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REVIEW

Postpartum Pain and Vaginal–Gut Microbiota Interactions: An Integrative Narrative Review with Implications for Maternal Recovery and Pain Modulation

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Background: Postpartum pain is a common and often underrecognized complication that adversely affects maternal recovery, quality of life, and psychological well-being. Emerging evidence suggests that alterations in the vaginal and gut microbiota during the puerperium may play a critical role in modulating these outcomes.

Objective: This integrative narrative review synthesizes available evidence on the associations between postpartum pain and the composition of the vaginal and gut microbiota, with a focus on mechanistic pathways and clinical implications for maternal recovery.

Methods: A systematic literature search was conducted in PubMed, Embase, and Web of Science for studies published between January 2017 and December 2025. Search terms encompassed postpartum pain, vaginal microbiota, gut microbiota, dysbiosis, and inflammation. Eligible studies included original research, clinical trials, and systematic reviews examining microbiota composition and pain-related outcomes in postpartum populations. Findings were synthesized narratively due to heterogeneity in study designs and outcome measures.

Key Findings: Following delivery, the vaginal microbiota frequently shifts from Lactobacillus-dominated communities toward more diverse, pathogen-enriched profiles, while the gut microbiota undergoes substantial remodeling. This microbial dysbiosis, compounded by impaired epithelial barrier function, is associated with increased risks of puerperal infection and enhanced local and systemic inflammation. Such inflammation may sensitize peripheral nociceptors and amplify pain signaling through immune–neural interactions. Furthermore, microbiota-driven inflammation appears to influence central pain processing via the gut–brain axis, including glial activation, which may contribute to pain persistence beyond the acute postpartum period. Key obstetric factors, including mode of delivery, episiotomy, antibiotic exposure, and breastfeeding practices, modulate both microbiota composition and postpartum pain outcomes.

Conclusion: Maternal vaginal and gut microbiota represent modifiable contributors to postpartum pain. Microbiota-informed strategies, such as probiotic supplementation or dietary interventions, may serve as adjunctive approaches to pain management. Well-designed longitudinal and interventional studies are needed to establish causality and guide clinical translation.

Keywords: postpartum pain, vaginal microbiota, gut microbiota, puerperal infection, microbiota–immune interactions, obstetric interventions

Introduction

The postpartum period is a critical phase for women's reproductive and mental health, during which pain represents one of the most common postpartum discomforts. Most women experience varying degrees of acute pain after childbirth, including perineal wound pain or cesarean section incision pain. In a subset of women, however, pain persists beyond the expected healing period and may develop into chronic pain. A study has reported that approximately 6%–18% of women

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after cesarean delivery and 4%–10% of women after vaginal delivery experience ongoing pain during the postpartum period.[1] Puerperal infections and wound-related complications constitute another major health concern after childbirth, affecting approximately 5%–7% of postpartum women and contributing substantially to maternal morbidity.[2] Globally, infections during the puerperium, including endometritis and postpartum wound infections, remain among the leading causes of maternal mortality, accounting for an estimated 10%–15% of postpartum deaths.[3] Clinically, postpartum pain and infection are closely interrelated: infectious complications may exacerbate and prolong pain, while severe or persistent pain may serve as an early clinical indicator of an underlying disease.[4]

In recent years, increasing attention has been directed toward the potential role of microbiota dysbiosis in linking postpartum infection and pain.[5] The vagina and the gut are the two anatomical sites in which postnatal microecological changes are most prominent. During pregnancy, hormonal and immunological adaptations induce characteristic alterations in vaginal and gut microbial communities; childbirth and the puerperium further disrupt this balance, leading to subsequent microecological remodeling.[6] These postpartum microbial shifts may influence epithelial barrier integrity, local and systemic immune responses, and susceptibility to infection, thereby potentially affecting pain perception and recovery trajectories.[7] Emerging evidence suggests that alterations in vaginal and gut microecology may contribute to postpartum pain through inflammation-related pathways, immune-mediated nociceptive sensitization, and cross-organ signaling mechanisms.[8] However, existing studies addressing postpartum pain, infection, and changes in the microbiota are heterogeneous in design, timing, and outcome definitions, and their findings have not been comprehensively integrated.[9,10] Emerging evidence also suggests that microbiota may influence pain perception through neuroimmune mechanisms, including modulation of inflammatory cytokines, disruption of epithelial barrier function, and activation of peripheral and central sensitization pathways via the gut–brain axis.[11,12]

Despite growing interest in mechanisms related to the microbiota, the specific role of vaginal–gut microbiota crosstalk in postpartum pain remains poorly understood.[13] Most existing studies have examined vaginal or gut microbiota independently, with limited attention to their bidirectional interactions and integrated effects on postpartum pain outcomes.[13,14] Furthermore, the clinical relevance of microbiota-driven mechanisms in shaping maternal recovery, pain modulation, and long-term well-being remains unclear.[15,16] Addressing these gaps is essential for developing a more comprehensive understanding of postpartum pain and identifying potential microbiota-targeted therapeutic strategies.

