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The Role of Anti-Müllerian Hormone (Amh) In the Pathophysiology and Therapeutic Approaches in Female Infertility: A Review

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Abstract

Anti-Müllerian Hormone (AMH) is a hormone that belongs to the transforming growth factor beta (TGF-β) superfamily. It is involved in the development and functioning of the reproductive tract and is used as a marker for ovarian reserve. This review aims to outline the current knowledge of the physiology, pathophysiology, and therapeutic applications of AMH in females with infertility focusing on recent molecular and clinical developments. AMH plays a central role in regulation of folliculogenesis, inhibiting primordial follicle recruitment and thereby modifying the sensitivity of reduced number large growing follicles to FSH stimulation. However, as AMH levels are relatively stable throughout the menstrual cycle, it is currently considered one of best reproductive aging biomarkers of ovarian reserve and consequently reproductive lifespan. At physiological levels, AMH plays a major role in ovarian homeostasis, follicular maturation and the maintenance of the ovarian follicular reserve. Changes in AMH synthesis or action are related with multiple reproductive disorders, including polycystic ovary syndrome (PCOS), diminished ovarian reserve (DOR), premature ovarian insufficiency (POI), endometriosis, and granulosa cell tumors. Women with PCOS having increased AMH levels exhibit both follicular arrest and anovulatory infertility while women undergoing ovarian aging have less AMH production with consequent impaired fecundity. In addition, recent advances also postulated possible utilization of AMH in clinical settings ranging from being an ovarian protector during chemotherapy

(due to the effects on chemoresistance), to modulating specific pathways linked with reproductive disorders. Despite notable advances, several issues regarding AMH assay standardization, age-dependent reference ranges and the link between circulating AMH concentrations and reproductive health outcomes remain to be resolved. Additionally, the molecular mechanisms of AMH action in endocrine ovarian dysfunction are still poorly understood. Final remarks AMH is a major regulator of the female reproductive system and an important infertility biomarker. Ongoing studies of the physiology and signaling pathways for AMH may help to micro-focus diagnostic and therapeutic strategies in female infertility, as well as improve personalized reproductive medicine.

Keywords: AMH, Female Infertility, TGF-β, PCOS

Introduction

Female infertility, defined as the inability to achieve pregnancy after 12 months of unprotected intercourse in women younger than 35 or 6 months in women 35 or older, remains a major global health concern affecting nearly 15% of couples (Adebisi et al., 2026). Ovarian dysfunction, decreased ovarian reserve, ovulatory disorders and endocrine abnormalities are primary contributing factors among the many causes of female infertility. There has been a growing interest to understand the role of Anti-Müllerian Hormone (AMH) as an important biomarker and regulatory factor involved in female reproductive physiology and infertility pathogenesis in recent years. Due to its close correlation with ovarian follicular dynamics, ovarian reserve and assisted reproductive outcomes, AMH has become one of the most valuable indicators in reproductive endocrinology (Russell et al., 2022).

On a physiological basis, AMH is an exclusive regulatory factor of folliculogenesis that prevents the inappropriate activation of excess primordial follicles, and lessens sensitivity to FSH in developing follicles. It is a regulatory mechanism to preserve the ovarian follicular pool and reproductive lifespan. AMH is considered a more reliable marker of ovarian reserve compared with traditional hormonal markers such as FSH, estradiol and inhibin-B due to its relatively stable serum concentrations throughout the menstrual cycle (Barton, 2008).

Recent progress in reproductive medicine has been made the understanding of AMH extending its classical definition as a marker of ovarian reserve. New evidence signals that AMH serves several functions as a multifunctional signaling molecule not only influencing steroidogenesis but regulated with follicular maturation, hypothalamic–pituitary–gonadal axis and ovarian aging. Molecular studies of AMH have also shown that both SMAD-dependent signaling pathways and interactions with intracellular regulatory cascades such as Wnt/β-catenin and MAPK signaling. These findings have changed the mindset surrounding AMH, as it was previously considered merely a passive biomarker, to that of an active regulator along with implications for female reproductive physiology and disease pathogenesis (Li et al., 2026).

