Journal of Andrology, Vol. 23, No. 6, November/December 2002

Possible Relationship Between Seminal Plasma Inhibin B and Spermatogenesis in Patients With Azoospermia

YEHIA F. EL GAREM*, ALI F. EL ARINI*, ADEL H. EL BEHEIRY*, SAMI A. ABOU ZEID* AND FRANK H. COMHAIRE†

*From the * Department of Andrology and Dermatology, Faculty of Medicine, Alexandria University, Alexandria, Egypt; and † Center for Medical and Urological Andrology, Ghent University, Ghent, Belgium.*

Correspondence to: Prof Dr Frank H. Comhaire, Ghent University Hospital, Center for Medical and Urological Andrology, De Pintelaan, 185, B-9000 Ghent, Belgium.

Received for publication December 19, 2001; accepted for publication May 29, 2002.

Abstract

Inhibin B is bidirectionally secreted by Sertoli cells, basal secretion into the circulation exerts negative feedback on follicle-stimulating hormone secretion, and serum inhibin B is considered a marker of spermatogenesis. The precise role of apical secretion is unknown. The objective of our work was to study the relationship between seminal inhibin B and spermatogenesis. Dimeric inhibin B was measured by immunoassay in seminal plasma of volunteers with normozoospermia (n = 10, group 1), in men after vasectomy (n = 10, group 2), and in men with azoospermia (n = 50, group 3). Testicular biopsy and testicular sperm extraction were performed in men with azoospermia. Seminal inhibin B levels were higher in men in group 1 than in men in groups 2 and 3 ($P < .0001$). In seminal plasma, inhibin B presents a positive correlation with alpha glucosidase activity ($r = .37$, $P = .002$). Seminal inhibin B is inversely related with serum FSH ($r = -.58$, $P < .001$), and presents a weak positive correlation with serum testosterone concentration ($r = .29$, $P = .03$). No difference was found between inhibin B levels in seminal plasma of patients with nonobstructive or obstructive azoospermia, and between positive or negative outcome of TESE. We conclude that inhibin B secretion by Sertoli cells is differentially regulated. The contribution of accessory sex glands limits the use of seminal plasma inhibin B as a marker of spermatogenesis.

Key words: Male infertility, Sertoli cell, testis biopsy, testicular sperm extraction

---

Inhibin is a glycoprotein hormone of gonadal origin, consisting of 2 dissimilar disulfide-linked subunits (α and either ßA or ßB), that has an inhibitory effect on pituitary gonadotropin production

(Burger and Igarashi, 1988). Recent studies have reported a strong negative correlation between serum follicle-stimulating hormone (FSH) and serum inhibin B in both fertile and subfertile men (Anderson et al, 1997). Serum inhibin B is considered a marker of Sertoli cell function and of spermatogenesis (Anawalt et al, 1996; Mahmoud et al, 1998a).

It has been shown that inhibin B, similar to other Sertoli cell products, is secreted bidirectionally (ie, into the seminiferous tubular lumen via the apex of Sertoli cells, and via the base of these cells into testicular interstitial fluid). It has been generally assumed that the latter is the main route by which inhibin reaches the blood stream because of the proximity of interstitial fluid to the vasculature (Maddocks and Sharpe, 1989).

Cultured Sertoli cells secrete bioactive inhibin, and the majority of secretion is into the adluminal compartment (Handelsman et al, 1990). The level of inhibin B in seminal plasma can reach 10 times the level in serum. This suggests that elevated levels are caused by secretion of inhibin B by Sertoli cells into the adluminal space (Byrd et al, 1998). A significant correlation has been reported between inhibin B concentrations in seminal plasma and in serum (Anderson et al, 1998).

The main action of inhibin B secreted into the circulation is to regulate serum FSH levels. The precise role of inhibin secreted in seminiferous tubules is unknown. Impure preparations have been demonstrated to inhibit spermatogonial mitosis in rat testes (de Kretser and McFarlane, 1996). Blocking the inhibin effect increases spermatogenesis in animals (Schanbacher, 1991; Bame, 1999).

