Medical Science

To Cite:
Rusak-Sobolewska A, Krych G. Inositol and its clinical influence on a human metabolism – A literature review. *Medical Science* 2026; 30: e165ms3964
doi:

Authors' Affiliation:
[1]The Nicolaus Copernicus Municipal Polyclinical Hospital in Olsztyn, ul. Niepodległości 44, 10-045 Olsztyn, Poland
[2]Provincial Complex Hospital named after Jędrzej Śniadecki in Białystok, ul. M. Curie-Skłodowskiej 26, 15-950 Białystok, Poland

ORCID List:
Aleksandra Rusak-Sobolewska 0009-0006-7366-3368
Gabriela Krych 0009-0005-6873-5358

***Corresponding author:**
Aleksandra Rusak-Sobolewska,
The Nicolaus Copernicus Municipal Polyclinical Hospital in Olsztyn, ul. Niepodległości 44, 10-045 Olsztyn, Poland,
ORCID: 0009-0006-7366-3368,
E-mail: olarusak008@gmail.com

Peer-Review History
Received: 16 February 2026
Reviewed & Revised: 09/March/2026 to 03/August/2026
Accepted: 16 August 2026
Published: 25 August 2026

Peer-review Method
External peer-review was done through double-blind method.

Medical Science
pISSN 2321–7359; eISSN 2321–7367

DISCOVERY SCIENTIFIC SOCIETY

Inositol and its clinical influence on a human metabolism – A literature review

Aleksandra Rusak-Sobolewska[1]*, Gabriela Krych[2]

ABSTRACT

Inositol is a cyclohexane-hexol carbohydrate present in nine stereoisomeric forms, best known as myo-inositol (MI) and D-chiro-inositol (DCI), with their phosphorylated derivatives - inositol hexakisphosphate (InsP6) and the inositol pyrophosphates. These molecules are membrane phospholipid components and intracellular second messengers that participate in insulin signaling, calcium regulation, DNA repair, and cellular energy transduction. Because of this function, inositol supplementation has been investigated across a broad range of metabolic and endocrine conditions. This review reveals current evidence on inositol's biochemistry and its clinical influence on human metabolism. MI and DCI act as insulin sensitizers by promoting GLUT4 translocation and glucose uptake; on the other hand inositol pyrophosphates regulate adipocyte thermogenesis, lipid handling, and insulin exocytosis in preclinical models. Meta-analysis pragmatically shows that the use of MI supplements is effective in lowering BMI conservatively, while it is very efficient in reducing the incidence of gestational diabetes mellitus and fasting insulin levels in PCOS patients. The evidence in non-alcoholic fatty liver disease is so far based on only one study in humans. By contrast, other reviews showed only low-quality evidence that MI improves live birth or clinical pregnancy rates in subfertile women with PCOS, and applications in thyroid disease, psychiatric disorders, and cancer chemoprevention remain preliminary. MI is, for the most part, well tolerated with mild gastrointestinal effects as the most common adverse event. Inositol shows the strongest support for glycemic outcomes in insulin-resistance. Its role in reproductive and other extra-metabolic applications requires further high-quality trials.

Keywords: myo-inositol, inositol phosphates, PCOS, gestational diabetes mellitus, human metabolism

1. INTRODUCTION

Inositol is a naturally occurring cyclohexane-hexol carbohydrate, structurally related to glucose, that exists in nine possible stereoisomeric forms generated by epimerization of its six hydroxyl groups (Chatree et al., 2020). Of these, myo-inositol (MI) is overwhelmingly the most abundant isomer in plants, animals, and human tissue, while D-chiro-inositol (DCI) — formed from MI through insulin-dependent epimerization of a single hydroxyl position — is the second most clinically studied form (Weinberg et al., 2021). Beyond its role as a free molecule, Inositol is a divided

