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Serum Interleukin-33 Levels and Their Association with Insulin Resistance and Hormonal Alterations in Women with Polycystic Ovary Syndrome: A Case–Control Study

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Abstract

Background: Polycystic Ovary Syndrome (PCOS) is the most common endocrine disorder associated with metabolic imbalances and chronic inflammation. Interleukin-33 (IL-33) has been suggested as a biomarker for inflammation in a number of diseases. **Aim of this study**: To assess serum IL-33 levels in women with PCOS compared to healthy controls and to analyze its predictive capabilities as diagnostic marker for PCOS. **Materials and Methods**: A total of 180 women participated aged (18-40) years in the study included 90 newly diagnosed with PCOS and 90 healthy control groups in a case-control study. Serum IL-33 level was measured using an enzyme-linked immunosorbent assay. Clinical parameters such as BMI, fasting insulin (FIN) and homeostasis model assessment of insulin resistance (HOMA-IR) were measured. Besides IL-33 levels, clinical parameters such as LH, FSH, total testosterone and estradiol were also measured, and significant differences and correlations were obtained, which were all suggestive of metabolic and reproductive dysfunction associated with PCOS. The Receiver Operating Characteristic (ROC) curve analysis was used to determine the diagnostic value of IL-33 levels, and Random Forest and Support Vector Machine (SVM) classifiers were used to see how well the levels of IL-33 could predict the disease. **Results**: The level of serum IL-33 was found to be significantly elevated in women with PCOS (75.4 ± 12.8 pg/mL) when compared with healthy controls (34.6 ± 8.5 pg/mL, $p < 0.001$). Positive correlations were observed between IL-33 level and BMI ($r = 0.681, p<0.001$), fasting insulin ($r = 0.575$, $p<0.001$),

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HOMA-IR (r = 0.752, p<0.001), LH (r = 0.493,p<0.001), FSH (r = 0.431,p = 0.005), LH/FSH Ratio (r = 0.484,p<0.001), Total Testosterone (r = 0.309,p<0.001), Free Testosterone (r = 0.526, p<0.001), Prolactin (r = 0.250,p = 0.062), Progesterone (r = 0.423,p = 0.007), Estradiol (E2) (r = 0.397,p<0.001) and Sex hormone binding globulin (SHBG) (r =-0.506,p<0.001) in women with PCOS group. ROC analysis was performed and resulted in an outstanding AUC value of 0.962 (95% CI: 0.934–0.990), which demonstrates excellent discrimination between PCOS and healthy subjects, with an optimal cut-off value of > 50.91 pg/mL, a sensitivity of 91.1% and a specificity of 90.0%. **Conclusions**: Overall, this study indicates a significant association between increased serum IL-33 levels and the inflammatory and metabolic changes seen in women with PCOS. Overall, these results highlight the promising potential of IL-33 as a valuable biomarker in the context of metainflammation in PCOS, given the high AUC values and the effectiveness of the machine learning models. These findings may have important implications for improving the diagnosis and management of PCOS and its associated complications.

Keywords: Interleukin -33, Insulin Resistance, Metainflammation, Polycystic Ovary Syndrome.

Introduction

Polycystic Ovary Syndrome (PCOS) is a complex endocrine disorder with a spectrum of symptoms, such as hyperandrogenism, polycystic ovaries and ovulatory dysfunction [1]. PCOS is a common cause of infertility in women of reproductive age (6-10%) and has been linked with other metabolic and psychological conditions, including obesity, insulin resistance and anxiety [2]. Diagnosis and management is challenging in PCOS, as there are many different aspects to this condition, therefore there is a need for a better understanding of the underlying pathophysiological mechanisms [3].

