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ROLE OF AMH, FSH, ANDROGEN AND ESTRADIOL AS MARKER OF PCOS FOLLICULOGENESIS: NARATIVE REVIEW

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Polycystic Ovary Syndrome (PCOS) poses a significant challenge in reproductive medicine due to its complex aetiology involving hormonal dysregulation, metabolic perturbations, and genetic predispositions. Central to PCOS pathogenesis is the disruption of folliculogenesis, the process vital for female reproductive health, characterized by the formation of numerous small antral follicles that fail to mature properly. This narrative review explores the roles of Anti-Müllerian Hormone (AMH), Follicle-Stimulating Hormone (FSH), androgens, and oestradiol as markers of PCOS folliculogenesis. AMH, prominently produced by granulosa cells, exhibits elevated levels in PCOS, contributing to excessive follicular recruitment and persistence. Dysregulation of FSH, androgen, and oestradiol further complicates follicular development, exacerbating PCOS-related abnormalities. Understanding the interplay between these markers is crucial for diagnosing and managing PCOS. However, challenges persist, including the lack of standardized serum AMH thresholds and technical limitations in testing methodologies. Further research is warranted to establish these thresholds and refine diagnostic approaches. Moreover, insights into hormonal mechanisms in PCOS folliculogenesis hold promise for developing targeted therapies to alleviate its impact on reproductive health.

Keywords: Folliculogenesis, Marker, Polycystic Ovary Syyndrom

INTRODUCTION

Polycystic Ovary Syndrome (PCOS) is a complex endocrine disorder characterized by aberrant folliculogenesis, contributing to reproductive dysfunction and infertility in affected individuals(Yadav and Malhotra, 2022). The pathogenesis of PCOS involves multifactorial interactions, including hormonal imbalances, metabolic

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disturbances, and genetic predispositions, culminating in disrupted follicular development(Armanini et al., 2022). Folliculogenesis, the process of follicle maturation within the ovaries, plays a pivotal role in female reproductive health (Bongrani et al., 2022; Naillat, 2022). In PCOS, folliculogenesis is dysregulated, marked by the formation of numerous small antral follicles that fail to mature properly. This dysregulated follicular development leads to the characteristic polycystic appearance of the ovaries observed in PCOS(Huang et al., 2021; Orisaka et al., 2021; Rad et al., 2022). Understanding the markers of folliculogenesis in PCOS is essential for both diagnosis and management of the syndrome(Bhide et al., 2019; Kałużna et al., 2020). Anti-Müllerian Hormone (AMH), Follicle-Stimulating Hormone (FSH), androgens, and oestradiol are key players in regulating follicular development and are increasingly recognized as important markers in PCOS folliculogenesis(Larsen et al., 2022; Lledó et al., 2022; Meena et al., 2021; Moolhuijsen and Visser, 2020; Saadia, 2020).

AMH, primarily produced by granulosa cells in preantral and small antral follicles, has emerged as a prominent marker in PCOS folliculogenesis(Kurniati et al., 2022; Moolhuijsen and Visser, 2020). Elevated levels of AMH in PCOS contribute to the excessive recruitment and persistence of small follicles, contributing to the characteristic phenotype of the syndrome(Kurniati et al., 2022; Moolhuijsen and Visser, 2020; Sahmay et al., 2018). Furthermore, alterations in FSH, androgen, and oestradiol levels also impact folliculogenesis in PCOS(Chauvin et al., 2022; Dewailly et al., 2019; Lledó et al., 2022; Tri Setiati et al., 2021). Dysregulation of these hormones disrupts the delicate balance required for follicular development, further exacerbating the folliculogenic abnormalities observed in PCOS(Chauvin et al., 2022; Tri Setiati et al., 2021).

In this context, elucidating the mechanisms underlying the role of AMH, FSH, androgens, and oestradiol in PCOS folliculogenesis is crucial for advancing our understanding of the syndrome and developing targeted therapeutic interventions aimed at restoring normal follicular development and improving reproductive outcomes in affected individuals.

METHODS

A comprehensive search was conducted of research articles published between 2020 and 2023, using PubMed, ScienceDirect, EMBAS, and Google Scholar. The keywords used included the topics "AMH", "FSH", "polycystic ovarian syndrome (PCOS)", "androgen", and "oestradiol", following the PICOT criteria. Duplicate literature was identified and excluded, while the remaining articles underwent a two-stage screening based on predefined inclusion criteria. Marker flow charting using Biorender application on biorender.combiorender("Scientific Image and Illustration Software | BioRender," n.d.)

