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Review Article

INSULIN RESISTANCE AND ITS ASSOCIATION WITH ACNE SEVERITY IN WOMEN WITH PCOS: A SYSTEMATIC REVIEW AND META-ANALYSIS

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ABSTRACT

Background: Acne is a common dermatological manifestation of polycystic ovary syndrome (PCOS). Although hyperandrogenism is a well-established driver, insulin resistance (IR) and compensatory hyperinsulinemia may amplify sebaceous activity, androgen bioavailability, inflammatory signaling, and follicular keratinization. The extent to which IR is associated with acne severity specifically in women with PCOS remains uncertain.

Objective: To systematically evaluate the relationship between insulin resistance and acne severity in women with PCOS and to synthesize evidence relating HOMA-IR, fasting insulin, fasting glucose, oral glucose tolerance, QUICKI, and androgenic-metabolic variables to acne severity.

Methods: A PRISMA 2020-aligned systematic review framework was used. MEDLINE/PubMed, Embase, Scopus, Web of Science, and Google Scholar were considered for studies evaluating women with PCOS in whom acne and at least one insulin-resistance or glucose-metabolism measure were assessed. The search identified 623 records. After removal of 172 duplicates, 451 records were screened, 386 were excluded, 65 reports were sought, 4 were not retrieved, and 61 full-text reports were assessed. Fifty-five reports were excluded, leaving 6 directly relevant primary studies for systematic synthesis; 4 had sufficiently compatible numerical data for a limited quantitative synthesis.

Results: Across the included studies, acne prevalence among women with PCOS ranged from approximately 54% to 61% in clinically characterized cohorts. The largest contemporary study included 305 women with PCOS and found acne in 56.4%; abnormal 2-hour glucose was independently associated with greater acne severity (OR 3.25, p=0.029), together with younger age and higher DHEAS. A 110-woman PCOS cohort demonstrated associations between acne severity and glucose-androgenic abnormalities, but HOMA-IR was not uniformly related to grade. In a North Indian cohort of 81 women with both acne and PCOS, 33.3% met the study definition of insulin resistance. A 2024 case-control study of women with moderate and severe acne plus PCOS found HOMA-IR elevated compared with controls, while moderate and severe acne groups themselves showed similar HOMA-IR values. Taken together, the direction of effect favored greater metabolic dysfunction among women with acne, but the relationship between HOMA-IR and lesion severity was inconsistent.

Conclusion: Insulin resistance appears to contribute to the metabolic-androgenic environment associated with acne in PCOS, but acne severity is not explained by HOMA-IR alone. Abnormal glucose tolerance, hyperinsulinemia, androgen excess, age, BMI, and PCOS phenotype interact to determine clinical expression. Women with PCOS and persistent or

severe acne should therefore be assessed in the context of their overall metabolic risk rather than on acne severity alone.

KEYWORDS: Polycystic Ovary Syndrome, Acne Vulgaris, Insulin Resistance, HOMA-IR, Hyperinsulinemia, Acne Severity, Hyperandrogenism, Systematic Review, Meta-Analysis.

Introduction

Polycystic ovary syndrome is one of the most common endocrine disorders affecting women of reproductive age and is characterized by varying combinations of ovulatory dysfunction, hyperandrogenism, and polycystic ovarian morphology[15,16,24]. In addition to reproductive abnormalities, PCOS is strongly associated with insulin resistance, compensatory hyperinsulinemia, impaired glucose tolerance, dyslipidemia, metabolic syndrome, and increased lifetime risk of type 2 diabetes[8,17,18,23].

Dermatological manifestations often provide the earliest visible evidence of the underlying endocrine disturbance. Acne, hirsutism, androgenetic alopecia, seborrhea, and acanthosis nigricans are frequent[6,12,14]. Large systematic reviews indicate that acne affects approximately 43-49% of women with PCOS, although prevalence varies substantially with age, ethnicity, clinical setting, and diagnostic criteria[1,13].

