SYSTEMATIC REVIEW OPEN ACCESS

Molecular Insights to Adiponectin in Pregnancy Pathologies: A Systematic Review on Physiological and Clinical Pathological Perspective

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ABSTRACT

Background: Adiponectin was seen to work as an important adipokine that originated from adipocytes to regulate metabolic and inflammatory processes. The concentration of adiponectin altered throughout pregnancy, linking it to different pregnancy-related medical complications. This review aimed to investigate how adiponectin affects pregnancy pathologies at the molecular level, while examining both normal physiological events and associated medical outcomes.

Methods: A thorough analysis of research was conducted through the examination of peer-reviewed studies in the PubMed, Scopus, and Google Scholar databases from 2010 to 2024. The research included studies examining how adiponectin functions during pregnancy, about gestational diabetes mellitus (GDM), preeclampsia, insulin resistance, and hypertension. The research included experimental studies, prospective studies, cohort studies, and case-control studies. The analysis excluded research papers that failed to mention definitive outcomes regarding adiponectin and those dealing exclusively with non-pregnancy medical conditions. This systematic review included 12 studies which received quality evaluation through application of the Evidence Project Risk of Bias Tool. Results were synthesized qualitatively.

Results: Among the 118 screened studies, 34 met the eligibility criteria after full-text review. 12 were finally included in the systematic review. The sample size ranged from 34 to 2503 participants. Pregnant women typically exhibited elevated adiponectin levels during early pregnancy, but this level decreased in cases of GDM and preeclampsia. Studies have shown that adiponectin regulates three key elements for pregnancy complications, which include insulin sensitivity, endothelial function, and inflammatory processes. The connection between impaired glucose metabolism in GDM patients and reduced vasodilatory and anti-inflammatory effects of adiponectin influences preeclampsia between these conditions. Research also indicated that adiponectin influenced fetal development by affecting the placenta's ability to support fetal nutritional needs.

Discussion: The potential existence of adiponectin represented a biomarker and therapeutic target for pregnancy complications.

Keywords: Adiponectin, Pregnancy, Gestational Diabetes, Preeclampsia, Molecular Mechanisms, Inflammatory Response, Metabolic Regulation.

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Doi: https://doi.org/10.36283/ziun-pjmd14-2/069

How to cite: Shaikh SN, Memon NUN, Zuhaib R, Rathore P, Naz T, Shaikh P, et al. Molecular Insights to Adiponectin in Pregnancy Pathologies: A Systematic Review on Physiological and Clinical Pathological Perspective. Pak J Med Dent. 2025 April ;14(2): 515-523 Doi: https://doi.org/10.36283/ziun-pjmd14-2/069.

Received: Tue, February 11, 2025 **Accepted:** Thu, March 27, 2025 **Published:** Sun, April 13, 2025

INTRODUCTION

The protein adiponectin, which is mainly derived from adipocytes, functions as a critical adipokine that governs multiple metabolic processes, including insulin sensitivity, inflammation, and vascular health[1]. Recent studies have confirmed that adiponectin functions as a vital factor for managing metabolic conditions such as obesity, type 2 diabetes, and pregnancy-related health problems. During pregnancy, the body experienced extensive systemic changes that affected the management of adiponectin levels[2]. The changing levels of adiponectin were connected with distinct pregnancy issues, which included gestational diabetes mellitus (GDM), preeclampsia and preterm birth, and other complications[3].

The bodily changes that occur throughout pregnancy cause intricate modifications to metabolic and immunological processes. The regulatory functions of insulin sensitivity, immune response, and endothelial function were directly managed by adiponectin[4]. Normal pregnancy started with elevated adiponectin levels, which decreased during later pregnancy stages, particularly when complications arose. Research now focused on establishing a clear understanding of how adiponectin worked at a molecular level, since this knowledge helped scientists to study pregnancy-related disorders[5]. Gestational diabetes mellitus (GDM) appeared as a frequent pregnancy-linked metabolic abnormality, which brought rise to elevated blood glucose levels because of insulin resistance. GDM patients with reduced adiponectin levels had compromised glucose metabolism and insulin resistance[6]. The decline in adiponectin regulatory functions, which improved insulin sensitivity and controlled inflammation, served to advance the development of GDM. Adiponectin played a crucial role in endothelial function in preeclamptic patients[7]. It helped control hypertension while reducing inflammation through its anti-inflammatory properties and vasodilatory effects. Medical research indicated that low adiponectin concentrations lead to endothelial dysfunction, which is marked preeclampsia[8].

