Introduction

Male infertility is equally contributing in reasons of infertility as female infertility that accounts for approximately 30 to 40%.[1] Semen analysis is the most important investigation to the detect the male infertility. As per semen analysis parameters of World Health Organization (WHO) 2010 criteria, different terminologies are used to define the abnormal sperm parameters - azoospermia, oligozoospermia, asthenozoospermia, teratozoospermia, oligoasthenoteratozoospermia (OAT). Abnormal sperm parameters can be defined as low sperm concentration of <15 million/mL (oligozoospermia), less total sperm motility of <40% with less progressive sperm motility of <32% (asthenozoospermia), low sperm morphology of <4% (teratozoospermia) and combination of all abnormal parameters (oligoasthenoteratozoospermia).[2] Male hormones - Follicle-stimulating hormone (FSH) and testosterone act through Hypothalamus–Pituitary–Testicular (HPT) Axis and work together synergistically to control spermatogenesis.[3] FSH stimulates the proliferation of Sertoli cells, supports germ cell development and the early stages of spermatogenesis and testosterone helps in maintaining the later stages and completion of spermatogenesis. Their levels are regulated by a negative feedback loop to balance the hormone production. The balanced levels of these hormones are essential for maintaining the sperm parameters. In case of excessive or deficiency of FSH and testosterone levels, sperm quantity and quality got affected and lead to male infertility. Supplementation of these hormones helps in treatment of male infertility in many cases. In this article, the essentiality of Follicle-stimulating hormone (FSH) and testosterone for spermatogenesis had been discussed along with its effect on sperm parameters and type of hormonal therapy which can be beneficial in these cases.

Hypothalamus–Pituitary–Testicular (HPT) Axis

Hypothalamus–Pituitary–Testicular (HPT) Axis is the key axis to regulate the spermatogenesis in males.[3] It starts in the hypothalamus that releases gonadotropin-releasing hormone (GnRH) in pulsatile manner and which stimulates the anterior pituitary gland to release the gonadotropins - follicle-stimulating hormone (FSH) and luteinizing hormone (LH). FSH stimulates the Sertoli cells in the testes through FSHR receptor and further stimulates spermatogenesis. LH acts on Leydig cells in the testes which helps in testosterone (T) production. The levels of GnRH, LH, and FSH are controlled by negative feedback mechanism through testosterone and aromatization of testosterone to estradiol (E2).[4]

Spermatogenesis

Spermatogenesis is a male reproductive process occurring in the seminiferous tubules of the testis that includes germ cell proliferation and differentiation. The tubules are made up of three important cells - peritubular myoid (PTM) cells, Sertoli cells and germ cells. PTM cells are located in external wall of the tubule and helps in sperm maturation and transport. Sertoli cells (SC) releases growth factors which are required for germ cells proliferation and differentiation. These are located at the base of the seminiferous tubules of the testis where they form an impermeable and immunological barrier called blood-testis barrier (BTB) and maintain the renewal of spermatogonia stem cells (SCC). SCC initiates the spermatogenesis process and produces differentiated spermatogonia which transforms into spermatocytes. Spermatocytes undergo meiosis to produce haploid round spermatids which after transformation to final spermatozoa are released in the lumen.[5] FSH is required for the proliferation of Sertoli cells and germ cells and particularly important in the early stages of spermatogenesis. Testosterone is primarily required for the completion of meiosis in spermatogenesis, sperm differentiation, maturation and release of sperms.

