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ASSISTED REPRODUCTION TECHNOLOGIES

# Serum Anti-müllerian hormone, follicle stimulating hormone and antral follicle count measurement cannot predict pregnancy rates in IVF/ICSI cycles

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**Abstract**
*Purpose* To investigate whether serum anti-müllerian hormone (AMH), follicle stimulating hormone (FSH), or antral follicle count (AFC) are predictive for clinical pregnancy in in vitro fertilization (IVF) patients.
*Methods* Serum AMH, inhibin B, FSH, luteinizing hormone (LH), estradiol (E2), prolactin, and thyroid stimulating hormone (TSH) levels and AFC of 189 women under 40 years of age were investigated. Pregnant and non-pregnant women were compared.
*Results* Forty-seven (24.8%) clinical pregnancies were observed in 189 women. There was no significant difference in terms of mean age, duration of infertility, body mass index, AMH, LH, FSH, E2, TSH, Inhibin B, AFC and total oocyte number between women who did and who did not become pregnant. Additionally, there was no significant difference in clinical pregnancy rates between the quartiles of AMH, FSH and AFC. (*P* values were 0.668, 0.071, and 0.252, respectively.)
*Conclusion* Serum AMH and FSH, and AFC cannot predict clinical pregnancy in IVF patients under 40; the pregnancy rate tends to increase as AMH increases, although this remains non-significant.

**Keywords** Antimüllerian hormone · Follicle stimulating hormone · Antral follicle count · Clinical pregnancy rate

*Capsule* Serum AMH and FSH, and AFC cannot predict clinical pregnancy in IVF patients under 40; the pregnancy rate tends to increase as AMH increases, although this remains non-significant.

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## Introduction

The reproductive capacity of a woman depends on many factors. Prediction of ovarian reserve has long been the golden key of reproductive endocrinology. Various endocrine [follicle stimulating hormone (FSH), inhibin B, estradiol (E2) etc.], and ultrasound tests [ovarian volume, antral follicle counts (AFC)] have been suggested to improve prediction of oocyte yield and pregnancy outcome following assisted reproductive technologies (ART) [1]. Currently, most in vitro fertilization (IVF) clinicians determine starting doses of gonadotropin in the first cycle of IVF based principally on the patient's age and basal FSH levels [2].

The established predictors of reproductive potential during infertility treatment are maternal age [3, 4], early follicular phase FSH concentrations [5, 6], and less popularly, serum inhibin B concentration [7]. None of these parameters is a particularly reliable predictor of the number or quality of oocytes remaining within the ovary, or the

likelihood of pregnancy from infertility treatment. Recently, interest in the use of anti-müllerian hormone (AMH) and AFC to predict patient response to ovarian stimulation has been intense [8].

A relatively new marker, AMH, was first identified as a specific protein in Sertoli cells of fetal testis, which inhibits the development of the mullerian duct [9]. AMH, a member of the transforming growth factor-beta super-family, is only produced by the granulosa cells surrounding preantral and small antral follicles in the ovary [10, 11]. AMH has been shown to decrease the sensitivity of preantral and small antral follicles to FSH [12], and its production is independent from that of FSH. AMH expression decreases during the FSH-dependent final stages of follicular growth [13] and atretic follicles do not express AMH [14]. Serum AMH levels decrease throughout reproductive life and are undetectable in postmenopause. Body mass index (BMI) does not seem to have an effect on serum AMH levels in reproductive age women, both with and without polycystic ovary syndrome (PCOS) [15].

There have been several studies about the relationship between AMH and oocyte or embryo quality [16–18]. The role of AMH in predicting pregnancy rates in normal responders has not been fully addressed in the literature. The aim of this cross-sectional study was to evaluate the association between AMH levels and pregnancy rates as well as to discover the highest pregnancy rates according to AMH levels.

## Materials and methods

Two-hundred consecutive women who were admitted to Istanbul University, Cerrahpasa School of Medicine, Department of Obstetrics and Gynecology, IVF Center with infertility from February 2009 to June 2010 were enrolled in our prospective cohort study. STROBE guidelines were followed.