The effect of adiponectin extended to fetal development while simultaneously affecting maternal metabolic health. Changes in adiponectin regulation influenced how the placenta operates, nutrients flow and fetal growth during pregnancy[9]. The concentrations of adiponectin in maternal blood streams influenced birth weight, which could lead to intrauterine growth restriction (IUGR) or fetal overgrowth development. Studies demonstrated that adiponectin held a critical position for managing pregnancy-related health for mothers and their growing fetuses[10].

Although research had expanded significantly, researchers still needed to understand fully how adiponectin operated at the molecular level to affect pregnancy-related diseases. Visible evidence suggested that adiponectin served both as an assessment marker and a therapeutic focus in managing pregnancy medical issues. This study conducted a comprehensive analysis of adiponectin's molecular mechanisms and its medical effects in pregnancy diseases while focusing on its influence in GDM diagnosis, preeclampsia progression, and fetal development. Industrial understanding of these molecular effects might help develop new therapeutic approaches that benefit both mothers and their fetuses during pregnancy.

METHODS

The researchers conducted this systematic review to understand how adiponectin affects pregnancy-related health conditions, specifically on molecular processes and clinical outcomes. The review implemented PRISMA guidelines to ensure complete reporting of research findings. The literature search was conducted using three databases: PubMed, Scopus, and Google Scholar, and included studies from 2010 up to 2024. The research design used Boolean operators with "adiponectin", "gestational diabetes", "preeclampsia", and "fetal growth" along with additional designated keywords. Researchers could only evaluate studies that appeared in English sources because they eliminated non-English reports due to translation restrictions.

The research included studies examining how adiponectin functions during pregnancy about gestational diabetes mellitus (GDM), preeclampsia, insulin resistance, and hypertension. The research included experimental studies, prospective studies, cohort studies, and case-control studies. The primary

outcomes for which studies were examined include: Insulin resistance, endothelial dysfunction, and fetal growth. And the data was extracted for study details, population size, pathology, mechanism and key findings. All of the measures, such as time points and analyses, which were explicitly tied to adiponectin's role in the specified outcomes were prioritized. The analysis excluded research papers that failed to mention definitive outcomes regarding adiponectin and those dealing exclusively with non-pregnancy medical conditions. The examination of full-text documents indicated that 34 studies fulfilled the eligibility criteria from an initial screening of 118 articles. 12 articles were included in the systematic review table. Studies were grouped based on outcomes (e.g., GDM, preeclampsia) for synthesis. Assumptions were included for standardized diagnostic criteria for maternal conditions (e.g., IADPSG for GDM) if they were unreported or missing. Quality assessment of selected articles employed a standardized assessment tool, i.e. Evidence Project Risk of Bias Tool. Two independent reviewers executed both the screening and data extraction tasks but resolved any differing opinions with the help of a third reviewer for consultation. As research utilized narrative synthesis, it was ensured that no study lacked data. If there was missing data present, either assumptions were made according to International Association of Diabetes and Pregnancy Study Groups (IADPSG) criteria or authors of the studies were contacted.

The research team extracted pertinent information from selected studies, such as study design, population, diagnosis of maternal conditions under examination, adiponectin functions, and study results. The authors combined data findings in a narrative framework, as the research presented heterogeneous results. The analysis used descriptive statistics for specific instances and conducted a qualitative synthesis to explain adiponectin's molecular mechanisms in insulin sensitivity control, inflammation reduction, vascular operation, and fetal developmental processes. For insulin resistance, mean differences in adiponectin levels and relations with insulin sensitivity were shown. Endothelial dysfunction was explored by presenting mean differences in adiponectin levels among preeclampsia and control populations and preeclampsia odds ratios. Fetal growth outcomes were analyzed using the correlation coefficients between the concentrations of maternal adiponectin and fetal characteristic indices such as birth weight. Data was tabulated using Excel 2020 and analyzed by theme. The research maintained acceptable ethical standards because all selected studies followed established ethical guidelines. The analysis groups research findings into major themes, demonstrating how adiponectin participates in gestational diabetes mellitus, preeclampsia, fetal growth, and other pregnancy complications. GRADE framework was used for the assessment of the certainty of evidence. Scientific research has shown adiponectin's potential as a biomarker for predicting pregnancy-related disorders and its therapeutic applications. However, more clinical evaluation is necessary for validating its use in managing pregnancy pathologies.