structure of molecules, not only a free molecule: the phosphorylated inositols, or inositol phosphates (IPs). These range from mono-phosphorylated forms through inositol hexakisphosphate (InsP6) and the energetic inositol pyrophosphates (InsP7 and InsP8), which carry high-energy phosphoanhydride bonds (Tu-Sekine and Kim, 2022). Bringing it all together, inositol and its phosphates participate in an unusually broad range of cellular processes: they serve as precursors of second messengers in calcium signaling, function as cofactors in DNA damage repair and chromatin remodeling, regulate nuclear mRNA export, and act as central nodes in the intracellular relay of the insulin signal (Chatree et al., 2020). This mechanistic breadth has motivated an unusually wide clinical research program, spanning metabolic syndrome and diabetes, polycystic ovary syndrome (PCOS), non-alcoholic fatty liver disease (NAFLD), obesity, thyroid disease, mood disorders, and cancer chemoprevention.

The objective of this paper is to emphasize the current understanding of inositol's metabolism and its molecular mechanisms of action influence on human metabolic regulation.

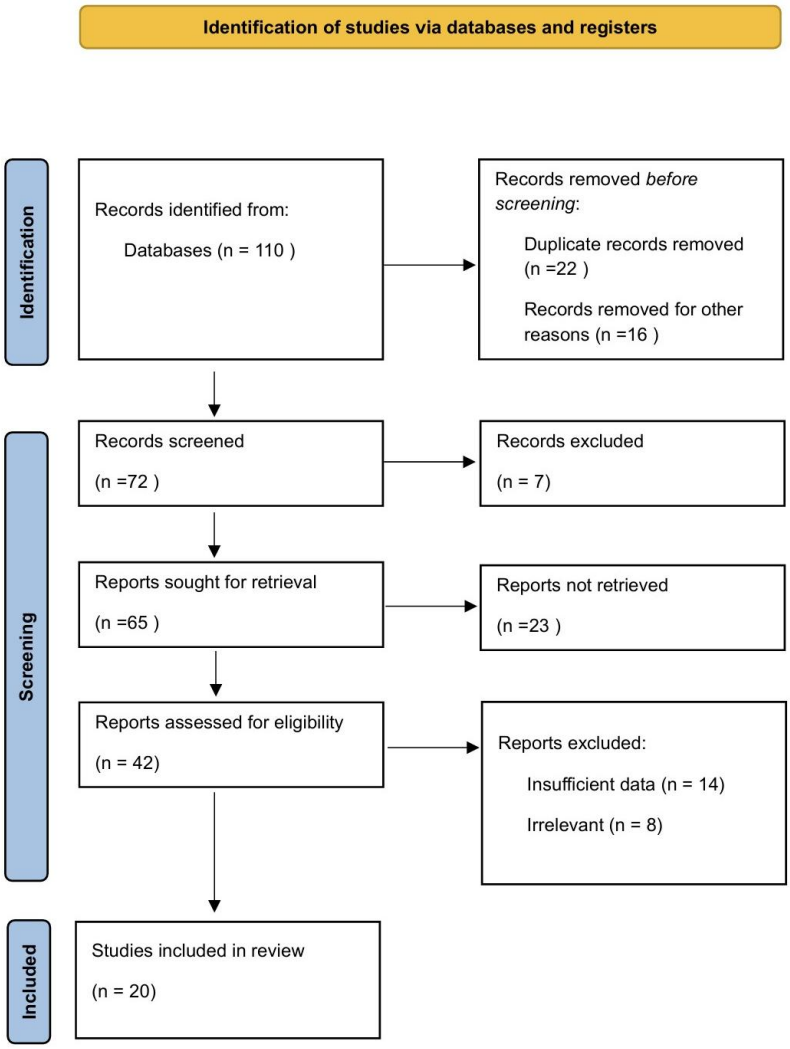

Figure 1. PRISMA CONSORT chart

2. REVIEW METHODS

This research was done through an extensive literature review of available scientific literature from several credible databases like PubMed, Mendeley, Google Scholar, and WHO guidelines. Some of the most important search terms used during the literature review include "myo-inositol," "PCOS," "inositol phosphates," "inositol safety," and "inositol characteristics." Only those articles written in English and available online were considered for this literature review, and the emphasis has been on post-2015 studies to have an up-to-date understanding of the latest clinically relevant research. Older literature was also taken into consideration for historical reasons

and to give basic knowledge about the topic. This paper is written by following the PRISMA guidelines for literature reviews (Figure 1). We aim to highlight the potential implications of inositol's effects on metabolic processes.