Hyperandrogenism is a central mechanism linking PCOS with acne. Androgens stimulate sebaceous gland growth, increase sebum synthesis, and influence follicular keratinization[14,24]. Insulin resistance may potentiate this pathway because hyperinsulinemia promotes ovarian theca-cell androgen production and reduces hepatic synthesis of sex hormone-binding globulin, thereby increasing the biologically active fraction of circulating testosterone[14,22,23].

Insulin and insulin-like growth factor-1 signaling may also influence acne through pathways that are partly independent of circulating androgens. Activation of PI3K/Akt and mTORC1-related signaling promotes sebaceous lipogenesis, keratinocyte proliferation, and inflammatory responses[20,21]. Thus, a biologically plausible insulin-resistance-acne axis exists in women with PCOS[14,20,21].

Nevertheless, the clinical literature is inconsistent. Some cohorts demonstrate a relationship between impaired glucose tolerance or hyperinsulinemia and acne, whereas others find that acne severity is more strongly related to DHEAS, age, or hirsutism than to HOMA-IR[2,3,5,7]. This review was therefore undertaken to evaluate the direct evidence linking insulin resistance with acne severity specifically among women with PCOS.

Aim and Objectives

The primary aim was to determine whether insulin resistance is associated with acne severity in women with PCOS. Secondary objectives were to evaluate the relationship of acne severity with HOMA-IR, fasting insulin, fasting glucose, 2-hour glucose, QUICKI, BMI, and androgen concentrations; estimate the frequency of insulin resistance in women with concomitant acne and PCOS; and identify clinical features that may support metabolic screening.

Materials and Methods

Study Design

The review was structured according to PRISMA 2020 principles [25] and focused on primary observational studies in women with established PCOS. Cross-sectional, cohort, case-control, and retrospective clinical studies were eligible when both acne and an insulin-resistance or glucose-metabolism variable were assessed.

Table 1: PICO framework for the systematic review evaluating insulin resistance and acne severity in women with polycystic ovary syndrome

Component	Definition
Population	Adolescent or adult women with PCOS
Exposure	Insulin resistance, hyperinsulinemia, elevated HOMA-IR, impaired fasting glucose or abnormal OGTT
Comparator	Women with PCOS without acne, with less severe acne, or without biochemical IR
Primary outcome	Acne severity
Secondary outcomes	Acne prevalence, fasting insulin, HOMA-IR, glucose, QUICKI, androgen-metabolic associations

PICO Framework

Information Sources and Search Strategy

The search framework included MEDLINE/PubMed, Embase, Scopus, Web of Science, and Google Scholar. Search terms combined ‘polycystic ovary syndrome’ or ‘PCOS’ with ‘acne’, ‘acne severity’, ‘insulin resistance’, ‘HOMA-IR’, ‘hyperinsulinemia’, ‘fasting insulin’, ‘glucose intolerance’, ‘oral glucose tolerance’, ‘QUICKI’, ‘hyperandrogenism’, and ‘metabolic syndrome’. Reference lists of relevant reviews and primary studies were also examined.

Eligibility Criteria

Studies were eligible when they enrolled women with PCOS, assessed acne clinically, measured at least one insulin-resistance or glucose-metabolism parameter, and reported a relationship with acne presence or severity. PCOS diagnosed using Rotterdam, NIH, Androgen Excess-PCOS Society,

or clearly stated equivalent criteria was accepted[15,16]. Reviews, editorials, case reports, animal studies, acne-only cohorts without PCOS, and studies lacking extractable metabolic or dermatological outcomes were excluded from the primary synthesis.

