FSH treatment for men with OTA: the impact on sperm microstructure and fertilization potential

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- Male factor is the sole cause of infertility in approximately 30% of infertility couples, and an additional 25% for causes of mixed factors
- Many aspects of male factor infertility are poorly understood, and not deeply investigated in the general practice.

- Most patients are classified as having idiopathic oligoteratoasthenozoospermia (OTA)

- The high fertilization and pregnancy rates achieved with ICSI have led to the almost complete discontinuation of efforts to improve sperm parameters in vivo.

- The wisdom of abandoning such efforts should be questioned because the importance of good sperm quality for embryonic development is only now beginning to be appreciated.

- ICSI bypasses physiological selection processes, allowing sperm with severe structural abnormalities and/or increased aneuploidy rates to fertilize oocytes.
- Moreover, an increased rate of De Novo chromosomal abnormalities in the offspring of the patients was observed in several reports.

In't Veld P. Lancet (letter) 1995; 346: 773
Bonduelle M et al. Hum Reprod. 1996; 11:131-159
Chandley AC, Hargreave TB. Hum Reprod. 1996; 11:930-932
Bonduelle M et al. Hum Reprod. 1998; 13:781-782

It is essential that the justified enthusiasm surrounding the use of ICSI as the treatment of choice for male factor infertility, should not prevent us from improving sperm quality and thereby achieving better results in both IVF and ICSI.

There is a general consensus on the need for FSH in regulating and maintaining quantitative and qualitative spermatogenesis in animals and humans

Matsumoto et al. J Clin Endocrinol Metab.1986;62:1184-1190
Matsumoto, 1989; Sharpe, 1989
Acosta et al Fertil Steril.1991;55:1150-6 & Hum reprod 1992;7:1067-72
Bartoov et al Fertil steril 1994;61:727-34
Maclachlan RI et al. Endocrinology.1995;136:4035-4043
Marshall GR et al. Endocrinology. 1995; 136:3504-3511
Kim Seung Bum, Fert Steril 2002; 78:S266-S267

Although the role of FSH treatment in hypogonadotropic, hypogonadal man is indisputable

Finkel et al; N Engl J Med 1985; 313:651-5
Comb et al;J clin Endocrinol Metab 1990; 70:3-7
Pierre Bouloux et al; Fert Steril 2002; 77:270-273
D. Warns et al; Fert Steril 2005; 84:S220
H. Okada; Fert Steril Sep 2005; 84:S222

Its effect in normogonadotropic, Normogonadal men with OTA is still controversial and even failed to demonstrate a beneficial effect.

Knuth et al; J clin Endocrinol Metab 1987;65:1081-7
Lunenfeld et al; Andrologia 1979; 11:331-6
Kamischke et al; Hum Reprod. 1998;13:596-603

The Failure is due in part to :

1. The use of a low dose of FSH
2. The use of classic sperm parameters as study end points
3. The inclusion criteria of subjects affected by hypospermatogenesis with maturative disturbances
    a. Homozygous males with inactivating mutation of FSH receptor
    b. Subjects affected by a mutation in the FSH-β subunit gene *(philip et al 1998; Lindstedt G et al 1998)*

FSH is responsible for the quality of the spermatozoa in general and for the sperm-zona pellucida interaction in particular; accounting for the improvement in the fertilization rates

European Journal of obstetrics & Gynecology and reproductive biology; 93 (2000): 105-108

FSH therapy may positively influence testicular function by improving:

a. Sperm parameters
b. Sperm ultrastructure

The increase in Fertilization rates after FSH therapy, may be related to improvement in subcellular components of the sperm. This treatment could restore defective spermatozoal maturation, mainly of the Acrosomal and nuclear regions.

Bartoov et al, Fertil steril 1994; 61: 727-34

long-term exposure in vivo to exogenous FSH of normogonadotropic, normogonadal men with OTA and proven low fertilization potential resulted in fourfold increase in their sperm fertilization potential.

