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The bridge between adiposity and insulin: Unmasking resistin's role in gestational diabetes

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Abstract

Impaired glucose tolerance due to increased insulin resistance and/or insufficient pancreatic β-cell compensation is the hallmark of gestational diabetes mellitus (GDM), an important metabolic disease of pregnancy. Among the various adipokines implicated in the pathogenesis of GDM, resistin has attracted considerable attention for its potential role in insulin resistance and glucose metabolism. This review aimed to evaluate the available evidence on the association of resistin with the development of GDM and its potential utility as a predictive biomarker. Available evidence suggests that resistin, secreted by both adipose tissue and the placenta, may contribute to pregnancy-induced insulin resistance and dysglycemia. Several studies have reported elevated maternal resistin concentrations in women with GDM, particularly during the second and third trimesters, whereas others found no significant association. These inconsistencies may reflect differences in study populations, obesity status, gestational age, and analytical methods. Nevertheless, prospective studies indicate that elevated resistin levels may precede the diagnosis of GDM, highlighting its potential as an early biomarker. Further large-scale prospective studies are required to clarify the precise role of resistin in GDM pathophysiology and prediction. *[J Assoc Clin Endocrinol Diabetol Bangladesh,* July 2026; 5 (2): e90630*]*

Keywords: Resistin, Gestational diabetes, Insulin resistance, Normal glucose tolerance

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Introduction

Gestational diabetes (GDM) results from complex and variable interactions among numerous factors, including pregnancy-induced factors, genetic aberrations, adipocytokines, inflammatory factors, autoimmune and environmental factors, etc.[1] The main underlying mechanism behind developing GDM is insulin secretory failure in the context of increased insulin resistance (IR).[2] In most cases, these abnormalities exist before pregnancy and can develop, increasing the risk of T2DM after pregnancy.[3] Insulin sensitivity was shown to be 20% lower in pregnant women with GDM compared to those with normal glucose tolerance (NGT).[4] As a result, GDM may be a temporary 'unmasking' of pre-existing latent metabolic abnormalities. In this review, we only highlight the role of one adipocytokine, resistin, in GDM.

Methods

We searched Google Scholar, PubMed, and ResearchGate using the keywords: gestational diabetes or GDM or hyperglycemia in pregnancy, resistin, adipose tissue, insulin resistance, and trimester variations in pregnancy, without any time or type restrictions, up to 30 April, 2026. 65 articles in PubMed and 47 articles in Google Scholar were identified. All abstracts were scrutinized; relevant original and review articles were included in this review. Only studies in humans were included. Duplicates, editorials, and other non-specified articles were excluded. To identify additional relevant research, we manually checked the reference lists of all listed papers and pertinent reviews.

Significance of adipose tissue in the GDM pathogenesis

The discovery of adipocyte-derived hormones, known as "adipokines," and their roles in regulating maternal metabolism, gestational IR, and β-cell dysfunction have ushered in a new era in the study of the pathophysiology of GDM. Changes in the expression and production of various adipokines, including resistin, leptin, retinol binding protein 4 (RBP-4), adiponectin, visfatin, apelin, vaspin, and omentin, have been reported in GDM patients.[5] Both higher concentrations of resistin and adipocyte fatty acid binding protein (AFABP) have been found to be positively correlated with insulin resistance in GDM.[6,7]

Adipocytokines: a new insight into understanding GDM

Although the relationship among obesity, insulin resistance, and GDM is well recognized, the key processes underlying this relationship remain poorly understood. Before 1950, adipose tissue was widely viewed as a passive reservoir for triglyceride storage and fatty acid release.[8] In 1987, adipose tissue was identified as a key site for the synthesis of the endocrine factor