FSH action

FSH is an important regulator of Sertoli cell (SC) proliferation and plays an important role in prepubertal preparation for spermatogenesis and pubertal spermatogenesis regulation.[6] SC are the somatic cells in seminiferous tubules which provides supporting environment for spermatogenesis. Its

number is determined by FSH action at neonatal and during pubertal age and also determines daily sperm production.[7]

FSH is a glycoprotein made up of two subunits - FSHα which is shared with other glycoproteins and FSHβ subunit which is peculiar to FSH. The human FSHβ is encoded by the *FSHB* gene located on chromosome 11p21 and its receptor FSH receptor (FSHR) is exclusively present on the cellular membrane of SC.[8] FSHR expression begins in the second half of gestation and but it activates in newborn when FSH secretion starts. At the time of puberty, pituitary FSH production increases which triggers Sertoli cell proliferation. The level of serum FSH determines the Sertoli cell number which further corelates with the quantity of sperm production and testicular size in adulthood.[9]

When FSH binds to the membrane-bound G-protein coupled receptor FSHR, conformational changes occur in FSHR which generates FSH signaling. FSHR recruits different types of G proteins to mediate different signaling pathways. There are five types of FSH signaling pathways occurring in SC - cyclic adenosine monophosphate (cAMP)/protein kinase A (PKA) pathway, extracellular-regulated kinase (ERK)/mitogen-activated protein kinase (MAPK) pathway, phosphoinositide 3-kinase (PI3K) pathway, calcium pathway and phospholipase A2 pathway which regulate spermatogonia pool maintenance, spermatogonia differentiation, entry into meiosis and positively regulates spermatocyte survival and limits their apoptosis.[6] SC also helps in germ cell coordination and metabolite exchanges via seminiferous epithelium through tight junctions which further helps in regulation of complex endocrine and paracrine function of spermatogenesis.[10] So, in males, FSH helps in maintaining the fertility. In the absence of FSHR function, oligospermia can happen and mutation in FSHβ subunit causes azoospermia and infertility.[11,12]

Testosterone action

Testosterone is the male reproductive androgen produced by the Leydig cell in the testis which help in regulation of qualitative spermatogenesis. Anterior pituitary gland secretes luteinizing hormone (LH) into the circulation which binds to its receptors on Leydig cells.[13] It increases the level of cAMP levels which help in expression of two proteins: StAR (the steroidogenic acute regulatory protein) and CYP11A1 (the cholesterol sidechain cleavage enzyme). StAR helps in the transfer of cholesterol from the outer to the inner mitochondrial membrane and CYP11A1 converts cholesterol to pregnenolone which is the precursor of all steroid hormones and results in testosterone production.[14]

Testosterone concentration is 50–100-fold higher within the testis than in peripheral circulation.[15] Testosterone diffuses into Sertoli cells and binds to the androgen receptor (AR) located in middle of the sperm in the mitochondrial region and are present in the cytoplasm and nucleus to complete the spermatogenesis.[16] So, both gonadotrophins LH and FSH along with high intratesticular testosterone concentration with direct action on AR in Sertoli cells are important for spermatogenesis.[17]

Testosterone directly act on specific androgen receptors and indirect act through intracellular conversion to dihydrotestosterone by 5-alpha reductase at cell level. Both testosterone and dihydrotestosterone bind to cell receptors and help in protein expression that maintains the BTB, supporting the completion of meiosis, the adhesion of elongated spermatids to Sertoli cells and the release of sperm through classical and non-classical signaling pathways.[18,19] Testosterone is also converted to estradiol by aromatase enzyme which stimulates pituitary FSH secretion and so indirectly helps in FSH mediated SC proliferation and modulates libido, erectile function and spermatogenesis.[20]

As we know testosterone binds strongly with AR, low hydroxyl group in testosterone leads its entry into the cell rather than binding to plasma membrane.[21,22] This helps in anti-apoptosis and sperm metabolism.[23,24] If testosterone levels go very high, there will be uncontrolled entry of testosterone inside the cell which alters the signal pathways causing inhibition

