



Original Research

Insulin resistance in Mexican women with acne vulgaris

Denisse Islas Cortes, M.D.¹, Patricia Carmona, M.D.², Eduardo Chuquiure Valenzuela, MHSc, M.D.³

¹ Internal Medicine Residency Program, Hospital Español de México, ² Private Dermatology Practice, Mexico City, ³ Cardiology, Instituto Nacional de Cardiología Ignacio Chávez

Keywords: acne vulgaris, insulin resistance, HOMA-IR

Journal of Integrative Dermatology

Relevance

Acne vulgaris is the most common dermatological condition defined as a chronic inflammatory disease of the pilosebaceous unit. Insulin could be part of the pathophysiology of acne through various mechanisms.

Objective

To determine the prevalence of insulin resistance in female patients with acne vulgaris.

Methods

We designed a descriptive observational study, in which we included a sample of 319 women with acne vulgaris. The diagnosis was established clinically by a dermatologist. Clinical data and biochemical parameters of serum insulin, fasting glucose, HOMA-IR and lipid profile were evaluated.

Results

319 women diagnosed with acne vulgaris were included, with a mean age of 30.45 years \pm 0.37 (18-69). 82.8% (264) of them were over 25 years old, classified as adult female acne. Laboratory results were: mean serum insulin 16.83 \pm 0.43 (2.61-54.30), mean glucose 90.74 \pm 0.83 (69-304), HOMA-IR 3.82 \pm 0.11 (0.53-14.08), and mean total cholesterol 173.92 \pm 1.88 (100-303). Of the 319 patients, 317 of them (99.37%) had a HOMA-IR >1.22.

Conclusion

Insulin resistance is underdiagnosed. It is essential that dermatologists diagnose and treat it promptly to improve the response to treatment and avoid the development of metabolic and cardiovascular complications.

INTRODUCTION

Acne vulgaris is the most common dermatological condition defined as a chronic inflammatory disease of the pilosebaceous unit.¹ The term insulin resistance (IR) refers to defects in glucose metabolism stimulated by insulin in tissues, in addition to impaired suppression of glucose production by the liver mediated by insulin.²

Classically, four main mechanisms have been described in the pathophysiology of acne, which include increased sebum production induced by androgens, alterations in keratinization, colonization by *Cutibacterium acnes*, and inflammation.³

Nevertheless, it has been shown that insulin could also be part of the pathophysiology of acne through various mechanisms that include the insulin-like growth factor 1 (IGF-1) and mechanistic target of rapamycin complex 1 (mTORC1) signaling pathways, which are stimulated by the Western lifestyle characterized by the consumption of high glycemic index foods and dairy products.^{4,5} Additionally, hyperinsulinemia is associated with increased androgen

production, gonadotropin-releasing hormone secretion, and decreased sex hormone-binding globulin.⁶ (Figure 1)

The gold standard test for evaluating insulin resistance is the hyperinsulinemic-euglycemic clamp, but as it is an invasive method and due to its complexity, its use is limited to clinical research studies.⁷ One of the most widely used methods in clinical practice is the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR), which has a good correlation coefficient value with the results of the hyperinsulinemic-euglycemic clamp.⁸ HOMA-IR estimates insulin resistance by evaluating the relationship between fasting insulin and glucose levels using a simplified formula: (fasting insulin [μ U/mL] \times fasting glucose [mmol/L]) \div 22.5.⁸ HOMA-IR cut-off points may vary according to age, gender, race, etc. A study conducted in a Mexican population without diabetes in 2018 by Alameda-Valdés et al reported that the cut-off point with the best sensitivity of the HOMA Index to diagnose insulin resistance was 1.22, with a sensitivity of 84.6% and a specificity of 48.8%.⁹