3. RESULTS AND DISCUSSION

Chemistry, Dietary Sources, and Metabolism

Myo-inositol can be synthesized endogenously through a two-step pathway in the kidney, in which glucose-6-phosphate is converted to myo-inositol-1-phosphate and subsequently dephosphorylated to free myo-inositol (Schneider, 2015). It is also obtained from the diet, with particularly high concentrations in legumes, whole grains, nuts, seeds, and certain fruits; nearly all ingested myo-inositol is absorbed from the gastrointestinal tract (Weinberg et al., 2021). Its phosphorylated storage form, InsP6, is the principal reservoir of phosphorus in plant seeds and bran and is itself absorbed and distributed to tissues, including across the blood–brain barrier Regarding the intracellular process, phospholipase C catalyzes phosphatidylinositol bisphosphate, giving rise to inositol trisphosphate that becomes phosphorylated to InsP6 and ultimately to energy-rich inositol pyrophosphates (InsP7, InsP8). The phosphate molecules control phosphate metabolism, chromatin remodeling, mRNA export, and energy metabolism (Chatree et al., 2020). Deficiencies of inositol availability, both due to nutrition and environmental factors, have been mechanistically connected to the onset of metabolic and cancerous diseases, as a Western-style diet with low content of fiber provides relatively small amounts of inositol and InsP6; hyperglycemia has been shown to accelerate inositol catabolism and elimination, while hindering its internal production and absorption.

Molecular Mechanisms in Metabolic Regulation

Myo-inositol and DCI act to improve insulin sensitivity largely through their role as precursors of inositolphosphoglycan mediators that propagate the intracellular insulin signal, by glucose transporter type 4 (GLUT4) translocation to the cell membrane and thereby facilitating cellular glucose uptake (Wei et al., 2022). At the cellular level, the pyrophosphate InsP7 has been shown to inhibit Akt signaling, which in animal models reduces body weight and fat mass, improves glucose tolerance and lipid profiles, and enhances the browning of white adipose tissue with a corresponding rise in energy expenditure. Inositol pyrophosphates are also involved in the regulation of insulin exocytosis and endocytosis in pancreatic beta cells as well as regulation of phosphate secretion via interaction with the XPR1 transport system (Nagpal et al., 2024). All these mechanistic studies have established the role of inositol phosphate pathway in regulating insulin sensitivity, fat cell thermogenesis, and lipids, although it is important to note that the molecular pathways by which the inositol pyrophosphates affect insulin resistance are not yet known completely (Tu-Sekine and Kim, 2022).

Insulin Resistance and Glycemic Metabolism

A meta-analysis from 2017 has shown inositol's positive effect on insulin resistance and found consistent improvements in fasting insulin and the homeostatic model assessment of insulin resistance (HOMA-IR) index across clinical populations (Unfer et al., 2017). Conclusions seemed to have practical correlates: myo-inositol has been reported to reduce jejunal glucose absorption and increase peripheral glucose uptake in animal models. It also to lowered post-load insulin levels in obese human subjects, indicating enhanced insulin sensitivity rather than simply lower baseline glucose (Schneider et al., 2015).

The findings from studies involving deletion of IP6K1 in animal models demonstrate the development of improvements in glucose tolerance with insulin resistance markers for muscle and liver; nevertheless, the reviews always emphasize that the application of this pathway of pyrophosphates to human physiology is not yet demonstrated with the certainty of myo-inositol pathway (Miñambres et al., 2018). A further layer of mechanistic complexity relevant to metabolic disease concerns the concept of "inositol resistance," in which cells become progressively less responsive to a given tissue concentration of inositol in a manner that parallels classical insulin resistance; reviewers of this literature have proposed that such acquired resistance, compounded by the reduced synthesis and increased clearance of inositol seen under hyperglycemia, may help explain why some insulin-resistant patients show a more robust clinical response to supplementation than others, although this remains a working hypothesis rather than an proven clinical entity (Lepore et al., 2021).