Ben-Rafael et al. Fertil Steril 2000; 73:24-30

Results of IVF after treatment FSH compared with control			
Result of IVF	Group 1 A (75 IU daily) (n=20)	Group 1 B (150 IU daily) (n=20)	Group 2 (control) (n=20)
Mean (±SD) no. of oocytes retrieved	6.2 ± 3.6	7.7 ± 3.1	8.9 ± 3.3
Mean (±SD) no. of oocytes fertilized	1.2 ± 2.0	1.8 ± 2.4	0.4 ± 0.6*
Mean (±SD) Fertilization rate	19.7 ± 23.0	20.5 ± 24.3	5.8 ± 9.9*
No. of pregnancies	1	2	0

P < .05 for group 2 vs group 1A and for group 2 vs. group B

Ben- Rafael. Fertil Steril 2000;73:24-30

Electron microscopy detected a significant increase of normal acrosomes, nuclei and Axonemes

The integrity of sperm cell subcellular organelles in patients before and after treatment with FSH according to their treatment group.

	Group 1A(n = 20)		Group 1B(n = 20)	
Subcellular Organelle	Before Treatment	After Treatment	Before Treatment	After Treatment
Acrosome	**30.5 ± 20.6***	**51.6 ± 22.8**	**26.2 ± 19.3***	**40.5 ± 25.9**
PAL	89.23 ± 26.0	83.7 ± 35.5	86.7 ± 29.1	87.8 ± 29.4
Nucleus	**17.0 ± 20.6***	**36.1 ± 24.5**	**37.4 ± 14.7**	**32.1 ± 18.1**
Karyoplasm	46.9 ± 23.9	55.1 ± 22.2	42.3 ± 23.3	41.6 ± 44.6
Neck	84.6 ± 5.4	84.7 ± 5.8	81.9 ± 8.3	82.0 ± 8.7
Mitochondria	91.8 ± 4.7	90.0 ± 8.1	90.8 ± 11.3	96.2 ± 3.0
Fibrous sheath	89.0 ± 8.6	83.7 ± 8.6	78.7 ± 13.3	72.8 ± 15.7
Axoneme	**17.0 ± 28.6***	**54.3 ± 30.9**	**26.3 ± 19.0**	**33.1 ± 33.2**

Note: values are percentage intact (± SD). PAL= postacrosomal lamina. * P< .01

Ben- Rafael. Fertil Steril 2000;73:24-30

Idiopathic oligozoospermic patients with Ⓝ FSH and Ⓝ Inhibin B plasma levels, and a testicular structure of moderate hypospermatogenesis without maturational arrest, are generally good responders to FSH therapy.