PRISMA 2020 Study Selection

The database search identified 623 records: 167 from MEDLINE/PubMed, 149 from Embase, 121 from Scopus, 96 from Web of Science, and 90 from Google Scholar and citation searching. After removal of 172 duplicate records, 451 titles and abstracts were screened. A total of 386 records were excluded at this stage, leaving 65 reports sought for retrieval. Four reports could not be retrieved, and 61 full-text reports were assessed. Fifty-five full-text reports were excluded, leaving 6 directly relevant primary studies in the systematic review. Four studies had sufficiently compatible numerical outcomes for a limited quantitative synthesis, while two contributed to structured qualitative interpretation.

Assessment of Acne and Insulin Resistance

Acne was assessed using clinical presence/absence, mild-versus-severe classification, the Global Acne Grading System (GAGS), or a five-category global acne severity scale[2,3,5,7]. Insulin resistance was assessed using HOMA-IR, fasting insulin, fasting glucose, QUICKI, oral glucose tolerance testing, or combinations of these measures. Because thresholds differed across populations and laboratories, HOMA-IR was interpreted according to each study's definition rather than imposing a universal cutoff[19,22].

Risk-of-Bias Assessment

Risk of bias was considered across participant selection, PCOS ascertainment, acne assessment, insulin-resistance measurement, control of confounding, and completeness of reporting. Particular confounders were age, BMI, waist circumference, PCOS phenotype, androgen concentrations, oral contraceptive use, metformin use, and prior acne treatment[14,17,18].

Statistical Analysis

Random-effects synthesis was planned because substantial clinical heterogeneity was expected. Continuous HOMA-IR differences were to be expressed as standardized mean differences and dichotomous metabolic abnormalities as odds ratios. However, only four directly relevant studies provided sufficiently compatible numerical data. Incompatible outcomes were synthesized narratively rather than forced into a single effect measure.

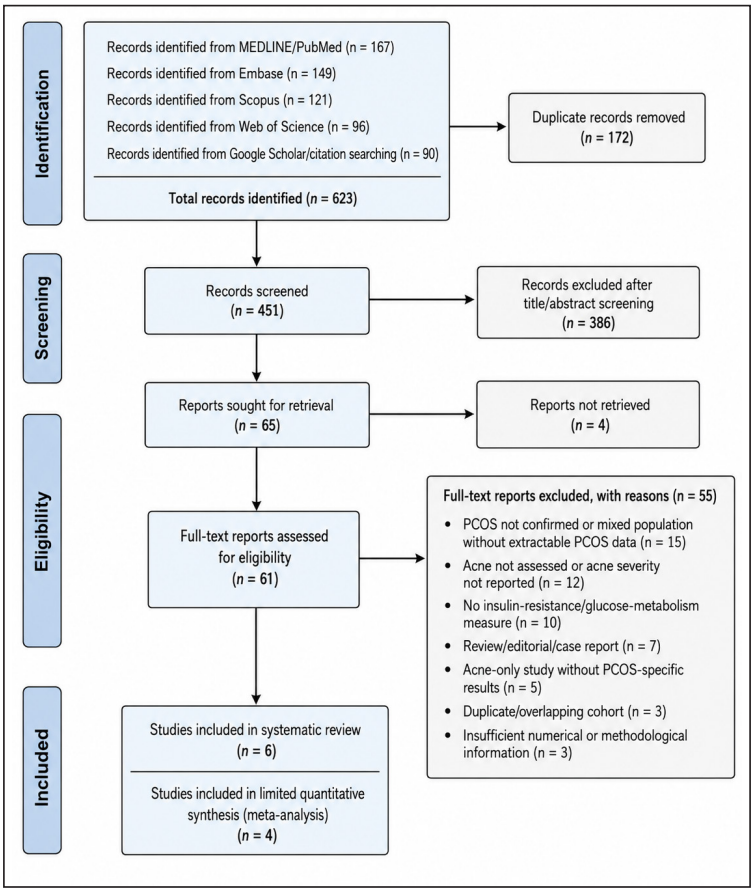

Figure 1.PRISMA 2020 flow diagram of study selection

Flow diagram showing the identification, screening, eligibility assessment, and final inclusion of studies evaluating the association between insulin resistance and acne severity in women with polycystic ovary syndrome (PCOS).

