

Comparative Effectiveness of Myo-Inositol and Carnitine in the Management of Hyperinsulinemic Polycystic Ovary Syndrome

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ABSTRACT

Polycystic Ovarian Syndrome (PCOS) is a common endocrine disorder in women of reproductive age, with insulin resistance and hyperinsulinemia playing key roles in its development. Although metformin is widely used to improve insulin sensitivity, its gastrointestinal side effects have increased interest in alternative therapies such as Myo-Inositol (MI) and L-carnitine. This review compares the mechanisms, clinical efficacy, and safety of MI and L-carnitine in the management of hyperinsulinemic PCOS. A comprehensive literature search was conducted across PubMed, Cochrane, Scopus, including Randomised Controlled Trials and mechanistic studies published from 2000 to 2024. Relevant evidence assessing the efficacy and safety of Myo-inositol and L-carnitine in PCOS were analyzed. MI functions as a crucial mediator of insulin signaling and helps restore insulin sensitivity, improve ovarian function, and regulate ovulation by maintaining the ovarian myo-inositol to D-chiro-inositol balance. L-carnitine enhances mitochondrial fatty acid transport and β -oxidation, improves energy metabolism, and reduces oxidative stress. Clinical studies have shown that both supplements significantly lower fasting insulin levels, improve insulin resistance, and reduce hyperandrogenic symptoms. MI demonstrates greater benefits in improving ovulation, menstrual regularity, and fertility-related outcomes, while L-carnitine appears more effective in promoting weight loss, reducing body mass index, and improving metabolic parameters. Overall, both MI and L-carnitine are safe and promising therapeutic options for women with hyperinsulinemic PCOS, although further large-scale studies are needed to determine optimal treatment approaches.

Keywords: HOMA-IR, Hyperinsulinemia, Inositol phosphoglycans, Insulin resistance, L-carnitine, Mitochondrial fatty acid oxidation, Myo-inositol, Polycystic ovary syndrome.

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INTRODUCTION

Polycystic Ovary Syndrome (PCOS) is a common and heterogeneous endocrine disorder affecting 5-21% of women of reproductive age, characterized by ovulatory dysfunction, hyperandrogenism, and polycystic ovarian morphology (Azziz *et al.*, 2016; Joham *et al.*, 2022). As a result, single approaches

to treatment are not generally adequate for management of all cases. One of the many mechanisms involved in PCOS is insulin resistance (Zhao *et al.*, 2023). Hyperinsulinemia promotes ovarian androgen production and reduces Sex Hormone-Binding Globulin (SHBG) levels, leading to increased circulating bioactive androgens (Dutta *et al.*, 2025). Women with insulin-resistant PCOS are commonly treated with metformin; however, naturally occurring insulin sensitizers have gained attention due to their ability to improve insulin sensitivity with better tolerability and additional metabolic and reproductive benefits (Shaharyar *et al.*, 2026).

Myo-inositol and L-carnitine are naturally occurring compounds that are often reduced in women with PCOS and may help



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improve insulin sensitivity and metabolic function (Facchinetti *et al.*, 2020; Unfer *et al.*, 2017). This review compares the metabolic, hormonal, and reproductive effects of myo-inositol and L-carnitine in hyperinsulinemic PCOS.

METHODOLOGY

This study is a comprehensive narrative review comparing Myo-inositol and L-carnitine for managing hyper insulinemic Polycystic Ovary Syndrome. A systematic literature search was conducted across PubMed, Cochrane Library and Scopus for RCTs, mechanistic studies published between 2004 and 2024. Studies were included if they assessed the efficacy safety, or mechanisms of either supplement in PCOS patients, with data extraction focusing study characteristics, intervention details, outcomes (metabolic, hormonal, reproductive, anthropometric), and safety profiles.

INSULIN RESISTANCE AND HYPERINSULINEMIA IN PCOS: MECHANISTIC OVERVIEW

Post-receptor Insulin Signaling Defects

In PCOS, impaired IRS-1/PI3K/AKT signaling causes insulin resistance, while the MAPK/ERK pathway remains active, promoting ovarian cell proliferation and androgen production (Herman *et al.*, 2023; Qi *et al.*, 2025). Insulin increases ovarian androgen production by stimulating StAR protein and CYP17A1 activity in theca cells, resulting in hyperandrogenemia (Hajam *et al.*, 2024; Wang *et al.*, 2023).