Table 1. Inclusion and Exclusion Criteria

Criteria	Inclusion	Exclusion
Population	Rat model	In vitro
Intervention	Letrozole	letrozol combination with other treated
Comparators	With control group	Without control group
Outcomes	Research shows PCOS phenotype	Does not form pcos phenotypes
Time	Within the past five years	More than the past five years
Study design	Experimental research	Analytical observational research
Language	Indonesian, English	Besides Indonesian and English

RESULTS AND DISCUSSION

Anti-Müllerian hormone (AMH)

AMH (Anti-Müllerian Hormone) plays an important role in the pathophysiology of polycystic ovarian syndrome (PCOS). In PCOS, increased AMH levels, accompanied by AMH and its receptor dysregulation (AMHR2), lead to an increased number of follicles and follicle cessation(Bhattacharya et al., 2022; Rudnicka et al., 2021; Teede et al., 2019). Excessive AMH production is also associated with the risk of a hyperandrogenic intrauterine environment in the offspring of pregnant women with PCOS (Dewailly et al., 2020; Meena et al., 2021; Monieum et al., 2019). The interaction of AMH with other hormones such as FSH, LH, androgen, and oestradiol affects the development of follicles and the function of other related organs such as the hypothalamus and the pituitary gland (Dewailly et al., 2016; Fu et al., 2021; Laven, 2019; Meng et al.,

2023; Oduwole et al., 2021; Saadia, 2020; Sova et al., 2019). In the pre-antral phase, AMH is induced by androgen and FSH but is inhibited by oestradiol after reaching a certain threshold (Dewailly et al., 2019; Tola et al., 2018; Tri Setiati et al., 2021). In the antral phase, AMH continues to inhibit the activity of the aromatase induction by FSH, but its expression gradually decreases, allowing for full expression of aromatase and oestradiol synthesis (Bhattacharya et al., 2022; Oduwole et al., 2021; Tri Setiati et al., 2021).

The measurement of serum AMH levels has several advantages in the diagnosis and treatment of PCOS (Meena et al., 2021; Rudnicka et al., 2021). AMH in serum reflects the secretion of AMH from all developing follicles, including those that are not visible on the ultrasound, and correlates with the severity of the PCOS symptoms (Dewailly et al., 2020; Sova et al., 2019; Tri Setiati et al., 2021). This measurement can also predict responses to various PCOS treatments (Bosch et al., 2023). An understanding of the interactions between AMH, FSH, androgens, and oestradiol provides insight into the regulation of folliculogenesis and PCOS pathophysiology (Bhattacharya et al., 2022; Dewailly et al., 2020). AMH expression in PCOS, which is affected by granulose cell dysregulation, interferes with normal ovarian function by affecting follicle recruitment (Bhattacharya et al., 2022), sensitivity to FSH (Tri Setiati et al., 2021), and production of oestradio (Tola et al., 2018; Tri Setiati et al., 2021)l. This dysregulation contributes to the characteristic characteristics of PCOS, such as an increased number of follicles and stopping the production of follicles (Fu et al., 2021; Monieum et al., 2019). Although the use of AMH serum is useful in the management of infertility in PCOS, the lack of standard thresholds for serum AMH levels and technical problems with current tests restrict its widespread use (Monieum et al., 2019; Teede et al., 2019). Therefore, each centre may need to set its own limit for serum AMH levels until consensus is reached.

Follicle-Stimulating Hormone (FSH)

Follicle-stimulating hormone (FSH) is a key player in the complex process of folliculogenesis, which is the development of ovary follicles in women. FSH functions by stimulating the growth and maturation of the follicle inside the ovaries, primarily acting on the granulose cells to promote their proliferation and differentiation (Lledó et

al., 2022; Saadia, 2020). This hormone plays an important role in recruiting primordial follicles into the growing follicle pool, eventually contributing to the maturation and production of oestrogen by granulosa cells (Dewailly et al., 2019; Tri Setiati et al., 2021). Furthermore, FSH is very important in the selection of the dominant follicle that will undergo ovulation during the menstrual cycle, stressing its importance in female reproductive health (Lledó et al., 2022; Saadia, 2020).

