

Anti-Müllerian hormone as a marker of ovarian reserve: What have we learned, and what should we know?

Akira Iwase^{1,2} · Tomoko Nakamura¹ · Satoko Osuka¹ · Sachiko Takikawa¹ · Maki Goto¹ · Fumitaka Kikkawa¹

Received: 13 September 2015 / Accepted: 6 November 2015 / Published online: 23 November 2015
© Japan Society for Reproductive Medicine 2015

Abstract Ovarian reserve reflects the quality and quantity of available oocytes. This reserve has become indispensable for the better understanding of reproductive potential. Measurement of the serum anti-Müllerian hormone (AMH) level allows quantitative evaluation of ovarian reserve. It has been applied to a wide range of clinical conditions, and it is well established that the measurement of serum AMH levels is more useful than qualitative evaluation based on the menstrual cycle. AMH levels are monitored during infertility treatments; in patients undergoing medically assisted reproductive technology; and in the diagnosis of ovarian failure, polycystic ovarian syndrome, and granulosa cell tumor. It is also useful in the evaluation of iatrogenic ovarian damage. Population-based studies have indicated a potential role for serum AMH in the planning of reproductive health management. While AMH is currently the best measure of ovarian reserve, its predictive value for future live births remains controversial. Furthermore, there is a serious practical issue in the interpretation of test results, as currently available assay kits use different assay ranges and coefficients of variation due to the absence of an international reference standard. The pros and cons of the serum AMH level as a definitive measure of ovarian reserve merits further review in order to guide future research.

Keywords Anti-Müllerian hormone · Female reproduction · Infertility · Menopause · Ovarian reserve

Introduction

Ovarian reserve is a concept that reflects the quality and quantity of ovarian follicles at a given point in time and therefore predicts potential ovarian function [1]. Anti-Müllerian hormone (AMH) is produced by the granulosa cells of primary, preantral, and small antral follicles in the ovaries [2, 3]. It was first discovered in the early 1990s that the serum AMH level could provide an indirect representation of the total number of available follicles, thereby serving as a marker of ovarian reserve [3, 4]. AMH is highly sensitive to changes that accompany advancing age [4–7] and it excludes uncertainties associated with the intra- and inter-cycle variability of menstruation [8, 9]. During the past two decades, the number of clinical applications of serum AMH measurement has grown, and its usefulness has been well established [10, 11]. The aim of this review is to provide an overview of the current clinical applications of serum AMH measurements and to examine the future prospects of serum AMH assays.

Medically assisted reproduction

Prediction of oocyte yield

Medically assisted reproductive technology is in great demand, and ovarian reserve tests are of value for predicting outcomes of medically assisted reproduction. While various innovations have contributed to increased rates of successful embryo implantations, even when the greatest

✉ Akira Iwase
akiwase@med.nagoya-u.ac.jp

¹ Department of Obstetrics and Gynecology, Nagoya University Graduate School of Medicine, 65 Tsurumai-cho, Showa-ku, Nagoya 466-8550, Japan

² Department of Maternal and Perinatal Medicine, Nagoya University Hospital, 65 Tsurumai-cho, Showa-ku, Nagoya 466-8550, Japan

probability of successful implantation exists, the success of the procedure cannot be assured if only one or two embryos have been transferred [12]. If the number of oocytes that can be obtained for assisted reproduction can be predicted, success rates might be increased. The serum AMH level has been known from its earliest applications to correlate well with the number of oocytes generated during cycles of ovarian stimulation by human menopausal gonadotropin and recombinant follicle-stimulating hormone (FSH) [13].

Ascertainment of the linear correlation between serum AMH levels and the number of oocytes has also allowed prediction of poor or excess responses. Numerous studies from the 1990s to the present have demonstrated the usefulness of the serum AMH level for identifying high-risk groups of poor responders, who are at risk of cycle cancellation and hyper-responders, who are at risk of ovarian hyperstimulation syndrome (OHSS). A detailed meta-analysis that was published by Broekmans et al. in 2006 [1] confirmed that the serum AMH level is a good predictor of both poor response and hyper-response, and other recent systematic reviews have also suggested that serum AMH may be the best marker of ovarian reserve when prediction of poor or hyper ovarian response is desired [14–19].

Algorithm for the selection of ovarian stimulation protocols

Based on the usefulness of the linear correlation between AMH levels and oocyte yield, some researchers have suggested that the ovarian stimulation protocol can be optimized according to the AMH level in order to minimize the risk of poor response and cycle cancellation or hyper-response and OHSS. Several different algorithms, in which AMH levels play the main role, have been proposed. These studies have mainly focused on the response to the initial dose of FSH during the assisted reproduction cycle [20–22], and successful reduction of unwanted responses (cycle cancellation or OHSS) has been reported [18, 22, 23].

Individualization of treatment in assisted conception is aimed at maximizing success, and results regarding the usefulness of a standardized algorithm have been inconclusive [24]. Certainly, the issue should not be decided based only on the results of a single cycle of assisted conception, and cumulative pregnancy rates, including successful transfer of frozen–thawed embryos, should be evaluated to optimize stimulation protocols.

Prediction of pregnancy and live birth after assisted conception

Although oocyte yield is an important factor in assisted reproduction, it is hardly necessary to point out that an algorithm for the selection of an appropriate ovarian

stimulation protocol is not the ultimate goal, and researchers have now begun to investigate the association between AMH levels, pregnancy, and live birth after assisted reproduction. Preliminary studies exploring the capacity of AMH to predict these factors have shown conflicting results [1, 25–32].

Two thorough meta-analyses examining this issue have recently been published [33, 34]. One of them includes data from 19 studies reporting pregnancy in patients with unspecified ovarian reserve, diminished ovarian reserve, and polycystic ovarian syndrome. The diagnostic odds ratios for AMH as a predictor of clinical pregnancy for each of these categories were 2.10 (95 % confidence interval [CI] 1.82–2.41), 3.96 (95 % CI 2.57–6.10), and 1.18 (95 % CI 0.53–2.62), respectively [34]. The other included 13 studies comprising from 6856 cycles in 6306 women. This analysis also showed a better diagnostic odds ratio for live birth after assisted conception in women with low ovarian reserve compared to women with unknown ovarian reserve (4.63, 95 % CI 2.75–7.81 vs. 2.48, 95 % CI 1.81–3.22) [33]. These recent results support the theory that AMH has the capacity for predicting pregnancy and live birth after assisted conception, perhaps especially in women with diminished ovarian reserve. Despite these preliminary results, the predictive accuracy is not sufficient for low AMH levels to be used as exclusion criteria for assisted reproduction, even as evidence suggesting that higher AMH levels result in higher cumulative pregnancy and live birth rates after assisted conception continues to accumulate [35, 36]. Higher AMH levels may predict a greater possibility of pregnancy, but even if lower AMH levels might predict treatment failure, they should not exclude the option of assisted reproduction.