Table-I: Different study findings of resistin levels in GDM

Study design	Sample size (case/control)	Gestational age	Major findings	Conclusion
Prospective [17]	14/14	Antenatal 11wks, 28 weeks, postnatal 8 wks	Resistin did not significantly differ between women with GDM and control women either during or after pregnancy	NS
Nested case-control study[18]	30/29	Early pregnancy (mean gestational age was 9.3 ± 2·6 weeks)	Mean resistin concentrations were not different between the two groups	NS
Prospective observational study[19]	60	11 to 13 weeks	Serum resistin is not significantly altered in GDM	NS
Meta-analysis (including 18 studies)[20]	1041/1292	All trimesters	The risk of GDM was associated with serum resistin level	-
Cross-sectional study[26]	20/20	3rd trimester	Serum resistin concentration was significantly higher in women with GDM than in controls before delivery. Serum levels of resistin significantly decreased after delivery in both the GDM group and controls	P < 0·001 for both
Cross-sectional observational study[28]	81/82, 25- healthy non-pregnant women	24 to 31 weeks	Resistin concentrations were significantly higher in the GDM than in the NGT group as well as in the non-pregnant women	P=0.047 (1st group) P<0.001 (2nd group)
Cross-sectional case-control study[30]	30/15	3rd trimester	Resistin levels were significantly higher in 3rd trimester in GDM than NGT group	P<0.01
Cross-sectional study[36]	81/86	All trimesters	Resistin is statistically and significantly higher in GDM than that of NGT group irrespective of gestational age	1st trimester (p<0.001), 2nd trimester (p<0.001), 3rd trimester (p<0.001)

GDM Gestational diabetes mellitus; NGT normal glucose tolerance; NS non-significant

adipsin and the metabolism of sex hormones.[9] The discovery of leptin in 1994, along with the subsequent identification and characterization of other bioactive peptides secreted from adipose tissue, firmly established it as an important endocrine organ.[10] Collectively, these bioactive peptides and proinflammatory cytokines are known as adipokines (leptin, adiponectin, visfatin, resistin, etc.). Leptin, resistin, and other adipokines have been linked to the emergence of insulin resistance, which underlies several harmful metabolic consequences, such as obesity, diabetes, and dyslipidemia.[11] In fact, they can modify insulin sensitivity in insulin-targeted organs either locally (autocrine/paracrine) or distally (endocrine), or they can act via neuroendocrine, autonomic, or immunological pathways.[12] Adipokines such as TNF-α, adiponectin, AFABP and leptin are considered the most likely candidates involved in the pathophysiology of GDM based on the available data.[13]

Resistin

The group of Dr. Mitchell A. Lazar first discovered Resistin in 2001.[14] Resistin is elevated in obesity, where it was initially identified as an adipokine that causes insulin resistance. According to the study, the adipokine also leads to inflammation, thrombosis, angiogenesis, smooth muscle cell dysfunction, and endothelial dysfunction.[15] Human circulating resistin levels are positively correlated with indicators of inflammation, insulin resistance, and obesity.[15] Circulating resistin in gestational diabetes has been quantified in several studies; however, the results are conflicting. These are described in **Table-I**. Most studies indicate no appreciable differences in resistin concentrations between pregnant women with GDM and healthy controls, while some studies have reported higher or lower resistin concentrations in GDM.[16] The risk prediction of GDM is not influenced by resistin, according to three independent prospective trials.[17-19] A meta-analysis linked higher maternal blood resistin levels to an increased risk of GDM, and the severity of GDM may also correlate with serum resistin levels.[20] The human placenta expresses resistin, and term placental tissue has greater resistin gene expression levels than chorionic tissue during the first trimester.[21] However, Resistin release from maternal subcutaneous adipose tissue, the placenta, fetal membranes, and skeletal muscles is not varied between patients with GDM and controls.[22]