In the context of polycystic ovarian syndrome (PCOS), hormonal imbalances interfere with the normal functioning of FSH and luteinizing hormone (LH) (Saadia, 2020), causing irregularities in the menstrual cycle and ovulation dysfunction(Elsayed et al., 2023). Relative increases in LH levels compared to FSH can inhibit ovulation, resulting in anovulation, a common characteristic of PCOS (Bosch et al., 2023; Tri Setiati et al., 2021). In addition, some individuals with PCOS may show resistance to the FSH, in which the ovaries do not respond effectively to the produced FSH (Dewailly et al., 2019; Saadia, 2020). This hormone imbalance contributes to irregular follicle growth, cyst formation, and disorders in the cycle of menstruation experienced by women with PCOS (Armanini et al., 2022; Chen and Pang, 2021). Furthermore, genetic variation in the genes of the receptor FSH (FSHR) has been associated with PCOS, suggesting that genetic factors can affect the ovarian response to FSH (Crespo et al., 2018; Gollapalli et al., 2022).

LH: FSH Ratio

In Polycystic Ovary Syndrome (PCOS), changes in the ratio of Follicle Stimulating Hormone (FSH) and Luteinizing Hormone (LH) play a key role in the pathophysiology of this condition (Lledó et al., 2022; Saadia, 2020). Increased LH:FSH ratios can interfere with normal folliculogenesis by inhibiting ovulation and causing anovulation in women with PCOS (Saadia, 2020). Furthermore, this hormonal imbalance can also lead to hyperandrogenism because increasing LH stimulates androgen production by the ovaries (Chappell et al., 2022; Rosenfield and Ehrmann, 2016), which is one of the characteristic features of PCOS, such as hirsutism and acne (Berga, 2021; Elsayed et al., 2023). An irregular follicle growth disorder can also occur because of an

unbalanced LH:FSH ratio, leading to the formation of ovarian cysts that are characteristic of PCOS(Larsen et al., 2022; Saadia, 2020).

Increased LH-FSH ratios in PCOS have a significant impact on ovulation, androgen production, and ovarian follicular growth(Chappell et al., 2022; Saadia, 2020). A thorough understanding of the role of the LH:FSH ratio in PCOS pathophysiology is essential for the development of appropriate management strategies for this condition (Lledó et al., 2022; Saadia, 2020), including targeted hormonal adjustments and therapies to address the hormonal imbalances underlying the symptoms of PCO S(Moolhuijsen and Visser, 2020; Walters et al., 2018).

Androgen

Androgen is a steroid hormone that is specifically produced by the ovaries as well as the adrenal glands in women (Abdelazim et al., 2020; Berga, 2021). This hormone plays an important role in the development of secondary sexual characteristics in women and is also a precursor of the hormone oestrogen in women, which is necessary for healthy reproductive function (Dewailly et al., 2019; Walters, 2020; Xu et al., 2021). In the ovaries, androgen plays a role in the production of oestradiol by the antral follicles, which is important for normal menstrual cycles (Chappell et al., 2022; Dewailly et al., 2019). Androgens play an important role in ovary folliculogenesis, especially in oestradiol production and the development of the antral follicle (Murat Altinkilic et al., 2023; Walters, 2020).

Androgens, such as testosterone, serve as estrogen precursors in the ovary (Abdelazim et al., 2020; Berga, 2021). In PCOS, there is an increase in luteinizing hormone (LH) secretion, leading to increased androgen production in the theca cells of the ovary(Orisaka et al., 2021; Shaaban et al., 2019; Weiss et al., 2019). Follicle-stimulating hormone (FSH) secretion is within the normal range in PCOS, but it does not upregulate aromatase activity in granulosa cells, resulting in overproduction of androgens (Larsen et al., 2022; Weiss et al., 2019). Insulin resistance and hyperinsulinemia are associated with increased androgen production in PCOS through insulin-like growth factors (IGFs)(Chen and Katznelson, 2022; Geng et al., 2019). Androgen receptors (ARs) play a role in folliculogenesis in the ovaries. Female mice lacking ARs exhibit profound

folliculogenesis defects, premature ovarian failure (POF)-like defects, and infertility (Aflatounian et al., 2020; Cara et al., 2023). The dysregulation of major regulators of folliculogenesis, including the gene encoding the kit ligand, is observed in mice (Berga, 2021; Walters, 2020). The ovarian follicles progress through primordial, primary, and secondary stages before forming antral cavities. Graafian follicles, which are the major source of cyclically secreted ovarian estrogen, are formed after puberty. Preovulatory LH surges trigger ovulation of Graafian follicles for fertilization (Rodriguez Paris and Bertoldo, 2019; Walters, 2020).

