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Insulin Resistance in Polycystic Ovary Syndrome and Its Association with Type 2 Diabetes Mellitus: A Systematic Review

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ABSTRACT

Aims: This systematic review aimed to investigate the relationship between polycystic ovary syndrome (PCOS), insulin resistance (IR), and type 2 diabetes mellitus (T2DM), with particular emphasis on the molecular mechanisms underlying impaired insulin signaling and metabolic dysfunction.

Methods: A literature search was conducted in PubMed, Scopus, Web of Science, and Google Scholar to identify relevant English-language publications from 2012 to 2026. Human studies, including original research articles, reviews, systematic reviews, and meta-analyses addressing PCOS-associated IR, glucose metabolism, T2DM, and related molecular pathways, were considered.

Findings: The reviewed evidence indicates that IR and compensatory hyperinsulinemia are major metabolic features of PCOS. Hyperinsulinemia may stimulate ovarian androgen production, aggravate hyperandrogenism, and contribute to impaired follicular development and ovulatory dysfunction. Alterations in insulin-related signaling pathways, particularly the PI3K/AKT pathway and downstream glucose transport mechanisms, may contribute to metabolic disturbances. Furthermore, women with PCOS have an increased susceptibility to impaired glucose regulation and T2DM, with obesity and ethnicity potentially influencing the extent of this risk.

Conclusion: IR represents an important link between the reproductive and metabolic manifestations of PCOS and contributes to increased susceptibility to T2DM. Integrating metabolic assessment into PCOS management and investigating the underlying molecular mechanisms may improve early identification of metabolic risk and support more targeted preventive and therapeutic strategies.

Keywords: Polycystic Ovary Syndrome; Insulin Resistance; Hyperinsulinemia; Type 2 Diabetes Mellitus; Insulin Signaling

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Introduction

Infertility is a major global reproductive health concern that extends beyond physical impairment, often affecting individuals' psychological, social, and economic well-being. It is defined as the failure to achieve pregnancy after at least 12 months of regular, unprotected sexual intercourse. Its etiology involves a complex interaction of environmental, lifestyle-related, and genetic factors, including advanced age, smoking, medication exposure, inherited disorders, and chromosomal abnormalities. Infertility may result from male, female, combined, or unexplained causes. In women, major contributing factors include ovulatory dysfunction, tubal abnormalities, reproductive tract disorders, and genetic or chromosomal alterations [1,2]. Polycystic ovary syndrome (PCOS) is a heterogeneous endocrine disorder affecting a substantial proportion of women of reproductive age worldwide [3]. Its estimated prevalence ranges from 4% to 21%, depending on the diagnostic criteria applied [4]. In 2003, the Rotterdam consensus established diagnostic criteria based on the presence of at least two of three features: hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology (defined by at least 12 follicles or an ovarian volume exceeding 10 mL) [5,6]. PCOS is frequently associated with metabolic disturbances, particularly insulin resistance and impaired insulin signaling, which substantially increase the risk of developing type 2 diabetes mellitus (T2DM). Beyond its reproductive manifestations, PCOS is characterized by hormonal and metabolic abnormalities, including disrupted insulin secretion and action, increased androgen production, and impaired ovulatory function. Insulin resistance and compensatory hyperinsulinemia play central roles in this metabolic dysfunction, potentially aggravating hyperandrogenism and contributing to long-term metabolic complications. Women with PCOS are therefore more susceptible to glucose intolerance, impaired glycemic regulation, and T2DM. These metabolic disturbances may coexist with menstrual irregularities, anovulation, and reduced fertility, highlighting the close relationship between reproductive dysfunction and metabolic abnormalities in PCOS [7,8]. Insulin resistance is a central metabolic feature of PCOS, linking reproductive dysfunction to an increased risk of type 2 diabetes mellitus. Impaired insulin signaling can promote ovarian androgen excess, disrupt follicular development, and contribute to anovulation and infertility. Moreover, persistent metabolic disturbances may impair glucose regulation and increase susceptibility to T2DM, highlighting the importance of understanding the interplay between insulin resistance and metabolic complications in women with PCOS.

Information & Methods

This study was designed as a systematic review to explore the association between polycystic ovary syndrome (PCOS), insulin resistance, and type 2 diabetes mellitus (T2DM), with particular emphasis on the molecular mechanisms involved in insulin signaling and metabolic dysfunction. A comprehensive literature search was conducted using PubMed, Scopus, Web of Science, and Google Scholar to identify relevant publications published between January 2012 and November 2026. The search strategy incorporated combinations of keywords and relevant Medical Subject Headings (MeSH), including "polycystic ovary syndrome," "PCOS," "insulin resistance," "hyperinsulinemia," "type 2 diabetes mellitus," "insulin signaling," "PI3K/AKT pathway," and "metabolic dysfunction." Publications were considered eligible if they addressed the relationship between PCOS and insulin resistance or T2DM, investigated metabolic abnormalities associated with PCOS, or examined molecular mechanisms and signaling pathways involved in insulin resistance. Human studies published in English between 2012 and 2026 were considered. Original research articles, review articles, and systematic reviews or meta-analyses relevant to the objectives of this study were also considered. Studies involving exclusively animal or in vitro models, publications in languages other than English, duplicate records, and articles without relevant information addressing the review objectives were excluded.

