International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Operating Publisher
SciFormat Publishing Inc.
ISNI: 0000 0005 1449 8214

2734 17 Avenue SW,
Calgary, Alberta, T3E0A7,
Canada
+15878858911
editorial-office@sciformat.ca

ARTICLE TITLE	IMPACT OF INSULIN RESISTANCE ON REPRODUCTIVE OUTCOMES IN WOMEN WITH PCOS
DOI	https://doi.org/10.31435/ijitss.2(50).2026.5515
RECEIVED	25 March 2026
ACCEPTED	11 June 2026
PUBLISHED	23 June 2026
LICENSE	The article is licensed under a **Creative Commons Attribution 4.0 International License.**

IMPACT OF INSULIN RESISTANCE ON REPRODUCTIVE OUTCOMES IN WOMEN WITH PCOS

Yuliya Hlukhava *(Corresponding Author, Email: yuliya.hlukhava@pimmswia.gov.pl)*
National Medical Institute of the Ministry of the Interior and Administration, Warsaw, Poland
ORCID ID: 0009-0002-7887-6937

Nadzeya Skadorva
Jan-Mikulicz Radziecki University Clinical Hospital in Wrocław, Wrocław, Poland
ORCID ID: 0009-0008-1267-1641

Vladimir Gurskii
University Clinical Hospital in Poznań, Poznań, Poland
ORCID ID: 0009-0001-3630-3738

Maryia Bohdzel
Medical University of Warsaw, Warsaw, Poland
ORCID ID: 0009-0006-0920-2225

Krystsina Babkova
National Medical Institute of the Ministry of the Interior and Administration, Warsaw, Poland
ORCID ID: 0009-0006-9580-4520

Iuliia Ilina
Our Lady of Perpetual Help Hospital, Wołomin, Poland
ORCID ID: 0009-0008-3240-1358

Alicja Korpacka
Our Lady of Perpetual Help Hospital, Wołomin, Poland
ORCID ID: 0009-0002-3638-5312

Patrycja Anna Borowiecka
Our Lady of Perpetual Help Hospital, Wołomin, Poland
ORCID ID: 0009-0009-4861-3053

Pavel Proborshch
Our Lady of Perpetual Help Hospital, Wołomin, Poland
ORCID ID: 0009-0005-7377-1695

Raman Sazon
Provincial Hospital in Bielsko-Biała, Bielsko-Biała, Poland
ORCID ID: 0009-0001-6460-8758

ABSTRACT

Insulin resistance (IR) is a key metabolic abnormality in polycystic ovary syndrome (PCOS) and is strongly linked to reproductive dysfunction. However, the specific impact of insulin resistance on reproductive outcomes remains unclear. This review explores the current evidence on the impact of insulin resistance on reproductive results in women with PCOS. A systematic search of PubMed/MEDLINE and Google Scholar was performed to identify relevant studies published through March 2025.

Research consistently demonstrates a correlation between insulin resistance, anovulation, ovulatory problems, and an increased risk of miscarriage. This could also raise the likelihood of developing gestational diabetes and may adversely impact results in assisted reproductive technology (ART). Although obesity is often seen as a contributing factor, many studies indicate that insulin resistance alone can considerably impact reproductive outcomes. Elevated insulin levels can increase ovarian androgen production, decrease sex hormone-binding globulin (SHBG), and disrupt normal follicular development. Interventions that enhance insulin sensitivity, including lifestyle modifications and, when appropriate, metformin treatment, can improve metabolic health and potentially lead to better reproductive outcomes. Insulin resistance plays a significant role in causing reproductive dysfunction in women with PCOS, highlighting how important it is to evaluate and manage metabolic health as a vital part of fertility treatment.