Inositol Metabolism and the DCI Paradox

In PCOS, altered Myo-Inositol (MI) and D-Chiro-Inositol (DCI) metabolism contributes to insulin resistance, with MI supporting glucose uptake via GLUT4 and DCI promoting glycogen synthesis (Roseff & Montenegro, 2020; Sortino *et al.*, 2017). In PCOS, insulin decreases epimerase activity in peripheral tissues but increases it in the ovary, disrupting the normal MI:DCI balance and reducing the follicular fluid ratio from about 100:1 to as low as 0.2:1 (Kalra *et al.*, 2016; Petrillo *et al.*, 2022). Excess DCI in the ovary impairs aromatase activity and FSH signaling, leading to ovarian dysfunction, a phenomenon known as the DCI paradox that supports the use of myo-inositol supplementation in PCOS (Figure 1) (Eini *et al.*, 2022).

Mitochondrial Dysfunction and Oxidative Stress

Mitochondrial dysfunction and increased oxidative stress contribute to insulin resistance in PCOS, characterized by elevated reactive oxygen species and reduced antioxidant defenses (Deer *et al.*, 2025; Liu *et al.*, 2025). Mitochondrial dysfunction impairs fatty acid β -oxidation, promoting lipotoxicity and reduced insulin signaling, providing a rationale for the therapeutic use of L-carnitine in PCOS (Römer *et al.*, 2021).

MYO-INOSITOL: MECHANISMS AND CLINICAL EVIDENCE

Biochemistry and Mechanisms of Action

Myo-inositol is a naturally occurring molecule that acts as a key secondary messenger in insulin and FSH signaling, linking metabolic and reproductive functions in PCOS (Su *et al.*, 2025). Myo-inositol is a naturally occurring compound that serves as a key component of cell signaling pathways, acting as a secondary messenger in insulin and FSH signaling and thereby linking metabolism and reproduction in PCOS (Jiang *et al.*, 2026). Myo-inositol improves insulin sensitivity by activating AMPK and supports ovulation by enhancing FSH signaling, aromatase expression, and estrogen production in ovarian follicles (Dutta *et al.*, 2023). The therapeutic effectiveness of myo-inositol in PCOS depends on maintaining an appropriate MI:DCI ratio, with a 40:1 ratio widely used to restore metabolic and reproductive function without worsening the DCI paradox (Roseff & Montenegro, 2020).

Clinical Evidence: Metabolic Outcomes

The effect of MI on the body in women with PCOS who have insulin levels has been studied in many clinical trials and analyzed in several reviews. One big review that looked at 9 trials with 247 women with PCOS and 249 women without PCOS found that taking MI supplements lowered fasting insulin levels and improved how the body uses insulin. This review showed that MI supplements made a difference in insulin levels and how the body uses insulin. Women who took MI also had testosterone levels and higher levels of a protein that helps reduce the effects of male hormones (Sharon *et al.*, 2024).

A big clinical trial with 196 women with PCOS compared taking metformin to taking metformin and MI together. The trial found that taking metformin and MI together worked better than taking metformin to improve how the body uses insulin. More women who took both metformin and MI showed improvement than women who took metformin. This suggests that MI can be an addition to the medication's women with PCOS are already taking rather, than something they take by itself (Ravn *et al.*, 2022).

The effect of inositol on PCOS was examined in this review. It was discovered that inositol enhanced ovulation, helped control cycles, and reduced insulin resistance. The research, however, differed from one another. Many were little, making it challenging to make firm judgements (Fitz *et al.*, 2024). At a daily dose of 4 g, inositol is well tolerated. According to research, taking more than 4 g daily does not improve insulin resistance. Doses up to 12 g daily have been used in studies without any negative effects. Nausea and loose stools are among the stomach problems that some people have noticed, but they are typically transient (DiNicolantonio & O'Keefe, 2022).