Role of resistin in GDM

Adipose tissue is the primary source of resistin, a cysteine-rich hormone considered crucial to insulin sensitivity. Resistin lowers insulin sensitivity, increases insulin resistance, and results in postprandial hyperglycemia during pregnancy, which can lead to GDM.[23] Additionally, the placenta produces and secretes resistin into the mother's bloodstream throughout pregnancy. The placenta is a significant source of resistin during pregnancy, compared with subcutaneous and abdominal adipose tissue.[23,24] Furthermore, insulin has been shown to play a significant role in the development of insulin resistance in pregnancy by stimulating the production of resistin from the human placenta.[22] Additionally, resistin has been connected to insulin resistance in obesity and type 2 diabetes.[25] Obesity and insulin resistance are related to the development of GDM, just as in type 2 diabetes.[26] Throughout the gestational period, the level of maternal resistin also fluctuates. The significance of resistin in the pathogenesis of GDM is demonstrated by the difference in resistin levels between GDM and normal pregnancy. Additionally, resistin is increasingly important as a possible biomarker. Resistin may be a biomarker for gestational diabetes, according to some researchers who found a link between resistin and glycated hemoglobin levels in GDM.[27] In a review article, Siddique & George (2017) 'the role of resistin in GDM' presented this in a schematic presentation (Figure-1).[28]

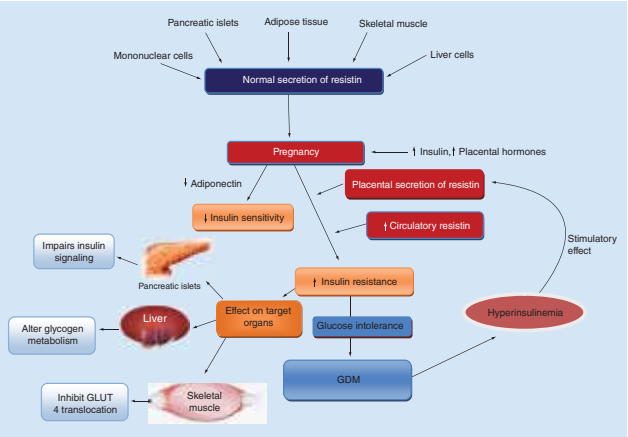

Figure-1: Schematic diagram showing the role of resistin in the development of gestational diabetes mellitus. Increased circulating resistin levels during pregnancy cause insulin resistance in insulin target organs such as the liver, skeletal muscle, and pancreatic islets. This leads to the development of gestational diabetes mellitus. The upward arrow (↑) in the text box indicates an increased level. Adopted with permission from Siddique K et al.[28]

Resistin and its association with GDM among

different trimesters of pregnancy

Compared with the nonpregnant condition, resistin levels rise during pregnancy and continue to rise up to the third trimester. It rises as gestational age advances.[29] Increased insulin resistance or increased fat deposition, particularly visceral fat during pregnancy and placental secretion, are the causes of this difference in resistin levels.[30] Additionally, the placenta secretes resistin, which is expressed more in the term placenta than in the chorionic villi of the first trimester.[31] However, when compared to normoglycaemic, healthy pregnant women in the third trimester, resistin levels are further elevated in GDM patients.[31] Compared to their matched control group, pregnancies complicated by GDM had significantly higher mean resistin values at the second and third trimesters.[7] Additionally, Bawah et al. evaluated the relationship between the development of GDM in the Ho municipality of Ghana and first-trimester leptin, resistin, visfatin, BMI, and maternal factors in a prospective case-control study. Many weeks prior to the disease's diagnosis, they discovered that resistin was independently higher in women with GDM. Additionally, they state that the increases in resistin were unrelated to parity, lipid levels, maternal age, or a family history of diabetes. These findings demonstrated that those who later developed gestational diabetes mellitus had greater serum resistin levels.[32] In contrast, at the second and third trimesters in one group and only the second trimester in another, maternal serum resistin was considerably higher in GDM women than in their matched controls.[7,29] Other researchers found that serum resistin levels did not differ significantly between pregnant women with GDM and healthy pregnant women at term, despite noticeably increased glucose and insulin levels.[33] The disparity in these results could be attributed to variations in the population under study or sampling period. It is important to note that Noureldeen et al. examined GDM patients whose gestational ages were nearly identical to those of a normal pregnancy.[7] In addition to insulin resistance being more pronounced in GDM than in control subjects, they were more obese than control during the second and third trimesters. Resistin has been shown to be down-regulated by anti-diabetic medications and up-regulated in both diet-induced and genetic obesity in vivo.[34] It was proposed that mRNA expression of resistin in adipocytes is elevated in obese individuals in humans,[34]; however, other studies failed to find any correlation between resistin expression and obesity.[35] This finding suggests that the enhanced insulin resistance associated with GDM may be significantly influenced by both obesity and elevated resistin concentrations. In a prior study, advances in gestation markedly increased maternal resistin in normal pregnant women but not in GDM.[7] As pregnancy progresses, there is an increase in the expression of the placental resistin gene.[21] Furthermore, resistin up-regulation was noted in the third trimester, which has been suggested to be involved in controlling the mother's energy metabolism during pregnancy.[21,30] According to a study conducted among pregnant women in Bangladesh, resistin levels are significantly higher in GDM than in NGT, regardless of gestational age, but are similar across the three trimesters.[36] Besides that, they discovered that while resistin levels during pregnancy are unrelated to insulin resistance or gestational weight gain, they do differ significantly from dysglycemia during pregnancy. The secretory abnormalities of pancreatic beta cells are linked to GDM in their population, since in GDM, HOMA-B is predictively associated with resistin.[36]