Gestational Diabetes Mellitus

A meta-analysis of seven randomized trials (1321 participants) found that consuming 4 g of myo-inositol daily showed significant reduction in the incidence of gestational diabetes mellitus (GDM) by approximately 70% relative to control. It was also noticed an

improvement in one-hour, and two-hour glucose on oral glucose tolerance testing, alongside reduced need for insulin therapy and lower rates of preterm delivery and neonatal hypoglycemia (D'Anna et al., 2015). It is worth noting that when a combination of 1.1 g myo-inositol and 27.6 mg DCI was used in a lower dosage, no statistical significance was obtained in any outcome measure (Wei et al., 2022). A broader review of inositol and antioxidant supplementation in pregnancy reached a complementary statement - characterizing inositol's insulin-sensitizing, anti-inflammatory, and antioxidant properties as well suited to counteracting pregnancy-related insulin resistance, while noting that diet, lifestyle, and insulin remain the only established first-line therapies for diabetes in pregnancy (Formoso et al., 2019). Myo-inositol stands as a promising adjunctive strategy for GDM prevention, but not a replacement for standard obstetric glucose treatment.

Obesity and Body Composition

In a meta-analysis of 15 trials (891 participants, mainly female in PCOS, metabolic syndrome, and type 1 diabetes), it was observed that inositol had a statistically insignificant decrease in BMI. Among the inositols, myo-inositol had the strongest effect compared to pinitol and other inositol forms. The effect was clinically modest, which is why the authors characterized inositol as an adjunct to standard weight management rather than a stand-alone intervention. Proposed mechanisms include increased energy expenditure and adipocyte thermogenesis, reduced fatty acid synthase activity, and — in IP6K1-deletion animal models — increased oxygen consumption and thermogenic gene expression independent of caloric intake (Zarezadeh et al., 2022).

Non-Alcoholic Fatty Liver Disease

A systematic review of NAFLD identified ten animal studies of myo-inositol or pinitol deficiency or supplementation alongside a single human randomized trial. Across the animal literature, inositol deficiency was consistently associated with increased hepatic fat accumulation, and the one human trial reported favorable effects of supplementation on liver-related and metabolic markers (Pani et al., 2020). The reviewers were explicit that this conclusion rests on a very limited human evidence base and called for larger, more rigorous trials — a gap between a strong preclinical signal and a sparse clinical one that recurs across several of inositol's proposed metabolic applications.

Polycystic Ovary Syndrome: Metabolic and Reproductive Outcomes

The most extensive investigation of inositol was done about its impact on PCOS, reflecting the syndrome's combination of insulin resistance, hyperandrogenism, and ovulatory dysfunction, and myo-inositol's dual role as insulin sensitizer and mediator of follicle-stimulating hormone signaling (Greff et al., 2023). A meta-analysis of nine randomized trials (247 women) found significant decreases in fasting insulin and HOMA-IR following myo-inositol supplementation, alone or with DCI (Unfer et al., 2017). Alterations in the follicular fluid MI:DCI ratio have themselves been proposed as part of PCOS pathophysiology, with hyperinsulinemia enhancing MI-to-DCI conversion and disturbing the normal balance between the isomers. The picture is considerably less favorable for reproductive endpoints. A Cochrane review of 13 trials (1472 subfertile women with PCOS) was done when comparing myo-inositol against placebo - including standard treatment, melatonin, metformin, clomiphene citrate, or DCI as pre-treatment to IVF or during ovulation induction.

