International Journal of Pharmacy and Pharmaceutical Research (IJPPR)
Volume 31, Issue 12, December 2025 ijppr.humanjournals.com ISSN: 2349-7203

Review on Cellular and Molecular Pathogenesis of PCOD

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Received: 15 November 2025 Revised: 29 November 2025 Accepted: 16 December 2025

ABSTRACT

Polycystic ovarian syndrome (PCOS) is a common condition in women of reproductive age. Its cause is not fully known, but anti-Müllerian hormone (AMH) seems to play an important role. Women with PCOS usually have higher levels of AMH. High AMH prevents normal follicle growth, which leads to anovulation. It also lowers estrogen and raises androgen levels, which can make insulin resistance worse. Because of this, AMH is linked to both reproductive and metabolic issues in PCOS. High AMH often connects to poor responses to treatments like weight loss, ovulation induction, and ovarian drilling. Patients generally show improvement after treatment when AMH levels are lower. Understanding how AMH works in PCOS could help develop new treatments and assist doctors in selecting better options for women with the condition.

Keywords: AMH, Androgen, PCOS, Anovulation, Insulin Resistance, Reproductive, Metabolic Issues

INTRODUCTION

Polycystic ovary syndrome (PCOS) is the most common hormone disorder in women who can have children. It affects about 5-10% of this group and is the main cause of ovulation problems [1]. The Rotterdam 2003 consensus states that to diagnose PCOS, at least two of the following three criteria must be present: irregular or absent ovulation, signs of high androgen levels, and/or polycystic ovaries as seen on an ultrasound [2]. PCOS often comes with insulin resistance, obesity, and changes in hormone secretion. Stein and Leventhal first described this syndrome in 1935 in women who experienced missed periods, infertility, excessive hair growth, and polycystic ovaries [3]. Ovarian hyperthecosis and higher androgen production are central to the hormonal issues associated with it. Both genetic and environmental factors are thought to play a role in its development [4]. Families often see clusters of cases, and certain genetic pathways have been linked to the metabolic and hormonal problems in PCOS [5]. Environmental factors also matter, as shown in studies with rhesus monkeys where exposure to androgens before birth resulted in PCOS-like traits in female offspring [6]. Despite years of research, the exact cause of PCOS is still unclear.

Anti-Müllerian hormone (AMH) is released by granulosa cells and plays a key role in the development of follicles [7]. Women with PCOS have serum AMH levels that are two to three times higher than those of women with normal ovulation, which reflects the larger number of small antral follicles [8]. It remains uncertain if AMH is just a marker of PCOS or if it actively contributes to its development. This review will look at the role of AMH in ovarian function and the increasing evidence linking AMH to the development of PCOS. A better understanding of this relationship may lead to new treatment options for PCOS.

1. AMH and Ovarian Function

Anti-Müllerian hormone (AMH), also known as Müllerian inhibiting substance (MIS), is a glycoprotein hormone in the transforming growth factor-β family [9]. Unlike other members of this family, AMH is mainly produced in the gonads and has an effect on reproductive organs [10]. The AMH gene is found on chromosome 19 [11]. In women, AMH is released by granulosa cells, decreases with age, and becomes undetectable after menopause [12]. Its levels stay relatively stable throughout the menstrual cycle [13].

AMH also contributes to male embryonic development by stopping Müllerian duct formation. If AMH is absent or if there are defects in its receptor, the uterus and Fallopian tubes can persist [14].

International Journal of Pharmacy and Pharmaceutical Research (IJPPR)
Volume 31, Issue 12, December 2025 ijppr.humanjournals.com ISSN: 2349-7203

During folliculogenesis, AMH is most abundant in pre-antral and small antral follicles (≤4 mm) and decreases in larger follicles, vanishing beyond 8 mm [15]. This drop is key for selecting the dominant follicle [16]. AMH slows down follicle growth: mice without AMH show quicker folliculogenesis [17]. In lab settings, AMH lowers FSH-stimulated aromatase activity and estradiol production [18].

AMH functions through type I and type II receptors that activate Smad signaling [19]. These receptors are present in granulosa cells, and AMHRII has also been found in theca and luteal cells, indicating paracrine signaling [20]. Overall, AMH stops premature follicle recruitment. As follicles grow larger, AMH decreases, which allows for FSH sensitivity, estrogen production, and ovulation (Figure 1).