The objective of this work was to study the relationship between seminal plasma inhibin B and spermatogenesis and the outcome of testicular sperm extraction (TESE) in men with azoospermia.

Materials and Methods

- Top
- Abstract
- Materials and Methods
- Results
- Discussion
- References

Semen samples were collected from 70 men attending the andrology outpatient clinics of Alexandria University Hospital (Alexandria, Egypt) and Ghent University Hospital (Ghent, Belgium). Samples were grouped as follow: group 1, samples from 10 volunteers with normozoospermia (ie, with semen parameters better than the World Health Organization [WHO] recommended reference values); group 2, samples from 10 men who had undergone successful vasectomy; and group 3, samples from 50 infertile men with azoospermia. The ethical committees of both hospitals approved the protocols for this study. An informed consent was obtained from all participants.

Semen Analysis

Semen samples were produced by masturbation and left for 30 minutes to liquefy, after which conventional semen analysis was performed according to WHO (1999) recommendations. Seminal plasma α glucosidase was measured by the colorimetric method using a commercial kit (EpiScreen, Fertipro, Beernem, Belgium; Mahmoud et al, 1998b).

Measurement of Inhibin B in Seminal Plasma

Dimeric inhibin B was measured in seminal plasma with a solid phase sandwich enzyme-linked immunosorbent assay kit (Serotec, Oxford, United Kingdom) as described previously (Mahmoud et al, 1998a). In normozoospermic controls, the measurement of inhibin B in undiluted seminal plasma revealed values in excess of the maximum level of the standard curve. Therefore, the samples were diluted up to 1:4 to obtain a reading. A possible effect of a matrix factor was excluded by assessing serial dilutions. The lower detection value of the method is 15 pg/mL; intraassay and

interassay coefficients of variation were 11.0% and 10.7%, respectively.

Testicular Biopsy

Under general anesthesia, a bilateral transverse incision was carried out across the layers of the scrotum over the midanterior surface of both testes. The testicular tissue was protruded from a small incision in the tunica albuginea. A biopsy was taken and submerged in 1 mL of Ham F10 medium with Hepes (N1387; Sigma Chemical Company, St Louis, Mo) for testicular sperm extraction. Another biopsy was fixed in Bouin fixative for histopathological examination. The incisions were closed in layers after adequate hemostasis.

Testicular biopsies were interpreted quantitatively according to a modified Johnsen score (Glander et al, 2000), and qualitatively according to the following 5 groups: normal spermatogenesis, hypospermatogenesis, maturation arrest, Sertoli cells only (SCO), and mixed patterns. Biopsies were classified into those with spermatogenic activity (ie, showing at least spermatocytes), and those without spermatogenic activity (presenting as SCO).

Testicular Sperm Extraction

After mincing and shredding testicular tissue in a Petri dish (BD Biosciences, Erembodegem, Belgium), the samples were examined with an inverted microscope (Hoffman Optics Zeiss Axiovert 135, Zeiss, Zaventem, Belgium) using 400x magnification. If no spermatozoa were detected, a chemical digestion of the testicular tissue was performed using collagenase type 1A (C2674; Sigma). Briefly, testicular tissue was incubated with collagenase for 2 hours at 37 °C, and then transferred to a Falcon tube (BD Biosciences) and centrifuged for 10 minutes at 3000 x *g*. The supernatant was removed and the pellet was resuspended. One drop was reexamined with a microscope using a Neubauer chamber (Laboroptik GmbH, Friedrichsdorf, Germany). If no spermatozoa were detected, 5 µL of the suspension was dispersed on a Petri dish and examined with the inverted microscope. The result was interpreted as positive or negative TESE according to the presence or absence of spermatozoa (Salzbrunn et al, 1996).

Serum Hormones

Serum concentrations of luteinizing hormone (LH) and FSH were measured by electrochemiluminescence immunoassay using Elecsys-LH and Elecsys-FSH kits (Roche, Germany), respectively. Testosterone was measured using the Orion Diagnostica (Spectria, Finland) radioimmunoassay test.