KEYWORDS

Polycystic Ovary Syndrome, Insulin Resistance, Insulin, Infertility, Miscarriage, Assisted Reproductive Technology, Obesity, Androgens, Sex Hormone-Binding Globulin, Metformin, Reproductive Outcomes, Pregnancy Complications

CITATION

Yuliya Hlukhava, Nadzeya Skadorva, Vladimir Gurskii, Maryia Bohdzel, Krystsina Babkova, Iuliia Ilina, Alicja Korpacka, Patrycja Anna Borowiecka, Pavel Proborshch, Raman Sazon. (2026) Impact of Insulin Resistance on Reproductive Outcomes in Women with PCOS. *International Journal of Innovative Technologies in Social Science*. 2(50). doi: 10.31435/ijitss.2(50).2026.5515

1. Introduction

Polycystic ovary syndrome is a widespread hormonal disorder affecting women of reproductive age, often associated with ovulation and fertility issues. Based on established international diagnostic criteria, it is characterized by hyperandrogenism, irregular ovulation, and/or polycystic ovarian morphology. Insulin resistance, accompanied by compensatory hyperinsulinemia, affects a substantial proportion of women with PCOS, including those with normal body weight. Elevated insulin levels promote ovarian androgen production, reduce SHBG concentrations, and interfere with normal follicular development. Together, these mechanisms establish a clear biological link between insulin resistance and reproductive dysfunction.

Given the increasing worldwide rates of metabolic disorders, it is essential to understand how insulin resistance affects reproductive outcomes in women diagnosed with PCOS.

2. Materials and Methods

This review is based on a thorough and systematic search and critical evaluation of the available evidence. A comprehensive search was performed in PubMed/MEDLINE and Google Scholar for publications up to March 18, 2025, using relevant keywords related to PCOS, insulin resistance, and reproductive outcomes.

The search identified 515 records, of which 215 were from PubMed/MEDLINE and 300 from Google Scholar. After eliminating duplicates, 445 records were available for subsequent screening. During this process, 305 records were excluded based on their titles and abstracts. The main reasons included the absence of PCOS populations (105 records), lack of insulin resistance data (90 records), failure to report reproductive outcomes (80 records), or inappropriate study design, such as case reports or review articles (30 records).

A total of 140 full-text articles were reviewed to determine their suitability. Of these, 65 were excluded because they were not original research (30), lacked sufficient data (25), or were duplicate publications (10). A total of 75 studies meeting the eligibility criteria were included in the qualitative analysis (Figure 1).

Studies were included if they satisfied the following criteria:

1. Included women diagnosed with PCOS according to established diagnostic criteria (Rotterdam, NIH, or AE-PCOS).
2. Assessed insulin resistance using validated methods, including the homeostatic model assessment of insulin resistance (HOMA-IR), fasting insulin levels, indices from the oral glucose tolerance test (OGTT), or the hyperinsulinemic–euglycemic clamp technique.
3. Reported at least one reproductive outcome, such as ovulation, pregnancy, live birth, miscarriage, results of assisted reproductive technologies, or complications related to pregnancy.
4. Were original research studies, such as randomized controlled trials, cohort studies, case-control studies, and cross-sectional studies.

The following data were extracted: author and year, study design, sample size, PCOS diagnostic criteria, method of insulin resistance assessment, reproductive outcomes assessed, and main findings.

3. Results

3.1. Definitions and Measurement of Insulin Resistance

The included studies were analyzed to evaluate the impact of insulin resistance on reproductive outcomes in women with PCOS. The results are presented according to key clinical domains, including ovulatory function, miscarriage risk, assisted reproductive technology outcomes, and pregnancy complications (Table 1).

Assessment methods for insulin resistance differed across the included studies, with the most commonly used methods being the homeostatic model assessment for insulin resistance (HOMA-IR) and fasting insulin levels. The HOMA-IR index is a standard method for evaluating insulin resistance based on measurements of fasting glucose and insulin levels. Various studies have proposed different cutoff points for diagnosing insulin resistance, with many suggesting that levels above 2.5 to 3.0 may indicate its existence. These thresholds can vary based on population traits and the methods used. Additionally, some studies used indices derived from the oral glucose tolerance test (OGTT), which provide further insight into dynamic insulin responses.

