Epigenetics in Gestational Diabetes Mellitus: From Fetal Programming to Metabolic Diseases in Adulthood

Epigenética no Diabetes Mellitus Gestacional: Da Programação Fetal às Doenças Metabólicas na Vida Adulta

Epigenética en la Diabetes Mellitus Gestacional: De lLa Programación Fetal a las Enfermedades Metabólicas en la Vida Adulta

RESUMO

Objetivo: Analisar criticamente as evidências sobre os mecanismos epigenéticos envolvidos na programação fetal induzida pelo diabetes mellitus gestacional (DMG) e suas repercussões metabólicas tardias na prole. **Método:** Realizou-se revisão integrativa da literatura, com buscas nas bases PubMed/MEDLINE, Scopus e Web of Science, abrangendo estudos publicados entre 2016 e 2026, além de trabalhos clássicos relacionados ao conceito das Origens Desenvolvimentistas da Saúde e da Doença (DOHaD). **Resultados:** As evidências indicam que alterações na metilação do DNA, modificações de histonas, RNAs não codificantes e perfis metabolômicos podem contribuir para mudanças persistentes na expressão gênica e no metabolismo energético. Contudo, esses efeitos resultam da interação entre ambiente intrauterino, predisposição genética e exposições pós-natais. **Conclusão:** A integração de dados epigenéticos e metabolômicos amplia a compreensão do DMG e pode favorecer a identificação precoce de risco e o desenvolvimento de estratégias preventivas de precisão.

DESCRITORES: Diabetes Gestacional; Epigênese Genética; Metabolômica; Desenvolvimento Fetal.

ABSTRACT

Objective: To critically analyze the evidence regarding the epigenetic mechanisms involved in fetal programming induced by gestational diabetes mellitus (GDM) and their long-term metabolic repercussions in the offspring. **Method:** An integrative literature review was conducted using PubMed/MEDLINE, Scopus, and Web of Science, including studies published between 2016 and 2026, as well as landmark studies related to the Developmental Origins of Health and Disease (DOHaD) concept. **Results:** The findings indicate that changes in DNA methylation, histone modifications, non-coding RNAs, and metabolomic profiles may contribute to persistent alterations in gene expression and energy metabolism. However, these effects result from the interaction between the intrauterine environment, genetic susceptibility, and postnatal exposures. **Conclusion:** The integration of epigenetic and metabolomic data broadens the understanding of GDM and may support early risk identification and the development of precision preventive strategies.

DESCRIPTORS: Diabetes, Gestational; Epigenesis, Genetic; Metabolomics; Fetal Development.

RESUMEN

Objetivo: Analizar críticamente las evidencias sobre los mecanismos epigenéticos implicados en la programación fetal inducida por la diabetes mellitus gestacional (DMG) y sus repercusiones metabólicas tardías en la descendencia. **Método:** Se realizó una revisión integradora de la literatura mediante búsquedas en las bases PubMed/MEDLINE, Scopus y Web of Science, incluyendo estudios publicados entre 2016 y 2026, además de trabajos clásicos relacionados con el concepto de los Orígenes del Desarrollo de la Salud y la Enfermedad (DOHaD). **Resultados:** Los hallazgos indican que las alteraciones en la metilación del ADN, las modificaciones de histonas, los ARN no codificantes y los perfiles metabolómicos pueden contribuir a cambios persistentes en la expresión génica y el metabolismo energético. Sin embargo, estos efectos resultan de la interacción entre el ambiente intrauterino, la susceptibilidad genética y las exposiciones posnatales. **Conclusión:** La integración de datos epigenéticos y metabolómicos amplía la comprensión de la DMG y puede favorecer la identificación precoz del riesgo y el desarrollo de estrategias preventivas de precisión.

DESCRIPTORES: Diabetes Gestacional; Epigénesis Genética; Metabolómica; Desarrollo Fetal.

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INTRODUCTION

Gestational diabetes mellitus (GDM) is one of the most common metabolic complications of pregnancy, characterized by glucose intolerance first diagnosed during pregnancy. Its incidence has been increasing worldwide, driven by obesity, advanced maternal age, and other metabolic risk factors, making it a public health problem due to its maternal, fetal, and neonatal repercussions [1].