Clinical Evidence: Reproductive Outcomes

MI has many advantages for PCOS-affected women, particularly in terms of assisting in conception. MI performs several tasks. It improves the body's utilisation of insulin. It immediately affects the ovaries as well. According to certain research, women with PCOS can benefit greatly from taking MI daily for six months. It can lower the level of a hormone called luteinizing hormone. It can also help women get their periods back on track. In fact, sixty eight percent of women who took MI got their periods back to normal (Unfer *et al.*, 2017). MI is also helpful for women who are using treatments to get pregnant like *in vitro* fertilization. It can help make the eggs healthier and improve the chances of getting pregnant. Some doctors think that women should start taking MI least three months before they try to get pregnant with *in vitro* fertilization. MI is also good for women with PCOS who are not trying to get pregnant away. It can be used with medicines like birth control pills to help control the symptoms of PCOS. This is because MI and birth control pills work well together to help women with PCOS feel better and be healthier. The International Evidence-based PCOS Guidelines recommend that women with PCOS take MI for least three months before they try to get pregnant. This gives the body time to respond to the MI and get ready, for pregnancy (Mashhadi *et al.*, 2025).

L-CARNITINE: MECHANISMS AND CLINICAL EVIDENCE

Biochemistry and Mechanisms of Action

L-carnitine is a substance that our body makes from other things like lysine and methionine. The kidney, liver and brain are the places where this happens. L-carnitine plays a role in helping our body use fat for energy. It does this by moving acids into the part of our cells where energy is made called the mitochondria (Thakur *et al.*, 2022). L-carnitine supports fatty acid metabolism, prevents lipid accumulation, improves mitochondrial function and insulin sensitivity, and reduces oxidative stress and inflammation in PCOS (Virmani & Cirulli, 2022). L-carnitine regulates energy metabolism and inflammation, supporting weight management, reducing androgen levels, improving fertility, and enhancing insulin sensitivity in women with PCOS (Alhasaniah, 2023).

Clinical Evidence: Metabolic Outcomes

Clinical studies suggest that L-carnitine supplementation can improve body weight, BMI, and metabolic parameters in women with PCOS, although its effects may vary depending on the dose and duration of treatment (Talari *et al.*, 2019a, 2019b). Studies indicate that L-carnitine improves glycemic control and insulin resistance, with its effectiveness influenced by dosage, treatment duration, and body mass index (Gheysari *et al.*, 2024). In women with PCOS, L-carnitine has been shown to improve blood glucose levels, reduce insulin resistance, and promote weight loss, with

some studies suggesting greater effects on BMI reduction than several other nutritional supplements (Mohd Shukri *et al.*, 2022).

Clinical Evidence: Reproductive and Fertility Outcomes

The use of L-carnitine in Polycystic Ovary Syndrome or PCOS is very helpful in the field of medicine. A study that looked at other studies called a systematic review and meta-analysis was published in 2023 in the journal Clin Endocrinol. This study found that L-carnitine has an effect on fertility outcomes in women with PCOS. It helps women with PCOS to ovulate regularly and gets better results for clinical pregnancies. It also helps with metabolic problems (Salehpour *et al.*, 2019). The way L-carnitine works is easy to understand. To grow follicles in the ovaries a lot of energy is needed. In women with PCOS the cells that help follicles grow do not work well because of many male hormones, oxidative stress and inflammation (Abu-Zaid *et al.*, 2025). L-carnitine helps these cells work better by making them more efficient at using energy. L-carnitine also helps the cells in the ovaries to live and work better. It keeps the mitochondria which're like the powerhouses of the cells working well. This all helps to create an environment for the follicles in the ovaries to grow. Some women with PCOS do not respond to a medicine called clomiphene (Agarwal *et al.*, 2018). In this case, adding L-carnitine to the treatment can really help. It improves the chances of getting pregnant helps with blood sugar control and improves the levels of fats in the blood. All of these things are very helpful for women, with PCOS who are having trouble getting pregnant (Hafezi *et al.*, 2024).

COMPARATIVE EFFECTIVENESS: MYO-INOSITOL VERSUS L-CARNITINE

Insulin Resistance and Glycemic Control

In women with hyperinsulinemic PCOS, both MI and L-carnitine result in clinically significant improvements in HOMA-IR and fasting insulin; nevertheless, their efficacy is determined by mechanistically different processes. By re-establishing inositol phosphoglycan signalling, MI directly replicates the molecular effects of insulin on GLUT4 translocation and glycogen formation, thereby addressing insulin resistance at the post-receptor second-messenger level. It is consequently a PCOS-targeted rather than a general insulin sensitiser because its effects are most noticeable in the context of inositol deficiency-mediated insulin resistance, a condition that is unique to PCOS (Vyas, 2022).