Foresta 2000. Mol Cell Endocrinol; 161:89-97

Caroppo 2003. Fertil Steril; 80 (No.6): 1398-1403

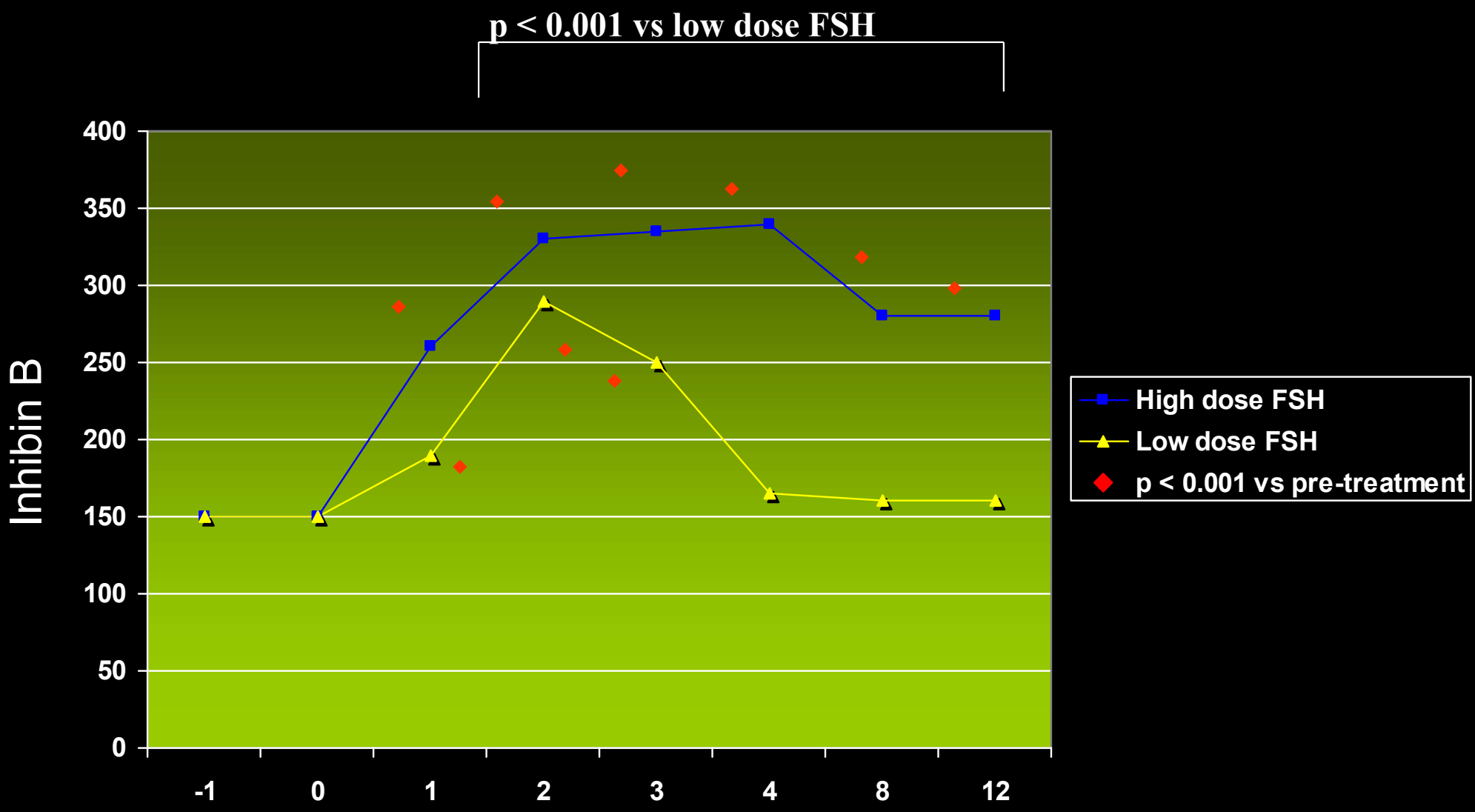
Foresta 2005. Fertil Steril; 84(No.3):654-661

Paradisi 2006. Fertil Steril; 86(No.3): 728-731

	n	FSH(IU/L)	Inhibin B (pg/ml)	Sperm number (x 10^6/ml)	Testicular volume (ml)	Sertoli index (%)	Spermatids (%)
Group **A**	77	3.5 ± 1.8	158.0 ± 81.9	10.4± 3.7	14.5 ± 2.5	279.0 ±220.1	43.2 ± 20.1
Group **B**	25	12.5 ± 5.8*	107.4 ± 36.6	3.5 ± 3.9*	13.2 ± 2.0	507.6 ±284.5	28.5 ± 11.6
Group **C**	33	18.9 ± 10.7*	50.2 ± 19.3*	1.9 ± 2.5*	10.8 ± 2.9*	727.5± 225.6*	17.7 ± 9.4*

