COALITION FOR METABOLIC HEALTH

PMOS and Metabolic Health: Why Insulin Resistance Matters Across a Woman's Lifespan

Polyendocrine metabolic ovarian syndrome (PMOS), previously known as polycystic ovary syndrome (PCOS), is one of the **most common endocrine disorders** affecting women of reproductive age,[1,2,3] and is commonly associated with infertility and reduced fertility.[4,5]

In 2026, an international multidisciplinary consensus formally adopted PMOS to move beyond a name that many experts viewed as inaccurate and better reflect the condition's impact on endocrine, ovarian, and metabolic health.[6]

This broader understanding has fueled growing interest in dietary approaches that target underlying metabolic dysfunction, with studies of low-carbohydrate and ketogenic therapies (diets typically defined as less than 130 grams of total carbohydrates per day and less than 50 grams of total carbohydrates per day, respectively) providing a valuable model for examining how nutritional strategies can alter the health trajectory for women with PMOS.

PMOS Is Common, Yet Often Missed

The most current U.S. data estimate that 8.7% of women ages 20–49 have been diagnosed with PMOS,[7] yet many women who meet diagnostic criteria may never receive a formal diagnosis. Recent research found that Black women have 69% higher odds of a missed diagnosis than non-Hispanic White women, and women covered by Medicaid or charity care are 90% more likely to experience a missed diagnosis than those with private insurance.[8] These diagnosis gaps are particularly concerning given the central role PMOS plays in shaping both reproductive and long-term metabolic health.

The Bridge Between Metabolic and Reproductive Health

PMOS is characterized by high levels of male sex hormones (androgens), ovulatory dysfunction, and an increased number of small fluid-filled sacs, known as follicles, in the ovaries.[6,9] While infertility is often the most visible manifestation, PMOS is increasingly understood as a multisystem condition that sits at the intersection of reproductive and metabolic health.[6,10,11]

A central feature of PMOS pathophysiology is insulin resistance, [11] which affects an estimated 35% to 85% of women with PMOS. [1] Insulin resistance is a metabolic state in which the body's cells become less responsive to insulin and require higher levels of the hormone to maintain normal blood glucose levels. Although insulin resistance can occur in women with PMOS of all body sizes, risks are often amplified in women who are overweight or obese.[4,5,12]

Elevated insulin levels (hyperinsulinemia) stimulate excess ovarian androgen production, a group of steroid hormones such as testosterone that can contribute to PMOS symptoms when levels become too high. At the same time, hyperinsulinemia reduces liver production of sex hormone-binding globulin (SHBG), a protein that regulates the number of active androgens circulating in the bloodstream. With less SHBG, increased concentrations of free androgens circulate in the bloodstream, worsening the hormonal balance that defines PMOS.[10,11,13,14]

Hyperinsulinemia also indirectly raises levels of luteinizing hormone (LH), a key reulator of ovulation. When LH levels become disproportionately high relative to follicle-stimulating hormone (FSH), a hormone that supports the growth and maturation of ovarian follicles, androgen production increases and normal follicular development can be impaired.[10,13,14] This can result in irregular or absent menstrual cycles, disrupted ovulation, and challenges with fertility.[3,10,13] The effects extend beyond fertility and into pregnancy. A large systematic review and meta-analysis involving more than 100,000 pregnancies found that women with PMOS face higher odds of miscarriage, gestational diabetes, gestational hypertension, and preeclampsia compared to women without the condition, even after accounting for age and body weight. [4]

Metabolic Consequences Beyond Reproductive Health

The effects of hyperinsulinemia are not limited to reproductive health. Insulin resistance and the metabolic dysfunction it drives significantly increase the risk of impaired glucose tolerance, type 2 diabetes, high cholesterol, and cardiovascular disease.[1,5,9,10] These findings underscore the far-reaching health consequences of insulin resistance in PMOS.

Why Nutrition Matters: What the Science Shows

Given the central role of insulin resistance in PMOS, dietary approaches that influence glucose and insulin sensitivity are a growing area of clinical and research interest. The evidence supporting low-carbohydrate and ketogenic therapies consistently demonstrates improvements in body measurements, metabolic health, hormone regulation, and reproductive outcomes.[2,5,13,14,15]

Across studies of women with PMOS, ketogenic interventions were consistently associated with reductions in body weight, BMI, visceral adiposity, and waist circumference.[3,5,12,13,15,16] Improvements in insulin resistance and glycemic regulation were also consistently observed, including reductions in fasting insulin and insulin resistance.[3,5,13,15,16,17] These findings have broader implications because excess body fat, particularly visceral adiposity, and insulin resistance are key contributors to many of the long-term metabolic complications associated with PMOS, including type 2 diabetes and cardiovascular disease risk factors.[1,9,10]

These metabolic changes are often accompanied by improvements in reproductive hormone profiles. Meta-analytic evidence has shown reductions in luteinizing hormone-to-follicle-stimulating hormone (LH/FSH) ratio and free testosterone alongside increases in SHBG following ketogenic interventions. Together, these changes reflect improvements in the hormonal imbalances that contribute to ovulatory dysfunction and other reproductive manifestations of PMOS.[14] Similar findings have been reported in individual intervention studies, including reductions in free testosterone, total testosterone, and clinical signs of hyperandrogenism.[3,16]