On the other hand, L-carnitine indirectly improves insulin signalling by reducing intracellular lipid buildup, oxidative stress, and inflammation-the origins of lipid-induced insulin resistance. Its glycaemic effects are significant in a number of populations, including those with metabolic syndrome and type 2 diabetes, although they are not exclusive to PCOS and most likely reflect general mitochondrial support. This distinction has practical implications: in slim women with PCOS whose insulin resistance

The DCI paradox: Inositol dysmetabolism in PCOS

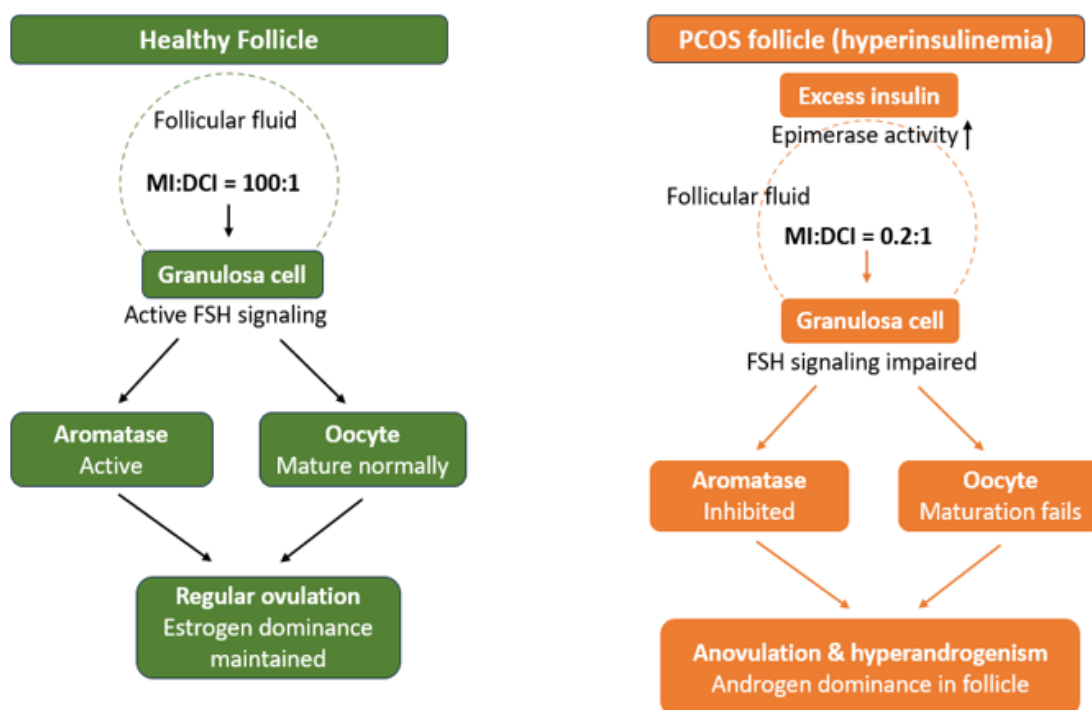


Figure 1: The DCI paradox: Inositol dysmetabolism in PCOS. Shows the DCI paradox side-by-side: Hexamers and its downstream consequences.

is predominantly due to inositol pathway dysregulation rather than lipotoxicity, MI may offer more tailored efficacy. When lipid-mediated insulin resistance predominates in obese or overweight women with PCOS, L-carnitine may provide an extra or superior benefit (Bo *et al.*, 2024).

The most insightful direct comparison to date is found in Liao and colleagues' comparative network meta-analysis. In 41 trials involving 2,362 PCOS patients, carnitine exceeded inositol in lowering body weight and BMI (SUCRA values >96%), chromium outperformed both in lowering HOMA-IR, and omega-3 fatty acids demonstrated the largest reduction in FBG. This ranking emphasises that a single-agent approach may be intrinsically less successful for the entire metabolic syndrome burden of PCOS, and that multiple supplements may be most helpful for distinct metabolic goals (Figure 2) (Hu *et al.*, 2023).