RESULTS

A total of 118 studies were gathered by searching through electronic databases such as PubMed, Scopus, and Google Scholar. 10 duplicates were removed leaving 108 articles to be screened via titles and abstract. 45 articles were excluded as the data was either irrelevant or insufficient. 63 were selected for full-text screening but 29 were unavailable, leaving 34 for full-text eligibility. In the last only 12 studies were left to be put into a systematic review as shown in **Figure 1.**

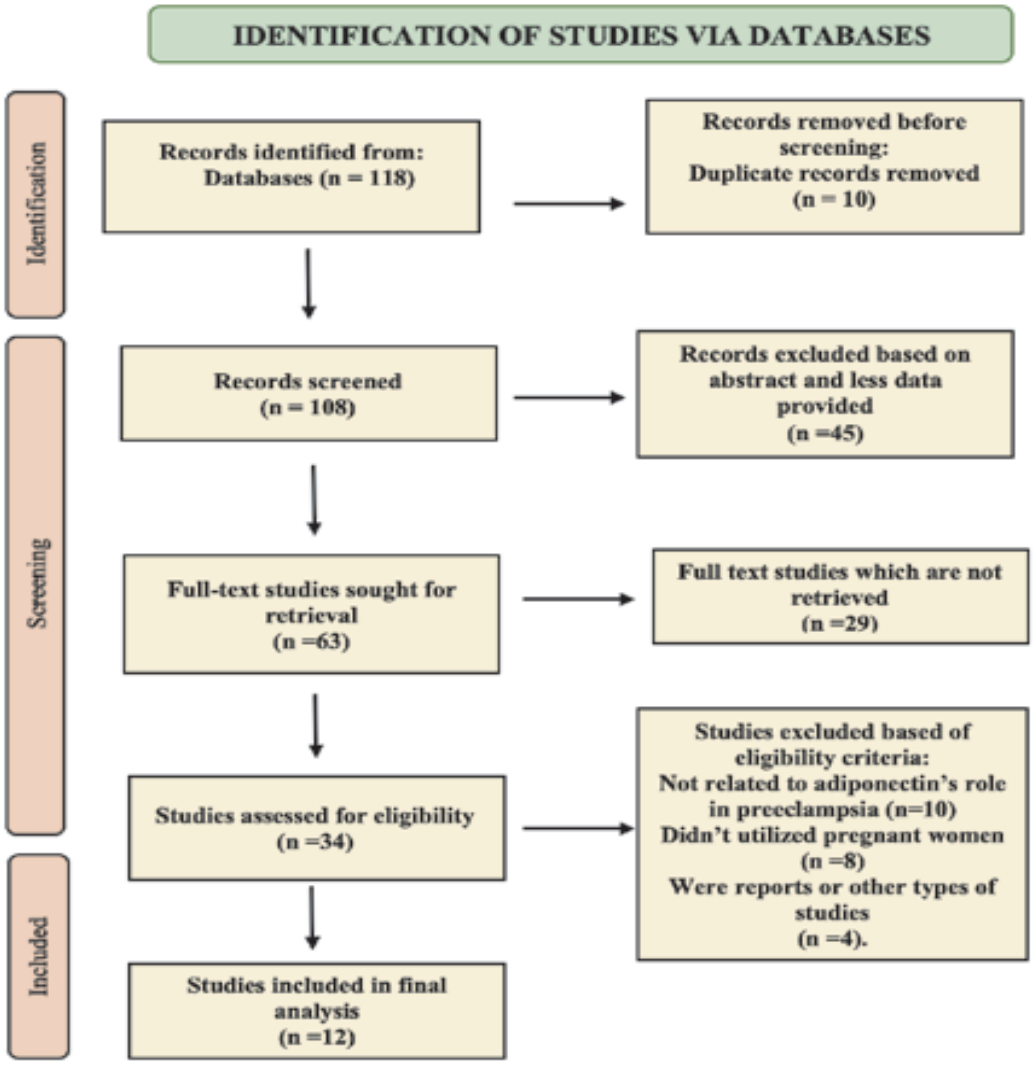

Figure 1: PRISMA Flow Diagram Demonstrating Filtering of Studies According to Inclusion and Exclusion Criteria. 16 Studies Were Selected for Systematic Review