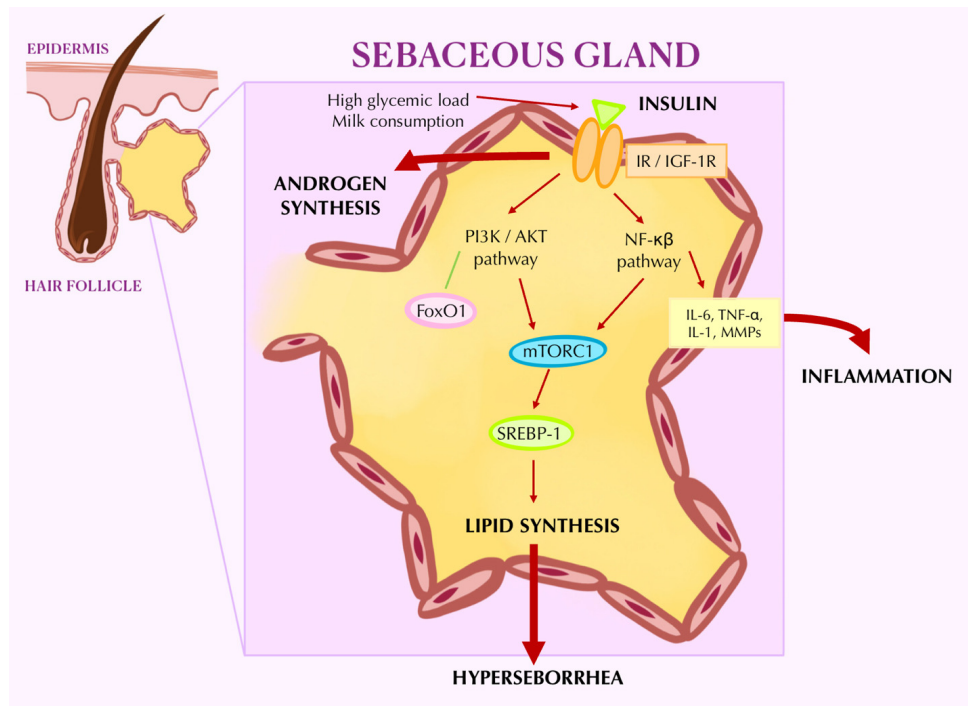


Figure 1. Main pathological effects of Insulin on the sebaceous gland.

IR: insulin receptor, IGF-1R: Insulin-like growth factor receptor, PI3K pathway: phosphoinositide 3-kinase pathway, mTORC1: mammalian target of rapamycin complex 1, SREBP-1: sterol regulatory element-binding protein-1, IL: interleukin, MMPs: matrix metalloproteinases, TNF: tumor necrosis factor.

MATERIALS AND METHODS

We conducted a descriptive observational study in a consecutive sample of 319 women who attended a dermatological consultation from January 1, 2021 to January 1, 2024 due to acne vulgaris. The diagnosis was established clinically by a dermatologist, and patients were selected according to the inclusion criteria which were: female patients with acne vulgaris over 18 years of age who were willing to participate in the study and undergo laboratory tests for follow-up with prior informed consent. Patients who did not meet the inclusion criteria or patients who received treatment in the last 6 months with drugs that affect insulin or glucose metabolism were excluded from this study.

Biochemical parameters of serum insulin, fasting glucose, HOMA-IR and lipid profile were evaluated, which were integrated in the clinical record along with clinical characteristics and findings in physical examination.

The demographic, clinical, and laboratory characteristics were entered into Microsoft Excel and analyzed using SPSS version 13.. All physicians declare strict adherence to ethical protocols in human studies.

RESULTS

319 women diagnosed with acne vulgaris were included in our study, with a mean age of 30.45 years \pm 0.37 (18-69). 82.8% (264) of them were over 25 years old, classified as adult female acne.

Laboratory results were as follows: mean serum insulin 16.83 \pm 0.43 (range 2.61-54.30), mean fasting glucose 90.74

\pm 0.83 (range 69-304), HOMA-IR 3.82 \pm 0.11 (range 0.53-14.08), and mean total cholesterol 173.92 \pm 1.88 (range 100-303). Of the 319 patients, 317 of them (99.37%) exceeded the cut-off point for the diagnosis of insulin resistance with a HOMA-IR >1.22, which suggests a high frequency of insulin resistance among women with acne vulgaris. (Table 1)

DISCUSSION

Insulin resistance significantly reduces the body's physiological responses, resulting in pathological conditions. If allowed to progress, IR results in tissue damage and manifests primarily as metabolic syndrome, non-alcoholic fatty liver disease, and type 2 diabetes.¹⁰

Currently, there are not sufficient studies available that investigate the association between insulin resistance and acne vulgaris, which is why we investigated this association in Mexican women.

