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Effect of Myo-Inositol in Treating Polycystic Ovary Syndrome (PCOS): A Review

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ABSTRACT

Background: Women of reproductive age are often affected by Polycystic Ovary Syndrome (PCOS), a condition that can cause infertility and metabolic problems. Hormonal changes contribute to PCOS's mechanism. It involves three interrelated symptoms, namely ovulation disorders, androgen excesses, and Polycystic Ovarian Morphology (PCOM), which should all be treated appropriately. Inositol therapy has been shown to play a significant role in PCOS in several studies. Despite this, there is no comprehensive discussion of Myo-Inositol (MI) and D-Chiro-Inositol (DCI) in relation to particular symptoms.

Aim: The purpose of this review is to demonstrate how well Myo-inositol treats PCOS symptoms. Additionally, the study emphasises on evaluating inositol consumption while considering their physiological characteristics and the process by which certain PCOS symptoms arise.

Methods: Using the databases PubMed and Google Scholar, a review of the literature was carried out using one of the following keywords: PCOS, myo-inositol, and insulin resistance.

Results: Multiple research studies have shown that the treatment of MI improved the function of ovaries and fertility in patients with Polycystic Ovarian Syndrome (PCOS), reduced symptoms of hyperandrogenism, including acne and hirsutism, beneficially affected metabolic aspects, and regulated a number of hormonal factors that are deeply connected to the function of the reproductive system and ovulation. Thus, using MI as a treatment has become a breakthrough approach to enhance spontaneous ovulation, stimulate ovulation, or minimise PCOS symptoms.

Conclusion: A physiological ratio of 40:1 between MI and DCI could prove to be advantageous for addressing the metabolic, hormonal, and reproductive components of PCOS, according to the existing clinical evidence.

Keywords:

Polycystic Ovary Syndrome, Inositol (PCOS), Myo-Inositol (MI), D-Chiro-Inositol (DCI), Ovulation, Androgen, Insulin resistance.

Abbreviations:

PCOS: Polycystic Ovary Syndrome; MI: Myo-Inositol; DCI: D-Chiro-Inositol; LH: Luteinizing Hormone; FSH: Follicle-Stimulating Hormone.

Introduction

The cause and management of Polycystic Ovarian Syndrome (PCOS) have drawn a lot of interest from the scientific and medical communities in recent years. Research has shown that the most prevalent cause of infertility in women who are of reproductive age is PCOS [1]. Treatments for patients with ovulation disorders, such as ovarian hippocampal signal path block theory, the theory of leptin, or inositol treatment, can be distinguished among the current treatments for PCOS, as can first-line therapies like lifestyle modification or oral contraceptive pills [2]. Most of these treatments have directional mechanisms of action, meaning that they have a greater effect on certain PCOS symptoms and less on others. The function of Myo-Inositol (MI) and D-Chiro-Inositol (DCI) in the treatment of PCOS-affected women in relation to specific symptoms is covered in detail in this study. Additionally, the author examines the available information about inositol usage while accounting for its physiological characteristics. Therefore, this paper's goal is to illustrate the significance of inositols as well as the process by which each unique PCOS symptom arises. The description, pathophysiology, and general details of PCOS are presented in the first section of the review, along with an overview of inositols and their use in medicine. The next section goes into great length on the pathophysiological basis of the main symptoms of PCOS and how important it is to treat them with MI and DCI.

Polycystic Ovarian Syndrome (PCOS)

In women of reproductive age, Polycystic Ovarian Syndrome (PCOS) affects approximately 6-15% [3-5]. This disorder is associated with menstrual disturbances, hirsutism, and female infertility [4]. Women with PCOS may also suffer from psychological (body image, depression, anxiety) [4,7,8], metabolic (insulin resistance, obesity, prediabetes, metabolic syndrome, type 2 diabetes, cardiovascular risk factors (dyslipidemia, hypertension). Among other complications, endometrial carcinoma, sleep apnea, pregnancy-related complications (gestational diabetes, preeclampsia, pregnancy-induced hypertension, postpartum hemorrhage and infection, preterm births, meconium aspiration, stillbirths, operative deliveries, shoulder dystocia) [9]. PCOS is therefore detrimental to reproductive health, sexual health, and quality of life [5].

The Rotterdam criteria are currently used to diagnose PCOS, which are two out of three of the following:

1. Oligo- or anovulation,
2. Clinical and/or biochemical signs of hyperandrogenism (hirsutism, alopecia, acne), and
3. Polycystic ovaries and exclusion of other etiologies (congenital adrenal hyperplasia, androgen-secreting tumors, Cushing's syndrome) [10].

