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The Role of Myo-Inositol in Complex Treatment in Correction of Insulin Resistance in Patients with Acne

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Abstract: Acne vulgaris is one of the most prevalent chronic inflammatory skin disorders affecting adolescents and adults worldwide. Recent studies have demonstrated a close relationship between insulin resistance, hyperinsulinemia, androgen excess, and the pathogenesis of acne. Insulin resistance contributes to increased production of insulin-like growth factor-1 (IGF-1), stimulation of sebaceous gland activity, follicular hyperkeratinization, and inflammatory responses. Myo-inositol, a naturally occurring carbocyclic sugar and an important intracellular signaling molecule, has gained significant attention as a therapeutic agent capable of improving insulin sensitivity and metabolic parameters. This review analyzes the role of myo-inositol in the complex treatment of acne patients with insulin resistance and discusses its potential mechanisms of action, clinical benefits, and therapeutic perspectives.

Keywords: Acne vulgaris, insulin resistance, myo-inositol, IGF-1, hyperinsulinemia, metabolic syndrome, dermatology.

Introduction: Acne vulgaris is a multifactorial inflammatory disease of the pilosebaceous unit characterized by comedones, papules, pustules, nodules, and, in severe cases, scarring. The condition affects approximately 85% of adolescents and a significant proportion of adults, especially women.

Traditional pathogenic factors include:

- Excessive sebum production;
- Follicular hyperkeratinization;
- Colonization by Cutibacterium acnes;
- Inflammatory reactions.

However, growing evidence indicates that endocrine and metabolic disturbances significantly contribute to acne development. Among these factors, insulin resistance has emerged as a critical component influencing disease severity and persistence.

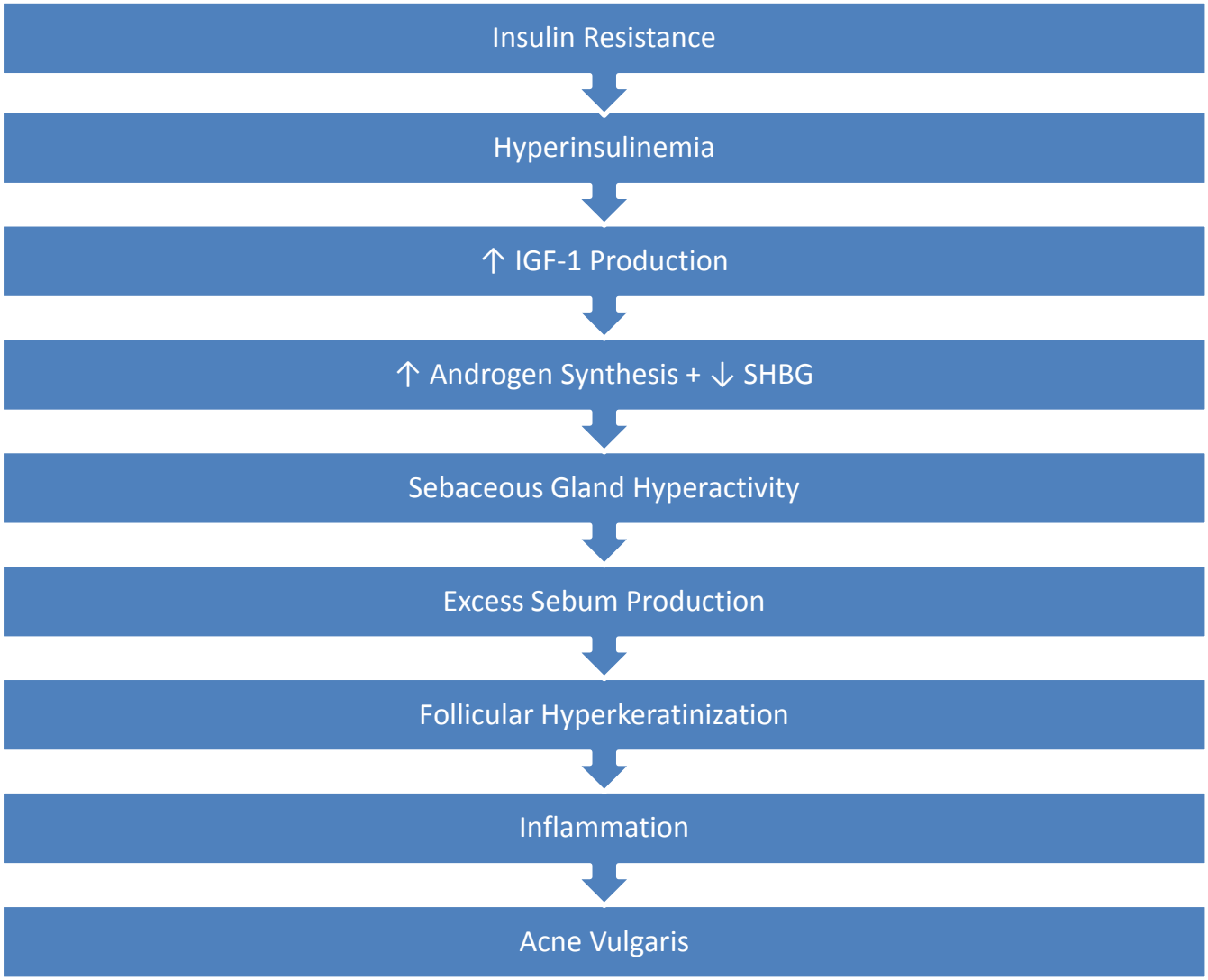
Insulin resistance is defined as a reduced biological response of peripheral tissues to insulin, resulting in compensatory hyperinsulinemia. Elevated insulin levels stimulate ovarian and adrenal androgen synthesis, suppress sex hormone-binding globulin (SHBG), and increase circulating free androgens, thereby aggravating acne lesions.

