REVIEW ARTICLE

Annals of Agricultural and Environmental Medicine
www.aaem.pl

Intestinal and vaginal microbiota as determinants of maternal and neonatal health in gestational diabetes mellitus – a narrative review

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Gorczyca K, Ziółkowski BA, Paszkowski M, Dąbrowski W. Intestinal and vaginal microbiota as determinants of maternal and neonatal health in gestational diabetes mellitus – a narrative review. Ann Agric Environ Med. doi:10.26444/aaem/218797

Abstract

Introduction and Objective. Gestational diabetes mellitus (GDM) is the most common metabolic complication of pregnancy, associated with adverse maternal and neonatal outcomes and increased long-term metabolic risk. Recent studies indicate that intestinal and vaginal microbiota play an important role in the pathogenesis and clinical course of GDM. The aim of the review is to summarize current evidence on alterations in maternal gut and vaginal microbiota in GDM and their clinical relevance.

Review Methods. A narrative review was conducted of observational, experimental, and meta-analytic studies indexed in PubMed and Scopus, focusing on microbiota composition, metabolic parameters, and perinatal outcomes in gestational diabetes mellitus.

Brief description of the state of knowledge. GDM is associated with reduced gut microbial diversity, depletion of short-chain fatty acid–producing bacteria, and increased abundance of Gram-negative species, contributing to inflammation and impaired glucose metabolism. Vaginal dysbiosis, characterized by reduced Lactobacillus dominance, is linked to bacterial vaginosis, preterm birth, and intrauterine infections. Both gut and vaginal microbiota alterations may influence neonatal microbial colonization and metabolic programming.

Summary. The available evidence indicates that gut and vaginal microbiota are key determinants of metabolic and reproductive health in women with gestational diabetes mellitus. Interventions based on probiotics, prebiotics, and lifestyle modification show potential in restoring microbial balance and improving glycaemic control, although further large-scale clinical studies are warranted. Assessment and targeted modulation of maternal microbiota may represent promising strategies for the prevention of GDM-related complications and for the development of personalized approaches in maternal care.

Key words

probiotics, gestational diabetes mellitus, gut microbiota, vaginal microbiota, pregnancy complications, maternal and neonatal health.

INTRODUCTION

Gestational diabetes mellitus (GDM) is currently recognized as the most common metabolic disorder complicating pregnancy, with its prevalence estimated between 5% – 22%, depending on maternal age, population characteristics, and diagnostic criteria applied [1, 2]. The diagnosis of GDM is strongly associated with an increased risk of adverse perinatal outcomes, such as foetal macrosomia, preterm delivery, as well as a higher probability of type 2 diabetes mellitus (T2DM) and metabolic syndrome in mothers later in life [3]. Evidence from observational studies and meta-analyses emphasizes that adequate glycaemic control during gestation, combined with timely nutritional interventions, reduces the incidence of complications for both mother and child. Nevertheless, continuous monitoring of insulin resistance and metabolic adaptations during pregnancy remains essential.

In recent years, growing attention has been directed towards the role of the intestinal and vaginal microbiota in the pathogenesis and clinical course of GDM, as well as in shaping the metabolic programming of offspring [4]. Microorganisms inhabiting the maternal gastrointestinal and genital tracts have been shown to influence glucose metabolism, insulin signaling pathways, and immune regulation. Consequently, disturbances in microbiota composition—referred to as dysbiosis—may contribute to increased intestinal permeability, endotoxin translocation,

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Received: 03.01.2026; accepted: 02.03.2026; first published: 01.04.2026

and activation of inflammatory cascades (e.g., TLR4/NF-κB), thereby exacerbating insulin resistance and impaired glucose homeostasis. Moreover, alterations in the vaginal microbiota, characterized by reduced *Lactobacillus* dominance and the overgrowth of potentially pathogenic bacteria, have been linked with intrauterine infections, premature rupture of membranes (PPROM), and preterm labour [5, 6]. These findings suggest that microbiota assessment and its modulation through targeted dietary strategies (prebiotics, probiotics) and lifestyle modifications (physical activity, healthy diet) could constitute an important element in preventing GDM and its complications. Furthermore, experimental studies highlight that maternal intestinal microbiota may directly affect neonatal microbial colonization and long-term metabolic outcomes. Such mechanisms of perinatal metabolic programming can determine susceptibility to obesity and metabolic disorders later in life. A comprehensive understanding of the interactions between maternal microbiota, carbohydrate metabolism, and immunity in GDM is therefore crucial for the development of personalized preventive and therapeutic strategies.