The inclusion criteria were as follows: <40 years old, FSH <15 mIU/mL, prolactin <50 ng/ml, thyroid stimulating hormone (TSH) <5.0 mIU/L. The exclusion criteria were current or past diseases such as hepatic, renal, adrenal or thyroid disorders, affecting ovaries or gonadotropin or sex steroid secretion, clearance, or excretion.

Eleven women were excluded during the study. Five (2.5%) women had no follicle above 18 mm at the end of the controlled ovarian hyperstimulation therefore no oocyte pick-up was performed. Ovarian hyperstimulation was encountered in 2 (1%) women, 2 (1%) couples requested cryopreservation, one (0.5%) woman withdrew her informed consent, one (0.5%) woman moved to another city. One hundred eighty-nine consecutive women were evaluated prospectively.

No woman reported use of any medication that could interfere with the normal function of the hypothalamic-pituitary-gonadal axis during the last three months. In all women, body weight and height were measured; BMI was calculated with electronic digital scales (Mercury, AMZ 14, Tokyo, Japan) and in light clothing; height was measured barefoot with a stadiometer (G-Tech International CO LTD, Kyonggi Province, Korea).

Blood samples were collected during the early follicular phase of menses in all women. AMH, inhibin B, FSH, luteinizing hormone (LH), E2, prolactin, and TSH were measured in all women.

AMH concentrations were measured with an enzymatically amplified two-sided immunoassay [DSL-10-14400 Active Müllerian Inhibiting Substance/AMH enzyme-linked immunosorbent assay (ELISA) kit, Diagnostic Systems Laboratories (DSL), Webster, TX]. The theoretical sensitivity of the method is 0.006 ng/ml, the intra-assay coefficient of variation for high values is 3.3%, and the interassay coefficient of variation for high values is 6.7%.

Serum inhibin B levels were determined by using a double antibody ELISA (Serotec, Varilhes, France). Functional sensitivity was 15 pg/ml, and intra-assay and interassay coefficients of variation were <6 and <9%, respectively.

Serum E2, LH, and FSH were measured on a Roche E-170 automated immunoassay analyzer. Between-batch coefficients of variation for these assays were 10%. 17-hydroxyprogesterone (OH-P) was measured by RIA with intra-assay CV less than 7% (DSL, Webster, TX). TSH was measured by colorimetric immunoassay (Dimension RxL clinical chemistry analyzer; Dade, Newark, DE) with a sensitivity of 0.01 mIU/l, a precision of less than 6.2% at all concentrations tested and calibrated for the range of 0.01–50 mIU/l. The manufacturer's reference range was 0.34–4.82 mIU/l.

Transvaginal ultrasound scans of the ovaries were performed by experienced sonographers who participated in the study. The presence of polycystic ovaries was diagnosed by the appearance of 12 or more follicles in each ovary measuring 2–9 mm in diameter and/or increased ovarian volume (>10 $cm^3$). The presence of polycystic ovary syndrome (PCOS) was diagnosed by the Rotterdam-2003 criteria.

All patients received gonadotropin-releasing hormone (GnRH) agonist, leuprolide acetate 1 mg/day s.c. (Lucrin®, Cedex, France) beginning on the 21st day of the previous cycle. Leuprolide acetate was reduced to 0.5 mg/day, and gonadotropin (Gonal F®, Serono, Swiss or Puregon®, Schering Plough, Istanbul) 150 IU for patients <30 years old and 225 IU for patients >30 years old was started daily IM. A transvaginal ultrasound scan was arranged on days 7 and 9 of ovarian stimulation and every 1 or 2 days thereafter, as required. The dose of the gonadotropin was changed according to the follicular growth. When more than 2 follicles were seen that were >17 mm, hCG (Pregnyl®, 10,000 IU, Schering Plough, Istanbul or Ovitrelle® 250 mcg, Serono, Swiss) was injected to induce final oocyte

maturation, and 36 h later, ovum pick-up (OPU) was performed. The embryos were transferred after 3 days if fertilization had occurred. The luteal phase was supported with progesterone 90 mg administered by the vaginal route once or twice a day (Progynex® jel, Kocak, Istanbul, or Crinone gel® 8%, Merk Serono, Istanbul) or by 100 mg progesterone injection daily IM (Progynex® ampule, Kocak, Istanbul) until the day of the pregnancy test 12 days after the embryo transfer. Measurements of AMH were determined in duplicate using the AMH/MIS enzyme-linked immunosorbent assay kit (Diagnostic Systems Lab, Webster, TX, USA). The sensitivity of the assay was 0.017 ng/ml. The intra- and inter-assay variations were 5% and 8%, respectively. The FSH and E2 concentrations were estimated using the immulite semi-automated assay system. The study was approved by the local ethical committee.