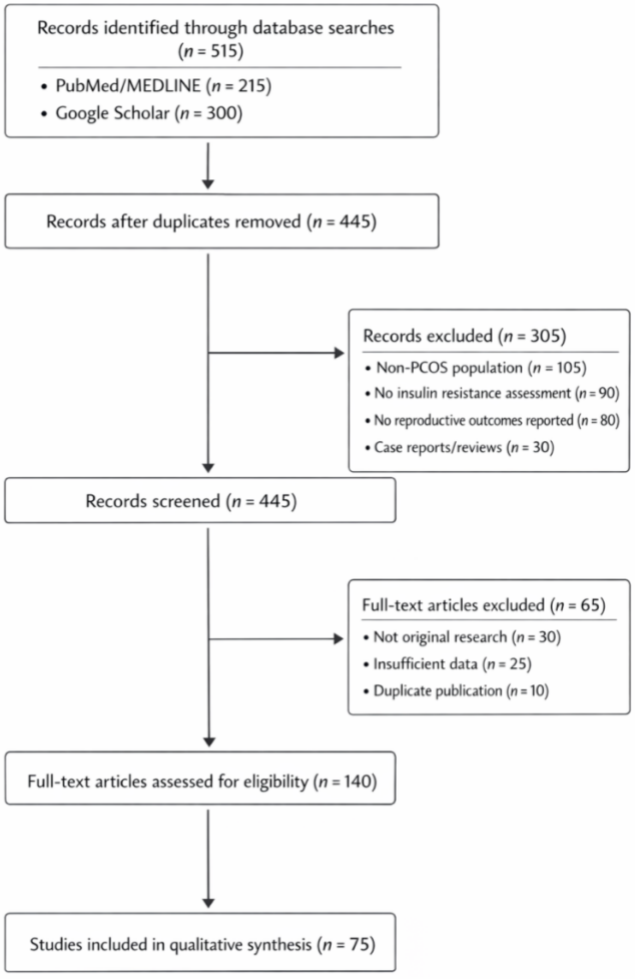

***Fig. 1**. PRISMA flowchart*

Table 1. Impact of Insulin Resistance Mechanisms in PCOS

Domain	Mechanism of Insulin Resistance
Ovulatory Function	Elevated insulin levels result in increased androgen synthesis and disrupt follicular development
Hormonal Imbalance	Insulin reduces SHBG levels and elevates free androgens
Endometrial Function	Elevated insulin level modifies endometrial receptivity and the process of implantation
Miscarriage Risk	Impaired glucose metabolism influences early pregnancy
ART Outcomes	IR affects oocyte quality and ovarian response
Pregnancy Complications	High insulin level causes systemic metabolic dysfunction and inflammation
Metformin treatment improves insulin sensitivity and reduces circulating insulin	
Independent IR vs obesity effects: IR occurs in lean and obese PCOS patients	

3.2. Insulin Resistance and Ovulatory Dysfunction

In women suffering from PCOS, insulin resistance, commonly evaluated using HOMA-IR, is strongly associated with disrupted ovulation. Multiple clinical studies suggest that HOMA-IR values above 2.5 to 3.0 are linked to decreased natural ovulation and a poorer response to ovulation induction treatments (Iuorno et al., 2002; Palomba et al., 2005).

In the PPCOS I randomized trial, women treated with clomiphene showed a significantly higher live birth rate of 22.5% compared to 7.2% in women who received only metformin. Those who received both treatments had an even greater rate of 26.8%. While the study did not stratify outcomes according to baseline HOMA-IR levels, metformin improved metabolic parameters, underscoring the role of insulin resistance in ovulatory dysfunction (Legro et al., 2007).

A Cochrane review indicated that metformin notably improved ovulation rates in comparison to a placebo (Odds Ratio 2.64; Confidence Interval 1.85–3.75). Nevertheless, its effect on live birth rates was modest and depended on individual patient characteristics (Tang et al., 2012).

Mechanistically, hyperinsulinemia promotes androgen production in ovarian theca cells by stimulating cytochrome P450c17α enzyme activity and reduces hepatic production of SHBG. This results in increased free circulating testosterone levels, which can lead to follicular arrest and chronic anovulation (Nestler et al., 1998; Dunaif, 1997).

3.3. Insulin Resistance and Miscarriage

Research indicates that women with PCOS and high insulin resistance face a greater likelihood of early pregnancy loss. An examination of women undergoing fertility treatment for polycystic ovary syndrome revealed that insulin resistance markedly raises the risk of miscarriage (OR 1.68; 95% CI 1.29–2.20). Importantly, even after accounting for body mass index (BMI), this correlation remained statistically significant even after adjusting for BMI (Sun et al., 2020).

Additional clinical data showed that higher HOMA-IR levels independently increased the risk of miscarriage after ART, regardless of BMI (Tian et al., 2007).