Based on present evidence, a key component of PCOS is insulin resistance and compensatory hyperinsulinemia.

One of the primary features of PCOS is the development of large polycystic ovaries with a high antral (2-8 mm) follicle number [11]. This characteristic ovarian shape is caused by endocrine abnormalities such as hyperandrogenemia, hypersecretion of LH, and hyperinsulinemia, which hamper follicular development and prematurely stop follicular expansion. In consequence of this, 60-80% of the patients experience anovulatory infertility together with irregular menstruation [12].

Etiology of PCOS

Determining the cause of PCOS remains a challenge even afteryears of research. While research has indicated a significant hereditary component, the PCOS mechanism also has an environmental foundation that is impacted by aspects related to embryogenesis [13].

Telomere shortening has been linked to PCOS pathogenesis and specific genes responsible for PCOS traits have been identified [14], particularly in individuals who are more likely to have metabolic comorbidities.

Environmental factors may be further classified as prenatal factors such as fetal developmental programming and postnatal factors, which are linked to harmful lifestyle choices and environmental contaminants, and prenatal influences, which include things like fetal developmental programming [15]. It is well known that the fetus is more susceptible to the mother's messages throughout pregnancy. Moreover, alterations in the expression of some genes may be linked to a higher risk of metabolic and reproductive problems in the postnatal period [16]. PCOS in humans and animals can be developed by exposure to increased levels of glucocorticoids and/or androgens during key stages of embryonic development [14,17].

It should be understood that continuous endocrinological changes that interact with one another cause PCOS. The function of the Hypothalamic-Pituitary-Gonadal (HPG) and Hypothalamic-Pituitary-Adrenal (HPA) gland axis is crucial to the mechanism of PCOS; disruptions in any one of these hormone levels can result in an excess of androgen or anovulation . Patients with PCOS have an endocrine profile that is characterised by increased levels of oestrogens (mostly oestrone) due to greater plasma concentrations of adrenal and ovarian androgens. In terms of physiology, LH causes ovarian theca cells to convert cholesterol to androstenedione, which is the primary building block of Testosterone (T) and oestrogen production. The stress response hormones Corticotrophin-Releasing Hormone (CRH) and Adrenocorticotropic Hormone (ACTH) increase the synthesis of steroids in the zona reticularis of the adrenal cortex. While adrenal involvement is more clearly shown by the rise of dehydroepiandrosterone sulphate, androstenedione and T have an ovarian origin. An higher frequency of LH pulses and an LH/Follicle-Stimulating Hormone (FSH) ratio are caused by the increased pulsatility of hypothalamic Gonadotropin-Releasing Hormone (GnRH). In PCOS, the stimulation of theca cells by LH to levels exceeding 8-12 mIU/mL resulted in an increase in androgen production and interruption of follicular formation [19]. Moreover, FSH promotes follicular maturation and affects androgen aromatisation. In the preantral and early antral phases, ovarian androgens promote follicle formation; but, in the later antral stages, their increased concentration can cause atresia. These hormonal alterations have been linked to elevated PRL and insulin levels, and they have been seen in people with PCOS who are slim or overweight as well as those who are overweight or obese [20,21]. It is commonly recognised that about 1% to 2% of the steroid hormones in the circulation are physiologically active due to their unbound state. In addition to the more glandular conversion, the decreased Sex Hormone-Binding Globulin (SHBG), which restricts the bioavailability and activity of sex hormones, is the reason for the rise in circulating steroids.

Table 1: Etiology of PCOS- Data of the Columns are adapted from Up to Date [18].

Heritable traits	Intra-uterine environment	Post-natal environment
Maternal Polycystic ovary syndrome	Congenital virilization	Insulin resistance
Polycystic ovary morphology	Disturbed fetal nutrition	Hyperandrogenism
Hyperandrogenemia		
Metabolic syndrome		
Genetic variation		
Epigenetic changes		

On the other hand, a relative surplus of free-circulating androgens is produced when hepatic SHBG production is reduced, allowing elevated androgen levels to self-regulate. Hirsutism may result from this process as well as the stimulation of peripheral conversion for Dihydrotestosterone (DHT) by increased androgen levels [21].