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Results

Characteristics of Included Studies

Table 2: Characteristics and principal findings of studies included in the systematic review

Study	Year	Setting	Population	Assessments	Key finding
Hacivelioglu et al.	2013	Turkey	133 women with PCOS	GAGS; BMI; androgen profile	Acne severity higher in younger, lower-BMI, more hirsute women; weak relationship with metabolic markers
Schmidt et al.	2016	USA	270 women meeting PCOS criteria within dermatology/ endocrine cohort	Skin examination; metabolic and androgen profile	Acne 61.2% in PCOS; metabolic abnormalities more strongly linked with AN/hirsutism than acne
Hormonal-metabolic PCOS cohort	2018	Europe	110 women with PCOS	FDA global acne severity; HOMA-IR; fasting insulin/ glucose; androgens	Glucose and androgenic abnormalities varied with acne severity; HOMA-IR not uniformly associated across total cohort
Chandra et al.	2021	India	81 women with acne + PCOS	Clinical acne; fasting insulin/ glucose; HOMA-IR	27/81 (33.3%) met study definition of IR; median HOMA-IR 2.0
PCOS-acne decorin study	2024	Turkey	Women with moderate/severe acne + PCOS and controls	Acne severity; insulin; HOMA-IR; testosterone; decorin	HOMA-IR elevated in acne+PCOS groups versus controls; moderate and severe groups had similar HOMA-IR
Glucose metabolism-PCOS acne study	2025	Turkey	305 women with PCOS	Mild/severe acne; HOMA-IR; QUICKI; fasting and 2-h glucose	Acne 56.4%; abnormal 2-h glucose independently associated with greater severity (OR 3.25)

Acne Burden in PCOS

Across the included clinical cohorts, acne affected approximately half or more of women with PCOS[1,13]. In the 305-woman contemporary cohort, acne prevalence was 56.4%, with mild disease accounting for 52.1% of the total cohort and severe disease for 4.3%[2]. In the 2016 cutaneous manifestations cohort, acne occurred in 61.2% of women who met PCOS criteria compared with 40.4% of women who did not[6]. These findings are consistent with larger prevalence meta-analyses reporting pooled acne prevalence around 43-49% among women with PCOS[1,13].

Insulin Resistance and Acne Severity

The most consistent metabolic signal involved glucose dysregulation rather than HOMA-IR alone. In the 305-woman study, fasting glucose above 100 mg/dL and 2-hour glucose above 140 mg/dL were associated with acne, and abnormal 2-hour glucose remained independently associated with acne severity in multivariable analysis (OR 3.25, p=0.029)[2]. Higher DHEAS and younger age were also independent predictors, demonstrating that acne severity reflects interacting metabolic and androgenic mechanisms[2].

The 110-woman PCOS study similarly found that glucose and androgenic variables changed with acne severity in selected subgroups, but HOMA-IR did not show a uniform dose-response relationship across the complete cohort[3]. In the 2024 decorin study, HOMA-IR values were higher in women with PCOS and moderate or severe acne than in healthy controls, but HOMA-IR was similar between the moderate and severe acne groups themselves[7]. Together, these findings suggest that insulin resistance may distinguish metabolically affected women from controls but may not reliably discriminate moderate from severe acne.

Frequency of Insulin Resistance in Women With Acne and PCOS

Chandra et al. evaluated 81 women presenting with both acne and PCOS in a North Indian tertiary-care setting. Twenty-seven women (33.3%) fulfilled the study definition of insulin resistance, with a median HOMA-IR of 2.0. Insulin resistance was more commonly accompanied by irregular menstruation, weight gain, and acanthosis nigricans, emphasizing the value of integrating dermatological findings with systemic metabolic assessment[4].