of anti-apoptotic properties leading to damage to sperm chromatin.[25,26] Low levels of testosterone maintain the sperm energy, anti-apoptotic properties and has positive effect on chromatin quality.[27] Its seems that though testosterone is critical for spermatogenesis, but low testosterone levels doesn't impact the semen quality much but opposite is not true. As per one study done on 853 patients with at least 5 million total motile sperm in which 116 had low TT (Testosterone) (<264 ng/dL) and 737 had normal TT (≥264 ng/dL), sperm parameters like semen volume, sperm cell count, progressive (A + B) motility and morphology (≥4% strict Kruger) were lower in the low testosterone group but not significant difference between low and normal TT groups.[28] In a study of unexplained infertility, low levels of testosterone were associated with abnormal sperm morphology and lower live birth rates.[29] Testosterone opposes the free radicals in cells by maintaining the genes of intracellular enzymes. In case of oxidative stress, both testosterone and estrogen activate signal pathways such as PI3K/Akt, MAPKs and manages mitochondrial electron transport chain, cell morphology, and gene expression.[30] Various studies showed that testosterone is very important for sperm parameters like motility and morphology.

Hormonal therapy

FSH, LH and testosterone are the important male reproductive hormones to control the testicular functions (sperm and androgen production). Spermatogenesis is physiologically regulated both by FSH and LH-dependent intra-testicular testosterone. So, reduced FSH levels results in low testosterone hormone secretion with low sperm production.

A meta-analysis showed that gonadotropin supplementation in subjects with hypogonadotropic hypogonadism (HHG) and azoospermia helps in spermatogenesis and testosterone replacement therapy (TRT) is not of use in these cases.[30] Various studies showed that FSH administration in HHG and normozoospermia improves the sperm parameters – count, motility, morphology and reduces DNA fragmentation, production of reactive oxygen species and aneuploidy.[31,32]

As it is known that LH acts on the Leydig cells stimulating testosterone production and activates spermatogenesis regulatory pathways. HCG can be given as a surrogate for LH in HHA and non-obstructive azoospermia (NOA).[33] HCG and LH both can bind to the same LHCGR receptor on the Leydig cells. But HCG can bind with greater affinity and has a longer half-life.[34] So, HCG is the good alternative for LH for hormonal treatment. **In one of the review,** HCG with or without hMG/FSH is the best suitable option in hypogonadal infertile male with avoidance of testosterone supplementation.[35,36]

Testosterone replacement therapy can suppress of FSH and LH that can lead to decrease in semen parameters and male infertility. As per study, short-acting testosterone preparations do not decrease serum FSH or LH to the same extent as longer-acting transdermal gels and injectables.[37] It was observed that many individuals showed low testosterone levels with normal sperm concentration.[38] Testosterone substitution therapy is not given nowadays. Instead, males should be treated with FSH and LH/HCG preparations which stimulate intratesticular testosterone production by the Leydig cells.[39]

Conclusion

FSH and testosterone are essential for the spermatogenesis. But it is observed that FSH supplements with or without LH/HCG supplements can maintain spermatogenesis even in presence of low levels of intratesticular testosterone. Also, high level of testosterone can damage the sperm and can lead to male infertility.

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FSH supplements with or without LH/HCG supplements can maintain spermatogenesis even in presence of low levels of testosterone.

References:

1. Leslie SW, Soon-Sutton TL, Khan MAB. Male Infertility. [Updated 2024 Feb 25]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK562258/

2. World Health Organization. Geneva: WHO Press; 2010. Laboratory Manual for the Examination and Processing of Human Semen, 5th ed.

3. Ramaswamy S, Weinbauer GF. Endocrine control of spermatogenesis: Role of FSH and LH/ testosterone. Spermatogenesis. 2015 Jan 26;4(2): e996025. doi: 10.1080/21565562.2014.996025. PMID: 26413400; PMCID: PMC4581062.

4. Klein CE. The Hypothalamic-Pituitary-Gonadal Axis. In: Kufe DW, Pollock RE, Weichselbaum RR, et al., editors. Holland-Frei Cancer Medicine. 6th edition. Hamilton (ON): BC Decker; 2003. Available from: https://www.ncbi.nlm.nih.gov/books/NBK13386/

5. Khanehzad M, Abbaszadeh R, Holakuyee M, Modarressi MH, Nourashrafeddin SM. FSH regulates RA signaling to commit spermatogonia into differentiation pathway and meiosis. Reprod Biol Endocrinol. 2021;19:4.