In addition to immediate obstetric complications, recent evidence shows that intrauterine exposure to hyperglycemia can induce persistent biological changes, altering fetal development and increasing the offspring's susceptibility to metabolic diseases. This concept is based on the *Developmental Origins of Health and Disease* (DOHaD) hypothesis, according to which environmental stimuli during critical periods of development exert a lasting influence on the structure and function of organs and systems [2,3].

Among the proposed mechanisms, epigenetic modifications—such as DNA methylation, histone modifications, and non-coding RNAs—stand out, as they are capable of modulating gene expression without altering its sequence. Identified in the placenta, umbilical cord blood, and fetal tissues, these changes are associated with insulin resistance, obesity, type 2 diabetes, and cardiovascular diseases in offspring [4,5].

Advances in metabolomics have broadened our understanding of maternal and fetal metabolic changes associated with GDM, enabling the identification of early biomarkers and pathways involved in intrauterine programming. The integration of epigenetics and metabolomics holds promise for elucidating pathophysiological mechanisms and supporting prevention, early diagnosis, and precision medicine [6,7].

Against this backdrop, this review aims to synthesize the scientific evidence regarding the role of epigenetic mechanisms in fetal programming induced by gestational diabetes mellitus, highlighting their interaction with metabolomic changes and the long-term metabolic repercussions for the offspring.

METHOD

This is an integrative literature review designed to synthesize and critically discuss the evidence regarding the role of epigenetic mechanisms in fetal programming induced by GDM and their interaction with metabolomic changes and long-term consequences for the offspring.

The literature search was conducted in the PubMed/MEDLINE, Scopus, and Web of Science databases, as they are recognized for the comprehensiveness and quality of their indexing of biomedical studies. Additionally, international guidelines and documents from scientific societies, including the *American Diabetes Association* (ADA) and the *International Federation of Gynecology and Obstetrics* (FIGO), were consulted when relevant to the topic.

Controlled descriptors (Medical Subject Headings, MeSH) and free-text terms were used, combined using Boolean operators, including: *Gestational Diabetes Mellitus*, *Epigenetics*, *DNA Methylation*, *Histone Modification*, *MicroRNA*, *Non-coding RNA*, *Placenta*, *Metabolomics*, *Fetal Programming*, *Developmental Origins of Health and Disease (DOHaD)*, *Offspring*, and *Cardiometabolic Disease*. The search strategies were adapted to the specific characteristics of each database.

Studies published between January 2016 and June 2026 in English, Portuguese, and Spanish were considered, including original articles, observational studies, clinical trials, experimental studies, systematic reviews, meta-analyses, and relevant narrative reviews on epigenetic mechanisms, metabolomic changes, fetal programming, or metabolic outcomes related to GDM, as well as seminal articles fundamental to the DOHaD concept. Duplicate studies, publications without full text, conference abstracts, letters to the editor, editorials, and works not directly related to the objective of this review were excluded.

The selected studies were analyzed qualitatively and organized into thematic areas: pathophysiology of fetal growth restriction; fundamentals of fetal programming; epigenetic mechanisms (DNA methylation, histone modifications, and noncoding RNAs); maternal, placental, and fetal metabolomic changes; long-term metabolic consequences for the offspring; and perspectives involving multi-omic approaches and precision medicine. The synthesis prioritized the most consistent and current findings, highlighting convergences, divergences, methodological limitations, and knowledge gaps.

As this is a literature review based on previously published secondary data, without direct contact with humans or animals, the study did not re-

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quire submission to a Research Ethics Committee.

RESULTS

Pathophysiology of Gestational Diabetes Mellitus

GDM is characterized by a state of glucose intolerance first diagnosed during pregnancy, resulting from the inability of pancreatic β-cells to compensate for the physiological increase in insulin resistance induced by pregnancy [1]. During normal pregnancy, placental hormones—such as human placental lactogen, placental growth hormone, progesterone, estrogen, and cortisol—promote a progressive state of insulin resistance, especially in the second and third trimesters, ensuring greater glucose availability for fetal growth. However, when pancreatic adaptation is insufficient, persistent maternal hyperglycemia sets in, a condition that characterizes gestational diabetes mellitus (GDM) [8].