Pharmacotherapy for PCOS

Pharmacological therapy for PCOS women includes oral contraceptives such as progestins, androgens receptor antagonists like spironolactone, progesterone derivatives, and aromatase inhibitors like letrozole [22] and metformin.

Inositol

Inositols are a family of chemical compounds known as cyclohexanols; they are a class of natural polyols (sugars) that

have hydroxyl groups connected to a cyclohexane ring. They are found in food in their natural form in fruits, cereals, beans, and nuts. Inositols are involved in numerous physiological processes, including transmission of signals, osmoregulation, and adult ion channel regulation. They are components of cell membrane phospholipids, plasma lipoproteins, and the phosphate forms in the nucleus [23]. They are essential for healthy foetal development during the first few weeks after giving birth [24].

Depending on where the hydroxyl groups are located, inositol can have nine stereoisomers. Myo-, scyllo-, muco-, neo-, and d-chiro-inositol are the five that arise naturally, whereas l-chiro-, allo-, epi-, and cis-inositol originate from myo-inositol (MI), which is synthesised by living cells [25]. Hexokinase phosphorylates glucose to produce d-Glucose-6-Phosphate (G6P), which is the initial stage of MI production. Next, Myo-Inositol-1L-Phosphate Synthase (MIPS) converts G6P to 1-l-myo-inositol-1-phosphate, which inositol monophosphatase dephosphorylates to produce free MI [26]. The human kidney is the primary site of MI production, and MIPS is the enzyme that limits this process's pace. The results of epimerising the hydroxyl groups of MI at the first and third carbons, respectively, are l-chiro-inositol and D-Chiro-Inositol (DCI). Insulin resistance causes a significant reduction in the activity of an enzyme called epimerase, which is responsible for regulating the levels of stereoisomer [25].

Mechanism of Action

Research has demonstrated that a malfunction in the Inositol Phosphoglycan (IPG) second messenger pathway is the cause of hyperinsulinemia [27]. At the level of the cellular membrane, glycosylphosphatidylinositol lipid hydrolysis produces IPG. These compounds take part in the stimulation of the intracellular pathway that governs glucose metabolism, both oxidative and non-oxidative, as well as glucose absorption by glucose transporter type 4 from the extracellular environment [28]. The normal ratio of DCI to MI varies among tissues, and the activity of an insulin-dependent epimerase that is reduced in insulin-resistant circumstances controls both stereoisomers. Thus, as insulin second messengers, inositols take involvement in several insulin-dependent activities. While DCI is transformed to an IPG insulin second messenger (DCI-IPG) and participates in glycogen formation, MI is converted to an Inositolphosphoglycan (IPG) insulin second messenger (MI-IPG) and is engaged in cellular glucose absorption [29]. While DCI-IPG is involved in insulin-mediated androgen synthesis, MI-IPG is involved in FSH signalling in the ovary. As a result, abnormalities in the ovarian MI-DCI ratio may deteriorate oocyte quality and hinder FSH signalling [30].

Myoinositol (MI) and D-chiroinositol (DCI), the isomers' inositol, are found in large amounts in follicular fluid and the ovaries, and they play distinct roles in follicular formation and insulin signalling. DCI is in charge of insulin-mediated androgen production and has the ability to inhibit aromatase, whilst MI acts as a second messenger by stimulating FSH signalling. The equilibrium between those two isomers sustains regular ovarian activity and hormone output in a healthy ovary. The MI/DCI ratio in follicular fluid is between 100:1 and 40:1 in plasma under normal circumstances [31,32]. Because elevated epimerase activity converts MI to DCI, hyperinsulinemia raises the DCI-to-MI ratio in individuals with PCOS and insulin resistance.31 Even while MI and DCI have similar chemical makeup and work in concert to increase insulin sensitivity, they have distinct effects on the ovary. When it comes to DCI, MI might have the opposite effect on aromatase activity. In this sense, lower MI/DCI ratios increase androgen synthesis in theca cells, whereas higher MI/DCI ratios encourage the action of aromatase in granulosa, raising oestrogen levels [33].

Role of MI in PCOS

Inositols have a number of advantageous effects on hormone control, glucose homeostasis, and follicular growth, all of which support their use as therapeutic agents in PCOS patients. Numerous investigations validate their beneficial impact on metabolic, hormonal, and reproductive disorders in PCOS, either in isolation or in conjunction with other drugs, augmenting their therapeutic effect and bioavailability. However, in contrast to other ovulation-induction therapy choices, MI treatment is safe and has relatively few negative effects. The 2015 International Consensus Conference on MI and DCI in Obstetrics and Gynaecology acknowledges that PCOS pathogenesis involves multiple biological pathways for both MI and DCI, and there is ample clinical evidence suggesting that supplementing with inositols may improve the disorder's metabolic and reproductive aspects [34]. Multiple studies conducted in the last several years have demonstrated their efficacy in PCOS patients [35].