Risk Factors and Etiology of PCOS

Polycystic ovary syndrome is a complex and multifactorial disorder whose precise etiology remains incompletely understood. Familial clustering and a higher prevalence among first-degree relatives of affected women suggest a substantial genetic contribution. Current evidence indicates that PCOS develops through interactions among genetic, epigenetic, and environmental factors [9].

Environmental factors: Maternal obesity, smoking, psychological stress, hypertension, diabetes, and excessive androgen exposure during pregnancy may influence fetal development and contribute to an increased susceptibility to metabolic and reproductive abnormalities later in life. These alterations may include insulin resistance, impaired glucose tolerance, and hyperandrogenism [10].

Epigenetic factors: Epigenetic mechanisms, including DNA methylation, histone modifications, and non-coding RNA regulation, can alter gene expression without changing the underlying DNA sequence. Abnormal DNA methylation patterns in genes involved in metabolic regulation, such as LEPR and ADIPOR2, have been reported in women with PCOS, suggesting a potential role for epigenetic dysregulation in disease development [11].

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Genetic factors: The genetic basis of PCOS is highly heterogeneous and involves multiple susceptibility genes rather than a single causative mutation. Genome-wide association studies (GWAS) have identified several genetic loci associated with reproductive and metabolic pathways. Among the reported candidate genes are CYP21, CYP19, INS, IRS1, IRS2, and FSHR, which are involved in steroidogenesis, insulin signaling, and gonadotropin regulation [12].

Mechanisms of Insulin Resistance

Insulin is a key metabolic regulator that coordinates nutrient utilization and storage across multiple tissues. When target cells become less responsive to this hormone, a condition known as insulin resistance develops, disrupting intracellular signaling and metabolic functions in both classical insulin-sensitive tissues, including the liver, skeletal muscle, and adipose tissue, and non-classical targets such as the ovaries and pituitary gland [13].

Under normal physiological conditions, insulin maintains glucose homeostasis by suppressing hepatic glucose production through the inhibition of gluconeogenesis and glycogenolysis while promoting glycogen synthesis. This inhibitory effect on gluconeogenesis is partly mediated by the suppression of FOXO1 transcriptional activity. Insulin also facilitates glucose uptake in skeletal muscle and adipose tissue, thereby maintaining normal blood glucose levels. Beyond glucose regulation, this hormone plays a major role in lipid metabolism by stimulating lipid synthesis in hepatic and adipose cells. In the liver, insulin activates sterol regulatory element-binding protein 1c (SREBP-1c), a transcriptional regulator that promotes lipogenesis. Dysregulation of these pathways under insulin-resistant conditions may contribute to excessive hepatic lipid accumulation and the development of metabolic dysfunction-associated steatotic liver disease (MASLD).

Similarly, insulin regulates lipid storage in adipocytes through IRS1/AKT signaling, which promotes de novo lipogenesis and suppresses triglyceride breakdown. However, impaired insulin responsiveness weakens this regulatory effect, resulting in increased lipolysis and the release of free fatty acids into circulation. The resulting elevation in circulating lipids can further aggravate metabolic disturbances and contribute to the progression of insulin resistance (Fig.1) [14].

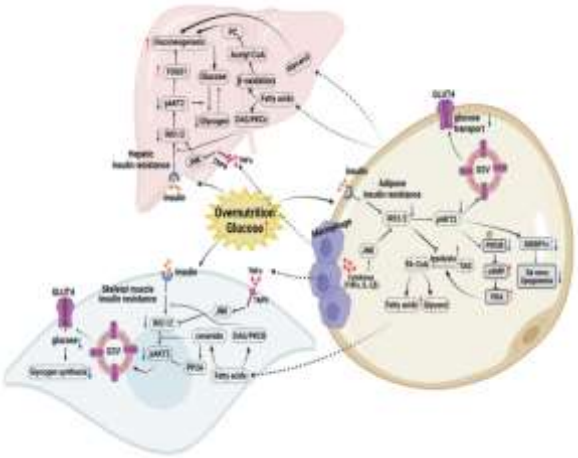


Figure1. Effects of Insulin Resistance on Different Tissues [14]