3.4. Insulin Resistance and Assisted Reproductive Technology Outcomes

Research indicates that increased insulin resistance may adversely affect assisted reproductive procedures, including IVF and ICSI. Women exhibiting elevated HOMA-IR levels can have lower implantation success rates and tend to generate fewer high-quality embryos. Furthermore, they are at a greater risk of early pregnancy loss. This could be a result of reduced oocyte quality caused by hyperinsulinemia, which affects the follicular environment by raising androgen levels and increasing oxidative stress (Li et al., 2024; Palomba et al., 2010).

Insulin resistance may affect endometrial receptivity and oocyte quality. Hyperinsulinemia has been associated with altered expression of key molecules involved in implantation, including integrins and HOXA10, which may impair endometrial decidualization and embryo implantation. Disruption of endometrial insulin signaling pathways could therefore independently contribute to the lower implantation rates seen in insulin-resistant women with PCOS (Cermik et al., 2003).

Insulin resistance might also influence the ovarian response to controlled stimulation. Elevated insulin levels may increase the ovaries' sensitivity to gonadotropins, leading to an excessive follicular response.

Women with PCOS already encounter an increased risk of ovarian hyperstimulation syndrome (OHSS), and metabolic dysfunction might further elevate this risk. However, the evidence is inconsistent and depends on the specific protocols used (Kotlyar et al., 2023; Tummon et al., 2005).

Interactions between insulin resistance and anti-Müllerian hormone (AMH) levels are also thought to play a role. Women with PCOS often exhibit higher AMH levels, indicating an increased number of antral follicles. Some studies indicate that high insulin levels may stimulate AMH production in granulosa cells, which can lead to follicular arrest and disrupt follicle development. However, the precise connection between insulin resistance, AMH levels, and ART results remains unclear (Pigny et al., 2003; Pai, 2024).

Together, these findings indicate that insulin resistance may impair ART success by affecting oocyte quality, endometrial receptivity, ovarian stimulation processes, and early embryo development.

3.5. Insulin Resistance and Pregnancy Complications

Women with PCOS are at an increased risk of pregnancy-related complications, including gestational diabetes, hypertensive disorders, and preterm birth. Meta-analyses indicate that women with PCOS are approximately three times more likely to develop gestational diabetes and have increased risks of hypertension and preterm birth during pregnancy (Boomsma et al., 2006).

Similarly, large population-based cohort studies have verified that women with PCOS face higher risks of gestational diabetes, preeclampsia, and very preterm delivery (Roos et al., 2011).

During the later stages of pregnancy, insulin sensitivity normally declines by approximately 50–60%. In women with pre-existing insulin resistance, the additional metabolic burden may surpass pancreatic compensatory capacity, increasing the risk of gestational diabetes and related complications (Barbour et al., 2007).

Multiple studies have shown that the increased risk of adverse pregnancy outcomes persists even after adjusting for body mass index, suggesting that insulin resistance may independently affect these risks beyond obesity (Roos et al., 2011).

However, due to the close relationship between insulin resistance and adiposity, it is difficult to completely separate their independent effects.

3.6. Effect of Insulin-Sensitizing Interventions

Lifestyle changes that lead to a 5–10% weight loss have been proven to significantly improve insulin sensitivity and may help restore ovulatory function in a substantial proportion of overweight women with PCOS (Legro et al., 2015).

Metformin is known to enhance insulin sensitivity and reduce androgen concentrations. Although it may enhance ovulation rates more effectively than a placebo, it has not demonstrated greater success than first-line treatments such as clomiphene in securing live births (Tang et al., 2012).

4. Discussion

This review emphasizes the significant impact of insulin resistance on causing negative reproductive outcomes in women with PCOS. This connection is biologically plausible and supported by consistent findings across mechanistic, clinical, and epidemiological research (Dunaif, 1997; Diamanti-Kandarakis and Dunaif, 2012). Increased insulin levels stimulate androgen production in ovarian theca cells by enhancing cytochrome P450c17α activity and decreasing hepatic SHBG synthesis. This leads to higher levels of free testosterone, which can disrupt follicle development, impair the function of granulosa cells, and result in ongoing anovulation. Together, these processes show how metabolic imbalances can affect reproductive health.