In this study, clinical pregnancy was defined as the ultrasound observation of fetal heart movements at 7–8 weeks of gestation.

## Statistics

The data have been presented as the arithmetical means and the standard deviations were calculated for each group as well. An independent sample *t*-test was performed for evaluating the statistical relations between the subgroups. A *p*-value <0.05 was considered statistically significant. All statistical analyses were performed using the Statistical Package for the Social Sciences (SPSS) software version 15.0.

## Results

Forty-seven (24.8%) clinical pregnancies were observed in 189 women. Mean AMH was 3.85±2.93 ng/mL in all women. The 25th and 75th percentiles were 1.81 ng/mL and 4.92 ng/mL, respectively. Mean FSH was 6,11± 2.08 ng/mL; the 25th and 75th percentiles were 4.9 ng/mL and 6.9 ng/mL, respectively. Mean AFC was 8.9±5.2; the 25th and 75th percentiles were 5 and 10.5, respectively.

There was no significant difference in terms of mean age, duration of infertility, BMI, AMH, LH, FSH, E2, TSH, Inhibin B, AFC and total oocyte number between women who did and who did not become pregnant (Table 1).

The number of transferred embryos was 1.83±0.70 and 2.44±1.24 in pregnant and non-pregnant women, respectively; and the number of transferred eight-cell grade I embryos was 1.90±0.94 in <25% AMH group (*n*=47), 2.11±1.07 in 25-75% AMH group (*n*=94), and 2.09 ±0.98 in >75% AMH group (*n*=48) (*p*=0.255). The fertilization rate was 62.5±36.36, 67.18±26.97, and 67.30±24.33 in <25%, 25-75% AMH, and >75% AMH groups, respectively (*p*=0.723).

**Table 1** Comparison of demographical and clinical parameters in pregnant and non-pregnant women

| | Pregnancy (+) (*n*=47) | Pregnancy (−) (*n*=142) | *P* value |
|---|---|---|---|
| Age (years) | 30.7±4.0 | 31.7±4.7 | 0.199 |
| Duration of infertility (years) | 6.6±4.2 | 7.0±4.2 | 0.593 |
| BMI (kg/m$^2$) | 24.0±2.8 | 24.2±2.8 | 0.673 |
| AMH (ng/mL) | 3.9±2.5 | 3.8±3.0 | 0.831 |
| LH (mIU/ml) | 3.8±1.9 | 4.2±2.4 | 0.278 |
| FSH (mIU/ml) | 5.7±2.1 | 6.2±2.0 | 0.116 |
| E2 (pg/ml) | 40.0±19.0 | 46.0±25.1 | 0.166 |
| TSH (mIU/l) | 1.6±0.8 | 1.6±1.0 | 0.904 |
| Inhibin-B (pg/mL) | 88.0±56.8 | 88.7±53.0 | 0.940 |
| AFC (*n*) | 9.6±5.1 | 8.7±5.3 | 0.340 |
| Total Oocyte | 9.2±4.5 | 9.0±5.1 | 0.793 |

AFC, antral follicle count; AMH, anti-müllerian hormone; BMI, body-mass index; E2, estradiol; FSH, follicle stimulating hormone; LH, luteinizing hormone; TSH, thyroid stimulating hormone

*P*>0.05 is significant

Also there was no significant difference in clinical pregnancy rates between the quartiles of AMH, FSH, and AFC (Table 2). The clinical pregnancy rate was 21% in the patients whose serum AMH level was lower than 1.81 ng/ml and 29.2% in patients whose serum AMH level was higher than 4.92 ng/ml. But this increase remained non-significant (*p*= 0.068). The lowest level of serum AMH was 0.45 ng/ml and 0.01 ng/ml in pregnant and non-pregnant groups, respectively. The clinical pregnancy rate was 23.8%, 20% and 22% in <25%, 25-75% and >75% FSH groups, respectively (*p*=0.071). The clinical pregnancy rate was 18.8%, 24.5% and 34.9% in <25%, 25-75% and >75% AFC groups, respectively (*p*=0.252). The distributions of AMH and AFC in pregnant and non-pregnant groups were shown in Figs. 1 and 2, respectively.