The evidence was rated as low to very low quality across nearly all comparisons, citing poor reporting, inconsistency, and frequent failure to report live birth or adverse events. The reviewers concluded they remained uncertain whether myo-inositol improves live birth or clinical pregnancy rates compared with standard treatment, and that no pooled evidence existed for several common comparisons or for ovulation induction at all (Showell et al., 2018). This divergence — reassuring evidence for metabolic markers, weak evidence for reproductive outcomes — is one of the more clinically important nuances in the PCOS literature, and patients should be counseled that a metabolic benefit does not necessarily translate into a reproductive one. While evaluating the mechanisms behind insulin and inositol resistance in patients with PCOS, researchers pointed out that one reason for the lack of success in translating metabolic benefits into improvements in oocyte quality and ovulation could be an inadequate transformation of MI into DCI in the ovary or an unbalanced MI:DCI ratio in the follicles, which has been the rationale for various trials on MI/DCI combinations with the same ratio as found in physiological ovarian conditions (Lepore et al., 2021).

Thyroid Physiology and Psychiatric Applications

Myo-inositol is known to be a precursor of the phosphoinositide pool, which is hydrolyzed downstream of TSH receptor activation, participating in thyroid hormone signaling. A review studied this physiology and proposed that inositol combined with selenium may be relevant to managing subclinical hypothyroidism (Nordio et al., 2017). But the argument is mostly based on physiological reasoning rather than controlled trials (Benvenga et al., 2021).

The research on inositol in psychiatry goes back to the discovery of the phosphatidylinositol pathway which plays an important role in the mechanisms of action of mood stabilizers. This topic was comprehensively reviewed in 2023. However, it was concluded that inositol, whether as monotherapy or adjunct, did not meaningfully has a clinical impact in mood or psychotic disorders. In any case, the use of inositol, both alone and in combination, did not have any significant effect on either mood or psychotic disorders. In this regard, inositol showed a more promising preliminary result regarding the treatment of panic disorder (Concerto et al., 2023). Both examples show the trend present in other research on inositol – plausible biological rationale but poor clinical translation.

Cancer Chemoprevention

A review discussing colitis-associated colorectal cancers reviewed data showing reduced incidence, multiplicity, and volume of tumors by myo-inositol and InsP6. Myo-inositol and InsP6 were used in mice with colitis, resulting in a reduction in biomarkers of nitro-oxidative stress and infiltration of macrophages (Weinberg et al., 2021). Mechanisms for their functions have been suggested to involve the antioxidative function of InsP6 through metal ion chelation, induction of DNA repair through Ku protein and DNA-dependent protein kinase, inhibition of the PI3K/AP-1 pathway that is linked to the development of cancer cells, and control of NK cell activity (Chen et al., 2023). Pilot clinical trials showed that myo-inositol treatment in patients with ulcerative colitis with low-grade dysplasia resulted in decreased activation of biomarkers in intestinal stem cells compared to placebo (Weinberg et al., 2021). However, the authors of the review emphasized the necessity for further research.

Nutritional Deficiency and Bioavailability

Western-pattern diet, according to a review focused on nutritional inositol deficiency, is low in fiber and rich in fat and sugar, which supplies substantially less dietary inositol and InsP6 than diets richer in whole grains and legumes, potentially compounding the diet's other metabolic risks. The same review highlighted a metabolic pathway leading to this deficiency: elevated glucose increases inositol degradation and excretion while inhibiting its synthesis and absorption. It raises the possibility of a self-reinforcing cycle in which insulin resistance depletes tissue inositol, further impairing insulin signaling. The authors were careful to note this remains a mechanistic hypothesis supported by indirect and epidemiological evidence rather than an established causal pathway, and called for further study of baseline inositol status in relation to metabolic, cardiovascular, and cancerous disease (Dinicola et al., 2017).