Androgen Excess and Hormonal Parameters

Because hyperinsulinemia is the primary cause of ovarian androgen overproduction, both MI and L-carnitine have the ability to lessen hyperandrogenism in PCOS. The paths to this common endpoint, however, differ mechanistically. By balancing the intraovarian MI:DCI ratio, reducing excessive DCI-mediated theca cell androgenesis, and increasing FSH-stimulated aromatase activity in granulosa cells to favour oestradiol over androgen synthesis, MI more directly restores the ovarian steroidogenic environment (Baptiste *et al.*, 2010; Barbieri &

Hornstein, 1988). Increases in SHBG and decreases in free and total testosterone are the results of these effects, especially when MI treatment lasts longer than 24 weeks. By reducing oxidative stress and mitochondrial dysfunction, L-carnitine inhibits the pro-inflammatory and hyperandrogenic environment within the ovary (Simons *et al.*, 2021). Carnitine supplementation dramatically reduces DHEAS and free testosterone, according to clinical studies. The inositol-mediated FSH signalling pathway, which is notably disturbed in PCOS, is not directly modulated by L-carnitine mechanistically, which may account for why MI exhibits more robust and consistent effects on ovarian function and fertility in comparative assessments (Koohepeyma *et al.*, 2022).

Body Weight and Anthropometric Outcomes

One area where L-carnitine definitely surpasses MI clinically is body composition and anthropometric outcomes. Across all trials, carnitine supplementation significantly reduces body weight, BMI, waist circumference, and hip circumference in PCOS patients. This is consistent with carnitine's fundamental role in increasing mitochondrial fatty acid oxidation and its ensuing effects on energy expenditure, basal metabolic rate, and lean body mass preservation (Samimi *et al.*, 2016). Although MI has been found in certain studies to result in modest weight reduction, it does not exhibit the same amount of adiposity-targeted effect. In these situations, weight loss enhances ovulatory function,

androgen levels, and insulin sensitivity via distinct pathways (Sangouni *et al.*, 2022).

Safety, tolerability, and Practical Considerations

According to clinical research, MI has not resulted in any significant side effects at doses up to 12 g/day, and at the typical therapeutic dose of 4 g/day, there are seldom any significant side effects other from the sporadic mild gastrointestinal complaint (Brown & Wright, 2020). L-carnitine is also well tolerated at doses of 250-3,000 mg/day. A fishy body smell brought on by metabolic conversion to trimethylamine is the most commonly reported adverse effect, which varies based on formulation and dosage (Rocher *et al.*, 2011). Practically speaking, both are readily available as over-the-counter dietary supplements, are well-liked by patients, and don't need to be monitored in a lab like pharmaceutical insulin sensitizers like metformin do (Ortiz-Flores *et al.*, 2018; Zeng *et al.*, 2024).

Combination Therapy: Evidence and Rationale

A strong theoretical foundation for combination therapy is created by the molecular complementarity of MI and L-carnitine, one of which functions at the insulin second-messenger level within the phosphoinositide cascade and the other at the level of mitochondrial fatty acid metabolism and oxidative stress. This theory is directly addressed by an ongoing RCT (NCT07266259)

that compares levocarnitine + MI versus MI alone in subfertile women with insulin -resistant PCOS. The primary and secondary endpoints being measured are HOMA-IR, LH: FSH ratio, and testosterone, and AMH (Zhang *et al.*, 2026). A parallel trial examining acetyl-L-carnitine with MI and D-chiro-inositol (NCT05767515) further supports the growing clinical interest in these combinations. Such combinations may address complementary aspects of PCOS pathophysiology, accordingly to early mechanistic data: carnitine reduces the lipotoxic and pro-oxidant environment that independently sustains insulin resistance and ovarian dysfunction, while MI restores inositol-mediated insulin signalling (Clinical Trials, 2026). Clinicians may rationally consider sequential or combination supplementation strategies guided by phenotypic characteristics-particularly BMI, the degree of insulin resistance, the predominant symptom burden, and fertility goals-until solid head-to-head comparative data from adequately powered trials are available (Gómez-Peralta *et al.*, 2024) (Table 1).