Why Nutrition Matters: What the Science Shows

Because elevated androgen levels and hormone imbalances are closely linked to ovulatory dysfunction in PMOS, improvements in these markers help support more normal reproductive function.[9,10,13] Consistent with this biological rationale, clinical reproductive outcomes have also shown encouraging signals, with studies indicating improvements in menstrual regularity, restoration of ovulatory cycles, and increases in ovulation frequency following ketogenic therapeutic approaches.[2,5,12,15,16]

In a 16-week randomized controlled trial, women assigned to a very-low-calorie ketogenic intervention experienced greater improvements in ovulation and hyperandrogenism compared with a Mediterranean low-calorie diet.[16] A few small studies have additionally reported spontaneous pregnancies among women with a history of infertility, findings that warrant further investigation in larger controlled trials.[2,12,17] Emerging evidence also suggests potential benefits in assisted reproduction, with one small study demonstrating improved embryo implantation and pregnancy rates among women with PMOS undergoing in vitro fertilization (IVF) treatments following a ketogenic therapeutic approach.[18]

Implications for Patients and Health Systems

The available evidence suggests that low-carbohydrate and ketogenic therapies may improve metabolic, hormonal, and reproductive outcomes in women with PMOS.

However, much of the current evidence is derived from relatively small studies with short follow-up periods and varying intervention protocols. Further research is needed to evaluate the impact of these nutritional approaches on outcomes such as fertility, pregnancy complications, and cardiometabolic disease. As understanding of the relationship between metabolic health and reproductive function continues to evolve, PMOS represents an important area for continued investigation into how metabolic interventions may influence women's health across the lifespan.

References

1. Amisi CA. Markers of insulin resistance in polycystic ovary syndrome women: an update. *World J Diabetes. 2022;13(3):129-149.*
2. Fleigle D, et al. Polycystic ovary syndrome and the effects of a ketogenic diet: a scoping review. *Nutrients. 2025;17(17):2893.*
3. Paoli A, et al. Effects of a ketogenic diet in overweight women with polycystic ovary syndrome. *J Transl Med. 2020;18(1):104.*
4. Bahri Khomami M, et al. Systematic review and meta-analysis of pregnancy outcomes in women with polycystic ovary syndrome. *Nat Commun. 2024;15:5591.*
5. Diha A, et al. Effects of ketogenic diet on metabolic, endocrine, and reproductive outcomes in overweight/obese women with polycystic ovary syndrome: a systematic review. *Cureus. 2026;18(2):e103615.*
6. Teede HJ, et al. Polyendocrine metabolic ovarian syndrome, the new name for polycystic ovary syndrome: a multistep global consensus process. *Lancet. 2026;407:2329-2339.*
7. Nugent C, et al. Endometriosis, polycystic ovary syndrome, and uterine fibroids in women ages 20–49: United States, 2022–2023. NCHS Health E-Stat. 2026 Jul;(121):1–5.
8. Silva EL, et al. Polycystic ovary syndrome underdiagnosis patterns by individual-level and spatial social vulnerability measures. *J Clin Endocrinol Metab. 2025;110(6):1657-1666.*
9. Teede HJ, et al. Recommendations from the 2023 international evidence-based guideline for the assessment and management of polycystic ovary syndrome. *J Clin Endocrinology & Metabolism. 2023;108(10):2447-2469.*
10. Dong J, et al. Polycystic ovary syndrome: pathophysiology and therapeutic opportunities. *BMJ Med. 2023;2:e000548.*
11. Dunaif A. Insulin resistance and the polycystic ovary syndrome: mechanism and implications for pathogenesis. *Endocrine Reviews. 1997;18(6):774-800.*
12. Cincione R, et al. Effects of mixed ketogenic diet in overweight and obese women with polycystic ovary syndrome. *Int J Environ Res Public Health. 2021;18(23):12490.*
13. Cannarella R, et al. Effects of ketogenic diets on polycystic ovary syndrome: a systematic review and meta-analysis. *Reprod Biol Endocrinol. 2025;23:74.*
14. Khalid K, et al. Effects of ketogenic diet on reproductive hormones in women with polycystic ovary syndrome. *J Endocr Soc. 2023;7(10)*
15. Arsenaki E, et al. The effects of ketogenic diet on polycystic ovary syndrome: a systematic review and meta-analysis. *Clin Nutr. 2026;56:106535.*
16. Pandurevic S, et al. Efficacy of very low-calorie ketogenic diet with the Pronokal® method in obese women with polycystic ovary syndrome: a 16-week randomized controlled trial. *Endocr Connect. 2023;12(7):e220536.*
17. Sharifi M, et al. The effects of portfolio moderate-carbohydrate and ketogenic diets on anthropometric indices, metabolic status, and hormonal levels in overweight or obese women with polycystic ovary syndrome: a randomized controlled trial. *Nutr J. 2024;23:152.*
18. Palafox-Gómez C, et al. Adding a ketogenic dietary intervention to IVF treatment in patients with polycystic ovary syndrome improves implantation and pregnancy. *Reprod Toxicol. 2023;119:108420.*