Increasingly, the importance of inflammation in the pathogenesis of PCOS has been studied [4]. Chronic low-grade inflammation due to metabolic disturbances is called metainflammation and is thought to play a role in the development and progression of

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PCOS [5]. This inflammatory state can lead to insulin resistance and hyperandrogenism, which can feed into each other and worsen insulin resistance [6]. The search for robust inflammatory markers in PCOS would be useful to understand better the mechanism and management of this disorder [7]. Interleukin-33 (IL-33) is a cytokine of the IL-1 family and a product of several cell types including endothelial cells, epithelial cells and immune cells [8]. It is involved in the regulation of Th2-mediated inflammation and plays a critical role in the immune response [9]. Recent studies have established the role of IL-33 in a wide range of metabolic and inflammatory disorders, and therefore made it a potential biomarker in the assessment of metainflammation [10]. Increased level of IL-33 is associated with obesity, diabetes, and cardiovascular diseases, reflecting its involvement in metabolic dysregulation [11].

For PCOS, the role of IL-33 in the inflammatory process related to the syndrome has only been somewhat investigated [12]. Initial research indicates women who have PCOS have different amounts of inflammatory markers (such as IL-33) than women who do not have PCOS(13). So far, however, IL-33 has been poorly studied with respect to the metabolic and reproductive characteristics of PCOS [14]. Thus, more research is necessary to establish if serum levels of IL-33 can be a valid marker for metainflammation in women with PCOS(15). This study aims to investigate the relationship between circulating IL-33 concentrations and metabolic and reproductive hormonal alterations in women with PCOS.

2: Materials and Methods

2.1 Study Design

This study employed a case-control design to investigate the serum levels of Interleukin-33 (IL-33) in 90 women newly diagnosed with PCOS by a specialist gynecologist according to the Rotterdam criteria [16] compared to 90 healthy fertile women as control group. The study was carried out during the period (March 2025 to May 2025) at Fertility center in AL Sader medical city in AL Najaf, Iraq. The study protocol was approved by the Institutional Review Board (IRB) (4122in 13/2/2025) and informed consent was obtained from all participants before including them in the study.

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- **Inclusion Criteria**:
  - Women between the ages of 18 and 40 years.
  - Newly diagnosed with PCOS (Rotterdam) – at least two of the following: Oligomenorrhea or amenorrhea, clinical and/or biochemical hyperandrogenism, polycystic ovary ultrasound.
  - Healthy fertile, controls who had regular menstruation and no history of endocrine disorders.
- **Exclusion Criteria**:
  - Other chronic disease and disorders including thyroid disorders or adrenal disorders.
  - Use of any type of anti-inflammatory or hormonal treatment in the past three months.
  - History of chronic inflammatory diseases.

2.2 Sample Collection

All participants were fasted overnight and blood samples were drawn between 8:00 AM and 10:00 AM. Venous blood was collected in sterile serum separator tubes and the blood was allowed to clot at room temperature for 30 minutes. The samples were then centrifuged at 3000 rpm for 10 minutes to isolate the serum which was kept at -80°C for further analysis.

2.3 Measurement of Interleukin-33

The level of IL-33 in the serum was measured by an available enzyme-linked immunosorbent assay (ELISA) kit (Elabscience Cat.No: E-EL-H2402 USA). An assay was carried out following the manufacturer's guidelines. The minimum detectable concentration of IL-33 was (15.63-1000 pg/mL).

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2.4 Assessment of Clinical Parameters

All participants were assessed on clinical parameters, which included:

Body Mass Index (BMI):

- **Body Mass Index (BMI)**: Calculated using the formula [17]:

  $BMI = \text{weight (kg)} / \text{height (m)}^2$.

- **Fasting Insulin Level**: Is measured using ELISA kit (Elabscience Cat.No: E-EL-H2665 USA), reference range of 0.78 - 50 μIU/mL.
- **Fasting serum Glucose Level**: Is a measurement obtained by the enzymatic colorimetric method (Biomeruix-franle kit) and has a normal range of 70-99 mg/dL.
- **HOMA-IR**: Estimated by Homeostasis Model Assessment of Insulin Resistance formula (using the following equation [18]:

  HOMA-IR=Fasting Insulin (μU/mL) ×Fasting Glucose (mg/dL)/405

Clinical parameters measured were LH, FSH, total testosterone, free testosterone, prolactin, progesterone, and estradiol, among others, including BMI, fasting insulin, and HOMA-IR.