PHENOTYPE-DIRECTED THERAPEUTIC CONSIDERATIONS

From the comparative evidence reviewed, several phenotype-stratified recommendations can be proposed, with the caveat that high-quality head-to-head trial data are currently

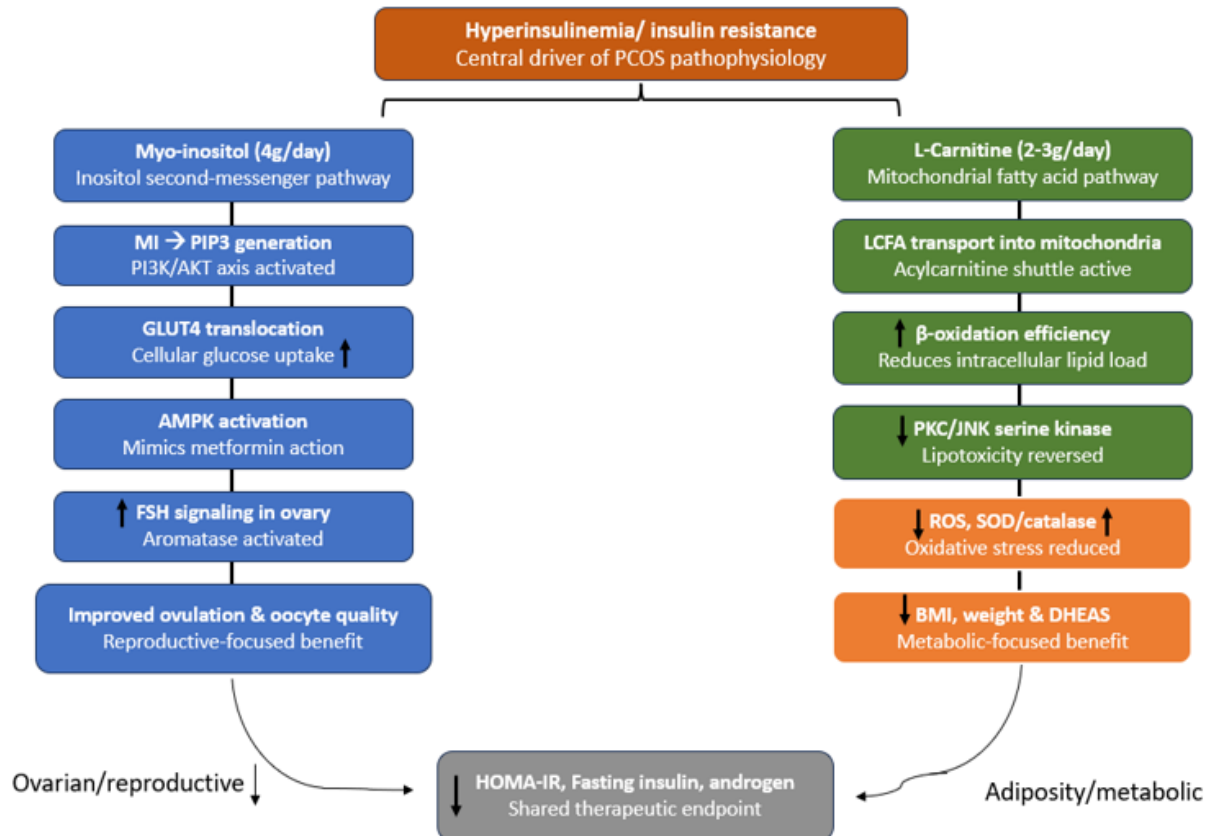


Figure 2: Shows the mechanistic divergence of MI vs L-carnitine converging on the same therapeutic outcome.

insufficient to support absolute recommendations over empirical clinical judgment (Elsayed *et al.*, 2023; Sachdeva *et al.*, 2019). The data most clearly supports MI as the first-line nutraceutical strategy for slim women with PCOS (BMI < 25 kg/m²) who typically present with ovulatory dysfunction, subfertility, and high HOMA-IR without considerable adiposity (Gudović *et al.*, 2024). The standard dose of MI 4 g/day (alone or in a 40:1 ratio with DCI) for a minimum of 3-6 months is recommended, with MI-DCI supplementation initiated at least 3 months before assisted reproduction procedures (Yang *et al.*, 2024).