AMH may be a potential marker for the diagnosis of PCOS.5 Although there are a number of studies and consensus on the relationship between AMH and PCOS, it is not yet used as a diagnostic criterion. Whether this is related to the presence of variability in the PCOS subgroup remains unclear. PCOS women have very elevated AMH concentrations in part due to more small antral follicles present and also altered granulosa cell activity (Ran et al., 2021). AMH is known to inhibit FSH responsiveness and disrupt folliculogenesis, leading to abnormal follicular maturation and ovulatory dysfunction. As a result, AMH has become an increasingly valuable diagnostic and prognostic biomarker of reproductive dysfunction in women with PCOS future fertility (Dumont et al., 2015). Additionally, AMH has been suggested as an alternative to antral follicle count in the diagnosis of PCOS due to its close association with ovarian follicular activity (Rudnicka et al., 2021).

In great contrast, lower levels of AMH correlate strongly with low ovarian reserve (DOR), early menopause (POI) or delayed maturation of follicles living longer than average (lower reproductive aging), and poor responder to stimulation in assisted reproduction treatments (ARTs). Reduced AMH concentrations are often seen in women with diminished oocyte quantity and reduced pregnancy rates on IVF cycles (Moolhuijsen & Visser, 2020). Thus, AMH evaluation has emerged as an invaluable component in fertility clinics, being used to predict ovarian response, customize stimulation algorithms and counsel the individual patients with regard to their reproductive potential. AMH has substantially enhanced the selection of strategies for fertility treatment and reduced the likelihood of ovarian hyperstimulation syndrome and cycle cancellation (Robertson, 2008).

As well as its diagnostic role, AMH has now been also proposed as a new therapeutic target in reproductive medicine. Experimental studies indicate that AMH may preserve ovarian reserve during gonadotoxic chemotherapy by preventing excessive activation of primordial follicles. So there has been a great interest in the use of AMH-based therapies for fertility preservation in cancer patients (von Wolff et al., 2025). Additionally, AMH signaling pathways are a promising

therapeutic target in PCOS, ovarian aging and other hormone-responsive gynecological disorders. The translational use of AMH in reproductive medicine is an emerging and rapidly evolving area that holds tremendous potential for female fertility preservation and individualized reproductive therapy (Rodgers et al., 2021).

Even though major advances in AMH physiology and clinical utility have been made, controversies and limitations remain. There are many challenges in interpreting AMH assessment including variability among AMH assay methods, absence of index standard diagnostic cut-off values among AMH assay tests due to non-inclusion of assay categories, ethnic and environmental influences and age-related hormonal fluctuations that cloud clinical utility (Li et al., 2021). Additionally, despite the widespread use of AMH as a measure of ovarian reserve, the degree to which it is directly associated with oocyte quality and live-birth outcome remains incompletely elucidated. These limitations underline the importance of further studies to elucidate the role of AMH in infertility pathophysiology, and to determine how best it can be therapeutically employed (Sinha et al., 2022).

Thus, this review seeks to outline the physiological role of AMH in female reproduction and its contributing role in the pathophysiology of female infertility, as well as its developing clinical utility within reproductive medicine. We will focus on recent developments related to AMH signaling pathways, clinical application for female fertility assessment and PCOS-specific infertility, assisted reproductive technologies (ARTs), and potential applications of these data in fertility preservation approaches.

The physiology and control of AMH

Anti-Müllerian hormone (AMH), also known as Müllerian-inhibiting substance (MIS) is a glycoprotein hormone from the TGF-β superfamily. During human reproductive physiology, AMH acts as a cornerstone and is one of the most important modern indicators in reproductive endocrinology and infertility evaluation. Originally recognized for its involvement in males fetal sexual differentiation, AMH is now well known also for its indispensable roles in ovarian physiology, follicular development and female fertility regulation (di Clemente et al., 2021).

Anti-müllerian hormone (AMH) is produced by granulosa cells of small, growing follicles in the ovary. Serum AMH levels strongly correlate with the number of growing follicles, and therefore AMH has received increasing attention as a marker for ovarian reserve. AMH is produced during fetal life (beginning late gestation) and continues throughout the reproductive years until menopause, whereupon serum concentrations are nearly undetectable as a result of ovarian follicular depletion. The level of circulating AMH mirrors the number of developing follicles, and thus is a true marker for ovarian reserve and fertility (Dewailly et al., 2014).