The pathophysiology of GSD is multifactorial and involves the interaction between genetic predisposition, obesity, low-grade chronic inflammation, oxidative stress, mitochondrial dysfunction, and changes in the secretion of adipokines and inflammatory cytokines [9,10]. Women with GSD often have elevated circulating levels of tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and C-reactive protein (CRP), associated with reduced insulin signaling in peripheral tissues. At the same time, there is a decrease in adiponectin and an increase in leptin, contributing to the worsening of insulin resistance and metabolic dysfunction [9].

The placenta plays a central role in this process, acting as a maternal-fetal exchange organ and as an endocrine and immunometabolic organ. In pregnancies with DMG, alterations in the expression of glucose (GLUT1), amino acid, and lipid transporters, combined with oxidative stress, inflammation, and placental vascular dysfunction, promote increased nutrient transfer to the fetus, contributing to fetal hyperinsulinism, macrosomia, and early alterations in energy metabolism [4,11].

Epigenetic Mechanisms Involved in Fetal Programming

Epigenetics encompasses potentially reversible—and sometimes mitotically stable—changes in the regulation of gene expression without modification of the DNA sequence. During fetal development, these mechanisms play a role in cell differentiation and organogenesis, a period of high biological plasticity and environmental susceptibility, during which maternal hyperglycemia associated with GDM can alter regulatory programs critical to the future health of the offspring [2,3].

This relationship is supported by the DOHaD concept, according to which environmental conditions during critical periods of development can induce persistent structural, physiological, and molecular adaptations. In GDM, the intrauterine environment—including hyperglycemia, fetal hyperinsulinemia, inflammation, oxidative stress, and an increased supply of energy substrates—can alter gene regulation during organ formation. Although initially adaptive, these responses may increase susceptibility to obesity, insulin resistance, type 2 diabetes, and cardiovascular changes, especially when the postnatal environment exacerbates the already established risk [2,3,4].

The main epigenetic mechanisms related to fetal programming include DNA methylation, post-translational modifications of histones, and the regulation of gene expression by non-coding RNAs, particularly microRNAs (miRNAs) and long non-coding RNAs (lncRNAs). These processes act in an interdependent and dynamic manner, converting metabolic and environmental signals into changes in gene activity related to fetal growth, angiogenesis, the inflammatory response, mitochondrial function, and insulin sensitivity [4,5].

DNA methylation is the most extensively studied mechanism in DMG. It involves the addition of methyl groups to the 5-position of cytosine, predominantly in CpG dinucleotides, and can alter chromatin accessibility and transcriptional activity; its effect depends on genomic location, cell type, and regulatory context. Studies of the placenta, umbilical cord blood, and other fetal tissues have demonstrated alterations in regions associated with glycemic homeostasis, adipocyte differentiation, insulin signaling, inflammation, and cardiovascular development [12,13,4].

Among the genes described are *LEP*, *ADIPOQ*, *IGF2*, *H19*, *PPARGC1A*, and *MEST*, which are involved in fetal growth, energy metabolism, adipogenesis, and regulation of insulin sensitivity. Altered expression of these genes may contribute to a metabolic phenotype that is more susceptible to fat accumulation, insulin resistance, and type 2 diabetes mellitus. However, these associations do not, on their own, demonstrate causality, lifelong persistence, or intergenerational transmission, as methylation patterns are also influenced by the tissue analyzed, cellular composition, maternal obesity, treatment of gestational diabetes, and postnatal exposures [12,13,4].

Post-translational modifications of histones—acetylation, methylation, phosphorylation, and ubiquitination—may also play a role in fetal programming by altering chromatin organization and DNA accessibility to transcription factors. In experimental models of gestational diabetes, hyperglycemia, oxidative stress, and lipotoxicity modify chromatin-regulating enzymes, affecting genes involved in lipid metabolism, inflammation, insulin signaling, and mitochondrial func-

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tion, which provides biological plausibility for the persistence of metabolic phenotypes, although evidence in humans remains limited [5].