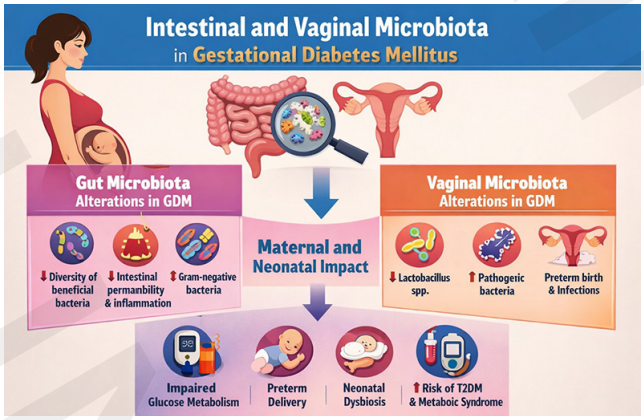

Figure 1. Intestinal and vaginal microbiota alterations in gestational diabetes mellitus and their impact on maternal and neonatal health

MATERIALS AND METHOD

A narrative review was conducted through a structured search of the PubMed and Scopus databases to identify studies addressing the relationship between gut and vaginal microbiota and GDM. The search covered publications from January 2010 – June 2025. The following key words and combinations were used: 'gestational diabetes mellitus', 'gut microbiota', 'intestinal microbiota', 'vaginal microbiota', 'pregnancy', 'probiotics', and 'maternal health.

The initial search yielded 154 records (PubMed: 98; Scopus: 56). After removal of duplicates (n = 21), 133 articles remained for title and abstract screening. Studies were excluded if they were conference abstracts, editorials, case reports, or animal-only studies without clinical relevance. After this stage, 74 articles were considered potentially relevant and underwent full-text evaluation, which resulted in the exclusion of 24 studies due to lack of direct relevance to gestational diabetes mellitus or insufficient microbiota-related outcomes. Ultimately, 50 articles were included in the final analysis, comprising observational studies, randomized controlled trials, systematic reviews, and meta-analyses.

Only articles published in English were considered. Additional relevant studies were identified through manual screening of reference lists of the selected publications.

Because the aim of this work was to summarize current evidence rather than conduct a systematic review, a formal risk-of-bias assessment was not performed.