In this study we used the HOMA-IR index to assess insulin resistance. At present time, there is no established cut-off value and there is a great variability in the parameters of the HOMA-IR index to establish the diagnosis of IR, depending on variables such as: ethnicity, sex, and age. We used a study realized in a Mexican population without diabetes in 2018 by Alameda-Valdés et al as a reference, which reported that the cut-off point with the best sensitivity (84.6%) of the HOMA Index to diagnose insulin resistance was 1.22, and a specificity of 48.8%.⁹ According to the results obtained in our study, we confirmed the possible association between insulin resistance and acne vulgaris, with 317 patients (99.37%) who exceeded the cut-off point for

Table 1. Laboratory test results.

Laboratory test	Mean	Standard error	Range
Plasma insulin (μ IU/mL)	16.83	\pm 0.43	2.61-54.30
Fasting plasma glucose (mg/dL)	90.74	\pm 0.83	69-304
HOMA-IR	3.82	\pm 0.11	0.53-14.08
Total cholesterol (mg/dL)	173.92	\pm 1.88	100-303

the diagnosis of insulin resistance with a HOMA-IR >1.22 . The high proportion of participants in our study who exceeded the HOMA-IR cutoff value (>1.22) may be partially explained by the high prevalence of metabolic risk factors in the Mexican population. Additionally, the relatively low HOMA-IR cut-off value used in our study (1.22), which prioritizes sensitivity over specificity, may have contributed to the very high prevalence of insulin resistance observed in our sample. According to the 2018 National Health and Nutrition Survey (ENSANUT), 75.2% of Mexican adults aged 20 years and older are classified as either overweight (39.1%) or obese (36.1%).¹¹ However, it should be considered that the diagnosis of insulin resistance is clinical and must integrate findings in the physical and laboratory examination that could indicate abdominal obesity, hyperinsulinemia, hyperglycemia, dyslipidemia, and comorbidities such as polycystic ovary syndrome, which have a high correlation with insulin resistance determined by hyperinsulinemic euglycemic clamp.¹²

Among the limitations of our study are the exclusive inclusion of female patients, the absence of validated HOMA-IR cutoff values specific to the Mexican population, which may affect the accuracy of insulin resistance classification, and the lack of a control group of participants without acne, which limits the generalizability of our findings to a broader population. Additionally, we were unable to analyze other relevant demographic and clinical variables, such as acne type and severity, or clinical signs of insulin resistance such as increased abdominal circumference or acanthosis nigricans, and laboratory parameters such as LDL and HDL cholesterol, which could have enriched our analysis and may serve as the basis for future research and treatment strategies in patients with acne vulgaris.

CONCLUSION

Insulin resistance is underdiagnosed despite its involvement in the pathogenesis of acne among other diseases. Skin is one of the first organs to clinically manifest insulin resistance; therefore, it is essential that dermatologists diagnose and treat it promptly to improve the response to treatment and avoid the development of metabolic and cardiovascular complications, which represent the main causes of morbidity and mortality and constitute a major public health problem. Our study allows new perspectives for future interventions to comprehensively treat patients with acne and is a call for preventive medicine. More studies are needed to achieve the generalization of these results according to cut-off points in more populations.

CORRESPONDING AUTHOR

Patricia Carmona Contreras, MD
Lamartine 408, Polanco, Polanco V Secc, Miguel Hidalgo,
11560 Mexico City, Mexico.
Telephone number: 55 7504 3471
E-mail: patriderma@gmail.com

CONFLICT OF INTEREST STATEMENT

The authors have no conflicts of interest to declare.

FUNDING SOURCES

None

Submitted: March 25, 2025 PDT. Accepted: January 19, 2026 PDT.