The included studies consisted of 7 case-control, three cohort, one prospective and one experimental study. The sample size ranged from 34 to 2503 participants. Adiponectin was measured using following methods: ELISA (75%, n=9) and molecular assays (25%, n=3). The focus of outcomes was insulin resistance, endothelial dysfunction and fetal growth.

Risk of bias was assessed using Evident Project Risk of Bias Tool, where it identified high selection bias in eight studies due to utilization of single center recruitment, moderate risk in six studies due to confounder and high missing data risk in 3 studies.

All studies resulted in low measurement bias. Key findings highlighted that there were reduced levels of adiponectin in gestational diabetes mellitus (GDM) and preeclampsia. Bawah et al. (2020) reported the lower levels of adiponectin during GDM, Xuan et al. (2019) noted significantly low adiponectin in severe preeclampsia compared to controls (5.08 ± 1.13 mg/L vs 11.67 ± 3.53 mg/L, $p < 0.001$). Adiponectin/leptin ratios) and placental STAT signaling were distinguished as predictive biomarkers for preeclampsia and function of placenta [11,17]. Fetal outcomes however were not consistent i.e. three studies linked low maternal adiponectin to intrauterine growth restriction while one study found no association [19,21].

Some studies were excluded despite them being closer to inclusion criteria e.g. Downs et al. (2024) was excluded because it extensively studied adiponectin's role in function of placenta and fetal growth without studying the details of molecular analysis of GDM or preeclampsia. Lopez-Jaramillo et al. (2018) study focused primarily on obesity and preeclampsia but lacked the specific insights into adiponectin's molecular mechanisms. Mihai et al. (2024) examined the role of adipokines in gestational diabetes but did not studied the adiponectin's signaling pathways, making it ineligible. The heterogeneity among studies resulted due to

differences in study design, population characteristics (sample size, BMI, ethnicity), methods of measuring adiponectin levels (ELISA, molecular assays) and the timings of assessment during pregnancy. Unfavorable diagnostic standards for GDM and preeclampsia were also a factor. Despite these factors, heterogeneity was discussed narratively.

GRADE assessments rated evidence of certainty as moderate for GDM associations, low to moderate for preeclampsia and very low for fetus-related outcomes due to heterogeneity in methodologies and measurements. **Table 1** presents a systematic review of various studies exploring the role of adiponectin in pregnancy-related pathologies such as gestational diabetes mellitus (GDM), preeclampsia (PE), and fetal growth. The table also summarizes the significance of adiponectin levels and adiponectin/leptin ratios as predictive biomarkers, highlighting the varying results across different studies.

Table 1: Systematic Review of Adiponectin's Role in Pregnancy Pathologies: Molecular Mechanisms and Clinical Implications