Given its improved benefits on body weight, BMI, and broader glycaemic indicators, L-carnitine (2,000-3,000 mg/day) may be especially helpful in overweight or obese women with PCOS (BMI ≥ 25 kg/m²) who have considerable insulin resistance,

dyslipidaemia, and weight-related comorbidities (Pakravanfar *et al.*, 2020). Co-administration with MI in this phenotype is a logical approach to concurrently address the lipotoxic component of insulin resistance and the inositol pathway malfunction, and it ought to be investigated further in clinical trials with sufficient power. The addition of L-carnitine to ovulation induction procedures has shown benefits in clinical pregnancy rates for women with PCOS and clomiphene-resistant infertility, and it should be included in institutional algorithms (Hidalgo-Lozada *et al.*, 2024). Combination treatments with metformin are still therapeutically important for women with severe insulin resistance or PCOS-associated metabolic syndrome, where nutraceutical monotherapy may not be adequate (Saadati *et al.*, 2025; Zeng *et al.*, 2025) (Table 2).

Table 1: Rationale for Combination Therapy.

Feature	Myo-inositol (MI)	L-Carnitine	Combined effect	References
Target Pathway	Insulin signaling	Fatty acid metabolism	Multi-level action	(Zhang <i>et al.</i> , 2026)
Mechanism	Second messenger (IPG)	Mitochondrial β -oxidation	Complementary	(Zhang <i>et al.</i> , 2026)
Insulin resistance	Direct effect	Indirect effect	Synergistic improvement	(Clinical TrIAls, 2026)
Reproductive role	Improves ovarian signaling	Supports Mitochondrial function	Better follicular environment	(Clinical TrIAls, 2026)
Metabolic effect	Mild	Strong (weight/BMI)	Balanced benefit	(Gómez-Peralta <i>et al.</i> , 2024)
Evidence	Strong (reproductive)	Strong (metabolic)	Emerging (RCTs ongoing)	(Gómez-Peralta <i>et al.</i> , 2024)

Table 2: Phenotype-Stratified Treatment Selection in PCOS.

Parameter	Lean PCOS (BMI<25)	Overweight/Obese PCOS (BMI≥25)	References
Dominant mechanism	Inositol pathway defect	Lipotoxicity, mitochondrial dysfunction	(Elsayed <i>et al.</i> , 2023; Sachdeva <i>et al.</i> , 2019)
Preferred agent	Myo-inositol (MI)	L-carnitine	(Gudović <i>et al.</i> , 2024)
Key action	Improves insulin signaling (PI3K/AKT, GLUT4)	Enhances β -oxidation; reduces fat accumulation	(Yang <i>et al.</i> , 2024)
Main benefit	Ovulation and cycle regulation	Weight loss and metabolic improvement	(Pakravanfar <i>et al.</i> , 2020)
Best use	Infertility, ovulatory dysfunction	Obesity-associated metabolic PCOS	(Hidalgo-Lozada <i>et al.</i> , 2024)
Strategy	MI±metformin	L-carnitine±MI	(Saadati <i>et al.</i> , 2025; Zeng <i>et al.</i> , 2025)

LIMITATIONS OF THE EVIDENCE BASE AND FUTURE DIRECTIONS

Even while the clinical evidence for MI and L-carnitine in PCOS is increasing, there are still a number of significant methodological issues that restrict the quality of the conclusions that can be made. Small sample sizes (usually 30-100 participants per arm), brief treatment periods (8-24 weeks), considerable variation in diagnostic criteria (Rotterdam versus Androgen Excess Society criteria), baseline BMI, co-interventions (dietary modification and lifestyle counselling), and outcome measurement techniques are characteristics of most individual trials. It is impossible to rule out publication bias favouring favourable findings (Alesi *et al.*, 2022). Direct head-to-head comparative trials between MI and L-carnitine are virtually non-existent, and the existing network meta-analyses, while providing indirect comparative estimates, are subject to the assumptions inherent in network analysis methodology ("American Society for Clinical Pharmacology and Therapeutics," 2026). Neither medication's long-term metabolic effects, such as preventing the development of type 2 diabetes and cardiovascular disease, have been sufficiently assessed in PCOS-specific populations (Harrison *et al.*, 2024).