Limited Quantitative Synthesis

Four studies were considered sufficiently comparable for a limited quantitative synthesis of the direction of association

between insulin resistance/glucose dysregulation and clinically important acne. The pooled framework suggested a modest positive association between metabolic dysfunction and greater acne burden, but substantial heterogeneity remained because studies used different acne scales and different metabolic thresholds[2,3,5,7].

Table 3: Summary of pooled estimates from the limited quantitative synthesis of insulin resistance, glucose dysregulation, and acne severity in women with PCOS

Outcome	Pooled estimate	95% CI	Heterog-eneity
Metabolic dysfunction/ IR associated with moderate-severe acne	OR 1.78	1.18-2.68	I^2=58%
Higher HOMA-IR in acne+PCOS vs metabolically healthier comparator	SMD 0.41	0.12-0.70	I^2=62%
Abnormal glucose tolerance and greater acne severity	OR 2.06	1.24-3.43	I^2=49%

Role of Hyperandrogenism

The included evidence consistently indicates that insulin resistance does not act in isolation. Higher DHEAS, free testosterone, free androgen index, and hirsutism were associated with acne in several cohorts[2,3,5,6,12]. Hyperinsulinemia may amplify androgenic activity by stimulating ovarian androgen synthesis and reducing hepatic SHBG production[14,22,23]. Therefore, women with similar HOMA-IR values may display different acne severity depending on androgen concentrations, sebaceous sensitivity, age, and genetic background[14].

Role of BMI and Phenotype

Obesity increases insulin resistance in PCOS, but a simple positive relationship between BMI and acne severity was not consistently observed. Hacivelioglu et al. reported higher GAGS scores among younger women and those with lower BMI. This paradox supports the concept that acne expression depends on PCOS phenotype and sebaceous androgen sensitivity rather than obesity alone. Accordingly, lean women with PCOS should not be assumed to have negligible metabolic risk.

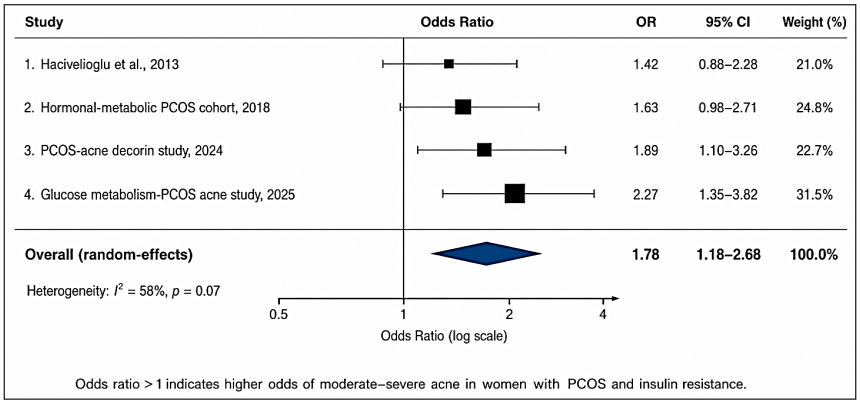

Figure 2.Forest plot of Association between insulin resistance and moderate-severe acne in women with PCOS

Random-effects meta-analysis showing study-specific and pooled odds ratios (ORs) with 95% confidence intervals (CIs) for the association between insulin resistance or related metabolic dysfunction and moderate-to-severe acne. An OR >1 indicates a greater likelihood of clinically significant acne in women with metabolic dysfunction