## Discussion

The value of AMH in the prediction of pregnancy has been investigated in various studies which showed inconsistent results. Some studies suggest that serum AMH level is associated with pregnancy rates [1, 19–23]; whereas others suggested that serum AMH levels are not associated with pregnancy outcomes [8, 24–27]. Other markers such as AFC and inhibin B, which were thought to predict pregnancy, were also evaluated in many studies. In our study we detected that day 3 serum AMH, AFC, and inhibin B

**Table 2** Pregnancy rates according to the quartiles of AMH, FSH and AFC

| | <25% | | %25–75 | | >75% | |
|---|---|---|---|---|---|---|
| | Range (ng/mL) | Pregnancy rate | Range (ng/mL) | Pregnancy rate | Range (ng/mL) | Pregnancy rate |
| AMH | <1.81 ($n$=47) | 21.3% | 1.81-4.92 ($n$=94) | 24.5% | >4.92 ($n$=48) | 29.2% |
| FSH | <4.92 ($n$=45) | 23.8% | 4.92-6.97 ($n$=90) | 20% | >6.97 ($n$=45) | 22.2% |
| AFC | <5 ($n$=32) | 18.8% | 5-10.5 ($n$=106) | 24.5% | >10.5 ($n$=43) | 34.9% |

$P$ values for: Quartiles of AMH: 0.668, quartiles of FSH: 0.071, quartiles of AFC: 0.252

AFC, antral follicle count; AMH, anti-müllerian hormone; FSH, follicle stimulating hormone

measurements in normal responder women were not found to be associated with pregnancy rates.

Hazout et al. [21] evaluated 109 women (<42 years old) and demonstrated that day 3 serum AMH level and IVF outcome were strongly associated, and higher AMH concentrations were associated with a higher clinical pregnancy rate; moreover, they showed that AMH might offer greater prognostic value than other currently available serum markers of ART outcome.

Elgindy et al. [20] prospectively evaluated 33 patients undergoing their first intracytoplasmic sperm injection treatment cycle with a long protocol and observed that midluteal and early follicular AMH may offer good prognostic value for clinical pregnancy. Eldar-Geva et al. [19] concluded that serum follicular or luteal phase AMH is the only predictor for the pregnancy that had a prospective design with 56 women. Wu et al. [23] detected that day 3 AMH and AFC were significantly higher in pregnant women compared to non-pregnant women (total of 60 infertile women). Multiple regression analysis for prediction of pregnancy showed day 3 AMH to be a good predictor of clinical pregnancy. The latter three studies had a common limitation: a small number of cases.

Majumder et al. [1] prospectively evaluated 162 infertile women and observed that both day 3 AMH and AFC had highly significant correlations with the number of oocytes retrieved and the number of oocytes fertilized. AMH was better than AFC in terms of predicting live birth, but both markers were more valuable in predicting the absence rather than the occurrence of live birth.

Several authors suggested that measurement of follicular fluid AMH and OPU day serum AMH could predict fertilization and clinical pregnancy rates [28–30]. However, prediction of pregnancy on day of OPU is too little too late since the IVF cycle has already been completed.

Broer et al. [31] performed a meta-analysis of 13 trials on AMH and 17 trials on AFC. They detected that sensitivities and specificities of AMH for the prediction of poor ovarian response varied between 40% and 91% and between 64% and 100%, respectively. Moreover, the receiver operating characteristic (ROC) curves do not suggest a clearly better predictive ability for AMH than for AFC, and the difference was not statistically significant ($P$=0.73). The authors concluded that AMH has at least the same level of accuracy and clinical value for the prediction of poor response and nonpregnancy as AFC.