Authors, Year	Study Design	Study Population	Pregnancy Pathology	Adiponectin Role/Mechanism	Key Findings
Rao S, 2021[11]	Prospective	60 PE, 60 controls	Pre-eclampsia	Adiponectin levels slightly higher in PE, not significant; leptin elevated; adiponectin/leptin ratio lower	Leptin (>23.3 ng/ml) and adiponectin/leptin ratio (<0.153) were significant PE biomarkers
Ghazali, 2023[12]	Case-control	50 PE (14 severe, 36 mild), 50 controls	Pre-eclampsia severity	Adiponectin higher in PE, no correlation with severity	PE had higher adiponectin, homocysteine, lower B12, and folic acid; no severity relation
Zhang, 2023[13]	Retrospective	118 severe PE, 90 controls	Severe PE, adverse outcomes	Adiponectin downregulated, negatively correlated with umbilical artery Doppler indices.	Doppler + adiponectin predicted adverse PE outcomes (AUC: 0.6545, specificity: 60.27%, sensitivity: 60.00%)
Song, 2016[14]	Case-control	74 PE, 79 controls	PE and adipokines	No significant adiponectin difference	Leptin, resistin were higher in PE; leptin correlated with BMI, inversely with birth weight; BMI ≥ 28 and leptin were independent PE risk factors.
Thagaard, 2019[15]	Cohort	2503 pregnancies (93 PE)	PE, adipocytokines, BMI	Lower adiponectin in PE among obese women, no association in normal-weight women	Severe obesity: lower adiponectin, leptin in 1st trimester; AUC 0.73 for PE prediction (sensitivity: 72.9%, specificity: 49%)
Bawah, 2020[16]	Case-control	90 PE, 100 controls	First-trimester adipokines, lipids in PE	Adiponectin predicted PE after adjusting for BMI, age, parity, and history	HDL was lower in PE; adiponectin, leptin, resistin, and visfatin differed; resistin best predictor after BMI adjustment, and adiponectin best after multiple adjustments
Dong, 2018[17]	Experimental	52 PE, 30 controls	Adiponectin in PE, molecular mechanism	Adiponectin regulates trophoblast function via p38 MAPK-STAT5	Adiponectin mRNA lower in PE; p-p38 high, p-STAT5 low; p-p38 negatively correlated with adiponectin, p-STAT5 positively correlated
Poniedziałek-Czajkowska, 2019[18]	Case-control	34 GH, 32 controls	Adipokines, endothelial dysfunction in GH	No significant adiponectin difference	Adiponectin is stable, suggesting a protective pregnancy mechanism against obesity effects
Perichart-Perera, 2017[19]	Prospective cohort	177 Mexican women	Obesity, weight gain, PE	Adiponectin is lower in overweight/obese women, linked to metabolic risk	Adiponectin is stable but lower in overweight/obese

Tang, 2019[21]	Case-control	74 PE (51 early-onset, 23 late-onset), 79 controls	PE	No significant adiponectin difference	Adiponectin is not linked to PE; leptin, resistin are higher in PE
Fisher, 2025[22]	Cohort	275 women (35 HDP, 55 complications, 19 recurrent, 186 controls)	HDP, preterm birth, GDM	Lower adiponectin linked to higher insulin resistance, inflammation post-complications	Women with complications had higher BP, insulin resistance, and leptin 9 years postpartum.

Table 2 evaluates the risk of bias in each of the studies included in the systematic review, using the Evidence Project Risk of Bias Tool. The table provides a visual representation of the methodological rigor of each study, highlighting potential biases related to selection, randomization, and follow-up. The "Yes" and "No" responses indicate whether the study met the specific criteria in each of the categories.

Table 2: Bias Table for The Included Studies Using the Evidence Project Risk of Bias Tool

Study	Cohort	Control/ Comparison Group	Pre/Post Intervention Data	Random Assignment to Intervention	Random Selection for Assessment	Follow-up Rate ≥ 80%	Groups Equivalent on sociodemographic	Groups Equivalent at Baseline on Disclosure
Rao S, 2021[11]	Yes	Yes	No	No	No	Yes	Yes	Yes
Ghazali, 2023[12]	Yes	Yes	No	No	No	Yes	Yes	Yes
Zhang, 2023[13]	Yes	Yes	No	No	No	Yes	Yes	Yes
Song, 2016[14]	Yes	Yes	No	No	No	No	Yes	Yes
Thagaard, 2019[15]	Yes	Yes	No	No	No	Yes	Yes	Yes
Bawah, 2020[16]	Yes	Yes	No	No	No	Yes	Yes	Yes
Dong, 2018[17]	No	Yes	No	No	No	No	No	No
Poniedziałek-Czajkowska, 2019[18]	Yes	Yes	No	No	No	Yes	Yes	Yes
Perichart-Perera, 2017[19]	Yes	Yes	No	No	No	Yes	Yes	Yes
Xuan, 2019[20]	Yes	Yes	No	No	No	Yes	Yes	Yes
Tang, 2019[21]	Yes	Yes	No	No	No	Yes	Yes	Yes
Fisher, 2025[22]	Yes	Yes	No	No	No	Yes	Yes	Yes