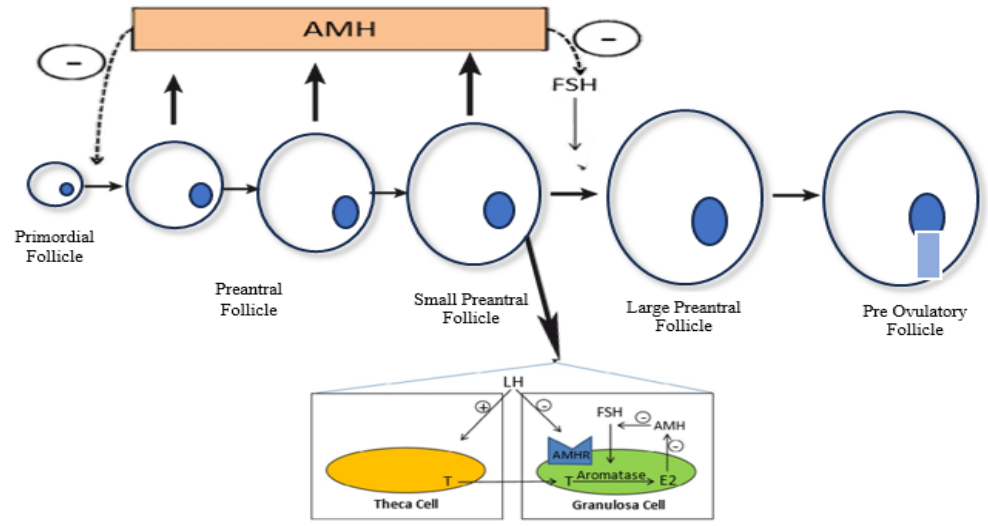

Figure 1: Anti-Müllerian hormone (AMH) production changes with follicular development. It primarily comes from pre-antral and small antral follicles. AMH has two main inhibitory effects. First, it prevents the FSH-independent recruitment of primary follicles from the primordial pool. Second, it hinders the FSH-dependent maturation and selection of the dominant follicle by reducing how responsive follicles are to FSH. At the molecular level, AMH interacts with both FSH and LH. Specifically, AMH reduces FSH-stimulated aromatase activity in granulosa cells. This limits the conversion of testosterone into oestradiol. As oestradiol levels drop, this feedback inhibits AMH itself. Meanwhile, LH not only encourages testosterone production in theca cells but also directly affects granulosa cells to decrease the expression of AMH receptor II (AMHRII). Abbreviations: AMHRII = anti-Müllerian hormone receptor II; E2 = oestradiol; T = testosterone.

2. Role of AMH in PCOS pathogenesis

PCOS ovaries have more pre-antral and small antral follicles, indicating a halt in follicle growth when AMH levels are normally highest [21]. Consequently, serum AMH levels are consistently higher in women with PCOS compared to those with normal ovaries [8]. In the follicular fluid, AMH levels in anovulatory PCOS women are about five times greater than in ovulatory women [22]. Pellatt et al. (2007a) demonstrated that granulosa cells from anovulatory PCOS produce about 75 times more AMH than normal granulosa cells. Catteau-Jonard et al. (2008) confirmed the increased AMH mRNA expression in polycystic ovaries. These findings imply that the overproduction of AMH in PCOS results from both the larger quantity of follicles and the higher AMH output per granulosa cell. Notably, AMH levels correlate with the severity of PCOS symptoms such as irregular cycles, hyperandrogenism, and polycystic ovarian morphology [23]. This supports the notion that AMH is not only a marker but also plays a role in the development of PCOS.

3. AMH and Hyperandrogenism in PCOS

Androgens are made in theca interna cells and changed into estrogens in granulosa cells by the action of aromatase [24]. Luteinizing hormone (LH) encourages this process by increasing androgen production in theca cells. In PCOS, higher serum AMH levels have been linked to increased androgen levels like testosterone and androstenedione [25]. This suggests that AMH may play a role in

hyperandrogenism. One theory is that AMH lowers aromatase activity in granulosa cells, which limits estrogen production and leaves more androgens unconverted [16]. Experimental studies support this: AMH decreased aromatase activity and mRNA expression in rat fetal ovaries [26]. In human granulosa-like cells, AMH also stopped CYP19 gene expression, which reduced FSH-induced estradiol production [18].

AMH may also influence theca cells, as AMHRII receptors are found there, possibly leading to improper androgen production in PCOS [27]. This suppression of FSH-induced aromatase activity may help explain abnormal follicular development in PCOS [28]. Genetic evidence backs this connection: Kevenaar et al. (2008) discovered that the AMH gene Ile49Ser variant was tied to androgen levels in PCOS, likely affecting aromatase regulation. All in all, current evidence points to AMH being a factor in hyperandrogenism in PCOS, although other metabolic and hormonal elements also play significant roles.