2.5 Statistical Analysis

SPSS, version 2016 was used to conduct statistical analyses. Demographic and clinical characteristics were expressed as descriptive statistics and continuous variables were expressed in terms of mean ± SD. Independent t test was used to compare the level of the IL-33 and clinical parameters in the PCOS group and healthy group. Pearson correlation analysis was performed to evaluate the correlation between IL-33 levels and clinical parameters. The p-value was considered significant if < 0.05. To account for potential confounding variables and assess independent associations, multivariable linear regression analyses were performed to evaluate the relationship between IL-33 levels and PCOS status

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after adjusting for age and BMI. Furthermore, additional models incorporating HOMA-IR and stratified analyses based on BMI categories (BMI < 25 vs. ≥ 25 kg/m^2) were conducted to dissect the interplay between obesity, insulin resistance, and IL-33.

2.6 Ethical Considerations

The study has been conducted in accordance with the Declaration of Helsinki. Both informed consent and maintaining the confidentiality of the data of all participants were preserved throughout the research process.

3: Results

3.1 Participant Characteristics

A total of 180 women were included in the study; 90 women were diagnosed with Polycystic (PCOS) and 90 healthy women. Demographic and clinical data for the subjects are presented in table 1. The PCOS group had significantly higher level of Body Mass Index (BMI), fasting insulin (FI), fasting glucose (FG), Serum level of IL-33, HOMA-IR, LH, FSH, LH/FSH Ratio, Total Testosterone, Free Testosterone, Prolactin, Progesterone and lower serum level of Sex hormone binding globulin (SHBG) than the healthy group. The present study investigated serum IL-33 concentrations and their associations with clinical and biochemical parameters in women with PCOS.

Table 1: Demographic and Clinical Characteristics of the Study Participants

Parameters	PCOS Group (N=90)	Healthy Group (N=90)	p-value
Age (years)	28.3 ± 5.4	27.9 ± 4.6	0.542
BMI (kg/m²)	29.7 ± 6.3	23.5 ± 3.2	<0.001
WHR	0.99±0.06	0.69±0.03	<0.001
LH (mIU/L)	9.5 ± 2.0	5.4 ± 1.0	<0.001
FSH (mIU/L)	6.3 ± 1.5	5.0 ± 0.8	0.012

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LH/FSH Ratio	1.51 ± 0.4	1.08 ± 0.2	<0.001
Total Testosterone (pg/mL)	56.4 ± 15.5	34.2 ± 10.0	<0.001
Free Testosterone (pg/mL)	1.5 ± 0.4	0.9 ± 0.2	<0.001
Prolactin (pg/mL)	14.500 ± 3,200	10.100 ± 2,500	<0.001
Progesterone (pg/mL)	5.000 ± 1,500	3.000 ± 800	<0.001
Estradiol (E2) (pg/mL)	180 ± 50	150 ± 40	0.045
SHBG (nmol/L)	40.6 ± 10.2	60.7 ± 9.5	<0.001
Free androgen index (FAI)	9.94	4.08±1.11	<0.001
Fasting glucose (mg/dL)	100.13±8.43	80.46±2.13	<0.001
Fasting Insulin (µU/mL)	13.64 ± 4.97	8.41 ± 2.13	<0.001
HOMA-IR	4.2 1± 1.53	1.50 ± 0.36	<0.001
Serum IL-33 (pg/mL)	75.4 ± 12.8	34.6 ± 8.5	<0.001

Note: Data are presented as Mean ± SD. WHR: Waist-to-Hip Ratio; LH: Luteinizing Hormone; FSH: Follicle-Stimulating Hormone; SHBG: Sex Hormone-Binding Globulin; HOMA-IR: Homeostasis Model Assessment of Insulin Resistance.

The results showed that the serum IL-33 level in the PCOS group (75.4 ± 12.8 pg/mL) was significantly higher than that of the healthy group (34.6 ± 8.5 pg/mL) ($p < 0.001$). Following multivariable adjustment for age and BMI, PCOS remained significantly associated with elevated serum IL-33 levels ($p < 0.001$). Stratified analysis based on BMI categories demonstrated that serum IL-33 levels remained significantly higher in women with PCOS compared to healthy controls across both non-obese (58.4 ± 10.2 vs. 32.1 ± 7.4 pg/mL, $p < 0.001$) and obese subgroups (84.2 ± 11.5 vs. 41.0 ± 8.9 pg/mL, $p < 0.001$). However, the inclusion of HOMA-IR in the regression model attenuated the effect size, suggesting a strong biological and mediating crosstalk between insulin resistance, metainflammation, and IL-33 expression. Correlation analysis identified associations

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between serum IL-33 concentrations and several metabolic and hormonal parameters in women with PCOS.