Statistics

Statistical analysis was performed using the MedCalc program (MedCalc Software, Mariakerke, Belgium) (Schoonjans et al, 1995). Significance of differences was assessed using the Kruskal-Wallis and Wilcoxon tests. Rank correlation was calculated to detect the relation between 2 variables.

Results

- Top
- Abstract
- Materials and Methods
- Results
- Discussion
- References

After diluting the normozoospermic samples, inhibin B concentrations in seminal plasma showed a range of 1592 to 10425 pg/mL. Inhibin B was measured in undiluted seminal plasma samples of patients with azoospermia and showed a range of 15 to 702 pg/mL.

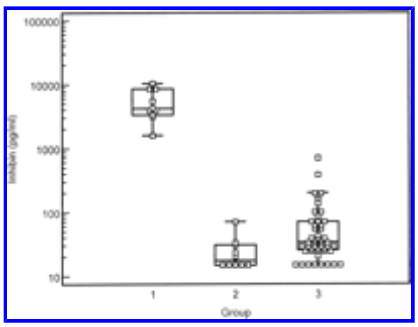
Table 1 shows the results of qualitative testicular biopsy and their corresponding levels of seminal plasma inhibin B. Inhibin B was detectable in the seminal plasma in all but 8 subjects with

azoospermia (group 3). Of the latter group, 2 research subjects showed maturation arrest at the spermatocyte stage, 4 exhibited SCO, and 2 had obstructive azoospermia. Inhibin B was undetectable in 5 of 10 postvasectomy subjects.

View this table:
[in this window]
[in a new window]

Table 1. *Seminal inhibin B levels in different qualitative pathological patterns in patients with azoospermia (group 3)*

The Figure shows that inhibin B in seminal plasma is significantly higher in normozoospermic samples than in postvasectomy and azoospermic samples ($P < .0001$). There was no significant difference in seminal inhibin B levels between obstructive azoospermic and postvasectomy samples ($P > .1$), nor between obstructive and nonobstructive (primary testicular) azoospermic samples ($P = .4$, data not shown). There was a tendency toward higher levels of inhibin B in azoospermic samples with spermatogenic activity than in those with SCO ($P = .1$). No difference was noticed between inhibin B levels in different qualitative pathological patterns of testicular biopsy (Table 1), nor was there a significant correlation between seminal inhibin B levels and testis biopsy score ($r = .17$, $P = .2$).



View larger version (15K):
[in this window]
[in a new window]

Seminal inhibin B levels in different study groups.

Seminal inhibin B showed a significant inverse correlation with serum FSH ($P = -.58$, $P < .001$), but no correlation with serum LH levels ($P = .1$).

Seminal α-glucosidase was significantly higher in group 1 subjects than in the other groups ($P < .001$), but the difference between samples of subjects with obstructive or nonobstructive azoospermia did not reach significance ($P = .1$). A strong positive correlation was found between seminal inhibin B and seminal plasma alpha glucosidase ($r = .37$, $P = .002$). There was a weak correlation between seminal inhibin B and serum testosterone ($r = .29$, $P = .03$).

There was no difference in seminal inhibin B concentration in azoospermic samples with positive or negative outcome of TESE (Table 2).

View this table:

Table 2. *Seminal inhibin B levels in positive and negative TESE*

[in this window]
[in a new window]

Discussion

Top
Abstract
Materials and Methods
Results
Discussion
References

Inhibin B was detectable in seminal plasma of all normozoospermic samples. This contrasts with the findings of Anderson et al (1998) who reported that inhibin B in seminal plasma was undetectable in 12% of samples of fertile men, although inhibin B was detectable in their serum. The cause of this discrepancy is unknown. We found seminal inhibin B to be significantly higher in normozoospermic than in azoospermic samples, sustaining the relation between inhibin B secretion and sperm concentration.