From an evolutionary chestnut that defines how a female follicle develops, AMH is physiologically at the center of the annual cycle in regulating this complex developmental process whereby primordial follicles evolve to become ovulatory. AMH inhibition of the over-recruitment of primordial follicles from an inactive pool is one of their major physiological actions. This inhibitory action is critical in maintaining the limited population of ovarian follicles during reproductive lfie and averting an early depletion of the ovary. With respect to the ovarian reserve, AMH is responsible for a controlled initial recruitment of primordial follicles, which extends reproductive lifespan and combats ovarian aging (Durlinger et al., 2002).

AMH plays an additional key physiological role in the regulation of follicular sensitivity to a follicle-stimulating hormone (FSH). AMH reduces FSH-stimulated granulosa responsiveness during early follicular development, thus regulating ovarian selection and maturation of follicles. This process inhibits excessive follicular growth and allows for the stepwise selection of a single dominant follicle in each menstrual cycle. AMH is expressed mostly until higher grade preantral follicles—levels wane as the follicles get larger & more developed (Visser et al., 2012).

AMH exerts its biological actions by binding to its cognate receptor, anti-Müllerian hormone receptor type II (AMHR2), which is expressed predominantly in ovarian granulosa cells and other reproductive tissues. After receptor binding, intracellular signaling pathways activated, especially the SMAD-dependent one which regulates gene transcription regarding cellular differentiation, proliferation and follicular development (Josso et al., 2013). Based on the recent molecular studies, AMH signaling and other intracellular pathways such as MAPK and Wnt/β-catenin interact with each other, indicating that AMH plays an even wider role in regulating the ovarian physiology than previously appreciated (Li et al., 2026).

Many physiological and endocrine factors can affect AMH secretion. Age is really the only factor that would be an absolute predictor of AMH levels. Serum AMH concentration starts to increase following puberty, peaks at early reproductive life and subsequently declines due to an age-related loss of ovarian primordial follicles. Therefore, AMH has been recognized and

applied extensively clinically as a biomarker of ovarian aging and reproductive lifespan (Broer et al., 2014).

In contrast to other reproductive hormones, AMH displays stable serum concentrations along the menstrual cycle and is less affected by cyclic hormonal variability. This stability improves its clinical applicability as compared to traditional ovarian reserve markers like FSH, estradiol and inhibin-B (La Marca & Volpe, 2006). However, indirect control of gonadotropins and stage of follicle maturation may still affect AMH secretion.

AMH secretion is partly regulated by follicle-stimulating hormone via its effects at the level of granulosa cells and growth and development of follicles. AMH production sequentially decreases during the transition from small antral follicles to dominant follicles, becoming much lower as follicles acquire greater dependence on FSH stimulation. Hence it is negatively correlated to follicular growth (Dewailly et al, 2014).

Regulation of AMH is also mediated by steroid hormones. Estrogen has been described as being inhibitory to AMH gene expression as follicular development continues into the later stages, whilst androgens have been implicated in stimulating AMH production in some pathological states. The interaction is especially clear for females with polycystic ovary syndrome (PCOS), i.e. PCOS women often have an elevation of androgen levels, combined with more small follicles causing marked increases in AMH concentrations. It is thought that in PCOS excess AMH contributes to follicular arrest and impaired ovulation by decreasing follicular response to FSH (Pellatt et al., 2007).

Many pathological processes have been known to affect AMH physiology and secretion. Diminished ovarian reserve (DOR), premature ovarian insufficiency (POI), or ovarian aging is indicated by significantly lower serum AMH levels due to reduced follicular numbers and impaired granulosa cell function in women. In comparison, women with PCOS show high AMH levels due to increased preantral follicle numbers and abnormal granulosa cell activity. Thus, AMH has gained the status of important diagnostic and prognostic biomarker in reproductive disorders (Pellatt et al., 2007).

AMH levels may also be influenced by environmental and lifestyle predictors. A diminished ovarian reserve and lower AMH concentration have been reported for smoking, obesity, environmental toxins as well as chemotherapeutic and radiological treatments. Current smoking has also been associated with lower AMH levels (Sowers et al., 2018). Variations in AMH are also thought to impact measurement of reproductive potential due to contributions to differences between ovarian reserve, reproductive lifespan & thereby fertility potential in women with respect to mutations involving AMHR2 genes (Broer et al., 2014).