4. AMH and Metabolic manifestation

Women with PCOS have a two- to three-fold higher risk of metabolic syndrome compared to healthy women of the same age group [29]. The most common metabolic issues are insulin resistance (IR) and obesity. These problems are more severe in anovulatory PCOS and also influence androgen regulation [30]. Although obesity worsens IR, women with PCOS are still more insulin resistant than expected based on obesity alone [31]. Some studies suggest that IR might affect AMH levels and contribute to hyperandrogenism [32], but the evidence is mixed. La Marca et al. (2004b) first reported a positive link between AMH and IR as measured by HOMA-IR. Similar findings were reported by Fonseca et al. (2014), Nardo et al. (2009), and Skalba et al. (2011). In contrast, other studies found no association, including those by Eldar-Geva et al. (2005), Caglar et al. (2013), Cassar et al. (2014), and Pigny et al. (2003). Several studies in Asian women also found no connection between AMH and IR [33], possibly due to their leaner body composition. Conflicting results may reflect differences in study populations. Since AMH has been variably correlated with BMI [34], obesity might act as a confounding factor. Larger studies that control for BMI and other variables are needed to clarify the relationship between AMH and IR.

Oxidative stress and advanced glycation end products (AGE) have also been linked to PCOS [35]. AGE are formed by nonenzymatic glycation of proteins, lipids, and nucleic acids, a process that speeds up in diabetes and IR [36]. Increased AGE levels have been found in the serum and ovaries of women with PCOS [35]. AGE can disrupt insulin signaling in granulosa cells [37]. Unlike RAGE, which mediates harmful effects, soluble RAGE (sRAGE) binds AGE in circulation, reducing their impact [38]. Diamanti-Kandarakis et al. (2009) reported a positive relationship between AMH and AGE, especially in anovulatory PCOS, suggesting they may both play a role in ovulatory dysfunction. Irani et al. (2014b, 2015) showed that vitamin D supplementation reduced AMH and increased sRAGE, leading to improvements in PCOS features. Merhi et al. (2015) found that AGE exposure in cumulus cells increased AMHRII expression and boosted AMH-induced signaling, effects reversed by vitamin D. Overall, these findings suggest that AGE enhance AMH activity in the ovary, contributing to ovulatory dysfunction and metabolic issues like IR. However, it remains unclear whether AMH directly affects insulin action locally or systemically, and this requires further investigation.

5. AMH and Infertility

Several studies suggest that AMH may affect subfertility related to PCOS, though the evidence is mixed. Weight loss programs for overweight and obese women with PCOS indicated that lower initial AMH levels predicted better menstrual and ovulation responses [39]. Similarly, ovulation induction studies showed that women with lower pretreatment AMH had better responses to clomiphene citrate. Thresholds like <1.2 ng/ml or <3.4 ng/ml predicted higher ovulation and pregnancy rates [40]. These results suggest that very high AMH levels, which indicate excessive granulosa cell activity, may hinder follicle formation and lower response to basic fertility treatments.

Evidence from IVF studies also highlights the complicated role of AMH in fertility with PCOS. Desforges-Bullet et al. (2010) found higher AMH levels in the follicular fluid of anovulatory PCOS women compared to those who ovulated, but lower levels in women who became pregnant. While AMH is a well-known predictor of ovarian response and oocyte yield [41], its link to outcomes like implantation and live birth remains uncertain. A meta-analysis by Iliodromiti et al. (2014) concluded that AMH was a weak predictor of live birth, and more data on PCOS populations continues to be inconsistent.

Further studies show this variability. Aleyasin et al. (2011) found AMH was linked to oocyte number and embryo transfer but not to pregnancy outcomes. Kaya et al. (2010) reported that day-3 AMH ≥3.2 ng/ml predicted clinical pregnancy with moderate accuracy, while Xi et al. (2012) saw lower implantation rates in women with high AMH despite similar fertilization outcomes. Sahmay et al. (2013) found no significant link between AMH and pregnancy rates in 150 PCOS women undergoing IVF. A meta-analysis by Tal et al. (2015) confirmed that AMH had weaker predictive value for pregnancy in PCOS compared to women with reduced ovarian reserve. This might be explained by the fact that higher AMH in PCOS shows increased production per follicle

rather than a greater number of follicles [42], connecting AMH more closely to disease severity than to reproductive success [43]. A clearer understanding of this relationship could help clarify the clinical importance of measuring AMH in PCOS subfertility.

6. AMH and folliculogenesis in PCOS

Previous studies suggest that AMH interferes with FSH activity in the ovaries, inhibiting folliculogenesis [44]. High AMH levels have been linked to ovulatory dysfunction. Laven et al. (2004) showed that anovulatory women, with or without PCOS, had higher AMH levels compared to ovulatory women, and AMH correlated with menstrual cycle length. Pellatt et al. (2010) proposed that PCOS can be split into ovulatory and anovulatory types based on AMH, with anovulatory PCOS showing 18-fold higher concentrations. Pigny et al. (2003) found that AMH positively correlated with small antral follicle count but negatively with FSH, suggesting that AMH contributes to follicular arrest. Dewailly et al. (2007) reported that excess 2–5 mm follicles were associated with more severe menstrual disorders, particularly amenorrhea. Increased AMH has also been observed in adolescents with oligomenorrhea [45], and Tal et al. (2014) showed its strong ability to predict amenorrhea. Overall, these findings suggest that excess AMH-producing small follicles create a microenvironment that limits FSH action and leads to anovulation, though direct evidence is still lacking.