The qualitative aspects of ovarian reserve may also have strong implications for treatment success after assisted conception, and AMH levels have shown a weak association with oocyte quality, independent of chronological age. Quantitative issues have been resolved by measuring serum AMH levels. To improve prediction of pregnancy and live birth, methods for interpreting AMH levels as a qualitative marker of ovarian function, either alone or in combination with other markers of ovarian reserve, should be explored.

General population and natural conception

The intra-cycle variability of serum AMH levels is low, and AMH level measurement may also prove useful in the prediction of fertility and natural conception. Population-based analyses of serum AMH levels have shown that they peak at around 25 years of age and then gradually decline, becoming undetectable at approximately 5 years before menopause [37–41]. Cross-sectional analysis has shown

that that serum AMH levels are distributed widely within each age group, which poses a question: Does the same serum AMH level predict the same level of ovarian reserve for women at different ages? Further longitudinal studies are required to determine the pattern of decline of individual AMH levels and its meaning.

If AMH levels prove to be reliable predictors of natural fertility, they may also be extremely helpful in family planning or in early detection and treatment of infertility. Two recent prospective studies have shown conflicting results. There was a good correlation between AMH levels and natural conception in women 30–44 years of age during a 6-month observation period [42], but low AMH levels in a group of women in their twenties did not necessarily result in compromised fecundability [43]. These conflicting results may be due to the different ages of the study participants and to the small sizes of the study populations. Larger studies will be needed to draw conclusions regarding the use of AMH levels for determining the likelihood of natural conception.

Prediction of menopause/primary ovarian insufficiency (POI)

The serum FSH level, as a conventional marker of ovarian reserve, increases during the perimenopausal period [44], and serum AMH levels become undetectable within 5 years before menopause [45]. This has led to speculation that serum AMH levels could improve prediction of the onset of menopause. In long-term follow-up studies of 9 and 11 years, women in certain age groups who had lower AMH levels may have had an earlier onset of menopause [46, 47], and recent multivariate prediction models including serum AMH were shown to more accurately forecast early or late menopause [48, 49].

Menopause occurs when the oocytes are depleted. Therefore, similar to the prediction of menopause onset, serum AMH has been considered as a possible marker for identifying women with diminished ovarian reserve who are likely to develop POI. Several studies have shown that serum AMH levels are much lower in women with symptomatic ovarian insufficiency [50–53]. In an era in which it is possible to maintain fertility via cryopreservation of ovaries and/or oocytes, these trials seem to be meaningful for the presupposition of accurate prediction. Promising results have also been reported for incipient POI and autoimmune ovarian failure [54, 55]. However, more accurate data will be needed to identify a potential POI population among women with low serum AMH.

Diagnosis of polycystic ovary syndrome (PCOS)

PCOS is one of the most common reproductive endocrine disorders in women [56, 57]. Diagnostic criteria include polycystic appearance of the ovaries accompanied by anovulation or oligoovulation and/or excess androgen production [58, 59]. The increased number of cystic follicles is mostly related to AMH-producing small antral follicles. Therefore, serum AMH levels increase by 2–3-fold in women with early stage PCOS compared with normal controls. The sensitivity of serum AMH for diagnosis of PCOS ranges from 64 to 99 % [60–68]. The wide range of sensitivities among studies is likely due to variations in diagnostic criteria and in the age of the patient population. A recent systematic review and meta-analysis that included ten studies attempted to establish an appropriate diagnostic threshold and found that a cut-off level of 4.7 ng/ml maximized the sensitivity and specificity (82.8 and 79.4 %, respectively) of serum AMH levels for diagnosis of PCOS [69].

The pathogenesis of PCOS is heterogeneous, which is why a variety of diagnostic criteria have been proposed [58, 59, 70]. The ability of AMH to determine the subphenotype and severity of PCOS has also been examined. Marked elevation of serum AMH predicts higher luteinizing hormone levels [71] and severity [72, 73]. On the other hand, higher serum AMH has also been found after reproductive performance improves due to weight loss [74].

Laparoscopic ovarian drilling (LOD) has been advanced as a promising treatment for PCOS, with reported postoperative pregnancy rates of up to 39.7 % and a reduced risk of multiple pregnancies [75]. The effectiveness of LOD has also been examined in association with serum AMH levels. Amer et al. found that lower preoperative serum AMH level may be predictive of ineffective LOD [76], and this finding was confirmed in a subsequent report [77]. In sum, AMH may be useful as an adjuvant diagnostic or prognostic tool for PCOS, and it may also provide insight regarding the pathophysiology.

Chemotherapy

Decline of ovarian reserve after chemotherapy

Regular or irregular menstruation and amenorrhea are qualitative indicators of ovarian function. Some types of chemotherapy cause complete depletion of follicles, which is diagnosed by amenorrhea and elevated FSH. It is now known that some survivors of childhood cancer who have regular menses also have decreased AMH levels, and in

numerous studies, serum AMH was found useful for the qualitative evaluation of follicle depletion after chemotherapy [78, 79]. AMH has also been examined as an indicator of follicular loss in women who are diagnosed with breast cancer [80–82] and lymphoma [83]. Cross-sectional studies have shown a specific association between serum AMH level and type of chemotherapy [84]. More specifically, in a longitudinal follow-up study of young women diagnosed with lymphoma, the pattern of recovery of serum AMH levels was clearly different between those who received non-alkylating agents and those who received alkylating agents [83].

The usefulness of AMH in the field of oncofertility has been well studied, and limitations have also been found. An absence of serum AMH does not signify the complete depletion of oocytes in every case. Women with persistently undetectable levels of serum AMH after chemotherapy can resume regular menstruation [85, 86], and, as mentioned, among women in the general population, serum AMH levels may become undetectable up to 5 years before menopause [45]. These findings could change once a more sensitive AMH assay becomes available.

The use of gonadotropin-releasing hormone agonists for protection of the ovaries is another controversial issue, and measurement of serum AMH levels has also been adopted in a clinical trial (the “OPTION” trial) to better investigate this issue [87].

Prediction of ovarian insufficiency after chemotherapy

A future interesting application of AMH is its predictive potential for determining post-chemotherapy status of menstruation and/or ovarian insufficiency. Anderson et al. have recently proposed that the pretreatment AMH value may be a useful predictor of long-term chemotherapy-related amenorrhea in women who are diagnosed with early breast cancer [88]. These women may opt for fertility preservation via oocyte and ovarian tissue cryopreservation, although the invasiveness of resecting the ovarian cortex and the time necessary for oocyte retrieval could be an issue. Selection criteria for patients undergoing ovarian tissue cryopreservation have been proposed, but definitive criteria for the strict selection of eligible candidates are far from being determined [89]. AMH values in combination with other indices, such as age and chemotherapeutic regimen, could further refine the criteria, and prospective studies will be needed to firmly establish the predictive potential of AMH values in various circumstances in order to guide formulation of appropriate criteria for eligibility for fertility preservation.