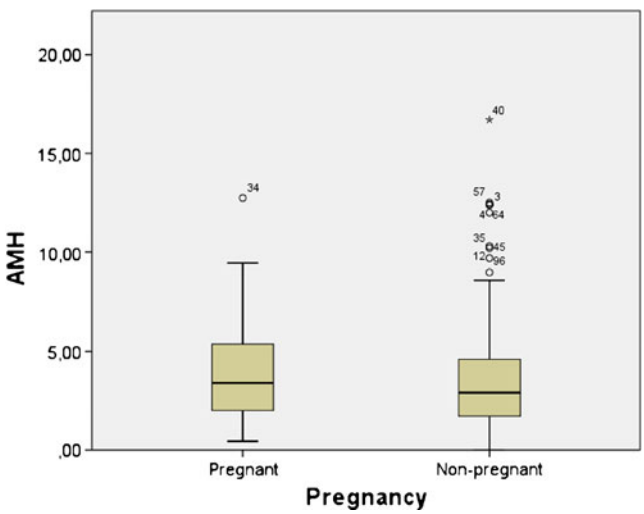


**Fig. 1** Distribution of AMH in pregnant and non-pregnant women

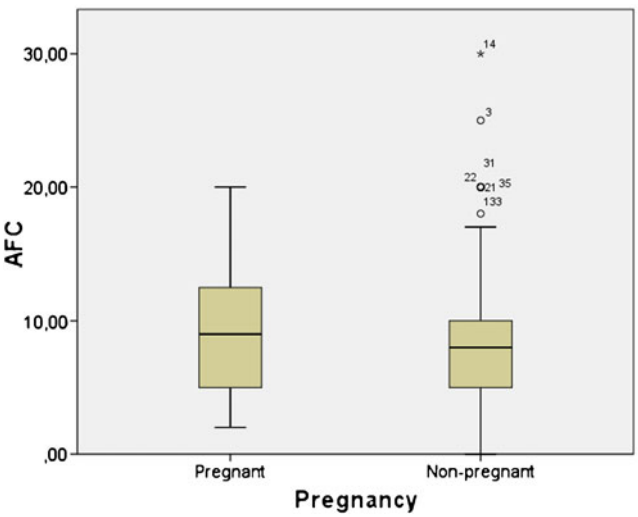


**Fig. 2** Distribution of AFC in pregnant and non-pregnant women

There are few studies which suggest that serum AMH is not associated with ongoing pregnancy rates. Penarrubia et al. [25] compared the data of 20 cancelled cycles and 60 controls and showed that basal and day 5 AMH serum concentrations were significantly lower in the cancelled than in the control group; and the capacity of day 5 AMH in predicting the likelihood of cancellation in an ART program was significantly higher than that for basal AMH measurement. However, in this study, AMH was not found to be beneficial in the prediction of pregnancy. Deffieux and Antoinne [24] suggested that day 3 AMH levels predict the number of oocytes retrieved, but the AMH level cannot predict the likelihood of pregnancy.

Previous studies on the relationship between pregnancy rates and serum level of AMH are summarized in Table 3.

Prediction of poor ovarian reserve is not the same thing as predicting ongoing pregnancy. In our previous study we found that an AMH cut-off level of 2 ng/ml could predict poor response with a sensitivity of 78.9% and specificity of 73.8% [32]. In another study we found that an AMH cut-off level of 3.3 ng/mL predicted ovarian hyperstimulation syndrome (OHSS) with a sensitivity of 90% and a specificity of 71%. [33] However, depending on the results of the present study, we may speculate that it is not possible to determine a certain threshold of AMH that can predict ongoing pregnancy.

In our previous studies, we have shown that body mass index does not seem to have an effect on AMH levels in reproductive age women, both with and without PCOS, [15]; and serum AMH level seems to be a quantitative marker of the ovary but not a quality factor. Serum AMH level does not seem to be a prognostic factor for ongoing pregnancy rated in IVF cycles [33].

Means of AMH differ in various studies. The most striking study on means of AMH in general population is the study of Tremellen and Kolo [27]. They evaluated a total of 1032 women aged between 18 and 43 years and found that the mean serum AMH level is relatively stable at approximately 30 pmol/l (4.1 ng/ml) (1 ng AMH is 7.143 pmol) in the under 30-year-old range; however, from 30 years of age onwards the serum AMH levels decline rapidly, halving in concentration to an average of only 14 pmol/L (1.95 ng/ml) in the 35- to 39-year-old age group. The design of the study of Wu et al. [23] was similar to our present study and mean AMH levels of pregnant and non-pregnant women were 4.3 ng/ml and 3.4 ng/ml, respectively. Our results were comparable to the other results in the literature (3.9± 2.5 ng/ml vs 3.8±3.0 ng/ml in pregnant and non-pregnant women, respectively). Exclusion of women with PCOS may lower the mean levels of AMH, as in the study of Hazout et al. [21], mean AMH levels of pregnant and non-pregnant women were 2.4 ng/ml and 1.1 ng/ml, respectively.