When subjects with obstructive azoospermia were compared to those without obstruction, there was no difference in seminal inhibin B concentrations. This finding contradicts the results of inhibin B measurement in serum, in which the level of inhibin B is higher in obstructive samples than in those without obstruction (von Eckardstein et al, 1999). Our finding can be explained by the fact that in subjects with obstructive azoospermia, the inhibin B secreted in seminiferous tubular fluid does not reach the ejaculated semen. The secretion of inhibin B in the circulation is independent of passage through the deferent ducts. At the other hand, the amount of inhibin B secreted may be lower in both serum and seminal plasma in subjects with nonobstructive azoospermia, due to Sertoli cell dysfunction.

We found a relatively high level of seminal inhibin B (100 pg/mL) in one patient with SCO with negative TESE and elevated serum FSH (51 IU/mL). In this patient, the presence of a high level of inhibin B and an absence of spermatozoa in testicular tissue could be explained by autonomous secretion of inhibin B by the Sertoli cells. This is in agreement with the report by Foresta et al (1999), who detected inhibin B in serum of 19% of cases with SCO. This autonomous secretion is either devoid of bioactivity, or there is peripheral resistance to its action, which explains the high level of FSH in these cases. The factors affecting autonomous inhibin B secretion are largely unknown (Raivio et al, 2000). Another possible explanation is that secretion of inhibin B from Sertoli cells may be under the regulation of factors other than germ cells, especially spermatids, that are proposed to exert paracrine control of inhibin B secretion (Andersson, 2000).

When comparing seminal inhibin B concentrations in subjects with azoospermia with or without spermatogenic activity, we found the level to be slightly higher in the former than in the latter. However, there was no correlation between inhibin B concentrations in seminal plasma and biopsy score. This contrasts with the results of Pierik et al (1998), who found a significant correlation between inhibin B in serum and biopsy score. Our data showed no difference in levels of inhibin B in seminal plasma related to the different qualitative pathological patterns, nor with the outcome of TESE. Similarly, Bohring and Krause (1999) and Von Eckardstein et al (1999) found that inhibin B in serum did not predict spermatogenic activity. The degree of overlap between different testicular pathologies appeared to limit the usefulness of inhibin measurement in predicting the result of TESE (Anderson, 2001). Indeed, in 2 of our subjects, spermatozoa were present in testicular tissue despite undetectable inhibin B in seminal plasma, whereas 2 subjects with seminal inhibin B values >100 pg/mL showed no spermatozoa upon TESE. These findings suggest that inhibin B in seminal plasma is not a useful marker for TESE. However, Ballesca et al (2000) and Brugo-Olmeda et al (2001) came

to the opposite conclusion, suggesting that inhibin measurement in seminal plasma is a useful, noninvasive predictor of spermatogenesis and TESE outcome.

Anderson and Sharpe (2000) reported high FSH serum concentrations to be associated with lower seminal plasma inhibin B concentrations in men with oligoazoospermia, but there was no correlation between seminal plasma inhibin B and serum FSH concentration (Anderson et al, 1998). In contrast, our results revealed a significant inverse correlation between seminal inhibin B concentration and serum FSH, which is in agreement with the report by Scott and Burger (1981), who measured bioactive inhibin in seminal plasma.

We found a significant, albeit weak positive correlation between serum testosterone concentration and seminal inhibin B. This may suggest a possible role for androgens in the regulation of seminal inhibin B. Recent studies provide evidence for the role of steroids in the control of inhibin B secretion from the Sertoli cells. Carlsen et al (1999) found a significant diurnal variation in serum inhibin B levels in normal men, lacking evidence for a role of FSH. However, the covariation with serum concentrations of testosterone and estradiol suggested that these hormones might play a role in the diurnal rhythm of inhibin B, although some other common influences could not be excluded (Sanford et al, 2002). Comhaire et al (1995), using an alpha subunit assay, recorded a similar diurnal variability in serum inhibin, and so did Kamischke et al (2001). The former authors noticed a general parallelism between changes in testosterone concentration and dihydrotestosterone on one hand, and serum inhibin on the other. Also, a greater inhibin secretion occurred after injection of human chorionic gonadotropin, in parallel with higher testosterone levels.