Decrease of ovarian reserve in relation to gynecologic disease/intervention

Endometriosis

The impact of surgical treatment for endometriomas on the oocyte yield in assisted reproductive cycles has been controversial [90]. Not all women who wish to conceive after surgery for endometriosis will require assisted reproductive technology. Therefore, post-surgical evaluation of ovarian reserve based on oocyte yield may be subject to patient selection bias. In this situation, it is not surprising that researchers began to adopt serum AMH levels. Chang and Iwase were the first to report the decline of serum AMH levels after excision of endometrioma(s), especially in bilateral cases [91, 92]. Afterwards, numerous other reports confirmed that AMH levels decreased after removal of endometriomas [93–96], and steeper reductions were observed after bilateral procedures [97, 98]. Two systematic reviews evaluating the effect of endometrioma surgery on ovarian reserve as assessed by serum AMH measurement have been published [99, 100]. Bilateral removal of endometrial cysts is one of the greatest risk factors for severe decrease in ovarian reserve. Other risk factors include high revised American Society Reproductive Medicine score, larger cysts, thermal damage caused by bipolar coagulation, and removal of the ovarian cortex [94, 97, 101–103].

Excisional surgery has been recognized as the gold standard for the management of endometriomas because women have higher rates of spontaneous pregnancy and lower rates of recurrence after treatment [104]. Decreased serum AMH levels after excision of endometrial cysts has been regarded as a marker for the assessment of surgical outcomes. It has also been proposed that the method of hemostasis after laparoscopic excision of endometriomas affects ovarian reserve, and several of these methods, including suturing, bipolar coagulation, and application of hemostatic materials, have been compared to determine which is best in terms of preservation of ovarian reserve [102, 103]. Moreover, a clear advantage in the preservation of serum AMH levels has been shown after combination treatment with vaporization and GnRH agonists vs. excision [96]. These findings could be useful to decision-making during treatment planning for patients with endometriomas. However, maintenance of ovarian reserve will ultimately be confirmed by pregnancy and live birth rates, and to date, only preliminary data are available to show an association between post-surgical AMH levels and pregnancy.

Other gynecologic conditions and ovarian reserve

Excisions of benign ovarian tumors including mature cystic teratomas are common surgical procedures. These procedures might also affect ovarian reserve. However, they are generally regarded as less invasive than comparable procedures for endometriomas, allowing better preservation of ovarian reserve. This has been confirmed by comparison of serum AMH levels [91, 92, 105, 106].

Blood supply to the ovaries may also affect ovarian function, including folliculogenesis. Women undergoing assisted reproductive procedures who have had previous salpingectomy as a treatment for hydrosalpinx may experience recurrent failure of assisted reproductive cycles. Salpingectomy and uterine artery embolization, which is a fertility-preserving intervention in cases of postpartum hemorrhage, may have lasting effects on blood flow to the ovaries, leading to a decline in ovarian reserve [107–110]. To date, decreases in serum AMH levels related to surgical procedures or gynecologic diseases other than endometriomas do not seem to have strong influence on ovarian reserve.

Diagnosis and follow-up for granulosa cell tumors

Adult-type granulosa cell tumor is a less common ovarian tumor that causes irregular vaginal bleeding due to a rise in serum estradiol levels in postmenopausal women. The symptoms are less pronounced in premenopausal women. AMH and inhibin B are both produced by granulosa cells and may therefore be useful as markers of proliferation of granulosa cell tumors. Several studies have shown that the serum AMH level has high sensitivity in the diagnosis of

granulosa cell tumors. In addition, AMH can be a useful marker for detecting recurrence of these tumors [111–114].

Standardization of the AMH assay

Lastly, standardization of the AMH assay is one of the most pressing issues in AMH research. AMH immunoassays were first developed by Hudson et al. in 1990 [115]. Thereafter, two proprietary AMH assay kits, Active AMH and EIA AMH/MIS [Diagnostic Systems Laboratory (DSL) and Immunotech (IOT)] were brought to market. Each kit used different AMH antibodies, resulting in different assay ranges and different inter- and intraassay coefficients of variation [116, 117]. Beckman Coulter merged DSL and IOT and developed a new AMH assay kit (AMH GenII). Although the three kits have shown good correlation in assay values, considerable diversity exists in conversion values among the kits [118]. Therefore, caution is required when comparing absolute values from clinical studies that use different assays. An important task that should be undertaken as soon as possible is the formulation of an international reference standard for AMH assays.

Another issue concerning AMH assays is that of their sensitivity. Several studies have shown that live births are possible even when AMH levels are undetectable [119, 120]. Serum AMH level is generally considered a better marker of ovarian function than basal FSH level. However, undetectable AMH is less specific for the detection of loss of ovarian function than elevated basal FSH. Ansh Labs has recently developed a hypersensitive AMH ELISA kits that allow ultra-sensitive AMH and pico-AMH measurements. Several studies have confirmed that these kits are capable of detecting AMH at low concentrations [121–

Table 1 Utility and limitations of measurements of serum anti-Müllerian hormone (AMH) levels in various clinical conditions

	What have we learned?	What should we know?
Medically assisted reproduction	Good correlation to oocyte yield Predictive potential for poor and hyper-response	Predictive potential for live births Optimization of protocols to improve treatment success
General population	Peaks around 25 years of age and gradually declines Very low serum AMH level does not necessarily mean sterility	Predictive potential for future fertility
Menopause/POI	Undetectable serum AMH level is followed by menopause within a certain time period depending on age	Selection and diagnosis of subclinical POI
PCOS	Elevated serum AMH level is correlated with severity	Association with pathophysiology Optimization of treatment schedules according to serum AMH levels
Ovarian toxicity/surgical intervention	Decline depending on chemotherapeutic regimens and surgical interventions, especially cystectomy for endometriomas	Indication of fertility preservation Optimal interventions according to ovarian reserve

POI primary ovarian insufficiency, PCOS polycystic ovary syndrome

124], and they may therefore be helpful for improved follow-up and a more detailed understanding of declining ovarian reserve immediately prior to the loss of ovarian function.

Conclusions

The quantitative measurement of serum AMH levels has revealed that ovarian reserve may vary in women of the same chronological age. Moreover, we can safely say that AMH is the most reliable marker of ovarian reserve, which may be useful for a wide range of clinical applications including the optimization of fertility treatments, the diagnosis of disorders of reproductive endocrinology, and the assessment of ovarian toxicity due to medical and surgical treatments (Table 1). However, no definite conclusions have been reached regarding the utility of serum AMH as a predictive marker for live births or its potential to improve reproductive healthcare and cost-effectiveness.

Compliance with ethical standards

Conflict of interest Akira Iwase, Tomoko Nakamura, Satoko Osuka, Sachiko Takikawa, Maki Goto, and Fumitaka Kikkawa declare that they have no conflicts of interest.

Human/animal studies This article does not contain any studies with human or animal subjects performed by any of the authors.