In the present study, we also evaluated the clinical pregnancy rates according to the quartiles of AMH. We observed that clinical pregnancy rates tended to increase with increasing quartiles of serum AMH, but differences between the pregnancy rates of 25%, 50%, and 75% AMH, FSH, and AFC groups were statistically non-significant. The pregnancy rate was 21% in the patients whose serum AMH level was lower than 1.81 ng/ml and 29.2% in patients whose serum AMH level was higher than 4.92 ng/ml. The lowest level of serum AMH was 0.45 ng/ml in the pregnant group. Considering our results, we may suggest that AMH <1 ng/ml do not definitely predict conception failure.

In our study we included women <40 years old. Wang et al [34] retrospectively investigated the relationship between IVF clinical pregnancy rates per initiated cycle and serum AMH tertiles stratified by age in 1558 women in all age groups and detected that age influenced the AMH & clinical pregnancy rate relationship. They found that for women

**Table 3** Summary of studies on the association between serum AMH levels and pregnancy rates

| Reference | Year | Design | Number of women | Age group | |
|---|---|---|---|---|---|
| Studies suggesting significant association between serum AMH levels and clinical pregnancy rates | | | | | |
| Hazout et al. | [21] | Retrospective | 109 women | <42 years | $p$=0.0017 |
| Nelson et al | [22] | Prospective | 340 patients | <37 years | $p$<0.001 |
| Eldar-Geva et al | [19] | Prospective | 56 women | <38 years | $p$<0.02 |
| Elgindy et al | [20] | Prospective | 33 women | <37 years | $p$=0.001 |
| Wu et al | [23] | Prospective | 60 women | <40 years | $p$=0.011 |
| Majumder et al | [1] | Prospective | 162 women | <40 years | $p$=0.002 |
| Wunder et al | [30] | Prospective | 276 women | <42 years | $P$=0.043 |
| Studies suggesting no association between serum AMH levels and clinical pregnancy rates | | | | | |
| Lekamge et al | [8] | Retrospective | 126 women | <41 years | NS |
| Tremellen & Kolo | [27] | Retrospective | 1032 women | <46 years | NS |
| Penarrubia et al | [25] | Case–control | 80 women | NA | NS |
| Smeenk et al | [26] | Prospective | 112 women | <42 years | NS |
| Fanchin et al | [28] | Retrospective | 342 women | <41 years | NS |

NS, non-significant; NA, not available; AMH, anti-müllerian hormone

$P$<0.05 is significant

aged ≥42 years with AMH ≤0.29 ng/ml, the clinical pregnancy rate was significantly lower than those of the middle and higher quartiles, whereas the clinical pregnancy rates for women in the middle and highest tertiles were not significantly different. However our results may only be appropriate for <40 years age group, since older women were excluded.

In conclusion, serum AMH and FSH, and AFC cannot predict clinical pregnancy in women under 40 who undergo IVF; the clinical pregnancy rate tends to increase as AMH and AFC increases, although this remains non-significant.

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**Conflict of interest** None.