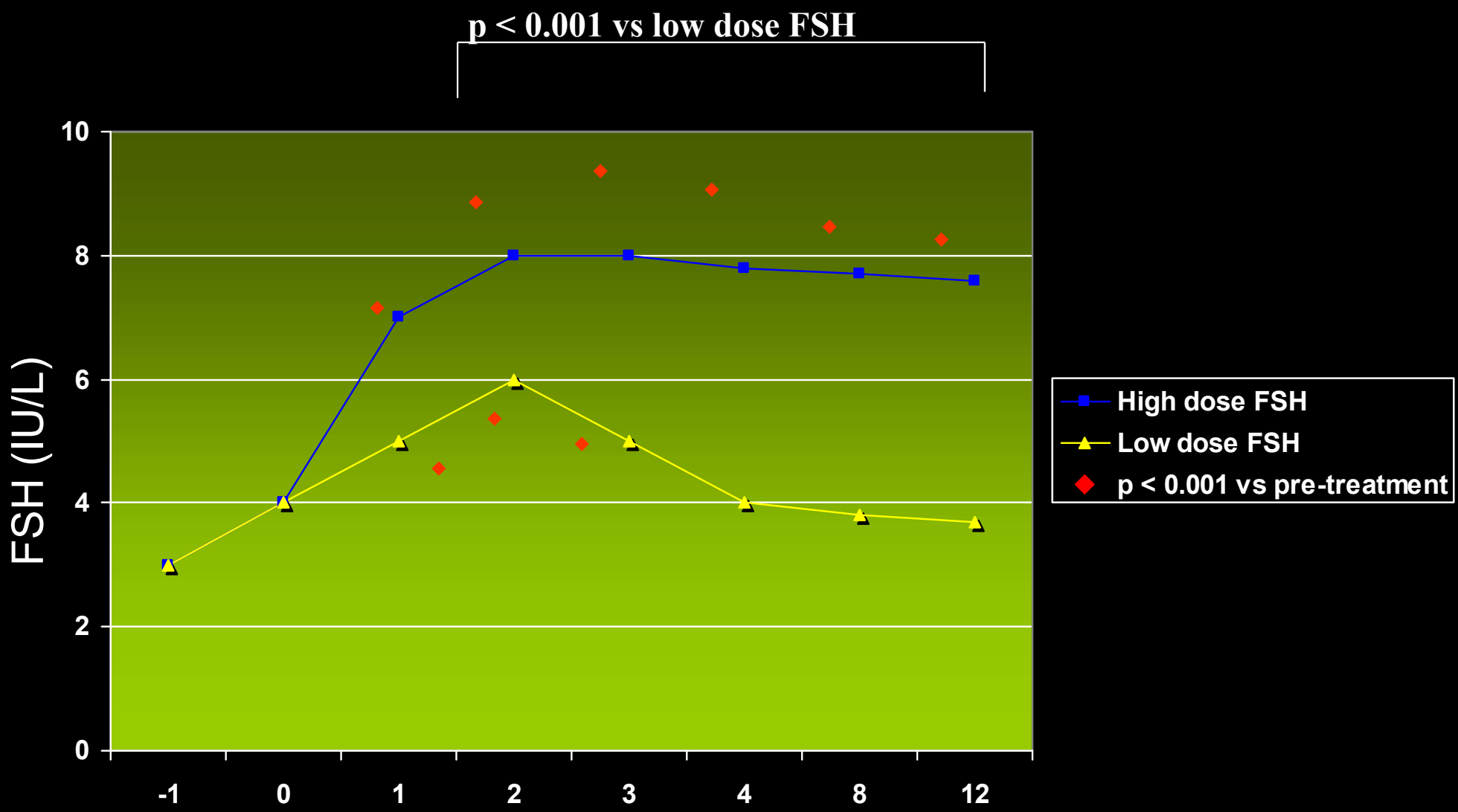
**P* < 0.01 versus group A.

C.Foresta et al. / Molecular and Cellular Endocrinology; 161 (2000): 89-97



Effects of FSH treatment on inhibin B plasma levels in oligozoospermic subjects of group A.

C.Foresta et al. / Molecular and Cellular Endocrinology ;161 (2000): 89-97



Effects of FSH therapy on FSH plasma levels in oligozoospermic subjects of group A.

C.Foresta et al. / Molecular and Cellular Endocrinology; 161 (2000): 89-97

FSH treatment increases the spermatogonial population in oligozoospermic subjects with FSH plasma levels associated with Ⓝ hypospermatogenesis without maturational disturbances

Foresta et al. Fertil Steril 1998; 69: 636-42

Foresta et al. Fertil steril 2002; 77:238-44

Cell Type	Controls	Pretreatment		Post-treatment	
	(n = 40)	Nonresponders (n = 40)	Responders (n = 20)	Nonresponders (n = 40)	Responders (n = 20)
Spermatogonia	1.4 ± 1.2	1.8 ± 1.2	1.2 ± 1.1	5.2 ± 1.2* †	5.3 ± 2.4* †
Spermatocytes	7.9 ± 3.6	8.3 ± 3.8	6.6 ± 3.4	17.2 ± 5.6*	12.0 ± 2.8 † ‡
Spermatids	41.9 ±12.9	61.6 ± 13.6 †	44.2 ± 11.4	68.0 ± 9.2	35.7 ± 15.6
Spermatic Index	48.8 ±13.2	28.3 ± 13.4 †	46.9 ± 13.7	9.4 ± 7.1 ♑	48.1 ± 9.8
Sertoli Index	30.4 ±11.6	88.3 ± 31.6 †	106.2±44.1 †	56.4 ± 25.1‡	68.3 ± 35.5*

Note. All values are means ± SD and percentages.