Non-coding RNAs represent another important aspect of epigenetic regulation in the DMG. miRNAs simultaneously regulate multiple messenger RNAs, influencing proliferation, cell differentiation, apoptosis, angiogenesis, and energy metabolism. Placental alterations in miR-21, miR-29, miR-132, miR-222, miR-330, and miR-518d are associated with insulin resistance, inflammation, endothelial dysfunction, and placental nutrient transport; however, their clinical application remains limited by the heterogeneity of the platforms and methods used [14,5] .

lncRNAs also regulate gene expression by interacting with transcription factors, chromatin-modifying proteins, and messenger RNA. Studies have identified differential expression of lncRNAs in the placentas of pregnant women with DMG, suggesting a role in trophoblastic proliferation and invasion, angiogenesis, the inflammatory response, and fetal metabolic adaptation, although their functional mechanisms remain only partially understood and have been poorly replicated across populations [15,16] .

The placenta occupies a central position at this interface between maternal exposure and fetal programming, acting as an endocrine, immune, and metabolic system responsive to maternal conditions. Placental epigenetic alterations can modify the expression of glucose, amino acid, and lipid transporters, as well as the production of cytokines, hormones, and angiogenic factors, thereby altering the availability of substrates to the fetus and the maturation of metabolically active tissues [5,17] .

The placenta also releases exosomes and other extracellular vesicles containing proteins, lipids, miRNAs, and non-coding RNAs. In pregnancies with DMG, changes in the concentration and composition of these vesicles may alter maternal-fetal communication and signaling pathways related to insulin, inflammation, endothelial function, and energy metabolism, although it has not been established whether they play a causal role or function merely as markers of placental dysfunction [5,17] .

In light of the DOHaD model, epigenetic mechanisms can be viewed as a molecular memory of exposures during development, but not as an immutable biological destiny, since the epigenome remains sensitive to nutrition, growth, and other environmental conditions after birth. The epigenetic marks associated with DMG therefore represent susceptibility factors that interact with genetic predisposition and postnatal exposures [2,3,4] .

Epigenetic mechanisms do not act in isolation: the interaction between genetic predisposition, maternal nutritional status, inflammation, the gut microbiome, and metabolomic changes produces a complex regulatory network responsible for the heterogeneity of phenotypes associated with DMG. This perspective underpins the use of multi-omic approaches, which integrate genomic, epigenomic, transcriptomic, proteomic, and metabolomic data to identify biological pathways and predictive biomarkers [6,7] .

Long-Term Metabolic Consequences in Offspring

Current epidemiological evidence indicates that children of women with DMG are more likely to develop metabolic abnormalities throughout their lives, including increased body fat, central obesity, insulin resistance, glucose intolerance, and greater susceptibility to type 2 diabetes. These associations tend to diminish after adjusting for maternal body mass index, gestational weight gain, genetic predisposition, and family environment, indicating that intrauterine exposure is part of a multifactorial network of cardiometabolic risk [18,19] .

Childhood obesity is the late-onset outcome most frequently associated with exposure to DMG. Longitudinal studies show that exposed offspring have higher body mass index, body fat percentage, and abdominal adiposity than children of normoglycemic mothers, an association that becomes more evident in late childhood and puberty [18,20] .

This relationship is also modulated by postnatal factors. In a recent prospective cohort study, greater severity of DMG was associated with a higher risk of obesity in preadolescence, but feeding practices during the first year of life modified this association, suggesting that metabolic risk is not necessarily irreversible and can be mitigated by dietary and lifestyle practices during childhood [21] .

In addition to adiposity, children exposed to DMG may exhibit reduced insulin sensitivity and early alterations in glycemic homeostasis, which are often subclinical until puberty. The combination of insulin resistance, excessive weight gain, and the limited compensatory capacity of pancreatic β-cells may promote progression to glucose intolerance and type 2 diabetes in susceptible individuals [20,19] .

The components of metabolic syndrome have also been investigated: in addition to abdominal adiposity and glycemic abnormalities, some studies report a higher prevalence of high blood pressure and unfavorable lipid profiles, with results that are less consistent than those for obesity and insulin resistance, possibly due to differences in diagnostic criteria and follow-up duration [19] .