In particular, HOMA-IR had the highest positive correlation ($r = 0.752$, $p<0.001$) with the variable of interest, indicating a strong association with insulin resistance. Furthermore, both fasting insulin ($r = 0.575$, $p < 0.001$) and BMI ($r = 0.681$, $p < 0.001$) were strongly associated with the variable, implying that the variable could be associated with a metabolic dysregulation in the studied population.

The other hormonal parameters, LH ($r = 0.493$), LH/FSH ratio ($r = 0.484$) and free testosterone ($r = 0.526$) showed significant positive correlations ($p < 0.001$), also supporting the hypothesis of hormonal interaction in the condition studied. Interestingly, the only parameter that showed a negative correlation was the Sex Hormone-Binding Globulin (SHBG) ($r = -0.506$, $p<0.001$), suggesting that the higher the level of the IL-33, the lower the level of SHBG and hence the high level of free androgens, which is a characteristic of hyperandrogenous state.

Elevated but less significant correlations were seen with other metabolic and endocrine measures, including estradiol ($r = 0.397$, $p < 0.001$), FSH ($r = 0.431$, $p = 0.005$), and fasting glucose ($r = 0.340$, $p = 0.009$). As shown Table 2, these associations were multiple, highlighting the relevance of the IL-33 in the pathophysiology of the disorder, and suggesting that the metabolic and hormonal pathways had associations.

Table 2: Correlations Between Serum IL-33 Concentrations and Metabolic and Hormonal Parameters in Women With PCOS.

Parameters	Sample Size (n)	Correlation Coefficient (r)	95% Confidence Interval (95% CI)	p-value
Age	90	0.342	[0.144, 0.513]	<0.01
BMI (kg/m²)	90	0.681	[0.551, 0.779]	<0.001
LH (mIU/L)	90	0.493	[0.318, 0.636]	<0.001
FSH (mIU/L)	90	0.431	[0.247, 0.585]	0.001

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LH/FSH	90	0.484	[0.308, 0.628]	<0.001
Total Testosterone (pg/mL)	90	0.309	[0.108, 0.485]	<0.003
Free Testosterone (pg/mL)	90	0.526	[0.357, 0.662]	<0.001
Prolactin (pg/mL)	90	0.25	[0.044, 0.436]	0.062
Progesterone (pg/mL)	90	0.423	[0.238, 0.579]	0.007
Estradiol (E2) (pg/mL)	90	0.397	[0.208, 0.558]	<0.001
SHBG	90	-0.506	[-0.646, -0.333]	<0.001
Free androgen index(FAI)	90	0.471	[0.293, 0.617]	0.001
Fasting glucose (mg/dL)	90	0.340	[0.142, 0.511]	0.009
Fasting Insulin (µU/mL)	90	0.575	[0.418, 0.700]	<0.001
HOMA-IR	90	0.752	[0.645, 0.830]	<0.001

3.2 ROC Curve Analysis

ROC curve analysis was performed to determine the diagnostic value of serum IL-33 levels. The analysis revealed an outstanding Area Under the Curve (AUC) of 0.962 (95% CI: 0.934–0.990, $p < 0.001$). The optimal cut-off value of > 50.91 pg/mL was established using Youden's index, yielding a sensitivity of 91.1%, a specificity of 90.0%, and a diagnostic accuracy of 90.6% in distinguishing women with PCOS from healthy controls as presented in Figure (1).

