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Myo-Inositol Effects on Pregnancy Outcomes and Fetal Growth

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Abstract

Myo-inositol (MI), a naturally occurring carbocyclic sugar and second messenger in insulin signaling, has emerged as a supplement of interest in pregnancy due to its potential metabolic and reproductive effects. This review summarizes current evidence on MI supplementation during pregnancy, with special emphasis on maternal outcomes (e.g., gestational diabetes mellitus [GDM], hypertensive disorders), neonatal outcomes like preterm birth, birthweight, and implications for fetal growth.

Keywords: Myo-Inositol, GDM, Fetal growth, Diabetes

Introduction

Myo-inositol was first isolated in 1850 by Johannes Joseph Scherer from muscle extracts, which inspired its name—myo being derived from "muscle." This early isolation identified myo-inositol as a sugar-like polyol distinct from common carbohydrates such as glucose

Subsequent work by Maquenne in 1887 greatly advanced the understanding of its structure. Maquenne purified myo-inositol from leaves and later from horse urine, establishing the molecule's cyclohexanol ring and physical properties, contrasting its inert chemical behavior with reducing sugars[1].Mid-20th-century research revealed that myo-inositol serves as the structural core for inositol phosphates and phospholipids, including key signaling molecules such as phosphatidyl-inositol and its polyphosphorylated derivatives. Subsequent biochemistry, including landmark reviews of the phosphoinositide cycle, underscored the central role of myo-inositol-derived lipids in cell signaling, membrane dynamics, and hormone action.[2]

Mechanism of action

At the biochemical level, myo-inositol forms the structural basis of inositol phospholipids and phosphates, which serve as key second messengers in multiple signal transduction pathways. Inositol derivatives such as phosphatidylinositol-4,5-bisphosphate (PIP_2) are hydrolyzed by phospholipase C into inositol-1,4,5-trisphosphate (IP_3) and diacylglycerol, both of which are critical mediators of intracellular calcium release and protein kinase activation. This phosphatidylinositol signalling cycle underpins hormone action and cellular responsiveness to external signals[3].Furthermore, myo-inositol metabolites participate in a complex network of inositol phosphates (IP_3, IP_4, IP_5, IP_6) that regulate metabolic and proliferative pathways[4]. A key clinical mechanism of MI is its insulin-sensitizing effect. MI enhances insulin signal transduction by improving post-receptor pathways, partly through activation of AMP-activated protein kinase (AMPK) and increased GLUT-4 translocation, thereby promoting cellular glucose uptake and improving insulin resistance[5].In reproductive tissues, MI supports FSH-mediated ovarian signaling, steroidogenesis, and oocyte maturation by regulating intracellular kinase pathways, including Akt and ERK signaling, which are critical for follicular development and reproductive competence [6].

Pharmacokinetics

Myo-inositol is readily absorbed after oral administration, mainly via sodium-dependent inositol transporters (SMIT1/SMIT2) in the small intestine, with peak plasma concentrations typically achieved within 1–3 hours. Its absorption may be partially saturable and

competitively inhibited by glucose. Once absorbed, myo-inositol is widely distributed in total body water and actively taken up into tissues through specific transporters, where it serves as a precursor for phosphatidylinositol and inositol phosphates involved in intracellular signaling. Endogenous synthesis, particularly in the kidneys, also contributes to circulating levels. Metabolism primarily involves incorporation into phosphoinositides and conversion to D-chiro-inositol via an insulin-dependent epimerase. Elimination occurs predominantly through renal excretion. Pharmacokinetic studies, mainly from intravenous administration in preterm infants, describe a one-compartment model with a volume of distribution of approximately 0.5–0.7 L/kg and an elimination half-life of about 5–8 hours; detailed oral pharmacokinetic parameters in adults remain limited but support rapid absorption and renal clearance [7,8,9].