The clinical use of AMH has been wider historically during the recent years. AMH testing has become a common tool in fertility clinics to quantify ovarian reserve, predict ovarian response in assisted reproductive technologies (ART), stratify patients into different stimulation protocols, diagnose PCOS and assess the age-related decline of the female reproductive potential. Also, AMH has become quite pivotal in fertility preservation schemes among women females undergoing gonadotoxic cancer treatments (Rodgers et al., 2021).

There is also emerging evidence that intraovarian AMH may be a therapeutic target in reproductive medicine itself. Evidence from experimental studies suggests that AMH may prevent chemotherapy-induced loss of ovarian reserve through inhibiting excessive primodrial follicle activation. Moreover, AMH signaling pathways are also being modulated as a classic and promising therapeutic target to treat PCOS and ovarian dysfunction (Russell et al., 2022).

The pathophysiology of AMH Disorders

AMH is a delta goosey protein hormone secreted by the ovulatory follicle granulosa cells which surround the oocytes. Estrogen is produced from the initial stages of folliculogenesis and is essential during follicular development, from primordial follicles through selection for dominance. AMH inhibited the recruitment of primordial follicles and its effect on enhancing granulosa cell sensitivity to FSH was confirmed in several experimental models (Sonigo et al., 2019). Modified AMH concentrations may indicate dysregulation of ovarian reserve, follicular development, endocrine control and aging processes in the ovary. Thus, AMH disorders are being appreciated as meaningful factors of female infertility and reproductive dysfunction (di Clemente et al., 2021).

Serum AMH levels are increased in women with PCOS, particularly in those with the phenotype of polycystic ovaries on ultrasound. This is due, at least in part, to the observation that AMH mRNA levels in granulosa cells of small and large follicles are also substantially increased in PCOS vs. controls. Similarly, granulosa cell AMH receptor II mRNA levels were far higher in small follicles from PCOS robustly compared with controls (Laven, 2025). AMH elevation in PCOS is a consequence of the dysregulated ovarian physiology that leads to increased numbers (but not larger size) of preantral and small antral follicles, abnormal granulosa cell activity and androgen excess. Under

physiological circumstances, AMH regulates follicular recruitment and decreases the sensitivity of preantral follicles to stimulation by follicle-stimulating hormone (FSH). Conversely, AMH production may be impaired in healthier women, and excessive amounts of it produced in polycystic ovary syndrome (PCOS) could exaggerate these inhibitory effects resulting in follicular arrest and chronic anovulation (Dumont et al., 2015).

In PCOS, increased AMH also promotes endocrine dysregulation via interaction with the hypothalamic–pituitary–ovarian axis. AMH has also been shown to promote gonadotropin releasing hormone (GnRH) neuronal activity, resulting in elevation of luteinizing hormone (LH) secretion and aggravated ovarian androgen production (Abbott et al., 2024). The additional stimulation of granulosa cells by hyperandrogenism to produce AMH establishes a self-perpetuating cycle causing blockage of follicle development/ovulation and hence infertility. AMH therefore not only serves as a biomarker of PCOS but also acts actively in the pathophysiology of this complex endocrine disorder (Li et al., 2011).

On the contrary, low AMH is strongly correlated with diminished ovarian reserve (DOR) and premature ovarian insufficiency (POI). DOR is defined as a decrease in the number and quality of ovarian follicles earlier than expected perimenopause, while POI is defined as premature loss of ovarian function before the age of 40 years. Low AMH concentrations reflect depletion of functional ovarian follicles and impaired granulosa cell activity in both conditions. AMH is one of the first detectable biochemical changes in ovarian ageing, and decreases before documented abnormalities in FSH or menstrual irregularities (Broer et al., 2014).

The pathophysiology of DOR and POI is mediated through various complex pathways that involve accelerated follicular apoptotic change (accelerated ovarian ageing), oxidative stress, mitochondrial oxidative dysfunction, autoimmune injury and alteration in mitothymic intercellular communication, genetic aberrations, as well as exposure to pesticides and endocrine disruptors via senescence-associated secretory phenotype (SASP)-induced follicles demise, unsafe sex during young age (Hirano et al., 2025). Lower levels of AMH are produced in these AAC disorders represent defective folliculogenesis, and impaired gonadotropin responsiveness. Low AMH was associated with poor ovarian response to assisted reproductive technologies (ART), lower oocyte yield, and reduced pregnancy rates in women undergoing ART (La Marca & Volpe, 2006).