DISCUSSION

Systematic review findings demonstrated how adiponectin played a vital role in pregnancy pathologies through complex molecular mechanisms and its potential clinical applications. Vascular function, insulin sensitivity regulation, and inflammatory pathways from core actions of adiponectin, which are essential in pregnancy-related disorders. Research demonstrated that abnormalities in adiponectin levels remained strongly linked to three major pregnancy complications, including gestational diabetes mellitus (GDM), preeclampsia, and fetal growth restrictions [23,24]. Additionally, decreased adiponectin levels are consistently associated with glucose metabolism problems in GDM patients and preeclampsia-induced endothelial dysfunction in pregnant mothers [25].

The review indicated that adiponectin showed promise as a biological indicator to detect pregnancy complications during early stages and guide their management. Numerous studies conducted during early pregnancy showed that reduced adiponectin levels acted as a risk indicator for preterm birth, GDM, and preeclampsia [26,27]. The research output agrees with previous studies, which indicated that adiponectin is activated as an initial marker for pregnancy-associated medical issues [28]. The molecular basis of adiponectin action through insulin sensitivity regulation and placental effects, alongside the inflammatory pathway, enhanced scientists' knowledge about this adipokine's role in maternal-fetal health [29,30].

This review strengthened the existing evidence about how adiponectin controlled maternal

metabolism and fetal development, drawing from previous research [31]. Multiple studies extracted results from diverse methods of design and measurement, producing obstacles for clear outcome comparison. Research findings showed mixed results about the relation between adiponectin levels and fetal growth, because different studies reached divergent conclusions [32,33]. The variability in results stemmed from contradictions between research groups that analyzed diverse populations through different study protocols, as well as changes when they monitored adiponectin levels during pregnancy[34,35]. Additional research was needed to standardize how measurements were conducted while examining multiple participant groups to improve research reliability.

Research indicated that adiponectin provided important clinical aspects relating to pregnancy pathologies. Adiponectin levels had the potential to be incorporated into regular prenatal screening as a reliable biomarker, which made it possible to find GDM, preeclampsia, and fetal growth restriction risks early [36]. Strategies focused on managing adiponectin levels provide new possibilities to mitigate disease development in women throughout pregnancy, with better results for maternal and fetal health [37,38].

More research was needed to fully understand how adiponectin influenced pregnancy outcomes, as the exact molecular processes had been unidentified [39]. Current research is needed to provide an in-depth analysis of these molecular processes while searching for appropriate treatment methods to control adiponectin amount and performing extensive clinical trials to establish adiponectin as an effective therapeutic option for pregnancy-related health conditions [40]. Research should be conducted to examine adiponectin's behavior across various populations with a focus on ethnic and genetic variations to validate it as universal biomarker and therapeutic target application.

CONCLUSION

Adiponectin regulates the metabolic, vascular, and inflammatory systems of maternal and fetal health throughout pregnancy. Pregnancy initiation raised adiponectin levels, but GDM or preeclampsia reduced them because decreased adiponectin levels lead to metabolic problems and endothelial malfunction. Through its action adiponectin influenced fetal growth together with placental functions. Scientists had not identified the molecular processes behind adiponectin's biomarker and therapeutic capabilities for pregnancy complications. Standardized clinical trials that involve large-scale testing would need completion before adopting adiponectin as a treatment approach for pregnancy-related conditions.

LIST OF ABBREVIATIONS

GDM – Gestational Diabetes Mellitus
PE – Preeclampsia
IUGR – Intrauterine Growth Restriction
ELISA – Enzyme-Linked Immunosorbent Assay
BMI – Body Mass Index
HOMA-IR – Homeostatic Model Assessment of Insulin Resistance
STAT – Signal Transducer and Activator of Transcription
MAPK – Mitogen-Activated Protein Kinase
PRISMA – Preferred Reporting Items for Systematic Reviews and Meta-Analyses
HDL – High-Density Lipoprotein

CONFLICT OF INTEREST

None

AUTHORS' CONTRIBUTIONS

All participants participated equally as per ICMJE.

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