Insulin sensitising, which enhances insulin resistance and is shown in a drop in the Homeostatic Model Assessment (HOMA-IR) index, is a critical effect that MI and DCI have on PCOS patients [36]. Subsequent research shown that MI therapy was beneficial in lowering oxidative, metabolic, and hormonal problems in PCOS patients by enhancing insulin resistance [37].

Other studies [38] that found a significant improvement in plasma insulin levels, glucose-to-insulin ratio, and HOMA index after 12 weeks of treatment along with a decrease in plasma LH, prolactin, testosterone levels, and LH/FSH ratio also supported the beneficial effect of MI on insulin sensitivity. Following combined MI + DCI therapy, improvements in ovulatory function and insulin resistance were also seen [39].

In another trial, MI considerably outperformed metformin in terms of its effects on fasting plasma glucose serum insulin levels, serum triglyceride levels, VLDL cholesterol levels, and the quantitative insulin sensitivity check index. When compared to metformin, MI supplementation also increased the expression of the peroxisome proliferator-activated receptor gamma (PPAR-) gene (p=0.002) [40].

Additionally, some research shows that combining MI with D-chiro-inositol at a physiological plasma ratio of 40:1 reduces LDL cholesterol, triglycerides, and the HOMA-index more effectively than MI alone [41,42]. The degree of hirsutism and the levels of total androgens, FSH, LH, and LDL cholesterol were significantly reduced in individuals with mild and moderate hirsutism after receiving 2g MI twice daily for six months [43].

According to certain studies, patients with PCOS who received MI treatment had increased ovarian function and fertility [44-55] reduced hyperandrogenism, acne, and hirsutism [43,48,56] positively impacted metabolic parameters, and modulated various hormonal parameters deeply involved in the function

of the reproductive axis and ovulation [49,57]. As a result, MI treatment became a novel way to improve ovulation induction [50-52] or spontaneous ovulation [45,58].

During the six months of MI therapy, there was a positive impact on the restoration and maintenance of a regular menstrual cycle, according to a research by Papaleo [45]. Despite the identical effects of both therapies on weight, BMI, waist and hip circumferences, the combination of MI and metformin demonstrated a greater effect on the menstrual cycle than metformin alone [59].

Clinical evidence has shown that MI and DCI effectively improved the metabolic and reproductive features of PCOS due to their insulin-sensitizing impact [34,60]. Research has demonstrated that FSH activity and glucose cellular absorption are enhanced by MI, and that insulin-induced overproduction of androgens in the ovary is facilitated by DCI, which is essential for glycogen synthesis [61,62]. Because of this, tissues that store glycogen, including fat, muscle, and liver, have high DCI levels, but tissues that require a lot of energy, like the brain and heart, have low concentrations [63]. It is important to emphasise that in these systems, the DCI level is always lower than the MI. DCI has decreased the amount of circulating insulin and androgens and returned normal insulin sensitivity in the usual insulin target tissues at low dosages [39]. Both ovulation frequency and ovarian cell activity are enhanced by these modifications. MI mostly affects insulin metabolism at the ovarian level, where it is concentrated. MI can therefore directly affect ovarian processes, such as steroidogenesis [64].

The effect of myo-inositol on androgrens

In theory, PCOS is a group of symptoms resulting from an overabundance of androgens. Features including hirsutism, acne, and female-pattern alopecia are clinical manifestations of hyperandrogenism, or high androgen levels. The production and release of androgen by theca or zona reticularis cells is also linked to hyperandrogenism [65]. Nonetheless, increased T and insulin concentrations in peripheral circulation, together with insulin resistance in ovarian tissue, are indicative of hyperandrogenemia in PCOS patients. There are two distinct methods that work in tandem to indirectly raise the amounts of free androgen in the blood, particularly T. Insulin resistance and high insulin concentrations cause the androgens to be secreted from the adrenal gland and ovaries, while the hepatic synthesis of SHBG is inhibited [66]. Premature follicular atresia, persistent anovulation, and gonadotropin imbalance which was demonstrated by elevated LH and decreased FSH levels, as well as an LH:FSH ratio more than 2.5 can result from these hormonal shifts [67].