7. Factors contributing to AMH overproduction

The reasons for high AMH in PCOS are still unclear, but several factors may play a role. Serum AMH has been linked to LH and androgen levels [46]. Pellatt et al. (2007a) showed that LH increased AMH production four-fold in granulosa cells from PCOS ovaries, but not in normal ovaries. Pierre et al. (2013) found that LH boosted AMH expression in granulosa cells of anovulatory PCOS women, suggesting LH contributes to AMH overproduction and follicular arrest. Androgens also promote early follicle growth [47], which might increase AMH production. However, Carlsen et al. (2009) found that long-term suppression of androgens did not lower AMH, indicating that other mechanisms likely maintain high AMH levels in PCOS.

7.1 Insulin and genetic influences

Insulin resistance may also raise AMH levels. La Marca et al. (2004a, 2004b) reported a link between HOMA-IR and AMH in women with PCOS, while Nardo et al. (2009) found AMH positively associated with fasting insulin in both PCOS and non-PCOS women. Park et al. (2010b) suggested that insulin might directly influence AMH secretion or indirectly increase AMH through androgen production. Genetic factors may also contribute. Kevenaar et al. (2009) identified an association between ACVR1 gene variants, AMH levels, and folliculogenesis, implicating ALK2 signaling in ovulatory disturbances. Stubbs et al. (2005) found fewer primordial follicles stained for AMH in anovulatory PCOS compared to ovulatory women but similar staining in pre-antral and antral follicles. This indicates reduced inhibition of primordial follicle recruitment in anovulatory PCOS, leading to the buildup of small follicles and AMH overproduction (Figure 2).

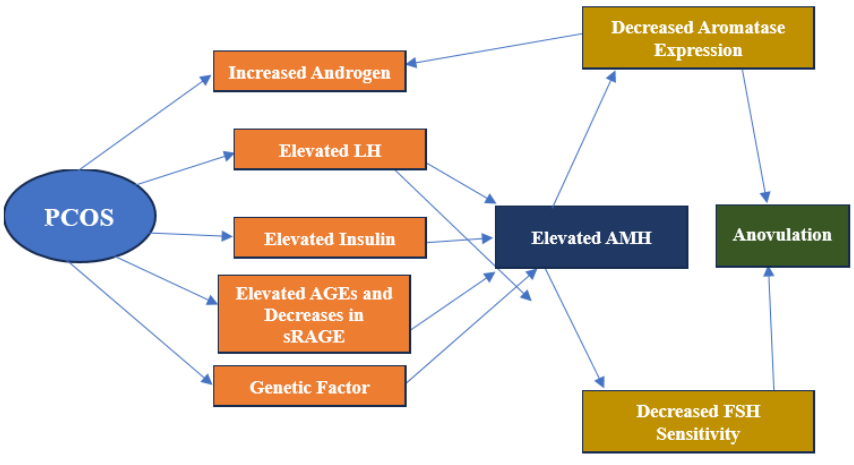

Figure 2: Shows the different factors that lead to high levels of anti-Müllerian hormone (AMH) and the effects of AMH overproduction. In polycystic ovarian syndrome (PCOS), AMH overproduction may arise from several factors that are unusually high, such as LH, androgens, insulin, and advanced-glycation end products (AGE), or unusually low, like the soluble receptor for AGE (sRAGE). Additionally, genetic factors, including variants of the ACVR1 gene, likely play a role in the overexpression of AMH. The rise in AMH reduces aromatase expression and dampens follicular sensitivity to FSH, which can lead to anovulation and hyperandrogenism. The exact link between AMH and insulin resistance is not yet completely understood.

Conclusion

AMH is significant in PCOS. It stops normal follicle growth, which results in anovulation. It may also play a role in high androgen levels and insulin resistance. High AMH levels are associated with a poor response to treatments like weight loss, ovulation induction, and ovarian drilling. In contrast, lower AMH levels often show improvement after therapy. More research is needed to fully understand how AMH affects PCOS and to investigate new treatment options.

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International Journal of Pharmacy and Pharmaceutical Research (IJPPR)
Volume 31, Issue 12, December 2025 **ijppr.humanjournals.com** ISSN: 2349-7203

How to cite this article:
Dr. R. Suresh et al. Ijppr.Human, 2025; Vol. 31 (12): 595-601.

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