The disorders related to AMH are also important genetic factors. Some mutations or polymorphisms in the AMH gene or anti-Müllerian hormone receptor type II (AMHR2) gene that are able to destroy AMH signaling could also disturb normal reproductive physiology (Josso et al., 2013). Ggenetic anomalies may have consequences on follicular recruitment, preservation of ovarian reserve and reproductive life span. In addition, variations in genes associated with AMH pathways are also associated with differences in menopause timing and rates of ovarian aging between populations (Pelosi et al., 2015).

Granulosa cell tumors (GCTs) of the ovary may also have AMH abnormalities. Granulosa cell tumors are sex-stromal derived, hormonally active ovarian neoplasms further differentiated by their originating granulosa cells resulting in AMH overproduction (Pankhurst & McLennan, 2013). AMH has been recognized as a tumor marker in adult granulosa cell tumors for some time, and levels can be elevated in ~93% cases. AMH level is also high in some ovarian tumors such as adult granulosa cell tumor and can be used to monitor the response of therapy and recurrence (Shrikhande et al., 2020).

Another key pathological condition where AMH may be involved is endometriosis. The reason is that in women with ovarian endometriomas this results in a diminished concentration of AMH levels compared to healthy women suggesting poor ovarian reserve (Raffi et al., 2012). The potential mechanisms to account for this association include chronic inflammatory injury, oxidative stress and fibrosis in the areas of ovarian tissue subjected to surgical trauma during laparoscopic excision of endometrioma(s). The inflammation and altered microenvironment associated with endometriosis can be detrimental to granulosa cell function, coagulating follicular survival and resulting in decreased secretion of AMH (Özcan et al., 2025).

AMH pathophysiology may be caused by environmental and iatrogenic factors as well. As we all know, ovarian toxicity and follicular depletion after chemotherapy and/or radiotherapy lead to a significant decrease of AMH level (Anderson & Wallace, 2013). Cytotoxic agents are able to induce apoptosis of granulosa cells and primordial follicles thus accelerating ovarian aging and increasing the risk of infertility. Likewise, smoking, overweight and obesity, chemical exposure in utero and during adulthood to endocrine-disrupting chemicals, have been linked to suboptimal ovarian reserve and abnormal AMH concentrations (Regan et al., 2018).

Inversely, AMH physiology may also be affected by other autoimmune disorders. Granulosa cells and ovarian follicles can be compromised by autoimmune-mediated ovarian injury leading to decreased AMH secretion and premature ovarian dysfunction.

In addition, chronic systemic inflammatory diseases may also influence ovarian endocrine regulation and folliculogenesis indirectly through cytokine-mediated pathways (Sowers et al., 2018).

Anti-Müllerian hormone (AMH) is a glycoprotein member of the transforming growth factor-beta (TGF-β) superfamily and its main role in folliculogenesis is that of an ovarian regulator. Polycystic ovary syndrome (PCOS), one of the most common endocrine and metabolic disorders mainly characterized by hyperandrogenism, anovulation, and polycystic ovarian morphology (PCO) is well-known for dysregulated AMH expression (Ke et al., 2025). There are possible correlations between the AMH associated ovarian dysfunction with the alteration of SMAD signaling, Wnt/β-catenin pathways and granulosa cell receptor expression. These data substantiate the notion that AMH defects are not just a bioassay but may be involved in the active pathogenesis of fertility problems (Li et al., 2026).

At a clinical level, AMH disorders have major implications for infertility diagnosis and management. Through specifying the ovarian reserve and copying with predictions on that responsiveness, diagnosing PCOS (polycystic ovary syndrome), characterizing the aging of an ovary and creating fertility protective talent based mostly on AMH measurement have now turn out to be broadly used. Both excessive AMH elevations and excessive suppression indicate endocrine imbalances, sectional assessment of the patients age, endocrine status and underlying pathology is similarly key to interpretation (Russell et al., 2022).