In clinical practice, it is frequently noted that elevated HOMA-IR levels correlate with a reduced number of spontaneous ovulations and a diminished response to ovulation induction treatment (Iuorno et al., 2002; Palomba et al., 2005). Although metformin can help improve insulin sensitivity and increase ovulation rates compared to placebo, studies have shown that it does not result in higher live birth rates than standard first-line ovulation induction medications (Legro et al., 2007; Tang et al., 2012). These results show that simply correcting metabolic issues may not be enough to fully restore reproductive function, especially in cases with more severe or varied presentations.

Insulin resistance may contribute to early pregnancy loss beyond just disrupting ovulation. Meta-analyses show that women with PCOS and insulin resistance have about a 60–70% higher risk of miscarriage, even after adjusting for BMI (Sun et al., 2020). In ART settings, higher insulin resistance is associated with reduced implantation rates and an increased risk of early pregnancy loss, indicating that its effects extends

beyond ovarian dysfunction and involve the endometrial receptivity and embryonic development (Tian et al., 2007; Li et al., 2024).

The link between obstetric complications and PCOS appears to be quite strong. Women diagnosed with PCOS have a nearly threefold increased risk of developing gestational diabetes, along with higher likelihoods of hypertensive conditions and preterm delivery (Boomsma et al., 2006; Toulis et al., 2009). These outcomes have been consistently supported by large population-based studies, even after adjustment for BMI and other factors (Roos et al., 2011). Since pregnancy naturally leads to a 50–60% reduction in insulin sensitivity in late gestation, women who already have insulin resistance before pregnancy may face additional metabolic difficulties. This, in turn, can raise their risk of developing gestational diabetes and endothelial dysfunction (Barbour et al., 2007).

Importantly, while obesity frequently co-occurs with insulin resistance in women with PCOS, the persistent association between insulin resistance and reproductive dysfunction even after adjusting for BMI indicates that insulin resistance isn't caused solely by excess body fat. PCOS affects metabolism in different ways, and insulin resistance isn’t limited to women who are overweight. Studies indicate that approximately 20–30% of lean women with PCOS exhibit signs of insulin resistance (Dunaif, 1997; Diamanti-Kandarakis and Dunaif, 2012). In women with PCOS, post-receptor insulin signaling impairments are frequently seen. These changes are mainly linked to reduced activity of the IRS-1–dependent PI3K/Akt pathway, whereas the MAPK pathway, which regulates mitogenic and growth-related processes, appears to remain largely unaffected. Insulin resistance linked to obesity primarily arises from dysfunctions in adipose tissue, including chronic inflammation, lipotoxicity, and changes in adipokine secretion. Although being overweight can worsen metabolic issues in PCOS, it doesn’t fully explain the insulin resistance seen in lean individuals.

The current evidence suggests a complex model in which insulin resistance affects reproductive function across ovarian, endometrial, and systemic metabolic levels.

5. Conclusions

Insulin resistance plays a significant role in reproductive problems linked to PCOS. This may lead to irregular ovulation, a higher likelihood of miscarriage, reduced success of fertility treatments, and an increased risk of metabolic complications during pregnancy. These findings emphasize the necessity of comprehensive metabolic evaluation and personalized interventions as crucial components of fertility treatment for women with PCOS.

However, most of the available evidence we have is observational. The different ways insulin resistance is defined, along with the variety of study designs, make it difficult to definitively determine cause-and-effect relationships. Going forward, it is crucial to conduct thorough prospective studies and carefully planned randomized controlled trials to increase our understanding of the underlying mechanisms, identify those at higher risk, and support the creation of more personalized treatment strategies.

Author’s Contribution:
Conceptualization: Yuliya Hlukhava, Nadzeya Skadorva
Methodology: Krystsina Babkova, Alicja Korpacka
Formal analysis: Vladimir Gurskii, Pavel Proborshch
Investigation: Raman Sazon, Maryia Bohdzel
Writing-rough preparation: Iuliia Ilina, Patrycja Anna Borowiecka
Writing-review and editing: Yuliya Hlukhava, Nadzeya Skadorva
Supervision: Yuliya Hlukhava, Nadzeya Skadorva

All authors have read and agreed with the published version of the manuscript.
Funding Statement: The authors did not receive special funding.
Conflict of Interest Statement: The authors declare no conflict of interest.