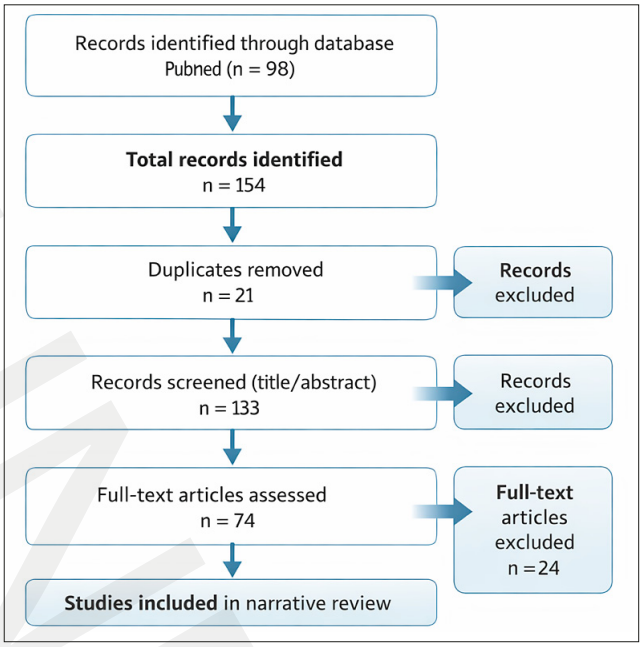

Figure 2. Flow diagram of study selection process

Gut microbiota alterations in pregnancy and gestational diabetes mellitus. Physiological pregnancy is characterized by dynamic alterations in the gut microbiota, including an increase in the abundance of *Proteobacteria* and *Actinobacteria,* accompanied by a decline in short-chain fatty acid (SCFA)-producing bacteria such as *Roseburia* and *Faecalibacterium prausnitzii* [7]. These changes are regarded as part of the maternal adaptation to the growing metabolic demands of the foetus and preparation for lactation, and are associated with enhanced energy bioavailability and progressive insulin resistance typical for late pregnancy [8]. In the context of GDM, these shifts are more pronounced, and the gut microbiota profile resembles that observed in individuals with type 2 diabetes mellitus (T2DM) [9]. Specific features of the intestinal microbiota in GDM include: an increased proportion of Gram-negative bacteria, e.g., *Parabacteroides, Prevotella, Haemophilus, Desulfovibrio,* decreased abundance of anti-inflammatory and SCFA-producing species, such as *Faecalibacterium, Ruminococcus, Akkermansia* [10], reduced microbial alpha diversity, which predisposes to chronic low-grade inflammation [11]. Dysbiosis observed in GDM contributes to increased intestinal permeability and the translocation of endotoxins (e.g., lipopolysaccharides, LPS), which activate inflammatory cascades such as TLR4/NF-κB, thereby promoting insulin resistance and impaired glucose regulation [12].

Clinical studies have also demonstrated correlations between altered gut microbiota composition and key metabolic parameters, including fasting glucose, serum insulin concentrations, and HOMA-IR indices [13]. Importantly, maternal microbiota is a determinant of the infant's microbial colonization and metabolic development.

During the perinatal period, these mechanisms can shape the child's long-term risk of obesity and metabolic diseases [14]. Interventions aimed at microbiota modulation, such as the use of probiotics (*Lacticaseibacillus rhamnosus, Bifidobacterium lactis*) or prebiotics, have demonstrated beneficial effects in improving glycaemic control and reducing the incidence of GDM, as confirmed by randomized controlled trials and meta-analyses [15, 16].

These findings indicate that gut microbiota may serve both as a predictive biomarker and a therapeutic target in GDM. However, further metagenomic studies and large-scale clinical trials remain necessary to optimize personalized approaches.

Beyond GDM, gut dysbiosis has been implicated in multiple chronic conditions, including T2DM, obesity, metabolic syndrome, and inflammatory bowel disease [17, 18]. Normal gut microbiota ferment dietary fibre to produce SCFAs, such as acetate, propionate, and butyrate, which improve insulin sensitivity, regulate incretin secretion (GLP-1), and exert anti-inflammatory effects through NF-κB signaling inhibition [19, 20]. In GDM and T2DM, reduced microbial diversity and depletion of SCFA-producing species are commonly observed, together with an over-representation of Gram-negative bacteria such as *Escherichia coli* [21]. Therapeutic strategies to restore eubiosis include dietary modification with increased fiber intake, supplementation with probiotics, prebiotics, and synbiotics, all of which have shown promise in lowering inflammatory markers and improving metabolic homeostasis [22]. Randomized clinical studies confirmed that supplementation with *Lacticaseibacillus rhamnosus* or *Bifidobacterium lactis* may improve glycaemic control, reduce CRP levels, and favourably modulate the microbiota composition in GDM and T2DM patients [23].

Several prebiotics have demonstrated beneficial metabolic effects, including inulin, fructooligosaccharides (FOS), and galactooligosaccharides (GOS). These compounds promote the growth of beneficial bacteria, such as *Bifidobacterium* and *Faecalibacterium,* enhance SCFA production (especially butyrate and propionate), and contribute to improved insulin sensitivity and glycaemic control [22]. Overall, intestinal microbiota disturbances exert significant clinical implications in metabolic and inflammatory disorders. Understanding these mechanisms is fundamental for developing personalized microbiota-based therapeutic strategies that aim to restore balance and improve maternal and neonatal health outcomes. Several probiotic strains, including *Lacticaseibacillus rhamnosus, Bifidobacterium lactis,* and *Limosilactobacillus reuteri,* have been shown to reduce inflammatory markers such as C-reactive protein (CRP) and pro-inflammatory cytokines. Similarly, prebiotics such as inulin and fructooligosaccharides (FOS) promote SCFA production and contribute to anti-inflammatory effects. Synbiotic combinations of *Bifidobacterium* or *Lactobacillus strains* with insulin or FOS have also demonstrated reductions in systemic inflammatory markers and improvements in metabolic profiles.