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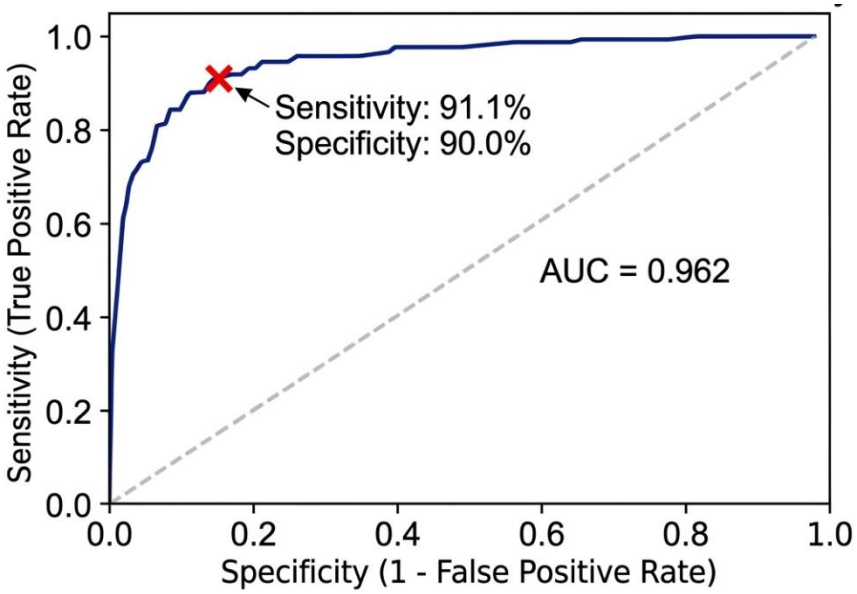


Figure 1. ROC Curve For IL-33 Levels in PCOS And Healthy Groups

4: Discussion

Increased levels of IL-33 in women with PCOS are consistent with previous studies which have associated the pathophysiology of PCOS with inflammation(19). IL-33 belongs to the interleukin-1 family and has been well identified for its function in promoting inflammatory responses in a Th2 mediated way. It is released from damaged tissues and serves as an alarmin which indicates the presence of injury or stress to the immune system [20]. The low-grade, long-lasting inflammatory state that is commonly associated with PCOS could be caused by metabolic dysfunction and abnormalities in fat tissue, which results in increased amounts of pro-inflammatory cytokines, such as IL-33 [29].

It is especially significant that the association of IL-33 with metabolic parameters has been identified. There is a correlation between BMI and IL-33 levels in women with PCOS, which indicates that greater adiposity in women with PCOS may lead to increased inflammation. Results were similar to those previously reported, with elevated inflammatory markers in women with PCOS, suggesting a significant role for systemic inflammation in the metabolic changes frequently seen in women with PCOS [22].

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The positive associations of IL-33 with glycemic indices, HOMA-IR, and selected reproductive hormones suggest a potential relationship between IL-33 and metabolic–endocrine alterations in PCOS [23]. Insulin resistance is a characteristic of PCOS and linked to the likelihood of developing type 2 diabetes and cardiovascular disease. Hyperinsulinism can worsen hyperandrogenism and lead to the general metabolic imbalance in PCOS [24].

The findings suggest that IL-33 may be a good marker for the severity of insulin resistance in women with PCOS, with higher levels of IL-33 corresponding to greater insulin resistance. This connection highlights the importance of continuing research to uncover the mechanistic pathways that mediate the effects of IL-33 on insulin sensitivity and metabolic health (Table 2).

A critical consideration in interpreting inflammatory biomarkers in PCOS is the frequent coexistence of obesity and insulin resistance, which can act as major metabolic confounders or biological drivers. Our multivariable regression and stratified analyses provide deeper clarity on this interaction. While unadjusted comparisons showed marked elevations in IL-33, adjusting for BMI and performing stratified weight-matched comparisons confirmed that PCOS is independently associated with increased IL-33. Concurrently, the strong correlation and regression attenuation observed with HOMA-IR reinforce the hypothesis that insulin resistance is not merely an external confounder, but an integral component of the 'metainflammation' loop in PCOS. Elevated insulin levels and adiposity synergistically stimulate inflammatory pathways (such as NF-*k*B or JNK signaling), upregulating alarmin cytokines like IL-33, which in turn exacerbate ovarian dysfunction and metabolic imbalance.