REFERENCES

1. Azziz, R., Carmina, E., Chen, Z., et al. (2016). Polycystic ovary syndrome. *Nature Reviews Disease Primers, 2*, Article 16057.
2. Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group. (2004). Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome. *Human Reproduction, 19*(1), 41–47.
3. Teede, H. J., Misso, M. L., Costello, M. F., et al. (2018). Recommendations from the international evidence-based guideline for the assessment and management of polycystic ovary syndrome. *Human Reproduction, 33*(9), 1602–1618.
4. Dunaif, A. (1997). Insulin resistance and the polycystic ovary syndrome: Mechanism and implications for pathogenesis. *Endocrine Reviews, 18*(6), 774–800.
5. Diamanti-Kandarakis, E., & Dunaif, A. (2012). Insulin resistance and the polycystic ovary syndrome revisited. *Endocrine Reviews, 33*(6), 981–1030.
6. Goodarzi, M. O., Dumesic, D. A., Chazenbalk, G., & Azziz, R. (2011). Polycystic ovary syndrome: Etiology, pathogenesis, and diagnosis. *Nature Reviews Endocrinology, 7*(4), 219–231.
7. Legro, R. S., Castracane, V. D., & Kauffman, R. P. (2004). Detecting insulin resistance in polycystic ovary syndrome: Purposes and pitfalls. *Obstetrical & Gynecological Survey, 59*(2), 141–154.
8. Moran, L. J., Misso, M. L., Wild, R. A., & Norman, R. J. (2010). Impaired glucose tolerance, type 2 diabetes, and metabolic syndrome in polycystic ovary syndrome: A systematic review and meta-analysis. *Human Reproduction Update, 16*(4), 347–363.
9. Palomba, S., de Wilde, M. A., Falbo, A., et al. (2015). Pregnancy complications in women with polycystic ovary syndrome. *Human Reproduction Update, 21*(5), 575–592.
10. Boomsma, C. M., Eijkemans, M. J., Hughes, E. G., Visser, G. H., Fauser, B. C., & Macklon, N. S. (2006). A meta-analysis of pregnancy outcomes in women with polycystic ovary syndrome. *Human Reproduction Update, 12*(6), 673–683.
11. Toulis, K. A., Goulis, D. G., Kolibianakis, E. M., Venetis, C. A., Tarlatzis, B. C., & Papadimas, I. (2009). Risk of gestational diabetes mellitus in women with polycystic ovary syndrome: A systematic review and meta-analysis. *Fertility and Sterility, 92*(2), 667–677.
12. Legro, R. S., Barnhart, H. X., Schlaff, W. D., et al. (2007). Clomiphene, metformin, or both for infertility in the polycystic ovary syndrome. *The New England Journal of Medicine, 356*(6), 551–566.
13. Tang, T., Lord, J. M., Norman, R. J., Yasmin, E., & Balen, A. H. (2012). Insulin-sensitising drugs (metformin) for women with polycystic ovary syndrome. *Cochrane Database of Systematic Reviews*, (5), Article CD003053.
14. Nestler, J. E., & Jakubowicz, D. J. (1996). Decreases in ovarian cytochrome P450c17α activity and serum free testosterone after reduction of insulin secretion in polycystic ovary syndrome. *The New England Journal of Medicine, 335*(9), 617–623.
15. Barber, T. M., McCarthy, M. I., Wass, J. A., & Franks, S. (2006). Obesity and polycystic ovary syndrome. *Clinical Endocrinology, 65*(2), 137–145.
16. Fauser, B. C. J. M., Tarlatzis, B. C., Rebar, R. W., et al. (2012). Consensus on women's health aspects of polycystic ovary syndrome. *Human Reproduction, 27*(1), 14–24.
17. Siristatidis, C. S., Sergentanis, T. N., Vogiatzi, P., et al. (2013). Assisted reproduction outcomes in women with polycystic ovary syndrome: A systematic review and meta-analysis. *Fertility and Sterility, 99*(7), 1929–1942.
18. Qiao, J., & Feng, H. L. (2011). Extra- and intra-ovarian factors in polycystic ovary syndrome: Impact on oocyte maturation and embryo developmental competence. *Human Reproduction Update, 17*(1), 17–33.
19. Glueck, C. J., & Goldenberg, N. (2019). Characteristics of obesity in polycystic ovary syndrome: Etiology, treatment, and genetics. *Metabolism, 92*, 108–120.
20. Pasquali, R., & Gambineri, A. (2006). Metabolic effects of obesity on reproduction. *Reproductive BioMedicine Online, 12*(5), 542–551.
21. Lord, J., Flight, I. H. K., & Norman, R. J. (2003). Metformin in polycystic ovary syndrome: Systematic review and meta-analysis. *BMJ, 327*, 951–953.
22. Cassar, S., Misso, M. L., Hopkins, W. G., et al. (2016). Insulin resistance in polycystic ovary syndrome: A systematic review and meta-analysis of euglycaemic-hyperinsulinaemic clamp studies. *Human Reproduction, 31*(11), 2619–2631.
23. Rojas, J., Chávez, M., Olivar, L., et al. (2014). Polycystic ovary syndrome, insulin resistance, and obesity: Navigating the pathophysiologic labyrinth. *International Journal of Reproductive Medicine, 2014*, Article 719050.
24. Franks, S. (1995). Polycystic ovary syndrome. *The New England Journal of Medicine, 333*(13), 853–861.
25. Iuorno, M. J., Jakubowicz, D. J., Baillargeon, J. P., et al. (2002). Effects of metformin on insulin and androgen levels in women with polycystic ovary syndrome. *The Journal of Clinical Endocrinology & Metabolism, 87*(2), 567–574.
26. Palomba, S., Orio, F., Jr., Falbo, A., et al. (2005). Prospective parallel randomized, double-blind, double-dummy controlled clinical trial comparing clomiphene citrate and metformin as first-line treatment for ovulation induction in nonobese anovulatory women with polycystic ovary syndrome. *The Journal of Clinical Endocrinology & Metabolism, 90*(7), 4068–4074.