This is an open-access article distributed under the terms of the Creative Commons Attribution 4.0 International License (CC0). View this license's legal deed at <https://creativecommons.org/publicdomain/zero/1.0> and legal code at <https://creativecommons.org/publicdomain/zero/1.0/legalcode> for more information.

REFERENCES

1. Eichenfield DZ, Sprague J, Eichenfield LF. Management of Acne Vulgaris: A Review. *JAMA*. 2021;326(20):2055-2067. doi:[10.1001/jama.2021.17633](https://doi.org/10.1001/jama.2021.17633)
2. Li M, Chi X, Wang Y, et al. Trends in insulin resistance: insights into mechanisms and therapeutic strategy. *Sig Transduct Target Ther*. 2022;7:216. doi:[10.1038/s41392-022-01073-0](https://doi.org/10.1038/s41392-022-01073-0)
3. Williams HC, Dellavalle RP, Garner S. Acne vulgaris. *Lancet*. 2012;379(9813):361-372. doi:[10.1016/S0140-6736\(11\)60321-8](https://doi.org/10.1016/S0140-6736(11)60321-8)
4. Melnik BC. Acne vulgaris: The metabolic syndrome of the pilosebaceous follicle. *Clin Dermatol*. 2018;36(1):29-40. doi:[10.1016/j.clindermatol.2017.09.006](https://doi.org/10.1016/j.clindermatol.2017.09.006). PMID:29241749
5. Cordain L, Lindeberg S, Hurtado M, Hill K, Eaton SB, Brand-Miller J. Acne vulgaris: a disease of Western civilization. *Arch Dermatol*. 2002;138(12):1584-1590. doi:[10.1001/archderm.138.12.1584](https://doi.org/10.1001/archderm.138.12.1584). PMID:12472346
6. Smith RN, Mann NJ, Braue A, Mäkeläinen H, Varigos GA. A low-glycemic-load diet improves symptoms in acne vulgaris patients: a randomized controlled trial. *Am J Clin Nutr*. 2007;86(1):107-115. doi:[10.1093/ajcn/86.1.107](https://doi.org/10.1093/ajcn/86.1.107). PMID:17616769
7. Tam CS, Xie W, Johnson WD, Cefalu WT, Redman LM, Ravussin E. Defining insulin resistance from hyperinsulinemic-euglycemic clamps. *Diabetes Care*. 2012;35(7):1605-1610. doi:[10.2337/dc11-2339](https://doi.org/10.2337/dc11-2339). PMID:22511259
8. Tahapary DL, Pratisthita LB, Fitri NA, et al. Challenges in the diagnosis of insulin resistance: Focusing on the role of HOMA-IR and Tryglyceride/glucose index. *Diabetes Metab Syndr*. 2022;16(8):102581. doi:[10.1016/j.dsx.2022.102581](https://doi.org/10.1016/j.dsx.2022.102581). PMID:35939943
9. Almeda-Valdés P, Bello-Chavolla OY, Caballeros-Barragán CR, et al. [Índices para la evaluación de la resistencia a la insulina en individuos mexicanos sin diabetes]. *Cac Med Mex*. 2018;154(Supp 2):550-S55. doi:[10.24875/GMM.18004578](https://doi.org/10.24875/GMM.18004578). PMID:30532124
10. Sadeghi A, Niknam M, Momeni-Moghaddam MA, et al. Crosstalk between autophagy and insulin resistance: evidence from different tissues. *Eur J Med Res*. 2023;28(1):456. doi:[10.1186/s40001-023-01424-9](https://doi.org/10.1186/s40001-023-01424-9). PMID:37876013
11. Instituto Nacional de Salud Pública (MX), Secretaría de Salud. *Encuesta Nacional de Salud y Nutrición 2018-19: Resultados Nacionales*. Instituto Nacional de Salud Pública; 2020.
12. Pollak-C F. RESISTENCIA A LA INSULINA: VERDADES Y CONTROVERSIAS. *Revista Médica Clínica Las Condes*. 2016;27(2):171-178. doi:[10.1016/j.rmcl.2016.04.006](https://doi.org/10.1016/j.rmcl.2016.04.006)