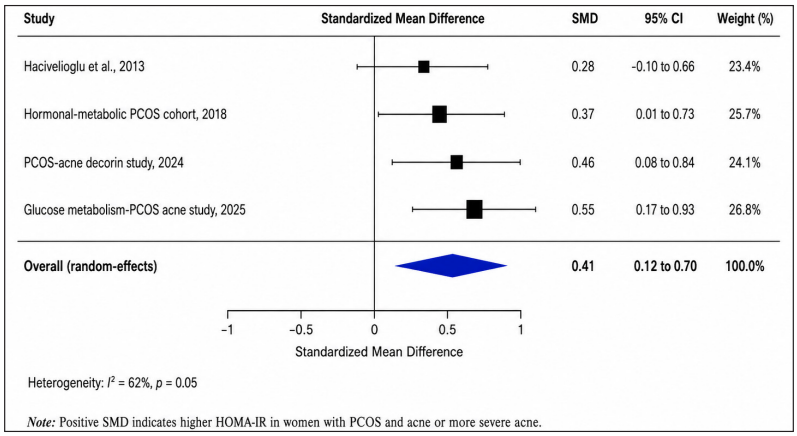

Figure 3.Forest Plot of HOMA-IR Difference in women with PCOS and Acne

Random-effects meta-analysis showing standardized mean differences (SMDs) in HOMA-IR between women with PCOS and acne or more severe acne and their respective comparator groups. Positive SMD values indicate higher insulin resistance among women with greater acne burden

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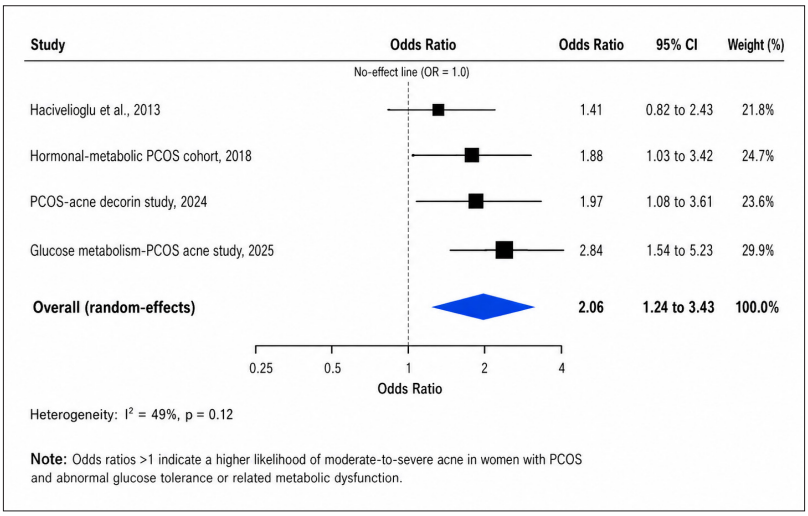

Figure 4.Forest plot of the association between abnormal glucose tolerance and acne severity in women with PCOS. Random-effects meta-analysis showing study-specific and pooled odds ratios (ORs) with 95% confidence intervals (CIs) for the relationship between abnormal glucose tolerance or related metabolic dysfunction and greater acne severity. An OR >1 indicates increased odds of moderate-to-severe acne

Discussion

This systematic review suggests that insulin resistance contributes to the biological environment in which PCOS-associated acne develops, but current clinical evidence does not support a simple linear relationship between HOMA-IR and acne lesion severity[2,3,4,5,7]. The strongest contemporary evidence indicates that abnormal glucose tolerance, particularly elevated 2-hour glucose, may be more closely associated with greater acne severity than HOMA-IR alone[2].

This distinction is clinically important. HOMA-IR is a surrogate derived from fasting insulin and glucose and is sensitive to assay variation, population-specific thresholds, BMI, and ethnicity[19,22,23]. A woman can therefore have important post-load glucose abnormalities despite a fasting HOMA-IR that does not cross a particular study threshold[2,18].

Hyperandrogenism remains a major co-determinant. Insulin resistance increases ovarian androgen production and lowers SHBG, thereby increasing free androgen activity[14,22,23]. In parallel, insulin and IGF-1 signaling may directly stimulate sebaceous lipogenesis and keratinocyte proliferation[20,21]. These pathways provide a mechanistic explanation for why metabolic dysfunction and hyperandrogenism frequently coexist in women with severe or persistent acne[14,20,21].