In vitro experiments revealed that the addition of testosterone to cultured Sertoli cells inhibits inhibin B production, whereas estrogen stimulates inhibin production (Depuydt et al, 1999). Martin et al (2000) produced evidence for the differential regulation of the 2 components of the bidirectional secretion of inhibin by Sertoli cells. They observed a rapid decline in seminal plasma inhibin B level with little change in serum inhibin B following administration of the combination of desogestrel and testosterone for contraceptive purposes. In agreement with data from animal studies (Handelsman et al, 1990), the available data suggest a possible role for sex steroids in the control of the (bidirectional) secretion of inhibin B the cells of Sertoli.

There are reasons to believe that inhibin in seminal plasma can originate from the accessory sex glands. In agreement with Anderson et al (1998), who found detectable values of seminal inhibin B in 2 of 20 postvasectomy samples, we detected inhibin in seminal plasma of 5 of 10 men who had successful vasectomy, and in 8 men with obstructive azoospermia. Scott and Burger (1981), using a bioactivity assay, found detectable amounts of seminal inhibin B in all postvasectomy samples and in 16 of 17 cases with obstructive azoospermia. This suggests that inhibin may be secreted into the prostatic fluid, the seminal vesicular fluid, or both (Vaze et al, 1980). Expression of messenger RNA for inhibin α and ß subunits was detected in the human prostate, indicating the capacity to produce all the known inhibins and activins (Thomas et al, 1998; Dowling and Risbridger, 2000). The accessory sex glands, however, appear unlikely primary sources of inhibin bioactivity because subjects with azoospermia with elevated plasma FSH levels had lower seminal plasma inhibin concentrations than those who had had vasectomy (Scott and Burger, 1981). Le Lannou et al (1979) have speculated that most of the inhibin in the rete testis fluid would be reabsorbed in the caput of the epididymis. However, we found a positive correlation between the concentrations of inhibin B and levels of α glucosidase in seminal plasma. This suggests a relation between seminal plasma inhibin B and the function of the epididymis, which seems contradictory to reabsorption of inhibin B from the epididymis. Our results rather suggest a possible role of inhibin B on epididymal function or a parallel effect of testosterone on both the epididymis and the Sertoli cells.

Conclusion

Seminal plasma inhibin B does not reflect the functional state of seminiferous epithelium, because there is no relation between seminal plasma inhibin B levels on the one hand, and spermatogenesis and TESE outcome on the other. This may be due to the contribution of accessory sex glands to the amount of inhibin secreted in the seminal plasma, to autonomous secretion of inhibin by the Sertoli cells, or to the differential regulation of inhibin B secretion into the adluminal and interstitial testicular compartments.

Footnotes

Supported by the Egyptian double channel system.

References

- Top
- Abstract
- Materials and Methods
- Results
- Discussion
- References

Anawalt BD, Bebb RA, Matsumoto AM, Groome NP, Illingworth PJ, McNeilly AS, Bremner WJ. Serum inhibin B levels reflect Sertoli cell function in normal men and men with testicular dysfunction. *J Clin Endocrinol Metab*. 1996;81:3341 -3345.[Abstract]

Anderson RA, Wallace EM, Groome NP, Bellis AJ, Wu FC. Physiological relationships between inhibin B, follicle stimulating hormone secretion and spermatogenesis in normal men and response to gonadotrophin suppression by exogenous testosterone. *Hum Reprod*.1997; 12:746 -751.[Abstract/Free Full Text]

Anderson RA, Irvine DS, Balfour C, Groome NP, Riley SC. Inhibin B in seminal plasma: testicular origin and relationship to spermatogenesis. *Hum Reprod*.1998; 13:920 -926.[Abstract/Free Full Text]

Anderson RA, Sharpe RM. Regulation of inhibin production in the human male and its clinical applications. *Int J Androl*. 2000;23:136 -144.[Medline]

Anderson RA. Clinical studies: inhibin in the adult male. *Mol Cell Endocrinol*.2001; 180:109 -116. [Medline]

Andersson AM. Inhibin B in the assessment of seminiferous tubular function. *Baillieres Clin Endocrinol Metab*.2000; 14:389 -397.