In healthy pregnant women, the gut microbiota is primarily dominated by bacteria belonging to the phyla *Firmicutes* and *Bacteroidetes,* with smaller contributions from *Actinobacteria* and *Proteobacteria*. Commonly reported genera include *Bacteroides, Prevotella, Faecalibacterium, Roseburia, Ruminococcus, Bifidobacterium,* and *Akkermansia*. Among SCFA-producing species, *Faecalibacterium prausnitzii* and members of the genera *Roseburia* and *Ruminococcus* play an important role in maintaining intestinal barrier integrity and regulating metabolic homeostasis. In addition, beneficial bacteria such as *Bifidobacterium adolescentis* and *Bifidobacterium longum* are frequently detected and contribute to carbohydrate metabolism and modulation of host immune responses.

Vaginal microbiota in postpartum women with GDM. The vaginal microbiota represents a crucial element of maternal health, especially during pregnancy, when its composition significantly affects pregnancy maintenance and perinatal outcomes [24]. Physiologically, the vaginal ecosystem is dominated by *Lactobacillus species,* such as *Lactobacillus crispatus, Lactobacillus jensenii, Lactobacillus gasseri,* and *Lactobacillus iners.* These microorganisms produce lactic acid, hydrogen peroxide (H_2O_2), and bacteriocins, which collectively maintain an acidic vaginal environment (pH 3.5–4.5) and protect against colonization by opportunistic pathogens [25, 26].

During pregnancy, an increase in estrogen and glycogen levels promotes *Lactobacillus* colonization and supports lactic acid production, thereby reducing microbial diversity but enhancing stability of the vaginal microbiota [27]. This physiological adaptation provides an effective defence against rising infections and premature rupture of membranes (PPROM). A predominance of *L. crispatus* has been particularly associated with reduced risks of bacterial vaginosis (BV) and preterm birth [28]. Pregnancy-related hormonal and immunological shifts exert a direct impact on the composition and resilience of vaginal microbiota. Elevated estrogen stimulates glycogen synthesis within the vaginal epithelium, which acts as a nutrient source for *Lactobacillus* growth, maintaining low pH and limiting pathogen proliferation [24, 29]. Compared to non-pregnant women, pregnant women demonstrate: reduced species diversity with dominance of protective *Lactobacillus strains* [30], increased microbial stability throughout pregnancy [31], higher prevalence of *L. crispatus, L. jensenii, L. gasseri*, and *L. iners* [32]. Such imbalance has been associated with BV, PPROM, preterm birth, and intrauterine infections including chorioamnionitis (33). Meta-analyses indicate that BV during pregnancy almost doubles the risk of preterm birth and increases the incidence of low birth weight infants [34]. When vaginal microbial balance is disrupted, the abundance of protective *Lactobacillus species* decreases, while anaerobic bacteria associated with bacterial vaginosis proliferate. Microorganisms most frequently associated with preterm birth include *Gardnerella vaginalis, Atopobium vaginae, Prevotella spp., Mobiluncus spp., Sneathia spp.,* and *Ureaplasma urealyticum*. These bacteria promote inflammatory responses within the reproductive tract and increase the risk of premature rupture of membranes and spontaneous preterm birth. Moreover, maternal vaginal microbiota strongly influences neonatal colonization during vaginal delivery. Early-life microbial exposure plays a pivotal role in shaping the infant's immune system, intestinal–immune axis, and susceptibility to allergic, metabolic, and infectious diseases [35, 36]. Strategies to support vaginal eubiosis include the use of oral or vaginal probiotics containing selected *Lactobacillus strains*, routine monitoring of vaginal microbiota as biomarkers of risk, and lifestyle modifications targeting intimate hygiene and reduction of

dysbiosis-promoting factors. Clinical trials confirm that supplementation with *Lacticaseibacillus rhamnosus GR-1 and Limosilactobacillus reuteri RC-14* reduces BV recurrence and lowers the risk of obstetric complications associated with vaginal dysbiosis [33, 37, 38]. These findings highlight that pregnancy-induced changes in vaginal microbiota, characterized by *Lactobacillus* dominance and reduced diversity, represent a protective adaptation. In contrast, disturbances of this balance increase the likelihood of adverse perinatal outcomes, reinforcing the importance of microbiota monitoring and targeted interventions in maternal care.