Role of Insulin Resistance in Polycystic Ovary Syndrome

Insulin resistance (IR) and its compensatory response, hyperinsulinemia, are widely recognized as major metabolic abnormalities contributing to the pathophysiology of PCOS. The association between hyperinsulinemia and hyperandrogenism in women with PCOS was first reported in 1980, prompting further investigations into the potential role of impaired insulin sensitivity in the development of this disorder. Despite substantial evidence supporting this relationship, current diagnostic criteria for PCOS primarily rely on reproductive and endocrine features, including hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology, rather than metabolic indicators of glucose regulation. Estimates suggest that insulin resistance affects up to 75% of women with PCOS, although its prevalence varies according to diagnostic methods and population characteristics. Similar to the metabolic changes observed in type 2 diabetes mellitus (T2DM), insulin resistance may initially be accompanied by compensatory hyperinsulinemia, which helps maintain normal blood glucose concentrations. Over time, however, pancreatic β-cell function may become insufficient to compensate for the increased insulin demand, leading to impaired glucose tolerance, prediabetes, or T2DM in susceptible individuals [15,16]. The relationship between insulin resistance and hyperandrogenism in PCOS is bidirectional. Compensatory hyperinsulinemia can stimulate androgen production by ovarian theca cells and enhance the effects of luteinizing hormone (LH), thereby aggravating hyperandrogenism. In addition, elevated insulin levels suppress hepatic production of sex hormone-binding globulin (SHBG), increasing the concentration of biologically available circulating testosterone. Insulin resistance is also associated with alterations in hypothalamic–pituitary–ovarian

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regulation and increased inflammatory activity, both of which may contribute to reproductive and metabolic disturbances. Collectively, these mechanisms may increase the long-term susceptibility of women with PCOS to T2DM and cardiovascular complications [17].

Insulin Signaling Pathway and Its Impairment in PCOS

Insulin resistance in PCOS involves impaired intracellular responses to insulin, particularly in pathways responsible for glucose utilization and metabolic regulation. Insulin, a peptide hormone released by pancreatic β-cells, regulates several cellular functions, including glucose uptake, glycogen and protein synthesis, and cell growth and survival. These effects are mediated through insulin receptor (INSR) activation and the subsequent recruitment and phosphorylation of intracellular insulin receptor substrates (IRS), which initiate downstream signaling cascades. Among these, the PI3K/AKT/mTOR pathway is particularly important in metabolic regulation and has been implicated in the pathophysiology of PCOS.

Following insulin binding to INSR, activation of IRS proteins stimulates PI3K and subsequently AKT, facilitating glucose transport and intracellular glucose utilization. In skeletal muscle and adipose tissue, AKT promotes the translocation of glucose transporter 4 (GLUT4) to the plasma membrane, thereby enhancing cellular glucose uptake. AKT also supports glycogen formation by inhibiting glycogen synthase kinase 3 (GSK3), which normally suppresses glycogen synthase activity. Consequently, alterations in this signaling cascade can compromise insulin-mediated glucose disposal and contribute to metabolic abnormalities associated with PCOS [18,19]. Given its involvement in insulin responsiveness and cellular metabolism, the PI3K/AKT/mTOR axis has attracted attention as a potential target for investigating therapeutic strategies in PCOS [20].

Type 2 Diabetes Mellitus and Risk Factors

Type 2 diabetes mellitus (T2DM) is a major metabolic disorder characterized by persistent hyperglycemia resulting from progressive pancreatic β-cell dysfunction, commonly accompanied by insulin resistance. Impaired insulin signaling reduces glucose uptake in peripheral tissues and disrupts metabolic homeostasis, while inadequate compensatory insulin secretion further contributes to elevated blood glucose levels. Several molecular components involved in insulin signaling, including the insulin receptor, IRS1/IRS2, and GLUT4, have been implicated in the development of T2DM [21,22].

The onset of T2DM is influenced by interactions between genetic susceptibility and environmental factors. Physical inactivity, unhealthy dietary patterns, smoking, and excess body weight are among the established modifiable risk factors, whereas genetic and epigenetic alterations may affect insulin secretion and sensitivity. These mechanisms are particularly relevant to PCOS, in which insulin resistance is frequently present and may increase susceptibility to impaired glucose regulation and eventual T2DM. Although estrogen has metabolic effects that can influence insulin sensitivity, its relationship with glucose homeostasis is complex and should not be interpreted as a direct or exclusive determinant of diabetes risk [23,24].