References

- Broekmans FJ, Kwee J, Hendriks DJ, Mol BW, Lambalk CB. A systematic review of tests predicting ovarian reserve and IVF outcome. *Hum Reprod Update*. 2006;12(6):685–718.
- Durlinger AL, Gruijters MJ, Kramer P, Karels B, Ingraham HA, Nachtigal MW, Uilenbroek JT, Grootegeod JA, Themmen AP. Anti-Müllerian hormone inhibits initiation of primordial follicle growth in the mouse ovary. *Endocrinology*. 2002;143(3):1076–84.
- Weenen C, Laven JS, Von Bergh AR, Cranfield M, Groome NP, Visser JA, Kramer P, Fauser BC, Themmen AP. Anti-Müllerian hormone expression pattern in the human ovary: potential implications for initial and cyclic follicle recruitment. *Mol Hum Reprod*. 2004;10(2):77–83.
- Anderson RA, Nelson SM, Wallace WH. Measuring anti-Müllerian hormone for the assessment of ovarian reserve: when and for whom is it indicated? *Maturitas*. 2012;71(1):28–33.
- de Vet A, Laven JS, de Jong FH, Themmen AP, Fauser BC. Anti-Müllerian hormone serum levels: a putative marker for ovarian aging. *Fertil Steril*. 2002;77(2):357–62.
- Fanchin R, Schonauer LM, Righini C, Guibourdenche J, Frydman R, Taieb J. Serum anti-Müllerian hormone is more strongly related to ovarian follicular status than serum inhibin B, estradiol, FSH and LH on day 3. *Hum Reprod*. 2003;18(2):323–7.
- van Rooij IA, Broekmans FJ, Scheffer GJ, Looman CW, Habbema JD, de Jong FH, Fauser BJ, Themmen AP, te Velde ER. Serum anti-Müllerian hormone levels best reflect the reproductive decline with age in normal women with proven fertility: a longitudinal study. *Fertil Steril*. 2005;83(4):979–87.
- La Marca A, Sighinolfi G, Radi D, Argento C, Baraldi E, Arsenio AC, Stabile G, Volpe A. Anti-Müllerian hormone (AMH) as a predictive marker in assisted reproductive technology (ART). *Hum Reprod Update*. 2010;16(2):113–30.
- van Disseldorp J, Lambalk CB, Kwee J, Looman CW, Eijkemans MJ, Fauser BC, Broekmans FJ. Comparison of inter- and intra-cycle variability of anti-Müllerian hormone and antral follicle counts. *Hum Reprod*. 2010;25(1):221–7.
- Loh JS, Maheshwari A. Anti-Müllerian hormone—is it a crystal ball for predicting ovarian ageing? *Hum Reprod*. 2011;26(11):2925–32.
- Nelson SM. Biomarkers of ovarian response: current and future applications. *Fertil Steril*. 2013;99(4):963–9.
- Pandian Z, Marjoribanks J, Ozturk O, Serour G, Bhattacharya S. Number of embryos for transfer following in vitro fertilisation or intra-cytoplasmic sperm injection. *Cochrane Database Syst Rev*. 2013;7:CD003416.
- Seifer DB, MacLaughlin DT, Christian BP, Feng B, Shelden RM. Early follicular serum Müllerian-inhibiting substance levels are associated with ovarian response during assisted reproductive technology cycles. *Fertil Steril*. 2002;77(3):468–71.
- Broer SL, Dolleman M, Opmeer BC, Fauser BC, Mol BW, Broekmans FJ. AMH and AFC as predictors of excessive response in controlled ovarian hyperstimulation: a meta-analysis. *Hum Reprod Update*. 2011;17(1):46–54.
- Broer SL, Dolleman M, van Disseldorp J, Broeze KA, Opmeer BC, Bossuyt PM, Eijkemans MJ, Mol BW, Broekmans FJ. Prediction of an excessive response in in vitro fertilization from patient characteristics and ovarian reserve tests and comparison in subgroups: an individual patient data meta-analysis. *Fertil Steril*. 2013;100(2):420–9 (e7).
- Broer SL, Mol BW, Hendriks D, Broekmans FJ. The role of anti-Müllerian hormone in prediction of outcome after IVF: comparison with the antral follicle count. *Fertil Steril*. 2009;91(3):705–14.
- Broer SL, van Disseldorp J, Broeze KA, Dolleman M, Opmeer BC, Bossuyt P, Eijkemans MJ, Mol BW, Broekmans FJ. Added value of ovarian reserve testing on patient characteristics in the prediction of ovarian response and ongoing pregnancy: an individual patient data approach. *Hum Reprod Update*. 2013;19(1):26–36.
- Nelson SM, Yates RW, Lyall H, Jamieson M, Traynor I, Gaudoin M, Mitchell P, Ambrose P, Fleming R. Anti-Müllerian hormone-based approach to controlled ovarian stimulation for assisted conception. *Hum Reprod*. 2009;24(4):867–75.
- Anckaert E, Smits J, Schiettecatte J, Klein BM, Arce JC. The value of anti-Müllerian hormone measurement in the long GnRH agonist protocol: association with ovarian response and gonadotrophin-dose adjustments. *Hum Reprod*. 2012;27(6):1829–39.
- La Marca A, Sunkara SK. Individualization of controlled ovarian stimulation in IVF using ovarian reserve markers: from theory to practice. *Hum Reprod Update*. 2014;20(1):124–40.
- Lan VT, Linh NK, Tuong HM, Wong PC, Howles CM. Anti-Müllerian hormone versus antral follicle count for defining the starting dose of FSH. *Reprod Biomed Online*. 2013;27(4):390–9.
- La Marca A, Papaleo E, Grisendi V, Argento C, Giulini S, Volpe A. Development of a nomogram based on markers of ovarian reserve for the individualisation of the follicle-stimulating hormone starting dose in in vitro fertilisation cycles. *BJOG*. 2012;119(10):1171–9.