In polycystic ovarian syndrome (PCOS), excess androgen can cause preantral and antral follicle dysfunction. Although there is a common view that androgen tends to impair normal ovary function, evidence suggests that androgen has a positive and important role in normal follicular growt(Dewailly et al., 2016; Walters et al., 2019)h. However, excess androgens can also inhibit proliferation and promote apoptosis in mature antral follicles, which can contribute to follicle loss. Exposure to excess androgen during foetal life or postnatal life can play a role in the development of PCOS, highlighting the possibility that PCOS is a developmental disorder in which "programming" by an excess of androgen plays a key role. Therefore, an in-depth understanding of the mechanisms of androgenic action in PCOS folliculogenesis can provide valuable insights into managing this condition(Dewailly et al., 2019; Rodriguez Paris and Bertoldo, 2019; Walters, 2020).

Oestradiol

Oestradiol (E2) plays an important role in regulating folliculogenesis, the process of ovarian follicle development. E2 is produced locally by the granulose cells in the ovarian follicle and is involved in several key events during the menstrual cycle in wome n(Tri Setiati et al., 2021; Xu et al., 2021). An increased concentration of E2 in the larger follicular fluid participates in the predominant preovulatory follicle selection and triggers an increase in the luteinizing hormone (LH) necessary for ovulation (Chauvin et al., 2022; Dewailly et al., 2019). As a result, E2 is considered an important marker of follicle quality(Walters, 2020; Xu et al., 2021). E2 works mainly through the activation of the ER□ and ER□ estrogen receptors, which are transcription factors activated by the ligands. These receptors regulate gene transcription through direct or indirect

mechanisms (Xu et al., 2021). The E2 signal path involves nuclear and extra-nuclear pathways, and receptors act mainly in the nucleus to regulate gene transcription (Walters, 2020).

The role of E2 in folliculogenesis includes promoting follicular growth, maturation, and predominant preovulatory follicle selection(Xu et al., 2021; Zhou et al., 2021). E2 also affects the fate of cells, especially in granulosa, and plays a role in preventing apoptosis of granulosa cells (Walters, 2020). Besides, E2 has genomic and non-genomic activity, affecting gene expression and triggering signal paths that are important for successful folliculogenesis. E2 is an important hormone that regulates important events in the menstrual cycle, folliculogenesis, and ovarian follicle maturation and selection (Xu et al., 2021; Zhou et al., 2021).

In the context of PCOS, changes in ER isoform expression and/or DNA accessibility may contribute to pathology(Zhou et al., 2021). There is also deregulation of expression of other steroid sex hormone receptors in PCOS, including unmodified or up-regulated androgen receptor expression (AR) and expression of progesterone a receptor in the mural granulosa cells of the PCOS follicle (Walters, 2020; Xu et al., 2021)s. Furthermore, PCOS is characterised by low levels of oestradiol and progesterone, which may be caused by abnormal expression and/or activity of various steroidogenic enzymes present in granulose cells(Xu et al., 2021; Zhou et al., 2021).

PCOS Folliculogenesis

In PCOS folliculogenesis, the complex interactions between the Anti-Müllerian Hormone (AMH), Follicle-Stimulating Hormone (FSH), Luteinizing Hormone (LH), androgen, and oestradiol play an important role in regulating ovarian follicle growth and contribute to the characteristics of the syndrome (Moolhuijsen and Visser, 2020; Saadia, 2020; Tri Setiati et al., 2021; Walters et al., 2019). To visualise the mechanism of the relationship between AMH, FSH, LH, androgen, and oestradiol in PCOS folliculogenesis, a mechanism circuit diagram can be used. This diagram will show how hormonal imbalances and disturbances in the regulation of folliculogenesis contribute to the characteristics of PCOS, as well as its impact on related organs in the reproductive

system. It will give a better understanding of the complexity of the syndrome and the potential for the development of more effective therapies.