Myo-inositol has recently attracted considerable interest due to its ability to improve insulin sensitivity and regulate metabolic pathways associated with acne pathogenesis.

Pathophysiological relationship between insulin resistance and acne

Insulin resistance induces a cascade of hormonal and metabolic changes contributing to acne progression.

Flowchart 1. Pathogenesis of Acne Associated with Insulin Resistance



Hyperinsulinemia increases the activity of the mammalian target of rapamycin complex 1 (mTORC1), a key regulator of cell growth and lipid synthesis. Overactivation of mTORC1 promotes sebocyte proliferation and increased sebum production, creating favorable conditions for acne development.

Numerous studies have reported significantly higher Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) scores in patients with moderate and severe acne compared with healthy controls.

Myo-inositol: biological characteristics and mechanisms of action in acne patients

Myo-inositol has recently gained considerable interest because of its ability to improve insulin sensitivity through modulation of intracellular signaling pathways. As a precursor of phosphatidylinositol and inositol phosphates, myo-inositol participates in numerous cellular processes including glucose metabolism, lipid regulation, calcium signaling, and hormone transduction. Deficiency or impaired metabolism of inositol compounds has been linked to insulin resistance and metabolic dysfunction.

The insulin-sensitizing effect of myo-inositol is primarily mediated through enhancement of insulin receptor signaling and promotion of glucose uptake in peripheral tissues. As insulin sensitivity improves, circulating insulin levels decrease, resulting in reduced stimulation of androgen synthesis and normalization of IGF-1 activity. Consequently, several key pathogenic mechanisms involved in acne development may be attenuated.

In addition to its metabolic effects, myo-inositol demonstrates anti-inflammatory and antioxidant properties. Experimental studies suggest that it may reduce oxidative stress markers and inflammatory cytokines, both of which contribute to acne lesion formation and progression. These effects provide additional support for the use of myo-inositol in patients with inflammatory acne associated with metabolic abnormalities.

Clinical investigations have demonstrated encouraging

results regarding the efficacy of myo-inositol supplementation in patients with insulin resistance and acne. Improvements have been observed in insulin sensitivity indices, hormonal profiles, and dermatological outcomes. Several studies involving women with polycystic ovary syndrome reported significant reductions in acne severity following treatment with myo-inositol either alone or in combination with conventional therapies.

Table 1. Clinical Effects of Myo-Inositol Supplementation in Patients with Acne and Insulin Resistance

Parameter	Before Treatment	After Treatment
Fasting insulin	Elevated	Reduced
HOMA-IR	Increased	Decreased
IGF-1 activity	Increased	Reduced
Free androgen index	Elevated	Reduced
SHBG level	Reduced	Increased
Sebum production	Increased	Reduced
Acne severity score	Moderate–Severe	Mild–Moderate
Inflammatory lesions	Numerous	Significantly decreased

Patients receiving myo-inositol in combination with dietary modification and conventional dermatological therapy demonstrated superior clinical improvement compared with standard treatment alone.