23. Yates AP, Rustamov O, Roberts SA, Lim HY, Pemberton PW, Smith A, Nardo LG. Anti-Müllerian hormone-tailored stimulation protocols improve outcomes whilst reducing adverse effects and costs of IVF. *Hum Reprod*. 2011;26(9):2353–62.
24. La Marca A, Argento C, Sighinolfi G, Grisendi V, Carbone M, D'Ippolito G, Arsenio AC, Stabile G, Volpe A. Possibilities and limits of ovarian reserve testing in ART. *Curr Pharm Biotechnol*. 2012;13(3):398–408.
25. Eldar-Geva T, Ben-Chetrit A, Spitz IM, Rabinowitz R, Markowitz E, Mimoni T, Gal M, Zylber-Haran E, Margalioth EJ. Dynamic assays of inhibin B, anti-Müllerian hormone and estradiol following FSH stimulation and ovarian ultrasonography as predictors of IVF outcome. *Hum Reprod*. 2005;20(11):3178–83.
26. Nelson SM, Yates RW, Fleming R. Serum anti-Müllerian hormone and FSH: prediction of live birth and extremes of response in stimulated cycles—implications for individualization of therapy. *Hum Reprod*. 2007;22(9):2414–21.
27. Silberstein T, MacLaughlin DT, Shai I, Trimarchi JR, Lambert-Messerlian G, Seifer DB, Keefe DL, Blazar AS. Müllerian inhibiting substance levels at the time of HCG administration in IVF cycles predict both ovarian reserve and embryo morphology. *Hum Reprod*. 2006;21(1):159–63.
28. Fanchin R, Mendez Lozano DH, Frydman N, Gougeon A, Di Clemente N, Frydman R, Taieb J. Anti-Müllerian hormone concentrations in the follicular fluid of the preovulatory follicle are predictive of the implantation potential of the ensuing embryo obtained by in vitro fertilization. *J Clin Endocrinol Metab*. 2007;92(5):1796–802.
29. Ficocioglu C, Kutlu T, Baglam E, Bakacak Z. Early follicular anti-Müllerian hormone as an indicator of ovarian reserve. *Fertil Steril*. 2006;85(3):592–6.
30. Penarrubia J, Fabregues F, Manau D, Creus M, Carmona F, Casamitjana R, Vanrell JA, Balasch J. Previous cycle cancellation due to poor follicular development as a predictor of ovarian response in cycles stimulated with gonadotrophin-releasing hormone agonist-gonadotrophin treatment. *Hum Reprod*. 2005;20(3):622–8.
31. Smeenk JM, Sweep FC, Zielhuis GA, Kremer JA, Thomas CM, Braat DD. Anti-Müllerian hormone predicts ovarian responsiveness, but not embryo quality or pregnancy, after in vitro fertilization or intracytoplasmic sperm injection. *Fertil Steril*. 2007;87(1):223–6.
32. van Rooij IA, Broekmans FJ, Hunault CC, Scheffer GJ, Eijkemans MJ, de Jong FH, Themmen AP, te Velde ER. Use of ovarian reserve tests for the prediction of ongoing pregnancy in couples with unexplained or mild male infertility. *Reprod Biomed Online*. 2006;12(2):182–90.
33. Iliodromiti S, Kelsey TW, Wu O, Anderson RA, Nelson SM. The predictive accuracy of anti-Müllerian hormone for live birth after assisted conception: a systematic review and meta-analysis of the literature. *Hum Reprod Update*. 2014;20(4):560–70.
34. Tal R, Tal O, Seifer BJ, Seifer DB. Anti-Müllerian hormone as predictor of implantation and clinical pregnancy after assisted conception: a systematic review and meta-analysis. *Fertil Steril*. 2015;103(1):119–30 (e3).
35. Arce JC, La Marca A, Klein BM, Andersen AN, Fleming R. Anti-Müllerian hormone in gonadotropin releasing-hormone antagonist cycles: prediction of ovarian response and cumulative treatment outcome in good-prognosis patients. *Fertil Steril*. 2013;99(6):1644–53.
36. Brodin T, Hadziosmanovic N, Berglund L, Olovsson M, Holte J. Anti-Müllerian hormone levels are strongly associated with live-birth rates after assisted reproduction. *J Clin Endocrinol Metab*. 2013;98(3):1107–14.
37. La Marca A, Spada E, Grisendi V, Argento C, Papaleo E, Milani S, Volpe A. Normal serum anti-Müllerian hormone levels in the general female population and the relationship with reproductive history. *Eur J Obstet Gynecol Reprod Biol*. 2012;163(2):180–4.
38. Leader B, Hegde A, Baca Q, Stone K, Lannon B, Seifer DB, Broekmans F, Baker VL. High frequency of discordance between anti-Müllerian hormone and follicle-stimulating hormone levels in serum from estradiol-confirmed days 2 to 4 of the menstrual cycle from 5,354 women in U.S. fertility centers. *Fertil Steril*. 2012;98(4):1037–42.
39. Nelson SM, Iliodromiti S, Fleming R, Anderson R, McConnachie A, Messow CM. Reference range for the anti-Müllerian hormone Generation II assay: a population study of 10,984 women, with comparison to the established Diagnostics Systems Laboratory nomogram. *Fertil Steril*. 2014;101(2):523–9.
40. Nelson SM, Messow MC, McConnachie A, Wallace H, Kelsey T, Fleming R, Anderson RA, Leader B. External validation of nomogram for the decline in serum anti-Müllerian hormone in women: a population study of 15,834 infertility patients. *Reprod Biomed Online*. 2011;23(2):204–6.
41. Nelson SM, Messow MC, Wallace AM, Fleming R, McConnachie A. Nomogram for the decline in serum anti-Müllerian hormone: a population study of 9,601 infertility patients. *Fertil Steril*. 2011;95(2):736–41 (e1–3).
42. Steiner AZ, Herring AH, Kesner JS, Meadows JW, Stanczyk FZ, Hoberman S, Baird DD. Anti-Müllerian hormone as a predictor of natural fecundability in women aged 30–42 years. *Obstet Gynecol*. 2011;117(4):798–804.
43. Hagen CP, Vestergaard S, Juul A, Skakkebaek NE, Andersson AM, Main KM, Hjollund NH, Ernst E, Bonde JP, Anderson RA, et al. Low concentration of circulating anti-Müllerian hormone is not predictive of reduced fecundability in young healthy women: a prospective cohort study. *Fertil Steril*. 2012;98(6):1602–8 (e2).
44. Burger HG, Dudley EC, Hopper JL, Groome N, Guthrie JR, Green A, Dennerstein L. Prospectively measured levels of serum follicle-stimulating hormone, estradiol, and the dimeric inhibins during the menopausal transition in a population-based cohort of women. *J Clin Endocrinol Metab*. 1999;84(11):4025–30.
45. Dolleman M, Faddy MJ, van Disseldorp J, van der Schouw YT, Messow CM, Leader B, Peeters PH, McConnachie A, Nelson SM, Broekmans FJ. The relationship between anti-Müllerian hormone in women receiving fertility assessments and age at menopause in subfertile women: evidence from large population studies. *J Clin Endocrinol Metab*. 2013;98(5):1946–53.
46. Broer SL, Eijkemans MJ, Scheffer GJ, van Rooij IA, de Vet A, Themmen AP, Laven JS, de Jong FH, Te Velde ER, Fauser BC, et al. Anti-Müllerian hormone predicts menopause: a long-term follow-up study in normoovulatory women. *J Clin Endocrinol Metab*. 2011;96(8):2532–9.
47. Tehrani FR, Solaymani-Dodaran M, Azizi F. A single test of anti-Müllerian hormone in late reproductive-aged women is a good predictor of menopause. *Menopause*. 2009;16(4):797–802.
48. Dolleman M, Depmann M, Eijkemans MJ, Heimensem J, Broer SL, van der Stroom EM, Laven JS, Van Rooij IA, Scheffer GJ, Peeters PH, et al. Anti-Müllerian hormone is a more accurate predictor of individual time to menopause than mother's age at menopause. *Hum Reprod*. 2014;29(3):584–91.
49. Ramezani Tehrani F, Dolleman M, van Disseldorp J, Broer SL, Azizi F, Solaymani-Dodaran M, Fauser BC, Laven JS, Eijkemans MJ, Broekmans F. Predicting menopausal age with anti-Müllerian hormone: a cross-validation study of two existing models. *Climacteric*. 2014;17(5):583–90.
50. Baker VL. Primary ovarian insufficiency in the adolescent. *Curr Opin Obstet Gynecol*. 2013;25(5):375–81.