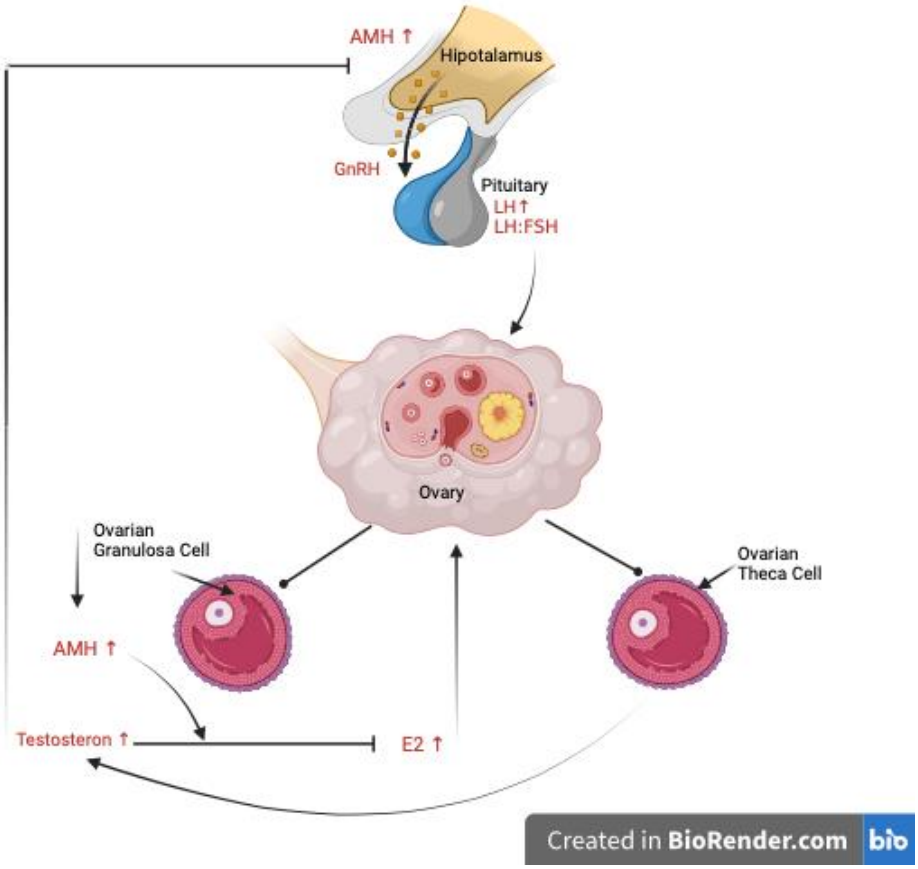

Figure 1. Mechanisms of AMH FSH LH androgen and estradiol in PCOS folliculogenesis

AMH, which is produced by small pre-antral and antral follicle granulosa cells, is increased in PCOS. Increased AMH leads to excessive follicular recruitment and persistence of small follicles, which contributes to polycystic ovaries (Bhattacharya et al., 2022; Kurniati et al., 2022; Moolhuijsen and Visser, 2020; Sahmay et al., 2018). FSH, which stimulates the growth of ovarian follicles, is also involved in the pathogenesis of PCOS. A decrease in FSH can lead to disturbances in the selection of the dominant follicle and proper follicular maturation, which is characteristic of PCOP (Laven, 2019; Oduwole et al., 2021; Orisaka et al., 2021). High LH in PCOS is also a characteristic, as it stimulates

ovarian hyperandrogenism, which affects the quality and quantity of the mature folicle(Orisaka et al., 2021; Saadia, 2020).

Hormonal imbalances affect androgen and estradiol production in PCOS folliculogenesis. Increased levels of androgen, such as testosterone, can lead to ovarian hyperandrogenism, which affects follicle growth and ovulation (Chappell et al., 2022; Walters et al., 2019). Estradiol, the main oestrogen hormone, is also involved in PCOS. Increased estradiol can exacerbate the hormonal disorders that occur in this syndrome, leading to greater imbalances in the regulation of folliculogenesis (Chauvin et al., 2022; Tri Setiati et al., 2021). Hormonal imbalances and folliculogenesis disorders in PCOS can also affect related organs, such as the uterus and hypothalamus-pituitary gland(Orisaka et al., 2021). Ovarian hormonal disorders in PCOS can affect menstrual cycles and the ability to conceive. The hypothalamus and the pituitary gland play an important role in regulating the production of FSH and LH, which contribute to the development of ovarian follicles (Dewailly et al., 2019; Orisaka et al., 2021). Therefore, hormonal imbalances in PCOS can interfere with complex interactions between related organs in the reproductive system (Dewailly et al., 2019; Merhi et al., 2019; Orisaka et al., 2021).

CONCLUSIONS

AMH (Anti-Müllerian Hormone) plays an important role in the pathophysiology of polycystic ovarian syndrome (PCOS) by increasing the number of follicles and contributing to the disturbance of folliculogenesis. FSH (Follicle-Stimulating Hormone) affects dominant follicle selection, while an unbalanced ratio of LH:FSH can lead to anovulation and hyperandrogenism. Androgens and estradiol also affect follicle growth and menstrual cycle regulation. A deeper understanding of the interactions between AMH, FSH, LH, androgen, and estradiol can help develop more effective PCOS management strategies. Further research is needed to establish standard thresholds for serum AMH levels and improve the technical tests. In addition, a better understanding of the mechanisms of hormonal action in PCOS folliculogenesis can help develop more targeted therapies.

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