Response is defined as a doubling of sperm count after treatment.

*P<0.01 (vs. pretreatment)

† P<0.01 (vs. controls)

‡ P<0.05 (vs. pretreatment)

♑ P<0.01 (vs. pretreatment)

Sperm Characteristics	FSH 75 IU on alternate day (*n* = 47)		FSH 75 IU on every day (*n* = 30)	
	Before	After	Before	After
Sperm number ($x10^6$/ml)	9.8 ± 3.9	14.0 ± 7.3	10.8 ± 3.6	15.6 ± 8.2
Total Sperm ($x10^6$)	16.6 ± 7.4	24.2 ±10.2	17.8 ± 8.2	28.5 ± 9.9
Normal Morphology (%)	33.6 ± 4.8	37.1 ± 5.2	37.4 ± 5.1	39.1 ± 5.6
Forward motility (%)	29.6 ± 5.5	31.8 ± 5.3	27.4 ± 4.9	33.8 ± 6.2

C.Foresta et al. / Molecular and Cellular Endocrinology; 161 (2000): 89-97

Clinical, hormonal, and seminal parameters of 23 infertile male patients before and after treatment with 150IU recombinant human FSH and of 10 untreated (controls)

	Treated patients		Controls	
parameter	Before treatment	After treatment	First evaluation	Second evaluation (3mo later)
Testicular volume (mL)	12.5 ± 4.77 (5-25)	15.1 ± 5.48 (6-25)+	12.3 ± 2.2 (9-15)	12.4 ± 2.06 (10-15)
Sperm count ($x10^6$/mL)	1.3 ± 2.2 (0.05-10)	3.8 ± 6.5 (0.1-22)*	2.54 ± 2.2 (0.1-6)	2.65 ± 2.1 (0.1-6)
No. motile sperm ($x10^6$/mL)	0.32 ± 0.57 (0-2)	0.79 ± 1.58 (0-6.7)*	0.9 ± 1.15 (0.01-3.2)	0.7 ± 0.7 (0.01-2.15)
% Normal morphology	23.9 ± 8.2 (5-40)	29.1 ± 9.34 (15-50)+	30 ± 12.9 (10-45)	27.3 ± 11.7 (10-40)
% Viable sperm	25.3 ± 15.5 (5-60)	37.04 ± 10.8 (20-65)++	22.2 ± 5.11 (15-30)	23.1 ± 8.5 (12-44)
FSH (IU/mL)	9.68 ± 6.05 (1.6-27)	Not assessed	10.53 ± 6.8 (2.5-27)	Not assessed
FSH (IU/mL)	6.01 ± 3.2 (0.8-12)	Not assessed	5.96 ± 3.8 (2.2-14)	Not assessed
T (ng/mL)	4.55 ± 1.29 (2-6.9)	Not assessed	4.25 ± 1.6 (2.3-7.3)	Not assessed
E-T ratio	5.95 ± 4.6 (25-40)	Not assessed	4.64 ± 2.7 (2-10.8)	Not assessed
Inhibin B (pg/mL)	90.5 ± 58.6 (21-242)	144 ± 196.97(20-937)	111.7 ± 56.9 (54-234)	100.1 ± 49.25 (48-200)

values are means ± SD, with range in parentheses, **P<.05 vs. pretreatment, +P<.01 vs pretreatment,* ++P<.001 vs. pretreatment.

Caroppo. FSH treatment for OAT patients before ICSI. Fertil Steril 2003

Clinical, hormonal, and seminal parameters of seven infertile male patients treated with 150 IU rhFSH before an ICSI cycle resulted in clinical pregnancy		
Parameter	Before treatment	After treatment
Testicular volume (ml)	14.5 ± 6.43 (8-25)	16.8 ± 6.08 (10-25)
Sperm count (x10^6/ml)	0.68 ± 0.84 (0.1-2)	4.15 ± 7.02 (0.2-18)
No. Motilc sperm (x10^6/ml)	0.18 ± 0.4 (0-1)	0.59 ± 1.18 (0-3)
% Normal morphology	25.7 ± 8.6 (15-35)	35 ± 9.12 (25-50)
% Viable sperm	29.42 ± 13.5 (15-55)	34.2 ± 14.9 (20-65)
FSH (IU/mL)	8.32 ± 4.89 (2.9-14.1)	Not assessed
LH (IU/mL)	7.05 ± 3.0 (2.8-10.3)	Not assessed
T (ng/mL)	5.17 ± 1.07 (4-6.7)	Not assessed
E-T ratio	6.0 ± 3.6 (2.76-13.75)	Not assessed
Inhibin B (pg/mL)	80.85 ± 55.69 (21-169)	99.8 ± 68.2 (20-223)

values are means ± SD, with range in parentheses, **P<.05 vs. pretreatment, +P<.01 vs pretreatment,* ++P<.001 vs. pretreatment.