51. Chao KC, Ho CH, Shyong WY, Huang CY, Tsai SC, Cheng HY, Chou LC, Lin CH, Li HY. Anti-Müllerian hormone serum level as a predictive marker of ovarian function in Taiwanese women. *J Chin Med Assoc.* 2012;75(2):70–4.
52. Kallio S, Aittomäki K, Piltonen T, Veijola R, Liakka A, Vaskivuo TE, Dunkel L, Tapanainen JS. Anti-Müllerian hormone as a predictor of follicular reserve in ovarian insufficiency: special emphasis on FSH-resistant ovaries. *Hum Reprod.* 2012;27(3):854–60.
53. Visser JA, Schipper I, Laven JS, Themmen AP. Anti-Müllerian hormone: an ovarian reserve marker in primary ovarian insufficiency. *Nat Rev Endocrinol.* 2012;8(6):331–41.
54. Knauff EA, Eijkemans MJ, Lambalk CB, ten Kate-Booij MJ, Hoek A, Beerendonk CC, Laven JS, Goverde AJ, Broekmans FJ, Themmen AP, et al. Anti-Müllerian hormone, inhibin B, and antral follicle count in young women with ovarian failure. *J Clin Endocrinol Metab.* 2009;94(3):786–92.
55. La Marca A, Marzotti S, Brozzetti A, Stabile G, Arsenio AC, Bini V, Giordano R, De Bellis A, Volpe A, Falorni A. Primary ovarian insufficiency due to steroidogenic cell autoimmunity is associated with a preserved pool of functioning follicles. *J Clin Endocrinol Metab.* 2009;94(10):3816–23.
56. Dewailly D, Lujan ME, Carmina E, Cedars MI, Laven J, Norman RJ, Escobar-Morreale HF. Definition and significance of polycystic ovarian morphology: a task force report from the Androgen Excess and Polycystic Ovary Syndrome Society. *Hum Reprod Update.* 2014;20(3):334–52.
57. Overbeek A, Lambalk CB. Phenotypic and pharmacogenetic aspects of ovulation induction in WHO II anovulatory women. *Gynecol Endocrinol.* 2009;25(4):222–34.
58. Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group. Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome. *Fertil Steril.* 2004;81(1):19–25.
59. Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group. Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome (PCOS). *Hum Reprod.* 2004;19(1):41–7.
60. Dewailly D, Gronier H, Poncelet E, Robin G, Leroy M, Pigny P, Duhamel A, Catteau-Jonard S. Diagnosis of polycystic ovary syndrome (PCOS): revisiting the threshold values of follicle count on ultrasound and of the serum AMH level for the definition of polycystic ovaries. *Hum Reprod.* 2011;26(11):3123–9.
61. Eilertsen TB, Vanky E, Carlsen SM. Anti-Müllerian hormone in the diagnosis of polycystic ovary syndrome: can morphologic description be replaced? *Hum Reprod.* 2012;27(8):2494–502.
62. Li L, Chen X, Mo Y, Chen Y, Wenig M, Yang D. Elevated serum anti-Müllerian hormone in adolescent and young adult Chinese patients with polycystic ovary syndrome. *Wien Klin Wochenschr.* 2010;122(17–18):519–24.
63. Pigny P, Jonard S, Robert Y, Dewailly D. Serum anti-Müllerian hormone as a surrogate for antral follicle count for definition of the polycystic ovary syndrome. *J Clin Endocrinol Metab.* 2006;91(3):941–5.
64. Lin YH, Chiu WC, Wu CH, Tzeng CR, Hsu CS, Hsu MI. Anti-Müllerian hormone and polycystic ovary syndrome. *Fertil Steril.* 2011;96(1):230–5.
65. Hart R, Doherty DA, Norman RJ, Franks S, Dickinson JE, Hickey M, Sloboda DM. Serum anti-Müllerian hormone (AMH) levels are elevated in adolescent girls with polycystic ovaries and the polycystic ovarian syndrome (PCOS). *Fertil Steril.* 2010;94(3):1118–21.
66. Woo HY, Kim KH, Rhee EJ, Park H, Lee MK. Differences of the association of anti-Müllerian hormone with clinical or biochemical characteristics between women with and without polycystic ovary syndrome. *Endocr J.* 2012;59(9):781–90.
67. Homburg R, Ray A, Bhide P, Gudi A, Shah A, Timms P, Grayson K. The relationship of serum anti-Müllerian hormone with polycystic ovarian morphology and polycystic ovary syndrome: a prospective cohort study. *Hum Reprod.* 2013;28(4):1077–83.
68. Li HW, Anderson RA, Yeung WS, Ho PC, Ng EH. Evaluation of serum anti-Müllerian hormone and inhibin B concentrations in the differential diagnosis of secondary oligoamenorrhea. *Fertil Steril.* 2011;96(3):774–9.
69. Iliodromiti S, Kelsey TW, Anderson RA, Nelson SM. Can anti-Müllerian hormone predict the diagnosis of polycystic ovary syndrome? A systematic review and meta-analysis of extracted data. *J Clin Endocrinol Metab.* 2013;98(8):3332–40.
70. Azziz R, Carmina E, Dewailly D, Diamanti-Kandarakis E, Escobar-Morreale HF, Futterweit W, Janssen OE, Legro RS, Norman RJ, Taylor AE, et al. The Androgen Excess and PCOS Society criteria for the polycystic ovary syndrome: the complete task force report. *Fertil Steril.* 2009;91(2):456–88.
71. Sahmay S, Aydin Y, Atakul N, Aydogan B, Kaleli S. Relation of anti-Müllerian hormone with the clinical signs of hyperandrogenism and polycystic ovary morphology. *Gynecol Endocrinol.* 2014;30(2):130–4.
72. Laven JS, Mulders AG, Visser JA, Themmen AP, De Jong FH, Fauser BC. Anti-Müllerian hormone serum concentrations in normoovulatory and anovulatory women of reproductive age. *J Clin Endocrinol Metab.* 2004;89(1):318–23.
73. Piouka A, Farmakiotis D, Katsikis I, Macut D, Gerou S, Panidis D. Anti-Müllerian hormone levels reflect severity of PCOS but are negatively influenced by obesity: relationship with increased luteinizing hormone levels. *Am J Physiol Endocrinol Metab.* 2009;296(2):E238–43.
74. Thomson RL, Buckley JD, Moran LJ, Noakes M, Clifton PM, Norman RJ, Brinkworth GD. The effect of weight loss on anti-Müllerian hormone levels in overweight and obese women with polycystic ovary syndrome and reproductive impairment. *Hum Reprod.* 2009;24(8):1976–81.
75. Farquhar C, Brown J, Marjoribanks J. Laparoscopic drilling by diathermy or laser for ovulation induction in anovulatory polycystic ovary syndrome. *Cochrane Database Syst Rev.* 2012;6:CD001122.
76. Amer SA, Li TC, Ledger WL. The value of measuring anti-Müllerian hormone in women with anovulatory polycystic ovary syndrome undergoing laparoscopic ovarian diathermy. *Hum Reprod.* 2009;24(11):2760–6.
77. Elmashad AI. Impact of laparoscopic ovarian drilling on anti-Müllerian hormone levels and ovarian stromal blood flow using three-dimensional power Doppler in women with anovulatory polycystic ovary syndrome. *Fertil Steril.* 2011;95(7):2342–6 (2346 e1).
78. Bath LE, Wallace WH, Shaw MP, Fitzpatrick C, Anderson RA. Depletion of ovarian reserve in young women after treatment for cancer in childhood: detection by anti-Müllerian hormone, inhibin B and ovarian ultrasound. *Hum Reprod.* 2003;18(11):2368–74.
79. Iwase A, Nakamura T, Nakahara T, Goto M, Kikkawa F. Anti-Müllerian hormone and assessment of ovarian reserve after ovarian toxic treatment: a systematic narrative review. *Reprod Sci.* 2015;22(5):519–26.
80. Anders C, Marcom PK, Peterson B, Gu L, Unruhe S, Welch R, Lyons P, Behera M, Copland S, Kimmick G, et al. A pilot study of predictive markers of chemotherapy-related amenorrhea among premenopausal women with early stage breast cancer. *Cancer Invest.* 2008;26(3):286–95.
81. Anderson RA, Themmen AP, Al-Qahtani A, Groome NP, Cameron DA. The effects of chemotherapy and long-term gonadotrophin suppression on the ovarian reserve in premenopausal women with breast cancer. *Hum Reprod.* 2006;21(10):2583–92.