## References

1. Majumder K, Gelbaya TA, Laing I, Nardo LG. The use of anti-Müllerian hormone and antral follicle count to predict the potential of oocytes and embryos. Eur J Obstet Gynecol Reprod Biol. 2010;150(2):166–70.
2. Borini A, Dal Prato L. (2005) Tailoring FSH and LH administration to individual patients. Reprod Biomed Online.;11:283–93.
3. Check JH, Peymer M, Lurie D. (1998) Effect of age on pregnancy outcome without assisted reproductive technology in women with elevated early follicular phase serum follicle-stimulating hormone levels. Gynecologic and Obstetric Investigation, **45**, 217–220.
4. Faddy MJ, Gosden RG. (1995) A mathematical model of follicle dynamics in the human ovary. Human Reproduction,**10**, 770–775.
5. Scott RT, Toner JP, Muasher SJ Oehninger S, Robinson S, Rosenwaks Z.(1989) Follicle-stimulating hormone levels on cycle day 3 are predictive of in vitro fertilization outcome. Fertility and Sterility, **51**, 651–654.
6. Toner JP, Philput CB, Jones GS, Muasher SJ. (1991) Basal follicle-stimulating hormone level is a better predictor of in vitro fertilization performance than age. Fertility and Sterility, **55**, 784–791.
7. Seifer DB, Lambert-Messerlian G, Hogan J, Gardiner AC, Blazar AS, Berk CA. (1997) Day 3 serum inhibin-B is predictive of assisted reproductive technologies outcome. Fertility and Sterility, **67, 110–114**
8. Lekamge DN, Barry M, Kolo M, Lane M, Gilchrist RB, Tremellen KP. (2007) Anti-Müllerian hormone as a predictor of IVF outcome. Reprod Biomed Online.;14(5):602–10
9. Josso N. (2008) Professor Alfred Jost: the builder of modern sex differentiation. Sex Dev.;2(2):55–63. Epub 2008 Jun 20.
10. Durlinger AL, Visser JA, Themmen AP. (2002) Regulation of ovarian function: The role of anti-müllerian hormone. Reproduction.;124: 601–9
11. La Marca A, Broekmans FJ, Volpe A, Fauser BC, Macklon N. (2009), ESHRE Special Interest Group for Reproductive Endocrinology–AMH Round Table: Anti-Mullerian hormone (AMH): what do we still need to know? Hum Reprod, 24:2264–2275.
12. Durlinger ALL, Gruijters MJG, Kramer P, Karels B, Kumar TR, Matzuk MM, Rose UM, de Jong FH, Uilenbroek JT, Grootegoed JA, Themmen AP. (2001) Anti-mullerian hormone attenuates the effect of FSH on follicle development in the mouse ovary. Endocrinology;142:4891–9.
13. Weenen C, Laven JSE, von Bergh ARM, Cranfield M, Groome NP, Visser JA, Kramer P, Fauser BC, Themmen AP. (2004) Anti-mullerian hormone expression pattern in the human ovary: potential implications for initial and cyclic follicle recruitment. Mol Hum Reprod;10:77–83.
14. Baarends WM, Uilenbroek JT, Kramer P, Hoogerbrugge JW, van Leeuwen EC, Themmen AP, Grootegoed JA. (1995) Anti-mullerian hormone and anti-mullerian hormone type 2 receptor messenger ribonucleic acid expressions in rat ovaries during postnatal development, the estrous cycle and gonadotropin-induced follicle growth. Endocrinology;136:4951–62.
15. Sahmay S, Guralp O, Senturk LM, Imamoglu M, Kucuk M, Irez T. Serum anti-mullerian hormone concentrations in reproductive age women with and without polycystic ovary syndrome: the influence of body mass index. Japan Society for Reproductive Medicine. 2011.
16. Ebner T, Sommergruber M, Moser M, Shebl O, Schreier-Lechner E, Tews G. (2006) Basal level of anti-mullerian hormone is associated with oocyte quality in stimulated cycles. Hum Reprod;21: 2022–6.
17. Fong SL, Baart EB, Martini E Schipper I, Visser JA, Themmen AP, de Jong FH, Fauser BJ, Laven JS. (2008) Anti-mullerian hormone: a marker for oocyte quantity, oocyte quality and embryo quality. Reprod Biomed Online;16: 664–70.
18. Irez T, Ocal P, Guralp O, Cetin M, Aydogan B, Sahmay S. Different serum anti-Müllerian hormone concentrations are associated with oocyte quality, embryo development parameters and IVF-ICSI outcomes. Arch Gynecol Obstet. 2011;284(5):1295–301.
19. Eldar-Geva T, Ben-Chetrit A, Spitz IM, Rabinowitz R, Markowitz E, Mimoni T, Gal M, Zylber-Haran E, Margalioth EJ. (2005) Dynamic assays of inhibin B, anti-Mullerian hormone and estradiol following FSH stimulation and ovarian ultrasonography as predictors of IVF outcome. Hum Reprod.;20(11):3178–83.
20. Elgindy EA, El-Haieg DO, El-Sebaey A. (2008) Anti-Müllerian hormone: correlation of early follicular, ovulatory and midluteal levels with ovarian response and cycle outcome in intracytoplasmic sperm injection patients. Fertil Steril.;89(6):1670–6.
21. Hazout A, Bouchard P, Seifer DB, Aussage P, Junca AM, Cohen-Bacrie P. (2004) Serum antimüllerian hormone/müllerian-inhibiting substance appears to be a more discriminatory marker of assisted reproductive technology outcome than follicle-stimulating hormone, inhibin B, or estradiol. Fertil Steril.;82(5):1323–9.
22. Nelson SM, Yates RW, Fleming R. (2007) Serum anti-mullerian hormone and FSH: Prediction of live birth and extremes of response in stimulated cycles–implications for individualization of therapy. Hum Reprod;22:2414–21.
23. Wu CH, Chen YC, Wu HH, Yang JG, Chang YJ, Tsai HD. (2009) Serum anti-Müllerian hormone predicts ovarian response and cycle outcome in IVF patients. J Assist Reprod Genet.; 26(7):383–9.
24. Deffieux X, Antoinne JM. (2003) Inhibins, activins and anti-Müllerian hormone: structure, signalling pathways, roles and predictive value in reproductive medicine. Gynecol Obstet Fertil;31 (11):900–11
25. Penarrubia J, Fabregues F, Manau D, Creus M, Casals G, Casamitjana R, Carmona F, Vanrell JA, Balasch J. (2005) Basal and stimulation day 5 anti- Mullerian hormone serum concentrations as predictors of ovarian response and pregnancy in assisted reproductive technology cycles stimulated with gonadotropin-releasing hormone agonist-gonadotropin treatment. Hum Reprod;20:915–22.
26. Smeenk JMJ, Sweep FCGJ, Zielhuis GA, Kremer JAM, Thomas CMG, Braat DDM. (2007) Anti-mullerian hormone predicts