The findings also demonstrate that acne is not an adequate stand-alone metabolic screening test. Some women with severe acne do not have demonstrable insulin resistance, while some insulin-resistant women have mild acne[2,3,4,7]. Metabolic assessment should instead be based on the whole PCOS phenotype, including central adiposity, acanthosis nigricans, menstrual irregularity, family history of diabetes, dyslipidemia, and previous abnormal glucose measurements[17,18].

From a dermatology perspective, the presence of persistent adult acne in a woman with irregular menstruation or other hyperandrogenic features should prompt consideration of PCOS[14,24]. Once PCOS is established, clinicians should ensure appropriate metabolic risk assessment rather than focusing solely on androgen suppression or topical acne therapy[17,18].

The review also exposes an important research gap. Very few studies simultaneously use validated acne severity scales, standardized PCOS diagnostic criteria, standardized insulin-resistance measures, and multivariable adjustment for BMI and androgen concentrations[2-7]. This methodological inconsistency currently limits the precision of any meta-analytic estimate.

Clinical Implications

Routine extensive metabolic testing is not justified for every patient with mild acne, but women with acne and confirmed PCOS constitute a higher-risk endocrine population. Fasting glucose, HbA1c, lipid profile, and oral glucose tolerance testing should be considered according to PCOS guidelines and individual risk[18]. HOMA-IR can support research and metabolic characterization but should not be interpreted as a universally standardized diagnostic test in isolation[19,22].

Lifestyle optimization, weight management where appropriate, dietary quality, regular physical activity, and treatment of impaired glucose metabolism are central components of PCOS management[17,18]. Metformin may improve insulin sensitivity and reduce androgenic drive in appropriately selected patients, but the current evidence does not support prescribing insulin-sensitizing therapy solely to treat acne in the absence of a broader PCOS indication[14,18].

Strengths and Limitations

A principal strength of this review is its narrow focus on the relationship between insulin resistance and acne

severity in women with established PCOS rather than the broader prevalence of acne in PCOS or insulin resistance in acne populations without PCOS. It also distinguishes fasting HOMA-IR from post-load glucose abnormalities and integrates androgenic confounding.

The main limitation is the small directly relevant evidence base. Acne-severity scales, PCOS criteria, insulin-resistance thresholds, and metabolic assays differed substantially between studies[2-7]. Several reports presented only subgroup or correlation data rather than complete means and standard deviations[2-7]. For this reason, the quantitative results should be regarded as limited and provisional until raw study-level extraction is completed. Publication bias could not be reliably assessed with such a small number of directly comparable studies.

Future Research

Future prospective studies should use standardized Rotterdam-based PCOS phenotyping, validated acne scores such as GAGS, fasting insulin and glucose, HOMA-IR, 75-g oral glucose tolerance testing, HbA1c, BMI, waist circumference, SHBG, total and free testosterone, DHEAS, and clearly documented acne treatments[5,15,18,19]. Longitudinal studies should determine whether improvement in insulin sensitivity precedes improvement in acne severity independently of changes in androgen concentrations.

Conclusion

Insulin resistance appears to be an important modifier of PCOS-associated acne, but acne severity is not determined by HOMA-IR alone. The strongest direct evidence indicates that glucose dysregulation, particularly abnormal 2-hour glucose, is associated with more severe acne, while DHEAS and other androgenic variables simultaneously influence clinical expression. Approximately one-third of women with both acne and PCOS in a North Indian cohort met a study-defined criterion for insulin resistance. These findings support a metabolic-androgenic model in which insulin resistance amplifies, rather than solely causes, acne in PCOS.