The long-term cardiovascular consequences remain uncertain: observational studies have identified subclinical alterations in endothelial function, arterial stiffness, blood pressure, and

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cardiac structure in individuals exposed to maternal diabetes; however, these studies had small sample sizes and insufficient follow-up to assess clinical events, which prevents an accurate estimation of the independent contribution of GDM to cardiovascular disease in adulthood [22,19].

Another emerging outcome is fatty liver disease: a systematic review and meta-analysis from 2024 identified an increased risk of fatty liver disease among offspring of pregnancies with GDM, but due to the limited number of studies and methodological heterogeneity, the association remains preliminary [23].

The analysis of long-term effects must also consider the treatment of GDM: although it reduces obstetric and neonatal complications, it is not established whether maternal glycemic control normalizes the metabolic risk in offspring, and studies using insulin and metformin show mixed results regarding growth and adiposity [24,25].

These uncertainties stem from methodological limitations: most of the evidence comes from observational studies, which are subject to residual confounding by maternal obesity, family diet, socioeconomic status, and genetic predisposition, as well as differences in diagnostic criteria and follow-up duration, which reduce comparability and hinder causal inferences[18,19].

DISCUSSION

Taken together, the reviewed findings support the biological plausibility of the DOHaD model as a framework for understanding the metabolic repercussions of DMG, demonstrating that epigenetic alterations—including DNA methylation, histone modifications, and non-coding RNAs—converge to modulate pathways related to glycemic homeostasis, adipogenesis, and vascular function [2–5].

However, most of this evidence is associative and observational, making it impossible to establish causality between specific epigenetic marks and metabolic outcomes. Heterogeneity across tissues, platforms, and normalization methods hinders the replication of these findings and limits their application as clinical biomarkers [12–14,16].

With regard to clinical implications, the association between DMG exposure and childhood obesity, insulin resistance, and glucose intolerance has been relatively consistent across studies, while cardiovascular and hepatic outcomes remain less well established, based on smaller samples and shorter follow-up periods [19,22,23]. Furthermore, the magnitude of these associations tends to decrease after adjusting for maternal and postnatal factors, such as body mass index, infant feeding practices, and socioeconomic status, reinforcing the notion that fetal programming interacts with—rather than replaces—environmental and behavioral determinants of metabolic risk[18,21].

Also noteworthy is the uncertainty regarding the impact of DMG treatment on the offspring's metabolic risk: although maternal glycemic control reduces adverse obstetric outcomes, there is insufficient evidence that it completely normalizes fetal programming, which reinforces the need for postnatal follow-up of the offspring regardless of the treatment instituted during pregnancy [24,25].

Given this scenario, the integration of epigenetic, metabolomic, and clinical data through multi-omic approaches emerges as a promising strategy for identifying early biomarkers and risk subphenotypes; however, its translation into clinical practice depends on methodological standardization across platforms and external validation in prospective cohorts with longer follow-up and greater population diversity [6,7].

CONCLUSION

Gestational diabetes mellitus goes beyond immediate obstetric complications, emerging as a condition capable of causing persistent biological changes with intergenerational repercussions. The reviewed evidence demonstrates that fetal exposure to hyperglycemia triggers a complex interaction between epigenetic mechanisms and metabolomic changes, resulting in lasting modifications in gene expression, energy metabolism, and the function of multiple organs, which explains, at least in part, the increased risk of obesity, insulin resistance, type 2 diabetes, metabolic syndrome, and cardiovascular diseases in offspring.

The integration of epigenetics and metabolomics, within the context of multi-omic approaches, represents a significant advance in understanding the heterogeneity of gestational diabetes mellitus and its pathophysiological mechanisms. In addition to expanding our knowledge of the origins of cardiometabolic diseases, these tools offer promising prospects for the early identification of biomarkers, risk stratification, and the development of individualized preventive and therapeutic strategies, in line with the principles of precision medicine.

Translating these advances into clinical practice still faces challenges, such as methodological standardization, external validation of biomarkers, and longitudinal studies that elucidate the interaction between genetic, epigenetic, environmental, and postnatal factors. Future research integrating clinical and multi-omic data across different populations will be essential to consolidate these approaches in maternal-fetal care and reduce the burden of metabolic diseases in offspring.

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