Caroppo. FSH treatment for OAT patients before ICSI. Fertil Steril 2003

Sperm parameters observed during the different periods of the study in responder and non responder groups (treated subjects) and in the infertile-control group (no treatment)

	Responder group (n=30)		Non-responder group (n=32)		Infertile-control group (n=50)	
	Sperm count (x10^6/mL)	Total Spermatozoa (x10^6)	Sperm count x10^6)	Total spermatozoa (x10^6)	Sperm count (x10^6 /ml)	Total spermatozoa (x10^6)
Basal	6.4 ± 3.6	17.1 ± 7.7	6.1 ± 3.3	14.8 ± 6.6	6.8 ± 3.2	16.5 ± 5.1
End of therapy period	19.8 ± 3.9 [a]	44.8 ± 8.6[a]	7.7 ± 4.8	16.1 ± 6.7	7.1 ± 3.5	17.3 ± 6.6
End of follow-up Period	16.4 ± 3.3 [b]	33.6 ± 6.6[b]	7.9 ± 5.1	17.0 ± 6.4	8.1 ± 3.6	17.4 ± 5.3
End of ART period	10.6 ± 4.5	21.1 ± 6.9	6.8 ± 4.4	15.1 ± 5.9	7.4 ± 4.1	16.1 ± 6.1

Therapy period: Rx Rec-FSH (100IU /IM) q other day x 3 months
F/up period: 3 months after withdrawal of therapy. ART: 3 months

Foresta. Fertil Steril 2005;84(issue3):654-661

Improvement in sperm motility, morphology and vitality after FSH therapy may indicate a beneficial action on sperm organelles

Strehler et al. J Androl 1997;18:439-47

Caroppo Fertil Steril 2003 ; 80(No.6): 1398-1403

Baccetti et al (1997) distinguished five categories of major sperm defects:

- Immaturity
- Infection
- Apoptosis
- Autoantibodies
- Azoospermia/spermatogenetic arrest

+ve responses were obtained in immature or apoptotic spermatozoa and infected sperm (when antibiotics were started before or with FSH therapy).

Effect of follicle stimulating hormone (FSH) therapy on the ejaculates of treated patients, divided into responders and non-responders, according to the formula of Baccetti et al. (1995)

	No. patients	Mean of the No. of healthy spermatozoa	Mean of the % of healthy spermatozoa
Before FSH therapy	**Responders (n = 47)**	**79 548**	0.163 [a]
	Non-responders (n = 19)	217 039	0.389
12 weeks FSH therapy	**Responders (n = 47)**	**570 024**	1.717[b]
	Non-responders (n = 19)	66 532	0.131
6-12 weeks after end of FSH therapy	**Responders (n = 34)**	**1.605×10^6**	1.829
	Non-responders (n = 14)	31 777	0.150

a,b values significantly different ($p < 0.05$, paired t-test)

B. Baccetti et al. Hum Reprod 1997;12(No.9):1955-1968

Effect of FSH therapy and placebo treatment upon patients with different categories of sperm defect

	No. patients	Mean no. spermatozoa/ml
Before FSH therapy	66	10 865 182
Responders:	47	10 576 596
Apoptosis	23	11 565 217
Infection	20	7 655 000
Immaturity	4	19 500 000
Non-responders	19	11 589 000
Before placebo treatment	15	11 672 133

After FSH therapy	66	12 716 666
Responders:	47	14 057 000
Apoptosis	23	16 091 000
Infection	20	10 030 000
Immaturity	4	22 500 000
Non-responders	19	9 400 000
After placebo treatment	15	10 542 312