Table 1. Characteristics of gut and vaginal microbiota in women with GDM

Aspect	Gut microbiota	Vaginal microbiota
Physiological changes in pregnancy	↑ *Proteobacteria*, *Actinobacteria*; ↓ SCFA-producing bacteria (*Roseburia*, *Faecalibacterium prausnitzii*) – adaptation to increased energy demand and insulin resistance [7, 8]	Dominance of *Lactobacillus species* (*L. crispatus*, *L. jensenii*, *L. gasseri*, *L. iners*); ↑ microbial stability; ↓ diversity – protective mechanism [25–27, 30–32]
Alterations in GDM	Profile similar to T2DM; ↑ Gram-negative bacteria (*Parabacteroides*, *Prevotella*, *Haemophilus*, *Desulfovibrio*); ↓ SCFA-producing species (*Faecalibacterium*, *Ruminococcus*, *Akkermansia*); ↓ alpha diversity [9–11]	Dysbiosis: ↓ *Lactobacillus species*↑ anaerobic bacteria; disturbed hormonal–immune balance; higher risk of BV, PPROM, preterm birth, intrauterine infections [28, 33, 34]
Metabolic consequences	↑ intestinal permeability, LPS translocation, activation of TLR4/NF-κB → low-grade inflammation, insulin resistance [12]; correlation with fasting glucose, insulin, HOMA-IR [13]; maternal microbiota influences neonatal colonization and metabolic programming [14]	BV nearly doubles the risk of preterm birth and increases incidence of low birth weight [34]; maternal vaginal microbiota strongly influences neonatal colonization → early immunity, gut–immune axis, risk of allergy, obesity, metabolic disorders [35, 36]
Protective mechanisms	SCFAs (acetate, propionate, butyrate): ↑ insulin sensitivity, regulation of GLP-1 secretion, anti-inflammatory effect via NF-κB inhibition [19, 20]	*Lactobacillus species.:* production of lactic acid, H_2O_2, and bacteriocins → low vaginal pH, bacteriostatic/ bactericidal effects [25, 26]
Therapeutic interventions	Prebiotics, probiotics (*Lacticaseibacillus rhamnosus, Bifidobacterium lactis*), synbiotics, high-fiber diet → improved glycaemic control, ↓ CRP, intestinal barrier support [15, 16, 22, 23]	Probiotics (*L. rhamnosus* GR-1, *Limosilactobacillus reuteri*), vaginal microbiota monitoring, hygiene modifications → restoration of eubiosis, ↓ BV and obstetric complications [33, 37, 38]

DISCUSSION

The intestinal and vaginal microbiota play pivotal roles in maternal and neonatal health, particularly in the context of gestational diabetes mellitus. Alterations in the composition of gut microbiota observed in GDM mirror those described in type 2 diabetes, with increased abundance of Gram-negative bacteria, decreased diversity, and reduced SCFA-producing strains. These changes contribute to impaired glucose metabolism, insulin resistance, and systemic low-grade inflammation. Furthermore, maternal microbiota has been shown to influence neonatal microbial colonization, thereby determining long-term metabolic programming and susceptibility to obesity and metabolic syndrome. Similarly, the vaginal microbiota, physiologically dominated by *Lactobacillus species*, undergoes pregnancy-related adaptations that protect against infections and obstetric complications. Dysbiosis in this compartment has been associated with BV, PPROM, preterm birth, and intrauterine infections, which significantly impact both maternal and neonatal outcomes. The evidence reviewed indicates that interventions targeting microbiota—such as probiotics, prebiotics, synbiotics, dietary modifications, and lifestyle interventions—may offer novel preventive and therapeutic strategies in GDM. Randomized clinical trials have already confirmed that selected probiotic strains improve glycaemic control, modulate inflammatory pathways, and support microbial balance in both GDM and T2DM patients. Nevertheless, further large-scale, well-designed studies are required to identify the most effective approaches, optimize strain selection, and establish recommendations tailored to individual patient profiles. In conclusion, the intestinal and vaginal microbiota should be regarded as important determinants of maternal and neonatal health in GDM. Their assessment and modulation represent promising elements of personalized medicine strategies aimed at reducing the risk of adverse pregnancy outcomes and improving long-term health trajectories for both mother and child.