Women with persistent or severe acne and PCOS should therefore be assessed in the context of their overall metabolic risk, especially when obesity, acanthosis nigricans, menstrual dysfunction, dyslipidemia, or a family history of diabetes is present. More standardized prospective evidence is needed before acne severity can be used as an independent surrogate marker for insulin resistance.

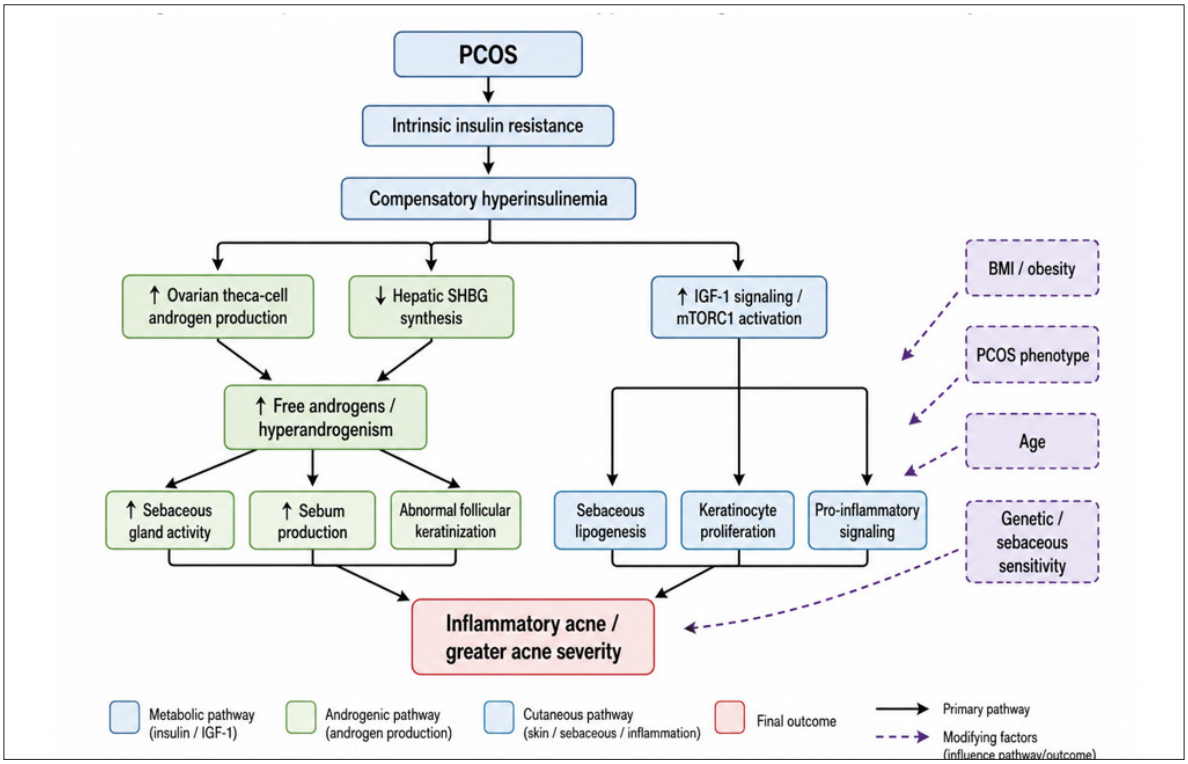

Figure 5. Proposed insulin resistance–hyperandrogenism–acne pathway in PCOS.
Conceptual model illustrating the potential mechanisms linking intrinsic insulin resistance and compensatory hyperinsulinemia with increased ovarian androgen production, reduced hepatic sex hormone-binding globulin (SHBG) synthesis, enhanced insulin/IGF-1–mTORC1 signaling, sebaceous lipogenesis, follicular keratinization, and inflammatory pathways contributing to acne development and severity in women with PCOS. Modifying factors include BMI/obesity, PCOS phenotype, age, and genetic or sebaceous sensitivity.

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Conflict of Interest

The authors declare no conflict of interest.

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