Introduction to Myo-Inositol in Pregnancy

Myo-inositol is involved in several intracellular signaling pathways, particularly insulin signal transduction, making it biologically relevant in pregnancy where insulin resistance naturally increases to support fetal nutrient transfer. Dysfunctional glucose metabolism in pregnancy can lead to GDM, a condition associated with adverse outcomes for both mother and fetus. These pathophysiological links have catalyzed research into MI's potential benefits in pregnant populations [10].

Myo-Inositol and Gestational Diabetes Prevention

Multiple systematic reviews and meta-analyses of randomized controlled trials indicate that supplementation with myo-inositol, especially at doses around four gram per day started in early pregnancy, is associated with a significantly lower incidence of GDM compared with placebo or no treatment, along with improved glucose tolerance test results (e.g., lower fasting, one hour, and two hour OGTT values), and reduced need for insulin therapy and some adverse pregnancy outcomes such as preterm delivery and neonatal hypoglycemia. In one meta-analysis including seven RCTs and over 1300 participants, daily four grams of myo-inositol reduced GDM incidence markedly (RR ≈ 0.30) and significantly improved OGTT glucose values compared with controls. Another meta-analysis similarly found that myo-inositol lowered the risk of GDM and preterm delivery, though not all secondary outcomes, like birth weight, were consistently affected. Analyses focused on high-risk groups (e.g., overweight/obese women) also support a preventive effect on GDM and related metabolic outcomes. However, the overall certainty of evidence is often rated low to moderate due to heterogeneity in study designs, doses, timing, and populations, and larger, well-powered trials are needed to confirm these findings and establish clinical guidelines [11,12].

Myo-Inositol and Pregnancy Outcomes in Hypertensive Disorders and Preterm Birth

Systematic reviews and meta-analyses of randomized controlled trials suggest that myo-inositol, particularly when started early in high-risk women, is linked to a lower incidence of gestational hypertension and a modest reduction in preterm birth, likely mediated through improved insulin sensitivity and endothelial function. However, evidence remains limited and heterogeneous, and current data are insufficient to recommend routine use specifically for the prevention of hypertensive disorders or preterm birth outside selected high-risk populations[13].

Fetal Growth and Neonatal Outcomes

Direct clinical evidence on myo-inositol's role specifically in preventing or treating fetal growth restriction (FGR) is very limited. Metabolic studies have explored inositol levels in growth-restricted fetuses, showing that brain myo-inositol ratios (e.g., myo-inositol: choline and myo-inositol: creatine) do not differ significantly between appropriately grown and growth-restricted fetuses, suggesting no major change in myo-inositol cerebral metabolism in FGR under preserved cerebral blood flow conditions. However, this research does not evaluate supplementation effects on fetal growth outcomes[14,15,16].

Myo-inositol has been widely studied as an insulin-sensitizing supplement in women with PCOS to improve ovulation and fertility, but its benefits during pregnancy in this population are not clearly established. A recent large, double-blind, randomized controlled trial in pregnant women with PCOS (2 g myo-inositol twice daily vs placebo from early pregnancy) found no significant reduction in a composite of gestational diabetes, preeclampsia, or preterm birth with myo-inositol supplementation versus placebo, indicating it did not improve major pregnancy outcomes in PCOS[17].{Citation}

Safety and Tolerability

MI supplementation appears well-tolerated in pregnancy, with no serious adverse events consistently reported. Safety assessment of inositol shows mild GI side effects possible at higher doses, with standard pregnancy doses generally well tolerated. However, dosing regimens vary widely across studies, and recommendations for routine use cannot be made until larger, high-quality trials clarify optimal dosing and long-term safety in diverse populations[8].

Conclusion

In summary, current evidence suggests that myo-inositol supplementation during pregnancy may reduce the incidence of GDM and possibly hypertensive disorders and preterm birth, particularly in high-risk populations. However, effects on fetal growth (birthweight, macrosomia) and broader neonatal outcomes remain uncertain. Discrepancies in results, especially those observed in specific subgroups like women with PCOS, underscore the need for larger, well-designed RCTs to guide clinical practice.

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