Although many studies consistently report reduced microbial diversity, decreased abundance of SCFA-producing taxa, and increased representation of Gram-negative bacteria in women with GDM, the reported microbial signatures vary considerably across studies. These discrepancies may partly reflect differences in study populations (ethnicity, BMI distribution, dietary patterns), timing of sample collection (first trimester, third trimester, or postpartum), and methodological approaches used for microbiome analysis, including 16S rRNA sequencing versus metagenomic sequencing. Furthermore, not all studies are adequately adjusted for potential confounding factors, such as antibiotic exposure, dietary intake, gestational weight gain, or mode of delivery. These methodological differences may contribute to inconsistencies in identifying specific microbial taxa associated with GDM.

From the public health and environmental perspective, maternal gut and vaginal microbiota may be considered modifiable biological mediators linking lifestyle, diet, and environmental exposures with metabolic health during pregnancy. Factors such as dietary patterns, antibiotic use, urbanization, and environmental stressors can influence microbiota composition, thereby affecting the risk of gestational diabetes mellitus and adverse pregnancy outcomes at the population level. Understanding these interactions may support the development of preventive strategies aimed at reducing the growing global burden of GDM and its long-term consequences for mothers and offspring.

Evidence linking maternal microbiota alterations in GDM with neonatal outcomes and long-term metabolic programming remains relatively limited. While several studies suggest that maternal gut and vaginal microbiota may influence early microbial colonization of the newborn, most available data are derived from observational cohorts that include heterogeneous populations and are not restricted to women with GDM. Consequently, conclusions regarding causal relationships between maternal dysbiosis in GDM and long-term metabolic outcomes in offspring should

be interpreted with caution. Longitudinal cohort studies that simultaneously analyze maternal microbiota, neonatal microbiome development, and metabolic outcomes during childhood are required to clarify these relationships.

Research gaps and clinical perspectives. Despite the growing body of evidence linking intestinal and vaginal microbiota with the pathogenesis and outcomes of gestational diabetes, several important research gaps remain. First, most available studies are cross-sectional, limiting causal inference between dysbiosis and metabolic complications. Longitudinal, prospective cohort studies are required to better elucidate temporal relationships and mechanisms. Second, most interventional studies investigating probiotics or prebiotics are limited by small sample sizes, heterogeneity of strains used, and short follow-up periods, which hinder the development of standardized clinical recommendations. Third, limited data are available on the combined impact of maternal gut and vaginal microbiota on neonatal colonization and long-term metabolic programming. From a clinical perspective, microbiota profiling may support risk stratification and preventive strategies in maternal health and could provide novel biomarkers for the early identification of women at risk of gestational diabetes and adverse perinatal outcomes.

Furthermore, microbiota-targeted therapies—including dietary interventions, probiotics, prebiotics, and synbiotics—hold promise as adjunctive strategies for optimizing glycaemic control and preventing complications. However, their widespread clinical implementation will require validation in large-scale, multicentre randomized controlled trials and the development of evidence-based guidelines tailored to specific patient populations.

CONCLUSIONS

Across the studies, consistent findings demonstrate that women with GDM present dysbiosis in both intestinal and vaginal microbiota**,** characterized by reduced diversity and diminished *Lactobacillus* or SCFA-producing strains. These disturbances contribute to insulin resistance, inflammation, and adverse pregnancy outcomes**,** while also influencing neonatal microbial colonization and long-term metabolic programming. Probiotics, prebiotics, and breastfeeding emerge as promising protective factors. However, evidence is still limited, and further large-scale, longitudinal studies are required to establish standardized microbiome-based strategies for the prevention and treatment of GDM.

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