27. Sun, X., Zhang, D., Zhang, W., et al. (2020). Insulin resistance is associated with increased risk of miscarriage in women with polycystic ovary syndrome: A systematic review and meta-analysis. *Reproductive Biology and Endocrinology, 18*, Article 76.
28. Tian, L., Shen, H., Lu, Q., et al. (2007). Insulin resistance increases the risk of spontaneous abortion after assisted reproduction in women with polycystic ovary syndrome. *Fertility and Sterility, 88*(2), 456–461.
29. Palomba, S., Falbo, A., Zullo, F., et al. (2009). Evidence-based and potential benefits of metformin in the polycystic ovary syndrome: A comprehensive review. *Endocrine Reviews, 30*(1), 1–50.
30. Matsuyama, S., Whiteside, S., & Li, S. Y. (2024). Implantation and decidualization in PCOS: Unraveling the complexities of pregnancy. *International Journal of Molecular Sciences, 25*(2), Article 1203.
31. Apparao, K. B. C., Lovely, L. P., Gui, Y., et al. (2002). Elevated endometrial androgen receptor expression in women with polycystic ovary syndrome. *Biology of Reproduction, 66*(2), 297–304.
32. Pigny, P., Merlen, E., Robert, Y., et al. (2003). Elevated serum anti-Müllerian hormone levels in polycystic ovary syndrome: Relationship to follicle number and insulin resistance. *The Journal of Clinical Endocrinology & Metabolism, 88*(12), 5957–5962.
33. Roos, N., Kieler, H., Sahlin, L., et al. (2011). Risk of adverse pregnancy outcomes in women with polycystic ovary syndrome: Population-based cohort study. *BMJ, 343*, Article d6309.
34. Legro, R. S., Dodson, W. C., Kunselman, A. R., et al. (2016). Benefit of delayed fertility therapy with preconception weight loss over immediate therapy in obese women with PCOS. *The Journal of Clinical Endocrinology & Metabolism, 101*(7), 2658–2666.
35. Cermik, D., Selam, B., & Taylor, H. S. (2003). Regulation of HOXA10 expression by testosterone in vitro and in the endometrium of women with polycystic ovary syndrome. *The Journal of Clinical Endocrinology & Metabolism, 88*(5), 238–243.