Complex treatment approach

The observed clinical improvements indicate that correction of metabolic disturbances may enhance the effectiveness of traditional dermatological treatment. Therefore, management of acne associated with insulin resistance should involve a multidisciplinary approach targeting both cutaneous manifestations and underlying metabolic dysfunction.

A comprehensive therapeutic strategy may include dietary intervention, physical activity, topical dermatological treatment, hormonal regulation when indicated, and metabolic correction using insulin-sensitizing agents such as myo-inositol.

Current evidence supports the hypothesis that insulin resistance contributes substantially to acne pathogenesis and that correction of metabolic disturbances may improve treatment outcomes. Myo-inositol appears particularly beneficial in patients presenting with concomitant insulin resistance, obesity, metabolic syndrome, or polycystic ovary syndrome. Its favorable safety profile and physiological mechanism of action make it an attractive therapeutic option for long-term management.

DISCUSSION

The growing recognition of acne as a systemic disease associated with metabolic dysfunction has expanded therapeutic possibilities beyond traditional dermatological approaches. Myo-inositol represents a promising adjunctive treatment due to its favorable safety profile and ability to target underlying pathogenic mechanisms.

Particularly in female patients presenting with acne, insulin resistance, obesity, or PCOS, myo-inositol may provide dual metabolic and dermatological benefits. Its role in improving insulin signaling and reducing hyperandrogenism makes it a valuable component of personalized treatment strategies.

Although current evidence is encouraging, further large-scale randomized controlled trials are needed to establish optimal dosing regimens, treatment duration, and long-term efficacy.

CONCLUSION

Insulin resistance plays a significant role in the pathogenesis and persistence of acne vulgaris. Hyperinsulinemia contributes to increased androgen production, enhanced IGF-1 activity, excessive sebum secretion, and inflammatory processes.

Myo-inositol is an effective insulin-sensitizing agent capable of correcting metabolic abnormalities associated with acne. Its incorporation into complex treatment regimens may improve insulin sensitivity, reduce androgen excess, decrease acne severity, and enhance overall therapeutic outcomes.

The available evidence supports the use of myo-inositol as a promising adjunctive therapy for patients with acne and insulin resistance, particularly in individuals with concomitant metabolic disorders such as PCOS.

REFERENCES

1. Nestler J.E., Jakubowicz D.J. Myo-inositol in the treatment of polycystic ovary syndrome. Current Opinion in Obstetrics and Gynecology. 2020;32(4):242–248.
2. D'Anna R., Scilipoti A., Giordano D. Myo-inositol supplementation and insulin resistance: clinical implications. Gynecological Endocrinology. 2021;37(6):501–507.
3. Del Prete M., Mauriello M.C., Faggiano A. Insulin resistance and acne: a systematic review. Journal of Endocrinological Investigation. 2022;45(3):419–429.
4. Melnik B.C. Linking diet to acne metabolomics, inflammation, and comedogenesis. Clinical Cosmetic and Investigational Dermatology. 2020;13:371–388.
5. Zaenglein A.L., Pathy A.L., Schlosser B.J. Guidelines of care for the management of acne vulgaris. Journal of the American Academy of Dermatology. 2024;90(2):305–329.
6. Diamanti-Kandarakis E., Paterakis T., Kandarakis H. The role of inositols in endocrine and metabolic disorders. International Journal of Endocrinology. 2023;2023:1–12.
7. Bhate K., Williams H.C. Epidemiology of acne vulgaris. British Journal of Dermatology. 2021;184(4):594–601.
8. Melnik B.C. Acne vulgaris and insulin resistance. Experimental Dermatology. 2022;31(8):1141–1152.
9. Unfer V., Facchinetti F., Orrù B. Myo-inositol effects in insulin-resistant conditions. European Review for Medical and Pharmacological Sciences. 2023;27(5):1824–1835.
10. Thiboutot D., Gollnick H., Bettoli V. New insights into acne pathogenesis and treatment. Dermatology Reports. 2024;16(1):45–57.