Safety, Dosing, and Limitations of the Evidence

Myo-inositol is generally described as well tolerated at trial doses of 2–4 g daily, with mild gastrointestinal symptoms — flatulence, diarrhea, nausea — the most common adverse effects and no clear dose-dependent increase in their frequency (Chatree et al., 2020). No adverse safety signals were noted among the studies included in this review, including those conducted among pregnant women (Formoso et al., 2019). However, there are several limitations to the methods used in this study that impact the clinical conclusions. The variability of isomers, dosages, and treatment duration poses a problem when combining studies (Dinicola et al., 2017). Several reviews — most notably the Cochrane review of PCOS-related subfertility — rated underlying trial evidence as low to very low quality, citing incomplete reporting and inconsistent or missing clinically important outcomes (Showell et al., 2018). Most of the mechanisms of action of pyrophosphates have been demonstrated in animal studies, leaving room for uncertainty regarding their relevance to human dosing and outcomes. Much of the pyrophosphate mechanistic evidence derives from animal studies, which is why a relevance to human dosing and outcomes remains unestablished. Also for thyroid disease, psychiatric disorders, and cancer chemoprevention, the evidence is present in small trials, pilot studies, or mechanistic reasoning rather than large randomized controlled trials (Concerto et al., 2023). These considerations argue for a condition-specific and appropriately cautious interpretation of inositol's clinical role. The summary of the study is mentioned in table 1.

Table 1. Summary of key findings

Clinical Domain	Key Findings	Evidence Strength
Insulin resistance/glycemia	Improves fasting and HOMA	Moderate-strong
Gestational diabetes	Cuts GDM by 70%	Moderate
Obesity	Modest BMI reduction	Moderate, small effect size
NAFLD	Defficiency worsens hepatic fat	Weak
PCOS – metabolic markers	Reduction in insulin fasting	Moderate
PCOS – reproductive outcomes	Uncertain effect on live bitrh	Low to very low
Thyroid	Plausible via TSH-receptor	Weak/theoretical
Psychiatric disorders	No clear benefit in mood disorders; possible in panic	Weak
Cancer preventions	Preclinical reduction in tumor incidence	Weak

4. CONCLUSION

Inositol and its phosphorylation products are essential players in human cell physiology, involved in insulin signaling pathways, lipid metabolism, energy metabolism, calcium signaling, and genome stability. The diversity of physiological roles of inositol has led to a correspondingly varied and not yet fully developed evidence base. There is a good evidential basis regarding metabolic parameters in insulin resistance conditions: myo-inositol has proven effective in preventing gestational diabetes, decreasing body mass index, improving fasting insulin levels and HOMA-IR in PCOS. Preliminary and insufficient data exist regarding fertility outcomes in PCOS-induced subfertility, as well as regarding other physiological targets (NAFLD, thyroid diseases, psychiatric disorders, and cancer prevention). There are several directions that require further investigation.

Larger, adequately powered randomized controlled trials with standardized inositol isomer, dose, and duration, and with pre-specified clinically meaningful primary outcomes — live birth rather than surrogate hormonal markers in PCOS-related infertility, for instance — are needed to resolve the uncertainty highlighted in some systematic reviews. Further human studies are also needed to determine whether the inositol pyrophosphate pathways identified in animal models could be compared to human metabolic regulation. Also it is crucial to know whether baseline inositol status, as shaped by diet and by glycemic control itself, is a modifiable risk factor for metabolic disease rather than simply a downstream marker of it.

Acknowledgments

We thank all co-authors and institutional staff for their support during the preparation of this review.

Authors' Contributions

Conceptualization, supervision, and project administration: Aleksandra Rusak-Sobolewska, Gabriela Krych

Methodology: Aleksandra Rusak-Sobolewska

Software, validation, formal analysis, investigation, resources, writing original draft preparation: Gabriela Krych

Writing review, editing, and visualization: Aleksandra Rusak-Sobolewska, Gabriela Krych.

All authors have read and agreed with the published version of the manuscript.

Informed consent

Not applicable.

Ethical approval

Not applicable. This article does not contain any studies with human participants or animals performed by any of the authors.

Funding

This research did not receive any external funding like specific grant from funding agencies in the public, commercial, or nonprofit sectors.

Conflict of interest

The authors declare that they have no conflicts of interest, competing financial interests or personal relationships that could have influenced the work reported in this paper.

Data and materials availability

All data associated with this study will be available based on the reasonable request to corresponding author.

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