Our results corroborate with the literature which emphasize inflammatory mediators in PCOS. The cytokines IL-6, TNF-α and CRP have been found to be increased in women with PCOS and to be associated with insulin resistance and obesity [25]. The present study brings a new inflammatory marker, IL-33 to the list of inflammatory markers associated with the syndrome.

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Furthermore, the role of IL-33 in metainflammation further underscores the importance of developing targeted therapeutic approaches that would aim at modulation of inflammatory pathways in women with PCOS. These interventions may have a beneficial effect on metabolic results and minimize the chance for long-term complications of the syndrome. The higher levels of IL-33 in women with PCOS than in healthy controls could be a result of the metabolic inflammation and hormonal derangements observed in PCOS patients, since the majority of correlations with clinical parameters were positive (except for SHBG which was inversely correlated, which could be due to low levels of androgen binding protein and high levels of free androgens typically seen in women with PCOS).

4.1 Limitations and Future Directions

Although we performed multivariable adjustments and stratified analyses to mitigate the confounding effects of obesity and insulin resistance, the cross-sectional nature of the study prevents definitive causal inference. Future large-scale longitudinal and interventional studies are warranted to precisely map the mediation pathways between insulin resistance, adiposity-driven metainflammation, and IL-33 dynamics in PCOS. Also, the number of samples was sufficient but could be extended with further studies to improve the generalisability of the results. Finally, further research is needed to elucidate the mechanisms by which IL-33 affects metabolic dysregulation. Knowing the biological pathways involved could help give insight into the possible therapeutic targets for controlling PCOS and its complications.

5: Conclusion

The serum levels of Interleukin-33 (IL-33) were found to be significantly elevated in women with Polycystic Ovary Syndrome (PCOS) compared to healthy controls (75.4 ± 12.8 pg/mL vs. 34.6 ± 8.5 pg/mL; $p < 0.001$). This marked significant increase highlights the possible role of IL-33 as a pro-inflammatory mediator in the immune-metabolic dysregulation seen in PCOS.

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Results from correlation analysis showed high positive correlation between IL-33 and several important metabolic and hormonal parameters. IL-33 was positively correlated highly with BMI (r = 0.681), fasting insulin (r = 0.575) and HOMA-IR (r = 0.752), suggesting its role in obesity and insulin resistance, which are hallmarks of the metabolic phenotype of PCOS. Additionally, IL-33 was positively associated with reproductive hormones such as LH (r = 0.493), FSH (r = 0.431), LH/FSH ratio (r = 0.484), total testosterone (r = 0.309), free testosterone (r = 0.526), estradiol (r = 0.397), progesterone (r = 0.423), and prolactin (r = 0.25). These interactions point to a more widespread part of the endocrine imbalance, in which cases IL-33 may also affect ovulatory dysfunction and hyperandrogenism, prevalent in PCOS.

Importantly, only SHBG (Sex Hormone-Binding Globulin) showed a negative correlation with IL-33 ($r = –0.506$, $p < 0.001$). This inverse relationship is biologically important because low levels of SHBG result in higher levels of free androgens in the blood and contribute to worsening clinical symptoms of PCOS, including hirsutism, acne and menstrual irregularities. The negative correlation indicates that IL-33 can be involved in the inhibition of hepatic SHBG production, either via inflammation signaling pathways (e.g., JAK/STAT or NF-κB).

Overall, these results suggest that IL-33 can be considered as a potential integrative biomarker representing both metabolic and reproductive aspects of PCOS. Given its high correlations with insulin resistance, adiposity, and hormonal changes, it is possible that IL-33 could be used as a useful tool for the identification of patients with a greater risk of worse metabolic or hormonal alterations. Additionally, the presence of IL-33 could improve diagnostic accuracy and patient stratification by being added to routine tests that are already used. In conclusion, there is a need for additional studies to further elucidate the mechanisms underlying the role of IL-33 in the pathogenesis of PCOS. Longitudinal and interventional studies are needed to assess the potential of targeting IL-33 pathways as a novel therapeutic approach, especially for patients with marked inflammatory or metabolic aspects. Finally, the use of IL-33 could help develop more personalized, targeted approaches for the diagnosis, monitoring and treatment of PCOS.

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