6-12 weeks After end of FSH therapy	48	13 664 583
responders:	34	14 441 000
Apoptosis	16	14 937 000
Infection	15	11 400 000
Immaturity	3	27 000 000
Non-responders	14	10 750 000

B. Baccetti et al. Hum Reprod 1997;12(No.9):1955-1968

The ultrastructure sperm components that respond to FSH therapy in the 3 groups are:

1. The nucleus, the chromatin and its compaction
2. The mitochondria, (Helix assembly)
3. The Axoneme (9 + 2 pattern; regular dynein arms)
4. Plasma membrane integrity

The acromosal complex and the fibrous sheath were modestly and variable sensitive to FSH

Variable	Study Group	Control Group
Mean (± SD) no.of embryos transferred	3.1 ± 1.8	3.8 ± 1.7
Mean (± SD) no.of grade A embryos	2.2 ± 1.6*	1.6 ± 1.6
Clinical pregnancy rate (%)	35.9	17.9
Implantation rate (%)	15.5 †	6.5

**P = .08 ,* † P <0.5

Ashkenazi. Purified FSH. Fertil Steril 1999

75 % of OTA normogonadotropic subjects treated with u-FSH x 6 months, showed a:

1. Bilateral significant increase in intratesticular blood flow
2. a decrease of resistance index
3. an increase of flow capacity of the same vessels
4. Enhancement of oxygen supply and consequently, reduction of germ cells exposure to reactive oxygen species (responsible for DNA breakage and lipid peroxidation)

F Causio et al. Fertil Steril 2002; 78: 1133-1135
Potts et al. Mutat Res 2000; 447:249-56

Successful Fertilization, in-vitro fertilization and spontaneous pregnancy rate in follicle-stimulating hormone treated and untreated patients.

Patients	Group	No	Fertilization per patient (%)	Fertilization per oocyte (%)	IVF pregnancy (%)	Spontaneous pregnancy (%)
Treated (Group I)	Severe OTA	32	66.4	29.4	6.5	28.1*
	Failed Fertilization	44	63.2	26.1	16.7	15.9
Untreated (Group II)	Control	102	72.8	38.4	12.7	4.9

M. Dirnfeld et al. / European Journal of Obstetrics & Gynecology and Reproductive Biology 93 (2000):105-108

Pregnancy rates during the follow-up and ART periods in responder and nonresponder groups (treated subjects) and infertile-control group (no treatment).

		ART period			
	Follow-up period spontaneous	**IUI**	**IVF-ET**	**ICSI**	**Cumulative**
Treated subjects (n= 62)	9.7 (6/62)	20.0 (3/15)	23.8 (5/21)	20.0 (4/20)	29.0 (18/62)
Responder group (n=30)	16.7 (5/30) [a]	**20.0 (3/15)**	**30.0 (3/10)**	-	36.7 (11/30)
Nonresponder group (n=32)	3.1 (1/32)	Nonsuitable	18.2 (2/11)	20.0 (4/20)	21.9 (7/32)
Infertile-control group (n=50)	4.0 (2/50)	Nonsuitable	22.2 (4/18)	20.0 (6/30)	24.0 (12/50)

Note : Data are expressed as percentage (number)

Foresta. Fertil Steril 2005;84(issue 3):654-661

Dosage & duration of Therapy

- FSH treatment protocols used in most studies were empiric in regard to both dosage and injection interval.
- Urinary, purified and rec-FSH were used for 30, 50, 71 days and for 3 to 6 months on alternate days or daily injections.
- The 3 months duration therapy is the most commonly used in the studies to affect all the stages of new whole spermatogenic and spermiogenic cycle and its normalization

Adamopoulos DA. Int J Androl 2000;23:320-331

The dose of 50 IU seems to be insufficient to induce any effect on the testicular tubal level, nor a modification of the sperm concentration

Foresta et al. Fertil Steril 2002; 77: 238-44

	Non treated patients		Treated Patients			
	(n = 15)		r-hFSH 50 IU n=15		r-hFSH 100 IU (n=15)	
	Basal	After 3 months	Pretreatment	Post-treatment	Pretreatment	Post-treatment
Spermatozoa ($x10^6$)	4.3 ± 2.1	5.6 ± 2.9	3.7 ± 1.8	5.8 ± 2.6	5.1 ± 2.2	9.6 ± 3.6[a]
Normal Morphology (%)	35.6 ± 8.8	36.8 ± 10.3	34.2 ± 10.1	33.5 ± 9.3	37.8 ± 9.4	43.2 ± 7.4
Forward motility (%)	28.6 ± 6.7	27.6 ± 8.5	30.4 ± 7.5	32.2 ± 9.6	35.5 ± 11.6	37.2 ± 8.8

[a] *p< .05 vs. pretreatment*

Foresta. R-hFSH in treatment of male factor infertility. Fertil Steril 2002

75 IU FSH is the minimum efficient dose able to influence human steroli cell functions in vivo and promote spermatogenesis.