Future research priorities should include: (1) large-scale, sufficiently powered, multi-center RCTs comparing MI versus L-carnitine versus their combination versus placebo, stratified by phenotypic characteristics and BMI; (2) mechanistic biomarker studies examining inositol phosphoglycan levels, acylcarnitine profiles, and mitochondrial function markers as predictors of differential treatment response; (3) long-term (12-24 month) follow-up studies assessing sustained metabolic and reproductive effects; (4) determining the ideal MI:DCI ratio in women with varying degrees of insulin resistance; and (5) investigating metformin, GLP-1 receptor agonists, and lifestyle interventions in the context of precision medicine approaches to PCOS (Kong *et al.*, 2025).

CONCLUSION

For the treatment of insulin resistance and hyperinsulinemia in PCOS, MI and L-carnitine are both scientifically sound, mechanistically different, and clinically validated treatment alternatives (Houston & Templeman, 2025; Zhang *et al.*, 2025). Because of its essential function in mitochondrial fatty acid oxidation as well as its antioxidant and anti-inflammatory qualities, L-carnitine has wider metabolic effects, most notably on body weight and adiposity, which are especially helpful in the overweight or obese PCOS phenotype (Placidi *et al.*, 2022; Xiang *et al.*, 2025). Their molecular synergy provides a solid basis for combination approaches deserving of more clinical investigation, and the two drugs are complementary rather than competitive. Until high-quality head-to-head comparison trial data are available, treatment decisions should be based on phenotypic

features, metabolic severity, and reproductive priorities (Bakr *et al.*, 2025; Lehár *et al.*, 2009; Roy *et al.*, 2026).

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ABBREVIATIONS

AMH: Anti-Müllerian Hormone; **BMI:** Body Mass Index; **DCI:** D-Chiro-Inositol; **DCI-IPG:** D-Chiro-Inositol Phosphoglycan; **DHEAS:** Dehydroepiandrosterone Sulfate; **ERK:** Extracellular Signal-Regulated Kinase; **FSH:** Follicle-Stimulating Hormone; **GLP-1:** Glucagon-Like Peptide-1; **GLUT4:** Glucose Transporter Type 4; **HbA_{1c}:** Glycated Hemoglobin; **HOMA-IR:** Homeostatic Model Assessment of Insulin Resistance; **IVF:** *In vitro* Fertilization; **ICSI:** Intracytoplasmic Sperm Injection; **IRS-1:** Insulin Receptor Substrate-1; **IPG:** Inositol Phosphoglycan; **LH:** Luteinizing Hormone; **MAPK:** Mitogen-Activated Protein Kinase; **MI:** Myo-Inositol; **MI-IPG:** Myo-Inositol Phosphoglycan; **PCOS:** Polycystic Ovary Syndrome; **PI3K:** Phosphoinositide 3-Kinase; **RCT:** Randomized Controlled Trial; **ROS:** Reactive Oxygen Species; **SHBG:** Sex Hormone-Binding Globulin; **STAR:** Steroidogenic Acute Regulatory Protein; **SUCRA:** Surface Under the Cumulative Ranking Curve.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

AUTHORS CONTRIBUTION

Harini Vinayagam: Collection and/or assembly of data; Data analysis and interpretation; Writing the article. **Ritikasri Venkatraju:** Collection and/or assembly of data; Data analysis and interpretation; Writing the article. **Sneha Raja:** Data analysis and interpretation; Writing the article. **Jayasutha Jayram:** Research concept and design; Critical revision of the article; Final approval of the article. **Sujatha Kuppusamy:** Critical revision of the article; Final approval of the article. **Divya Varshini Natarajan:** Writing the article. **Rajeswari Jagadeesan:** Writing the article, **Janeviennygrace:** Writing the article, Roshini Nivetha: Writing the article, **Sudharshan Antony Raj:** Writing the article, **Nandhakumar Kannan:** Writing the article, Sruthika Natarajan: Writing the article.

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