ovarian responsiveness, but not embryo quality or pregnancy, after in vitro fertilization or intracytoplasmic sperm injection. Fertil Steril; 1:223–6.
27. Tremellen K, Kolo M. (2010) Serum anti-Mullerian hormone is a useful measure of quantitative ovarian reserve but does not predict the chances of live-birth pregnancy. Aust N Z J Obstet Gynaecol.;50 (6):568–72.
28. Fanchin R, Schonauer LM, Righini C, Guibourdenche J, Frydman R, Taieb J. (2003) Serum anti mullerian hormone is more strongly related to ovarian follicular status than serum inhibin B, estradiol, FSH and LH on day 3. Hum Reprod;18:323–7.
29. Takahashi C, Fujito A, Kazuka M, Sugiyama R, Ito H, Isaka K. (2008) Anti-Müllerian hormone substance from follicular fluid is positively associated with success in oocyte fertilization during in vitro fertilization. Fertil Steril.;89(3):586–91.
30. Wunder DM, Guibourdenche J, Birkhäuser MH, Bersinger NA. (2008) Anti-Müllerian hormone and inhibin B as predictors of pregnancy after treatment by in vitro fertilization/intracytoplasmic sperm injection. Fertil Steril.;90(6):2203–10.
31. Broer SL, Mol BW, Hendriks D, Broekmans FJ. (2009) The role of antimullerian hormone in prediction of outcome after IVF: comparison with the antral follicle count. Fertil Steril.;91(3):705–14.
32. Sahmay S, Cetin M, Ocal P, Kaleli S, Senol H, Birol F, Irez T. Serum anti-Müllerian hormone level as a predictor of poor ovarian response in IVF patients. Reprod Med Biol. 2011; 10:9–14.
33. Ocal P, Sahmay S, Cetin M, Irez T, Guralp O, Cepni I. Serum anti-Müllerian hormone and antral follicle count as predictive markers of OHSS in ART cycles. J Assist Reprod Genet. 2011;28 (12):1197–203.
34. Wang JG, Douglas NC, Nakhuda GS, Choi JM, Park SJ, Thornton MH, Guarnaccia MM, Sauer MV. The association between anti-Müllerian hormone and IVF pregnancy outcomes is influenced by age. Reprod Biomed Online. 2010 Dec;21(6):757–61.