Following the administration of MI [38,54], DCI [68], or both [39], a drop in the free T (fT) level was seen, which may indicate modifications to the menstrual cycle and fertility. The use of both stereoisomers in combination to treat PCOS symptoms has been the subject of a fair amount of study. Research by Januszewski et al. revealed a 10:1 ratio of substantial increases in SHBG plasma concentration and decreases in fT, FSH, and LH when compared to the levels before to MI and DCI administration. During the course of the three-month therapy, these hormonal alterations were linked to an improvement in skin condition [31]. Nonetheless, the optimal option for inositol treatment appears to be the concurrent delivery of both substances at a physiological plasma ratio of 40:1. It has been noted that obese women with PCOS who received a 40:1 ratio of MI and DCI showed improvements in their endocrine profiles in comparison to those who received a placebo. Furthermore, in response to the combination, the concentrations of fT and LH were lowered while those of oestradiol (E2) and SHBG were raised [31].

The effect of myo-inositol in ovulation disorders and infertility

It is widely accepted that MI administration has had a significant impact in raising the success rate of assisted reproduction procedures in PCOS [33,69]. Studies utilising folic acid and MI given to infertile women over a period of two to three months have produced data. Better embryo quality and a higher rate of fertilization/pregnancy roughly 15% of all the women in the study were the outcomes of this treatment. Even when the patients were given a dose of 4000 mg MI per day, no notable negative effects were reported [44]. Thus, in many PCOS-affected women, inositol therapy can aid in restoring spontaneous ovarian activity, including menstrual cyclicity and spontaneous ovulation, and subsequently, fertility. Additionally, it has been proposed that MI excess in the ovary raises FSH sensitivity and enhances PCOS-affected women's fertility and embryo quality [70]. The outcomes demonstrated a lower quantity of retrieved oocytes in the MI group and a decreased likelihood of hyperstimulation syndrome, confirming the benefits of using the MI to enhance IVF procedures for PCOS patients [44].

Women with increased HDL cholesterol concentrations and insulin sensitivity saw an approximately two-fold increase in ovulation instances following the administration of DCI or MI [71]. In obese women with PCOS, treatment with MI at a dosage of 2 g per day led to a drop in the LH:FSH ratio and metabolic parameters [45,64] Nevertheless, in contrast to one-fold MI, recent research has demonstrated that both MI and DCI, at a normal plasma ratio of 40:1 can restore the hormonal characteristics sooner. The particular actions of these substances in a combination therapy are explained by the roles played by DCI in reducing peripheral hyperinsulinemia and MI in improving ovulatory function. Nordio and Proietti [41] examined the benefits of ovarian function improvement between MI monotherapy and combination MI and DCI treatment. In PCOS-affected individuals, ovarian function was positively impacted by both MI and DCI. Following DCI treatment, there have been positive effects on ovulation and metabolic parameters in PCOS by increasing insulin sensitivity [72]. Comparing the combined treatment to therapy utilising just DCI, Colazingari et al. [73] demonstrated a clear benefit of the ratio of MI and DCI of 40:1 on the quality of the oocyte. In order to increase the quality of the oocytes and embryos being transferred, as well as the chances of pregnancy, women with PCOS who are having IVF with embryo transfer should choose for combination treatment [74].

The effect of myo-inositol in polycystic ovarian morphology

It was advised that while evaluating the ovaries, care should be

taken to note how they appeared sonographically, specifically the number of antral follicles in each ovary [75]. Each ovary has 12 or more follicles with a diameter of 2 to 9 mm when PCOS is present. Additionally, elevated AMH concentration and ovarian volume which are often elevated in PCOS are diagnostically significant. The granulosa cells of the ovary's preantral and tiny antral follicles generate Antimicrobial Hormone (AMH). Granulosa cells in PCOS-affected women produce AMH at much greater amounts than in both healthy ovulatory women and ovulatory women whose ovaries are polycystic [76]. An enhanced conversion of MI to DCI and a decrease in MI in the follicles were the ovarian phenotypes observed in women with PCOS [61]. Along with the existence of developed and fertilised follicular oocytes, there was a positive connection found between the amount of follicular fluid and its MI concentration [70]. As a result, MI monotherapy has greatly impacted the quantity of follicular fluid in a fertilised egg or the number of normal mature follicles in a patient with PCOS, as well as the quality of the oocyte and embryo [41,39]. The concurrent use of MI and DCI throughout therapy had the greatest clinical outcomes [77]. Data collected after mice were exposed to continuous light revealed developed ovaries with morphological characteristics common to PCOS in humans as well as decreased gonad activity. The scientists detected a high ratio of theca/granulosa cell layer thickness (TGR) in mice as an indicator of PCOS. In these mice, a decrease in reproductive capacity was associated linearly with an increase in TGR. Treatment of the animals with 40:1 ratios of MI and DCI therefore resulted in full healing of the ovaries from PCOS symptoms to normal histological characteristics, appropriate TGR ratio, and reproductive restoration [78].