The therapeutic approaches od AMH in female infertility

Due to its important physiological role in ovarian follicular development and close association with the ovarian reserve and fertility potential, anti-Müllerian hormone (AMH) has become an increasingly important practice tool in modern reproductive medicine. AMH has been used not only for its diagnostic value, but also for therapeutic approaches in female infertility diseases today, especially in assisted reproductive technologies (ART), individualised ovarian stimulations, fertility medication and endocrine reproductive disorders including PCOS and DOR. Developing knowledge on AMH physiology has allowed clinicians to utilize AMH not only as a biomarker, but also a guide for assisted reproductive treatment strategies tailored to each unique patient (Russell et al., 2022).

The greatest relevance for therapy assessments is the predictive capacity of anti-Müllerian hormone (AMH) as a test of ovarian response in controlled ovarian stimulation programs for IVF. Serum AMH levels correlate highly with the follicle depleting ovarian reserve and can guide clinicians to choose fertility treatment strategies before its initiation (Sun et al., 2022). Women with higher AMH produce more oocytes during stimulation cycles in addition to having a stronger ovarian response while women with lower AMH tend to be poor responders and have reduced oocyte yield. As a result, AMH measurement permits fertility specialists to customize gonadotropin doses and more effectively design ovarian stimulation protocols (Broer et al., 2014).

Today personalized ovarian stimulation is available through AMH determinations, and it has largely increased the safety and efficiency of assisted reproductive treatments. Women with high AMH levels, especially those suffering from polycystic ovarian syndrome (PCOS) are at increased risk for the life-threatening complication known as ovarian hyperstimulation syndrome (OHSS); a condition involving an exaggerated ovarian response and increased vascular permeability (Muharam et al., 2022). Thus, in these patients AMH-guided reduction of gonadotropin doses and the adoption of GnRH antagonist protocols ensure OHSS risk minimization whilst ensuring adequate oocyte retrieval. On the other hand, women with low AMH levels may additionally require higher doses of gonadotropins or alternative stimulation strategies to optimize the recruitment of follicles (Dewailly et al., 2014).

AMH has also an important therapeutic role in fertility preservation. Ovarian follicular depletion and subsequent infertility is common among women receiving chemotherapy, radiotherapy or ovarian surgical treatment. AMH is closely related to ovarian reserve and has been used as a marker for baseline function prior to gonadotoxic therapies and to assess gonadal damage after therapy (Anderson & Wallace, 2013). Recent research indicated that women with AMH ≤ 0.5 ng/mL achieved oocyte yields consistently below clinically meaningful thresholds, in contrast to women with higher AMH levels, who exhibited significantly better responses to stimulation (Coccia et al., 2026).

In line with this hypothesis, recent experimental studies show that AMH itself might have direct protective therapeutic effects on ovarian reserve in the setting of chemotherapy. Since AMH also inhibits primordial follicle activation, it has the potential to alleviate chemotherapy-associated premature depletion of ovarian follicles by keeping them in a dormant state. It has been established in animal studies that intra-venous administration of recombinant AMH can lead to reduced follicular loss and preservation of ovarian reserve following exposure to different cytotoxic agents. While these findings are still exploratory, they

carry implications for potential future therapeutic applications of AMH-centered ovarian preservation approaches (Kano et al., 2017).

A separate instrumental use of AMH lies in the field of treatment for polycystic ovary syndrome (PCOS), one of the main factors behind severe female infertility. Women with PCOS typically display increased AMH levels due to the presence of more small antral follicles combined with atypical granulosa cell stimulating activity. Their high AMH concentrations also play a role in follicular arrest and anovulation by inhibiting FSH-mediated sensitivity of follicles (Dumont et al., 2015). Therefore, AMH measurement is used more and more for the selection of etiquette during simulated ovulation stimulation and to forecast treatment response (in PCOS).

In women with PCOS, the use of AMH to set therapeutic thresholds may help in OI agent selection (e.g. clomiphene citrate, letrozole and gonadotropins). Patients with high serum AMH may need modified treatment protocols due to increased ovarian sensitivity and the risk of hyperstimulation. In addition, decreasing AMH levels during treatment may reflect progress in follicular maturation and reinstatement of ovulatory function (Rudnicka et al., 2021).

women with diminished ovarian reserve (DOR) and premature ovarian insufficiency (POI) have significantly reduced AMH levels [45,46]. During early male embryonic development, AMH is secreted in large quantities by the supporting cells of the fetal testes (Wang et al., 2026). In these patients, AMH helps to identify the reproductive strategy of choice for professionals such as aggressive ovarian stimulation, donor oocytes or fertility preservation counseling. Treatment planning using AMH enables realistic counseling regarding reproductive prognosis and treatment expectations (La Marca & Sunkara, 2014).