6. Wang, JM., Li, ZF., Yang, WX. *et al.* Follicle-stimulating hormone signaling in Sertoli cells: a licence to the early stages of spermatogenesis. *Reprod Biol Endocrinol* **20**, 97 (2022). https://doi.org/10.1186/s12958-022-00971-w

7. Sharpe RM, McKinnell C, Kivlin C, Fisher JS. Proliferation and functional maturation of Sertoli cells, and their relevance to disorders of testis function in adulthood. Reproduction. 2003 Jun;125(6):769-84. doi: 10.1530/rep.0.1250769. PMID: 12773099.

8. Das N, Kumar TR. Molecular regulation of follicle-stimulating hormone synthesis, secretion and action. J Mol Endocrinol. 2018 Apr;60(3):R131-R155. doi: 10.1530/JME-17-0308. Epub 2018 Feb 7. PMID: 29437880; PMCID: PMC5851872.

9. Allan CM, Garcia A, Spaliviero J, Zhang FP, Jimenez M, Huhtaniemi I, et al. Complete Sertoli cell proliferation induced by follicle-stimulating hormone (FSH) independently of luteinizing hormone activity: evidence from genetic models of isolated FSH action. Endocrinology (2004) 145:1587–93. 10.1210/en.2003-1164

10. Zimmermann C, Stévant I, Borel C, Conne B, Pitetti JL, Calvel P, Kaessmann H, Jégou B, Chalmel F, Nef S. Research resource: the dynamic transcriptional profile of sertoli cells during the progression of spermatogenesis. Mol Endocrinol. 2015 Apr;29(4):627-42. doi: 10.1210/me.2014-1356. Epub 2015 Feb 24. PMID: 25710594; PMCID: PMC5414748.

11. Layman LC, Porto AL, Xie J, da Motta LA, da Motta LD, Weiser W, Sluss PM. FSH beta gene mutations in a female with partial breast development and a male sibling with normal puberty and azoospermia. J Clin Endocrinol Metab. 2002;87:3702–7.

12. Zheng J, Mao J, Cui M, Liu Z, Wang X, Xiong S, Nie M, Wu X. Novel FSHβ mutation in a male patient with isolated FSH deficiency and infertility. Eur J Med Genet. 2017;60:335–9.

13. Walker WH. Testosterone Signaling and the Regulation of Spermatogenesis. *Spermatogenesis* (2011) 1(2):116–20. doi: 10.4161/spmg.1.2.16956

14. Miller WL. Early steps in androgen biosynthesis: from cholesterol to DHEA. Baillieres Clin Endocrinol Metab. 1998 Apr;12(1):67-81.

15. *Roth M.Y., Lin K., Amory J.K., Matsumoto A.M., Anawalt B.D., Snyder C.N., Kalhorn T.F., Bremner W.J., Page S.T. Serum LH correlates highly with intratesticular steroid levels in normal men. J. Androl. 2010;31:138–145. doi: 10.2164/jandrol.109.008391.*

16. Mruk D.D., Cheng C.Y. Sertoli-Sertoli and Sertoli-germ cell interactions and their significance in germ cell movement in the seminiferous epithelium during spermatogenesis.

17. Sofikitis N, Giotitsas N, Tsounapi P, Baltogiannis D, Giannakis D, Pardalidis N. Hormonal Regulation of Spermatogenesis and Spermiogenesis. *J Steroid Biochem Mol Biol* (2008) 109(3–5):323–30. doi: 10.1016/j.jsbmb.2008.03.004

18. Grande G, Barrachina F, Soler-Ventura A, Jodar M, Mancini F, Marana R, Chiloiro S, Pontecorvi A,

Oliva R, Milardi D. The Role of Testosterone in Spermatogenesis: Lessons From Proteome Profiling of Human Spermatozoa in Testosterone Deficiency. Front Endocrinol (Lausanne). 2022 May 19;13:852661. doi: 10.3389/fendo.2022.852661. PMID: 35663320; PMCID: PMC9161277.