Ballesca JL, Balasch J, Calafell JM, Alvarez R, Fabregues F, de Osaba MJ, Ascaso C, Vanrell JA. Serum inhibin B determination is predictive of successful testicular sperm extraction in men with non-obstructive azoospermia. *Hum Reprod*.2000; 15:1734 -1738.[Abstract/Free Full Text]

Bame JH, Dalton JC, Degelos SD, et al. Effect of long-term immunization against inhibin on sperm output in bulls. *Biol Reprod*. 1999;60:1360 -1366.[Abstract/Free Full Text]

Bohring C, Krause W. Serum levels of inhibin B in men with different causes of spermatogenic failure. *Andrologia*,1999; 31:137 -141.[Medline]

Brugo-Olmedo S, De Vicentiis S, Calamera JC, Urrutia F, Nodar F, Acosta AA. Serum inhibin B may be a reliable marker of the presence of testicular spermatozoa in patients with nonobstructive azoospermia. *Fertil Steril*.2001; 76:1124 -1129.[Medline]

Burger HG, Igarashi M. Inhibin: definition and nomenclature, including related substances. *Mol Endocrinol*.1988; 2:391 -392.[Medline]

Byrd W, Bennett MJ, Carr BR, Dong Y, Wians F, Rainey W. Regulation of biologically active dimeric inhibin A and B from infancy to adulthood in the male. *J Clin Endocrinol Metab*.1998; 83:2849 -2854. [Abstract/Free Full Text]

Carlsen E, Olsson C, Petersen JH, Andersson AM, Skakkebaek NE. Diurnal rhythm in serum levels of inhibin B in normal men: relation to testicular steroids and gonadotropins. *J Clin Endocrinol Metab*. 1999;84:1664 -1669.[Abstract/Free Full Text]

Comhaire FH, Rombauts L, Vereecken A, Verhoeven G. Inhibin and steroid responses to testicular stimulation in normal men. *Hum Reprod*. 1995;10:1740 -1744.[Abstract/Free Full Text]

de Kretser DM, McFarlane JR. Inhibin in the male. *J Androl*. 1996;17:179 -182.[Free Full Text]

Depuydt CE, Mahmoud AM, Dhooge WS, Schoonjans FA, Comhaire FH. Hormonal regulation of inhibin B secretion by immature rat Sertoli cells in vitro: possible use as a bioassay for estrogen detection. *J Androl*. 1999;20:54 -62.[Abstract/Free Full Text]

Dowling CR, Risbridger GP. The role of inhibins and activins in prostate cancer pathogenesis. *Endocr Relat Cancer*.2000; 7:243 -256.[Abstract]

Foresta C, Bettella A, Petraglia F, Pistorello M, Luisi S, Rossato M. Inhibin B levels in azoospermic subjects with cytologically characterized testicular pathology. *Clin Endocrinol (Oxf)*.1999; 50:695 -701.[Medline]

Glander HJ, Horn LC, Dorschner W, Paasch U, Kratzsch J. Probability to retrieve testicular spermatozoa in azoospermic patients. *Asian J Androl*. 2000;2:199 -205.[Medline]

Handelsman DJ, Spaliviero JA, Phippard AF. Highly vectorial secretion of inhibin by primate Sertoli cells in vitrol. *J Clin Endocrinol Metab*. 1990;71:1235 -1238.[Abstract]

Kamischke A, Simoni M, Schrameyer K, Lerchl A, Nieschlag E. Is inhibin B a pharmacodynamic parameter for FSH in normal men? *Eur J Endocrinol*. 2001;144:629 -637.[Abstract]

Le Lannou D, Chambon Y, Le Calve M. Role of the epididymis in reabsorption of inhibin in the rat. *J Reprod Fertil Suppl*. 1979;26:117 -121.