Role of insulin over resistin

In one study, insulin was identified as a major regulator of resistin gene expression.[37] In a separate study, insulin triggered the human placenta to secrete resistin.[38] These findings suggest that both normal pregnancy and GDM are associated with hyperinsulinemia and increased insulin resistance. Furthermore, evidence that insulin promotes resistin secretion supports a potential role for resistin in mediating the increase in insulin resistance that occurs with advancing gestation and in the greater insulin resistance observed in GDM relative to normal pregnancy.[7] Insulin probably has a biphasic effect on resistin release in humans. At low concentrations, insulin greatly increases resistin release; however, when the placenta is exposed to higher insulin concentrations, resistin release recovers to basal levels, suggesting that resistin expression is downregulated in a high-insulin medium.[33] Bawah et al. concluded that the precise role of resistin in GDM is still unknown, but women who later developed GDM had higher resistin levels between weeks 11 and 13 of pregnancy, suggesting that this adipokine may be involved in the pathophysiology of GDM and that high resistin concentration may occur before the onset or diagnosis of GDM. This may cause early insulin resistance that results in glucose intolerance and may decrease or stabilize to levels similar to those

of healthy pregnancies once GDM has started. This could account for the disparities in the findings reported by various researchers.[32]

Resistin as a predictor of GDM

The area under the receiver operating characteristic (ROC) curve (AUC) is commonly used to assess a test's or marker's accuracy. The AUC reflects how effectively resistin predicts GDM. Resistin may be a promising predictor of GDM, as suggested by a prospective study in Ghana and a cross-sectional observational study in Bangladesh.[32] In the Bangladeshi study, the optimal resistin cut-off, determined using the Youden index, was 7.01 pg/mL, yielding a sensitivity of 100% and a specificity of 84%. The reported AUC for fasting resistin was 0.856 ($p < 0.001$), demonstrating excellent predictive performance for GDM.[36] The AUC for fasting resistin is very similar to that reported in another study, which was 0.836.32 During pregnancy, resistin levels differ significantly with dysglycemia and are a reliable predictor of GDM, according to ROC curve analysis, which can be applied comparably to the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) to predict GDM.[36] However, the clinical implications of resistin as a predictive marker of GDM are not yet established. On the other hand, resistin is not currently available for routine investigation. It is costly and used only for research purposes. Therefore, more extensive research is required on a large scale across the 1st to 3rd trimesters of pregnancy and the postpartum period to determine the predictive role of resistin in GDM risk.

Conclusions

The role of Resistin in GDM has not yet been established. Available studies are controversial. Resistin may serve as a biomarker for the early detection of GDM, though more widespread studies are required for a clear understanding.

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