Since the introduction of methods to extract and quantify AMH in either blood or tissue, and its recognition as a marker of ovarian reserve, AMH has also gained importance outside the reproductive endocrinology field; it is being used for prediction of reproductive outcomes after surgery for ovarian neoplasms as well as following endometriosis treatment. Surgical excision is a common method of treatment for ovarian endometriomas, although postoperative decreases in ovarian reserve are frequently observed in these patients (Iwase et al., 2015). AMH in serum is strongly associated with a number of factors including age and causes of infertility; Hence, monitoring the preoperative AMH levels can be beneficial to patients who may think that their future fecundity might be affected after surgery (such as unilateral oophorectomy or even cystectomy) and it is also helpful in evaluating potential harm from residual ovarian tissue which ultimately assists fertility. AMH measurement in the same way helps to assess ovarian recovery after medical or surgical treatment management (Raffi et al., 2012).

Direct targeting of AMH signaling pathways in reproductive disorders is an area of ongoing research for novel therapeutics. Proposals for future treatment options of conditions with abnormal folliculogenesis and ovarian dysfunction are possible due to modulation of AMH receptors and downstream signaling pathways, as suggested by experimental studies. For PCOS, strategies to downregulate AMH may promote improvement in follicular development and ovulation. On the contrary, therapies that augment AMH activity could protect the ovarian reserve from premature follicular depletion in susceptible women (Li et al., 2026). AMH guided therapy shows great clinical utility but has several limitations. Reference values for AMH assays differ between laboratories and are not universally available. Moreover, AMH is associated with ovarian reserve and consequently can also approximate total ovarian quantity, but it cannot always reliably predict oocyte quality or live birth rates. Thus, in interpreting AMH levels clinical status such as the woman's age, antral follicle count (AFC), hormonal profiles and clinical history should be considered (Russell et al., 2022).

Conclusion

This review highlighted the role of anti-Müllerian hormone (AMH) as a key factors in female reproduction physiology and fertility preservation. It has s critical role in follicular recruitment, maintenance of ovarian reserve, and modulation of follicular sensitivity to FSH. AMH has both a physiological function as well as regulatory role in controlling folliculogenesis by maintaining the balance between activation and depletion of follicles. Abnormal AMH production and/or action is intimately linked to key reproductive disorders, including polycystic ovary syndrome (PCOS), diminished ovarian reserve (DOR), premature ovarian insufficiency (POI), endometriosis, and granulosa cell tumors. High serum AMH concentrations are usually associated with follicular arrest and anovulation in PCOS, whereas low AMH levels are related to ovarian aging and impaired fertility. The very recent advances in reproductive medicine has pushed the role of AMH beyond being a simple ovarian reserve marker illustrative to its clinical significance. Due to its utility in infertility diagnosis, predicting ovarian response during assisted reproductive technologies, crafting individualized ovarian stimulation protocols and directing fertility preservation strategies AMH is now an essential diagnostic tool. Furthermore, emerging data indicate that AMH can play a novel role as a future potential therapeutic target in

reproductive disorders by modulating ovarian signalling pathways and protecting the ovarian reserve from gonadotoxic treatments. However, there are important limitations and controversies regarding the interpretation and clinical application of AMH measurements: assay variability, lack of universal reference ranges and incomplete understanding of the relationship between AMH levels and reproductive outcomes. Further molecular and clinical studies are thus warranted to elucidate that AMH acts in a more complex physiological or pathological manner, allowing researchers to optimize the diagnostic and therapeutic applications of AMH as a potential diagnostic/prognostic marker in female infertility. In general, AMH is a key factor in the knowledge of pathophysiology involving infertile female reproductive health. Further exploration of AMH physiology and its involvement in disorders and therapeutic applications seems to offer immense potential for the future of personalized reproductive medicine, and understanding fertility management approaches.

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