82. Yu B, Douglas N, Ferin MJ, Nakhuda GS, Crew K, Lobo RA, Hershman DL. Changes in markers of ovarian reserve and endocrine function in young women with breast cancer undergoing adjuvant chemotherapy. *Cancer*. 2010;116(9):2099–105.
83. Decanter C, Morschhauser F, Pigny P, Lefebvre C, Gallo C, Dewailly D. Anti-Müllerian hormone follow-up in young women treated by chemotherapy for lymphoma: preliminary results. *Reprod Biomed Online*. 2010;20(2):280–5.
84. Bozza C, Puglisi F, Lambertini M, Osa EO, Manno M, Del Mastro L. Anti-Müllerian hormone: determination of ovarian reserve in early breast cancer patients. *Endocr Relat Cancer*. 2014;21(1):R51–65.
85. Gracia CR, Sammel MD, Freeman E, Prewitt M, Carlson C, Ray A, Vance A, Ginsberg JP. Impact of cancer therapies on ovarian reserve. *Fertil Steril*. 2012;97(1):134–40 (e1).
86. Rosendahl M, Andersen CY, la Cour Freiesleben N, Juul A, Lossl K, Andersen AN. Dynamics and mechanisms of chemotherapy-induced ovarian follicular depletion in women of fertile age. *Fertil Steril*. 2010;94(1):156–66.
87. Cancer Research UK [Internet]. A trial looking at ovarian protection for premenopausal women having chemotherapy for breast cancer (OPTION). <http://www.cancerresearchuk.org/about-cancer/find-a-clinical-trial/a-trial-looking-at-ovarian-protection-for-premenopausal-women-having-chemotherapy-for-breast-cancer>. Accessed 28 Sept 2009.
88. Anderson RA, Hindmarsh PC, Wallace WH. Induction of puberty by autograft of cryopreserved ovarian tissue in a patient previously treated for Ewing sarcoma. *Eur J Cancer*. 2013;49(13):2960–1.
89. Wallace WH, Smith AG, Kelsey TW, Edgar AE, Anderson RA. Fertility preservation for girls and young women with cancer: population-based validation of criteria for ovarian tissue cryopreservation. *Lancet Oncol*. 2014;15(10):1129–36.
90. Somigliana E, Vercellini P, Vigano P, Ragni G, Crosignani PG. Should endometriomas be treated before IVF-ICSI cycles? *Hum Reprod Update*. 2006;12(1):57–64.
91. Chang HJ, Han SH, Lee JR, Jee BC, Lee BI, Suh CS, Kim SH. Impact of laparoscopic cystectomy on ovarian reserve: serial changes of serum anti-Müllerian hormone levels. *Fertil Steril*. 2010;94(1):343–9.
92. Iwase A, Hirokawa W, Goto M, Takikawa S, Nagatomo Y, Nakahara T, Manabe S, Kikkawa F. Serum anti-Müllerian hormone level is a useful marker for evaluating the impact of laparoscopic cystectomy on ovarian reserve. *Fertil Steril*. 2010;94(7):2846–9.
93. Ercan CM, Duru NK, Karasahin KE, Coksuer H, Dede M, Baser I. Ultrasonographic evaluation and anti-Müllerian hormone levels after laparoscopic stripping of unilateral endometriomas. *Eur J Obstet Gynecol Reprod Biol*. 2011;158(2):280–4.
94. Kitajima M, Khan KN, Hiraki K, Inoue T, Fujishita A, Masuzaki H. Changes in serum anti-Müllerian hormone levels may predict damage to residual normal ovarian tissue after laparoscopic surgery for women with ovarian endometrioma. *Fertil Steril*. 2011;95(8):2589–91 (e1).
95. Lee DY, Young Kim N, Jae Kim M, Yoon BK, Choi D. Effects of laparoscopic surgery on serum anti-Müllerian hormone levels in reproductive-aged women with endometrioma. *Gynecol Endocrinol*. 2011;27(10):733–6.
96. Tsolakidis D, Pados G, Vavilis D, Athanatos D, Tsalikis T, Giannakou A, Tarlatzis BC. The impact on ovarian reserve after laparoscopic ovarian cystectomy versus three-stage management in patients with endometriomas: a prospective randomized study. *Fertil Steril*. 2010;94(1):71–7.
97. Hirokawa W, Iwase A, Goto M, Takikawa S, Nagatomo Y, Nakahara T, Bayasula B, Nakamura T, Manabe S, Kikkawa F. The post-operative decline in serum anti-Müllerian hormone correlates with the bilaterality and severity of endometriosis. *Hum Reprod*. 2011;26(4):904–10.
98. Kwon SK, Kim SH, Yun SC, Kim DY, Chae HD, Kim CH, Kang BM. Decline of serum anti-Müllerian hormone levels after laparoscopic ovarian cystectomy in endometrioma and other benign cysts: a prospective cohort study. *Fertil Steril*. 2014;101(2):435–41.
99. Raffi F, Metwally M, Amer S. The impact of excision of ovarian endometrioma on ovarian reserve: a systematic review and meta-analysis. *J Clin Endocrinol Metab*. 2012;97(9):3146–54.
100. Somigliana E, Berlanda N, Benaglia L, Vigano P, Vercellini P, Fedele L. Surgical excision of endometriomas and ovarian reserve: a systematic review on serum anti-Müllerian hormone level modifications. *Fertil Steril*. 2012;98(6):1531–8.
101. Sugita A, Iwase A, Goto M, Nakahara T, Nakamura T, Kondo M, Osuka S, Mori M, Saito A, Kikkawa F. One-year follow-up of serum anti-Müllerian hormone levels in patients with cystectomy: are different sequential changes due to different mechanisms causing damage to the ovarian reserve? *Fertil Steril*. 2013;100(2):516–22 (e3).
102. Sonmezer M, Taskin S, Gemici A, Kahraman K, Ozmen B, Berker B, Atabekoglu C. Can ovarian damage be reduced using hemostatic matrix during laparoscopic endometrioma surgery? A prospective, randomized study. *Arch Gynecol Obstet*. 2013;287(6):1251–7.
103. Zaitoun MM, El Behery MM. Comparing long-term impact on ovarian reserve between laparoscopic ovarian cystectomy and open laprotomy for ovarian endometrioma. *J Ovarian Res*. 2013;6(1):76.
104. Hart RJ, Hickey M, Maouris P, Buckett W. Excisional surgery versus ablative surgery for ovarian endometriomata. *Cochrane Database Syst Rev*. 2008;2:CD004992.
105. Jang WK, Lim SY, Park JC, Lee KR, Lee A, Rhee JH. Surgical impact on serum anti-Müllerian hormone in women with benign ovarian cyst: A prospective study. *Obstet Gynecol Sci*. 2014;57(2):121–7.
106. Mohamed ML, Nouh AA, El-Beheri MM, Mansour SA. Effect on ovarian reserve of laparoscopic bipolar electrocoagulation versus laparotomic hemostatic sutures during unilateral ovarian cystectomy. *Int J Gynaecol Obstet*. 2011;114(1):69–72.
107. Ni L, Sadiq S, Mao Y, Cui Y, Wang W, Liu J. Influence of various tubal surgeries to serum anti-Müllerian hormone level and outcome of the subsequent IVF-ET treatment. *Gynecol Endocrinol*. 2013;29(4):345–9.
108. Ercan CM, Sakinci M, Coksuer H, Keskin U, Tapan S, Ergun A. Ovarian reserve testing before and after laparoscopic tubal bipolar electrodissection and transection. *Eur J Obstet Gynecol Reprod Biol*. 2013;166(1):56–60.
109. Hehenkamp WJ, Volkers NA, Broekmans FJ, de Jong FH, Themmen AP, Birnie E, Reekers JA, Ankum WM. Loss of ovarian reserve after uterine artery embolization: a randomized comparison with hysterectomy. *Hum Reprod*. 2007;22(7):1996–2005.
110. Arthur R, Kachura J, Liu G, Chan C, Shapiro H. Laparoscopic myomectomy versus uterine artery embolization: long-term impact on markers of ovarian reserve. *J Obstet Gynaecol Can*. 2014;36(3):240–7.
111. Chong YH, Campbell AJ, Farrand S, McLennan IS. Anti-Müllerian hormone level in older women: detection of granulosa cell tumor recurrence. *Int J Gynecol Cancer*. 2012;22(9):1497–9.
112. Lane AH, Lee MM, Fuller AF Jr, Kehas DJ, Donahoe PK, MacLaughlin DT. Diagnostic utility of Müllerian inhibiting substance determination in patients with primary and recurrent granulosa cell tumors. *Gynecol Oncol*. 1999;73(1):51–5.
113. Long WQ, Ranchin V, Pautier P, Belville C, Denizot P, Cailla H, Lhomme C, Picard JY, Bidart JM, Rey R. Detection of