Assessment of Type 2 Diabetes Risk in Women with PCOS

Women with polycystic ovary syndrome (PCOS) have a higher susceptibility to glucose metabolism abnormalities and type 2 diabetes mellitus (T2DM) than women without the syndrome. Previous studies have reported an increased prevalence of T2DM in this population, with some analyses suggesting an approximately fourfold higher risk compared with controls [25-27]. This association is largely attributed to the frequent coexistence of insulin resistance and compensatory hyperinsulinemia in PCOS, which may progressively impair glucose homeostasis and increase the likelihood of developing T2DM. The coexistence of these metabolic disturbances may also aggravate reproductive complications, potentially affecting fertility and pregnancy outcomes [28].

Given this increased metabolic risk, early identification of insulin resistance in women with PCOS is important, even before overt hyperglycemia develops. Several approaches are available to assess insulin sensitivity; among them, the homeostasis model assessment (HOMA) provides a practical method based on fasting glucose and insulin concentrations. HOMA-IR is used to estimate systemic insulin resistance, whereas HOMA-β provides an estimate of pancreatic β-cell function. Although these indices are useful in metabolic research and clinical assessment, they should be interpreted alongside other clinical and biochemical findings rather than used as standalone diagnostic tests [29,30].

Discussion

Polycystic ovary syndrome (PCOS) is increasingly recognized as a condition involving interconnected reproductive and metabolic disturbances, with insulin resistance representing one of the major mechanisms underlying this relationship. Impaired insulin sensitivity and compensatory hyperinsulinemia may contribute to ovarian androgen excess and abnormal follicular development, thereby aggravating ovulatory dysfunction and reproductive complications. The interaction between hyperandrogenism and insulin resistance may also create a self-reinforcing metabolic environment that contributes to the persistence of PCOS manifestations [13,15,16].

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At the molecular level, alterations in insulin-related signaling pathways provide a potential explanation for the metabolic abnormalities observed in PCOS. The PI3K/AKT pathway plays an essential role in mediating insulin responses, including glucose uptake and intracellular metabolic regulation. Impairment of this pathway, together with changes in insulin receptor substrate activity and downstream signaling components, may contribute to reduced insulin responsiveness in different tissues. Evidence also suggests that abnormalities in PI3K-related signaling may influence autophagy and apoptosis, indicating that insulin resistance in PCOS extends beyond glucose metabolism and may affect cellular functions relevant to ovarian physiology [18-20].

The metabolic consequences of PCOS are particularly important because affected women appear to have an increased susceptibility to impaired glucose tolerance and T2DM. Evidence from systematic reviews and meta-analyses indicates that the prevalence of glucose abnormalities and T2DM in women with PCOS is influenced by factors such as ethnicity and obesity, although an increased metabolic risk has been consistently reported [14].Furthermore, the relationship between PCOS and T2DM may be bidirectional, as studies have also investigated the occurrence of PCOS among women with established T2DM [23]. These findings emphasize the importance of considering metabolic health as an integral component of PCOS assessment rather than focusing exclusively on reproductive symptoms.

From a clinical perspective, identifying metabolic disturbances before the development of overt diabetes may provide an opportunity for earlier intervention [31]. Assessment of glucose regulation and insulin resistance can therefore contribute to the identification of women who may require closer metabolic follow-up. Although indices such as HOMA-IR are useful in research and clinical investigations, their interpretation should take into account differences in population characteristics and assessment methods [29,30]. In addition, variations in age, body mass index, ethnicity, and PCOS phenotype should be considered when comparing metabolic findings across studies.

Despite progress in understanding the relationship between PCOS and insulin resistance, further research is needed to clarify the molecular events that determine individual susceptibility to metabolic complications. In particular, investigations into regulatory mechanisms involving insulin signaling, autophagy, apoptosis, and gene-expression networks may help identify potential biomarkers and therapeutic targets. Longitudinal studies involving well-characterized populations are also necessary to determine how early metabolic abnormalities evolve into impaired glucose tolerance and T2DM.

Conclusion

Insulin resistance is a major metabolic feature of PCOS that connects reproductive dysfunction with an increased susceptibility to T2DM. Through disturbances in insulin signaling, altered glucose metabolism, and increased ovarian androgen production, this condition may contribute to both infertility and long-term metabolic complications. Early assessment of metabolic status, alongside appropriate reproductive evaluation, is therefore important in the clinical management of women with PCOS. Further studies focusing on the molecular mechanisms of insulin resistance and its regulatory pathways may improve understanding of disease progression and support the development of more effective strategies for early risk identification and individualized management.

Ethical Considerations

This study is a review article based on the review and analysis of previously published sources and studies. In this study, no intervention or primary data collection was performed from patients, and no personal or identifiable information of patients was used; therefore, the principles of confidentiality, privacy, and individual rights were respected, and obtaining a code of ethics was not necessary for this study.

Conflict of Interests

There was no conflict of interest in this study.

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