19. Smith LB, Walker WH. The regulation of spermatogenesis by androgens. Semin Cell Dev Biol. 2014 Jun;30:2-13. doi: 10.1016/j.semcdb.2014.02.012. Epub 2014 Mar 2. PMID: 24598768; PMCID: PMC4043871.

20. Nassar GN, Leslie SW. Physiology, Testosterone. [Updated 2023 Jan 2]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK526128/

21. Freeman ER, Bloom DA, McGuire EJ. A brief history of testosterone. J Urol. 2001 Feb;165(2):371-3. doi: 10.1097/00005392-200102000-00004. PMID: 11176375.

22. Oren I, Fleishman SJ, Kessel A, Ben-Tal N. Free diffusion of steroid hormones across biomembranes: a simplex search with implicit solvent model calculations. Biophys J. 2004 Aug;87(2):768-79. doi: 10.1529/biophysj.103.035527. PMID: 15298886; PMCID: PMC1304487.

23. Tesarik J, Martinez F, Rienzi L, Iacobelli M, Ubaldi F, Mendoza C, Greco E. In-vitro effects of FSH and testosterone withdrawal on caspase activation and DNA fragmentation in different cell types of human seminiferous epithelium. Hum Reprod. 2002 Jul;17(7):1811-9. doi: 10.1093/humrep/17.7.1811. PMID: 12093844.

24. Zalata AA, Mokhtar N, Badawy Ael-N, Othman G, Alghobary M, Mostafa T. Androgen receptor expression relationship with semen variables in infertile men with varicocele. J Urol. 2013 Jun;189(6):2243-7. doi: 10.1016/j.juro.2012.11.112. Epub 2012 Nov 28. PMID: 23201383.

25. Sanocka D, Kurpisz M. Reactive oxygen species and sperm cells. Reprod Biol Endocrinol. 2004 Mar 23;2:12. doi: 10.1186/1477-7827-2-12. PMID: 15038829; PMCID: PMC400757.

26. Vasconsuelo A, Pronsato L, Ronda AC, Boland R, Milanesi L. Role of 17β-estradiol and testosterone in apoptosis. Steroids. 2011 Nov;76(12):1223-31. doi: 10.1016/j.steroids.2011.08.001. Epub 2011 Aug 10. PMID: 21855557.

27. Mohammadzadeh M, Montazeri F, Poodineh J, Vatanparast M, Rahmanian Koshkaki E, Ghasemi Esmailabad S, Mohseni F, Talebi AR. Therapeutic potential of testosterone on sperm parameters and chromatin status in fresh and thawed normo and asthenozoospermic samples. Rev Int Androl. 2023 Jul-Sep;21(3):100352. doi: 10.1016/j.androl.2023.100352. Epub 2023 May 25. PMID: 37244225.

28. Di Guardo F, Vloeberghs V, Bardhi E, Blockeel C, Verheyen G, Tournaye H, Drakopoulos P. Low Testosterone and Semen Parameters in Male Partners of Infertile Couples Undergoing IVF with a Total Sperm Count Greater than 5 Million. J Clin Med. 2020 Nov 26;9(12):3824. doi: 10.3390/jcm9123824. PMID: 33255908; PMCID: PMC7761260.

29. Trussell JC, Coward RM, Santoro N, Stetter C, Kunselman A, Diamond MP, Hansen KR, Krawetz SA, Legro RS, Heisenleder D, Smith J, Steiner A, Wild R, Casson P, Coutifaris C, Alvero RR, Robinson RB, Christman G, Patrizio P, Zhang H, Lindgren MC; Reproductive Medicine Network. Association between testosterone, semen parameters, and live birth in men with unexplained infertility in an intrauterine insemination population. Fertil Steril. 2019 Jun;111(6):1129-1134. doi: 10.1016/j.fertnstert.2019.01.034. Epub 2019 Apr 12. PMID: 30982604; PMCID: PMC6557574.