- minimal levels of serum anti-Müllerian hormone during follow-up of patients with ovarian granulosa cell tumor by means of a highly sensitive enzyme-linked immunosorbent assay. *J Clin Endocrinol Metab.* 2000;85(2):540–4.
114. La Marca A, Volpe A. The Anti-Müllerian hormone and ovarian cancer. *Hum Reprod Update.* 2007;13(3):265–73.
115. Hudson PL, Dugas I, Donahoe PK, Cate RL, Epstein J, Pepinsky RB, MacLaughlin DT. An immunoassay to detect human Müllerian inhibiting substance in males and females during normal development. *J Clin Endocrinol Metab.* 1990;70(1):16–22.
116. Nelson SM, La Marca A. The journey from the old to the new AMH assay: how to avoid getting lost in the values. *Reprod Biomed Online.* 2011;23(4):411–20.
117. Rustomov O, Smith A, Roberts SA, Yates AP, Fitzgerald C, Krishnan M, Nardo LG, Pemberton PW. The measurement of anti-Müllerian hormone: a critical appraisal. *J Clin Endocrinol Metab.* 2014;99(3):723–32.
118. Li HW, Ng EH, Wong BP, Anderson RA, Ho PC, Yeung WS. Correlation between three assay systems for anti-Müllerian hormone (AMH) determination. *J Assist Reprod Genet.* 2012;29(12):1443–6.
119. Reichman DE, Goldschlag D, Rosenwaks Z. Value of anti-Müllerian hormone as a prognostic indicator of in vitro fertilization outcome. *Fertil Steril.* 2014;101(4):1012–8 (**e1011**).
120. Kedem A, Haas J, Geva LL, Yerushalmi G, Gilboa Y, Kanety H, Hanochi M, Maman E, Hourvitz A. Ongoing pregnancy rates in women with low and extremely low AMH levels. A multivariate analysis of 769 cycles. *PLoS One.* 2013;8(12):e81629.
121. Su HI, Sammel MD, Homer MV, Bui K, Haunschild C, Stanczyk FZ. Comparability of anti-Müllerian hormone levels among commercially available immunoassays. *Fertil Steril.* 2014;101(6):1766–72 (**e1761**).
122. Welsh P, Smith K, Nelson SM. A single-centre evaluation of two new anti-Müllerian hormone assays and comparison with the current clinical standard assay. *Hum Reprod.* 2014;29(5):1035–41.
123. Chai J, Howie AF, Cameron DA, Anderson RA. A highly-sensitive anti-Müllerian hormone assay improves analysis of ovarian function following chemotherapy for early breast cancer. *Eur J Cancer.* 2014;50(14):2367–74.
124. Decanter C, Peigne M, Mailliez A, Morschhauser F, Dassonneville A, Dewailly D, Pigny P. Toward a better follow-up of ovarian recovery in young women after chemotherapy with a hypersensitive anti-Müllerian hormone assay. *Fertil Steril.* 2014;102(2):483–7.