Foresta and Varotto 1994

- It is conceivable that the daily use of FSH is more effective, since bio-FSH has a half life of 13.4 hours.
  - Jockenhovel et al; Clin Endocrinol (Oxf) 1990;33:573-84
  - Out HJ et al; Hum Reprod 1996;11:61-3
  - Achkenazi et al; Fertil Steril 1999; 72:670-3
- Similar effects were shown by other investigators on an alternate-day administration but failed to maintain higher FSH and Inhibin B levels 4 weeks after cessation of therapy.

  Foresta et al; Molecular and Cellular Endocrinology;161(2000);89-90

No difference between 150 IU rhFSH daily and 300 IU q other day because serum FSH reaches the maximal pharmacologic effect within 3-4 days with rhFSH therapy (in a dose-proportional fashion), maintaining thereafter new steady levels throughout the whole treatment and even few weeks after withdrawal

R Paradisi et al. Fertil Steril 2006; 86(issue 3): 728-731

To Remember

- Normal FSH value does not exclude severe derangement of spermatogenesis

  Bergmann et al.Clin Endocrinol1994 ;40:133-136

- Elevated FSH concentration is insufficient to indicate a damaged germinal epithelium nor Sertoli-cell syndrome

  Bar.On et al. Fertil Steril 1995;64:1043-1045

  Marin-du-Pan and Bischof. Hum Reprod (1995); 10:1940-1945

- Inhibin B levels are not always correlated to spermatogenic function nor predict the type of spermatogenetic damage

Anderson et al. J Clin Endocrinol metab. 1998;83:4451-4458
Kardstein S et al. J Clin Endocrinol Metab. 1999;84:2496-2501

- Inhibin B negatively correlates with T that controls its secretion by the steroli cell.

The negative control of Inhibin B with FSH, LH and Testo suggest its intact role in the feedback regulation

E Caroppo et al. Fertil Steril 2003; 80:1398-1403

FSH plays a critical role in stimulating mitotic and meiotic DNA synthesis in spermatogonia and preleptotene spermatocytes through its action on Sertoli cells

Stanton et al. Endocrinology 1992; 130:2820-32

Krishnamurthy et al. J Androl 2000;21:316-27

Summary
FSH Therapy

1. Promotes the expression of FSH-receptors on Sertoli and germ cells-surface
   - Post-receptor response ➡ increased
   - Spermatogenesis ➡ improves
   - Inhibin B secretion ➡ increases
2. Pretreatment Inhibin B levels ➡ a result of the functional status of H-P-T Axis

Integrity of H-P-T Axis is not essential for good fertilization rate if Rx is established.

3. Post-treatment Inhibin B levels ➡ a consequence of testicular paracrine activity in response to exogeneous Rec FSH stimuli
4. +ve correlation between post-treatment inhibin B levels, and Fertilization rate could imply that improved sperm structure is the outcome of an enhanced testicular microenvironment.

- Rec-FSH may be a valuable pretreatment for oligozoospermic patients undergoing ICSI by improving sperm parameters, sperm structure and Fertilization rate.
- The improvement of Inhibin B levels after therapy is the result of Rec-FSH action on testicular paracrine activity.

Conclusion

FSH therapy in Normogonadotropic men with OTA (moderate hypospermatogenesis) without maturational disturbances, is proved to be beneficial.

a. FSH and Inhibin B levels increases.
b. Qualitative and quantitative sperm are improved specially the integrity of sperm organelles that are crucial for embryonic growth.
c. Defective spermatozoal maturation, mainly the Acrosome and nuclear regions are mostly restored which may influence fertilization and implantation rates and potential before IVF/ICSI.

MIDDLE EAST FERTILITY SOCIETY

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14th ANNUAL MEETING

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