30. Mancini A, Raimondo S, Persano M, Di Segni C, Cammarano M, Gadotti G, Silvestrini A, Pontecorvi A, Meucci E. Estrogens as antioxidant modulators in human fertility. Int J Endocrinol. 2013;2013:607939. doi: 10.1155/2013/607939. Epub 2013 Nov 20. PMID: 24363671; PMCID: PMC3863713.

31. Valenti D, La Vignera S, Condorelli RA, Rago R, Barone N, Vicari E, Calogero AE. Follicle-stimulating hormone treatment in normogonadotropic infertile men. Nat Rev Urol. 2013 Jan;10(1):55-62. doi: 10.1038/nrurol.2012.234. Epub 2012 Dec 11. PMID: 23229508.

32. Cannarella R, La Vignera S, Condorelli RA, Mongioì LM, Calogero AE. FSH dosage effect on conventional sperm parameters: a meta-analysis of randomized controlled studies. Asian J Androl. 2020 May-Jun;22(3):309-316. doi: 10.4103/aja.aja_42_19. PMID: 31274479; PMCID: PMC7275804.

33. Shiraishi K, Matsuyama H. Gonadotoropin actions on spermatogenesis and hormonal therapies for spermatogenic disorders [Review]. Endocr J. 2017 Feb 27;64(2):123-131. doi: 10.1507/endocrj.EJ17-0001. Epub 2017 Jan 19. PMID: 28100869.

34. Esteves SC, Humaidan P. The role of luteinizing hormone activity in spermatogenesis: from physiology to clinical practice. Reprod Biol Endocrinol. 2025 Jan 13;23(Suppl 1):6. doi: 10.1186/s12958-024-01333-4. PMID: 39800700; PMCID: PMC11726975.

35. Ho CC, Tan HM. Treatment of the Hypogonadal Infertile Male-A Review. Sex Med Rev. 2013 May;1(1):42-49. doi: 10.1002/smrj.4. Epub 2015 Oct 21. PMID: 27784559.

36. Casarini L, Crépieux P, Reiter E, Lazzaretti C, Paradiso E, Rochira V, Brigante G, Santi D, Simoni M. FSH for the Treatment of Male Infertility. Int J Mol Sci. 2020 Mar 25;21(7):2270. doi: 10.3390/ijms21072270. PMID: 32218314; PMCID: PMC7177393.

37. Masterson TA, Turner D, Vo D, Blachman-Braun R, Best JC, Westfield G, Bryson N, Ramasamy R. The Effect of Longer-Acting vs Shorter-Acting Testosterone Therapy on Follicle Stimulating Hormone and Luteinizing Hormone. Sex Med Rev. 2021 Jan;9(1):143-148. doi: 10.1016/j.sxmr.2020.07.006. Epub 2020 Sep 8. PMID: 32912767.

38. Greenberg DR, Hudnall MT, Goyette BN, Lai JD, Hayon S, Bennett NE, Brannigan RE, Halpern JA. Assessing the Prevalence of Low Testosterone and Elevated Follicle Stimulating Hormone Among Men Presenting for Fertility Evaluation Without Oligospermia. Urology. 2023 Oct;180:130-134. doi: 10.1016/j.urology.2023.07.011. Epub 2023 Jul 22. PMID: 37482101.

39. Casarini L, Lispi M, Longobardi S, Milosa F, La Marca A, Tagliasacchi D, et al. LH and hCG action on the same receptor results in quantitatively and qualitatively different intracellular signalling. *PLoS ONE.* (2012) 7:e46682. doi: 10.1371/journal.pone.0046682