Maddocks S, Sharpe RM. The route of secretion of inhibin from the rat testis. *J Endocrinol*.1989; 120:R5 -R8.[Abstract/Free Full Text]

Mahmoud AM, Comhaire FH, Depuydt CE. The clinical and biologic significance of serum inhibins in subfertile men. *Reprod Toxicol*. 1998a;12:591 -599.[Medline]

Mahmoud AM, Geslevich J, Kint J, Depuydt C, Huysse L, Zalata A, Comhaire FH. Seminal plasma alpha-glucosidase activity and male infertility. *Hum Reprod*.1998b; 13:591 -595.[Abstract/Free Full Text]

Martin CW, Riley SC, Everington D, Groome NP, Riemersma RA, Baird DT, Anderson RA. Dose finding study of oral desogestrel with testosterone pellets for suppression of the pituitary-testicular axis in normal men. *Hum Reprod*.2000; 15:1515 -1524.[Abstract/Free Full Text]

Pierik FH, Vreeburg JT, Stijnen T, De Jong FH, Weber RF. Serum inhibin B as a marker of spermatogenesis. *J Clin Endocrinol Metab*. 1998;83:3110 -3114.[Abstract/Free Full Text]

Raivio T, Saukkonen S, Jaaskelainen J, Komulainen J, Dunkel L. Signaling between the pituitary gland and the testes: inverse relationship between serum FSH and inhibin B concentrations in boys in early puberty. *Eur J Endocrinol*.2000; 142:150 -156.[Abstract]

Salzbrunn A, Benson DM, Holstein AF, Schulze W. A new concept for the extraction of testicular spermatozoa as a tool for assisted fertilization (ICSI). *Hum Reprod*.1996; 11:752 -755. [Abstract/Free Full Text]

Sanford LM, Price CA, Leggee DG, Baker SJ, Yarney TA. Role of FSH, number of FSH receptors and testosterone in the regulation of inhibin secretion during the seasonal testicular cycle of adult rams. *Reproduction*.2002; 123:269 -280.[Abstract]

Schanbacher BD. Pituitary and testicular responses of beef bulls to active immunization against inhibin alpha. *J Anim Sci*.1991; 69:252 -257.[Abstract]

Schoonjans F, Zalata A, Depuydt CE, Comhaire FH. MedCalc: a new computer program for medical statistics. *Comput Methods Programs Biomed*. 1995;48:257 -262.[Medline]

Scott RS, Burger HG. An inverse relationship exists between seminal plasma inhibin and serum follicle-stimulating hormone in man. *J Clin Endocrinol Metab*.1981; 52:796 -803.[Abstract]

Thomas TZ, Chapman SM, Hong W, Gurusingfhe C, Mellor SL, Fletcher R, Pedersen J, Risbridger GP. Inhibins, activins, and follistatins: expression of mRNAs and cellular localization in tissues from men with benign prostatic hyperplasia. *Prostate*.1998; 34:34 -43.[Medline]

Vaze AY, Thakur AN, Sheth AR. Levels of inhibin in human semen and accessory reproductive organs. *Andrologia*.1980; 12:66 -71.[Medline]

von Eckardstein S, Simoni M, Bergmann M, Weinbauer GF, Gassner P, Schepers AG, Nieschlag E. Serum inhibin B in combination with serum follicle-stimulating hormone (FSH) is a more sensitive marker than serum FSH alone for impaired spermatogenesis in men, but cannot predict the presence of sperm in testicular tissue samples. *J Clin Endocrinol Metab*. 1999;84:2496 -2501.[Abstract/Free Full Text]

World Health Organization. *WHO Laboratory Manual for the Examination of Human Semen and Sperm-Cervical Mucus Interaction*. 4th ed. Cambridge, United Kingdom: Cambridge University Press;1999 .

This article has been cited by other articles:

Abstract FREE
Full Text (PDF)
Alert me when this article is cited
Alert me if a correction is posted

Services

Similar articles in this journal
Similar articles in PubMed
Alert me to new issues of the journal
Download to citation manager

Citing Articles

Citing Articles via HighWire
Citing Articles via Google Scholar

Google Scholar

Articles by Garem, Y. F. E.
Articles by Comhaire, F. H.
Search for Related Content

PubMed

PubMed Citation
Articles by Garem, Y. F. E.
Articles by Comhaire, F. H.

HOME HELP FEEDBACK SUBSCRIPTIONS ARCHIVE SEARCH TABLE OF CONTENTS