The effect of myo-inositol in metabolic abnormalities

Among PCOS patients, compensatory hyperinsulinemia and insulin resistance are often noted dysfunctions [63]. According to estimates, 95% of obese people have insulin resistance, compared to 60–80% of PCOS patients who have the same condition [19]. Anovulation, early follicular atresia, gonadotropin imbalance, and hyperandrogenism were the results of both illnesses' stimulation of androgen secretion and suppression of SHBG synthesis [38]. Numerous cardiometabolic illnesses, including obesity, poor glucose tolerance, type 2 diabetes, dyslipidaemia, hypertension, metabolic syndrome, and cardiovascular disease, have been linked to insulin resistance [80]. For overweight PCOS patients, lifestyle changes such as exercise and nutrition management appear to be crucial and have to be the first line of therapy [81]. It is even more significant to note that endocrine alterations in the regulation of adipokines, which take place during weight gain and the development of inflammation in the adipose tissue, may be the cause of increased ovarian androgen production in addition to other symptoms [82]. It has been shown that altering one's lifestyle in addition to receiving medication therapy improves insulin resistance, menstruation problems, and other symptoms of PCOS in addition to lowering body mass index [83]. After four months of treatment, Nybacka et al. [84] found that the delivery of food and exercise reduced T and raised SHBG levels. Moreover, menstrual cycle restoration was reported by 69% of women. Programs for lifestyle adjustment have been demonstrated in systematic reviews and meta-analyses to lower insulin and glucose levels, which may have a positive impact on overweight or obese women receiving PCOS therapy [85].

It has been discovered that isothiols have a favourable impact on the metabolic parameters in PCOS-affected women due to their insulin-sensitizing action. Insulin resistance in PCOS may be caused by reduced tissue availability or changed inositol or IPG metabolism, particularly the IPG insulin second messenger route [86]. MI-IPG and DCI-IPG have a role in the activation of enzymes that regulate the metabolism of glucose. Because of the improved cellular response to the metabolic pathways, inositol treatment is therefore a suitable alternative in terms of the metabolic parameters [72].

Additional research has demonstrated that the combination of DCI and MI treatment improved the metabolic profile of PCOS-affected women. Following MI and DCI therapy, the levels of TG, HDL, and LDL were altered in a physiological ratio of 40:1 [42]. When glucose was given orally to obese PCOS women, the administration of DCI also reduced their insulin response [67]. There have been improvements in blood pressure, plasma lipid content, and insulin resistance [68]. After six months of therapy, decreases in body weight and insulin levels were seen in the PCOS patients, who were diagnosed based on the Rotterdam criteria [31]. When combined in a 40:1 ratio, the MI and DCI considerably decreased fasting insulin, which is useful in determining insulin sensitivity in young, obese PCOS-affected women [64]. Patients with PCOS may benefit from MI's recently identified insulin-sensitizing action in preventing Gestational Diabetes Mellitus (GDM) [87].

Conclusion

PCOS is a multifactorial condition that involves a combination of heredity, genetic factors and various environmental influences factors throughout pregnancy and adulthood. The beneficial effects of inositols in treating certain PCOS symptoms has been explored in this literature review. Results for every symptom taken into consideration were most effective when both MI and DCI were administered in a 40:1 ratio. In order to effectively lower the metabolic indices and induce a clinical change of PCOS, this combination therapy need to be employed as the initial line of treatment for overweight people with PCOS. Researchers have noted that the effectiveness of this combination inositol treatment depends on how well both substances work that is, how much MI improves ovulatory function and DCI reduces peripheral hyperinsulinemia. thus, this strategy improves insulin resistance, the endocrine profile, and lowers the risk of metabolic syndrome. Using inositol appears to be a suitable option among the many alternative treatment techniques available for women with PCOS, since it has a high efficacy and comparatively low adverse effects.

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