Therefore, this integrative narrative review synthesizes current evidence to summarize interactions between postpartum pain and vaginal and gut microbiota. To provide a comprehensive overview, we first characterize the distinct microbial shifts in the vaginal and gut environments during the puerperium. We then detail the mechanistic pathways, including immune-mediated sensitization and the gut-brain axis, through which this dysbiosis amplifies pain. Finally, we explore the clinical implications of these interactions, focusing on how microbiota-targeted interventions may serve as adjunctive approaches to postpartum pain management.

Materials and Methods

We conducted a structured literature search and narrative synthesis. Databases including PubMed, Embase, and Web of Science were searched for English-language literature published from January 1, 2017, to December 31, 2025. Search terms included: (postpartum OR postnatal OR puerperal) OR (pain OR “postpartum pain” OR “perineal pain” OR “cesarean section pain”) AND (“vaginal microbiota” OR “vaginal microbiome” OR “gut microbiota” OR “gut microbiome”) AND (infection OR “postpartum infection” OR endometritis OR “wound infection” OR “surgical site infection”). The search strategy and the number of records retrieved from each database are summarized in Table 1.

Inclusion criteria were: (a) study participants were puerperal women or cohorts in the postpartum period; (b) studies explored associations between postpartum pain and vaginal or gut microbiota, including infection-related outcomes; (c) study types included clinical observational studies, randomized controlled trials, mechanistic animal or in vitro studies, and relevant review articles; and (d) full text was available in English. Exclusion criteria were inaccessible abstracts, irrelevant topics, or duplicate data.

Two independent reviewers screened the studies and extracted key findings based on thematic relevance. Supplementary searches were conducted by tracing references. A total of 813 records were identified, and 84 studies were included in the narrative synthesis. A total of 813 records were identified, and 80 studies were included for narrative synthesis (Figure 1).

Table 1 Search Strategy and Number of Records Retrieved from Each Database

Database	Search Strategy	Records Identified
PubMed	(postpartum OR postnatal OR puerperal) OR (pain OR "postpartum pain" OR "perineal pain" OR "cesarean section pain") AND ("vaginal microbiota" OR "vaginal microbiome" OR "gut microbiota" OR "gut microbiome") AND (infection OR "postpartum infection" OR endometritis OR "wound infection" OR "surgical site infection")	251
Embase	("postpartum" OR "postnatal" OR "puerperal") AND ("pain" OR "postpartum pain" OR "perineal pain" OR "cesarean section pain") AND ("vaginal microbiota" OR "vaginal microbiome" OR "gut microbiota" OR "gut microbiome") AND ("infection" OR "postpartum infection" OR "endometritis" OR "wound infection" OR "surgical site infection")	303
Web of Science	TS=((postpartum OR postnatal OR puerperal) OR (pain OR "postpartum pain" OR "perineal pain" OR "cesarean section pain")) AND TS=("vaginal microbiota" OR "vaginal microbiome" OR "gut microbiota" OR "gut microbiome") AND TS=(infection OR "postpartum infection" OR endometritis OR "wound infection" OR "surgical site infection")	235

Given the heterogeneity of study designs, postpartum time windows, pain assessments, and microbiological detection methods, we conducted a narrative synthesis to integrate the evidence. This manuscript is a narrative rather than a systematic review; although a structured search was conducted across PubMed, Embase, and Web of Science, the review did not aim to capture all eligible literature exhaustively or to perform formal quality or risk-of-bias assessments. Instead, included studies were selected to provide a comprehensive, thematically organized synthesis of current knowledge on postpartum pain, vaginal and gut microbiota, and associated immune and mechanistic pathways. Results were organized by key themes, including associations between postpartum infection and pain, vaginal and gut microecology, inflammatory and neural sensitization mechanisms, cross-organ microbiota–immune interactions, and clinical implications for intervention.

Clinical Associations and Disease Burden of Postpartum Pain Related to Infections and Wound Complications

Postpartum infections (also known as puerperal infections) and delivery-related wound complications are closely associated with maternal pain, and the two are often mutually reinforcing, exacerbating the postpartum disease burden.[17] Clinical observations have found that women with infectious complications usually experience more severe and persistent pain. However, not all studies report consistent findings, and the strength of this association varies across study designs, populations, and definitions of postpartum pain. For example, when an infection occurs in an episiotomy or perineal laceration wound, local erythema, swelling, and pain are markedly aggravated, wound healing is prolonged, and persistent pain can seriously affect the mother's life.[18] The reported incidence of postpartum perineal wound infections varies widely across populations, ranging from 0.1% to 23.6%, and the incidence of wound dehiscence is about 0.21% to 24.6%.[19] This significant variation reflects the influence of different obstetric interventions, hygienic conditions, and reporting standards. For example, some studies have indicated that among various types of perineal lacerations, surgically incised (episiotomy) wounds have the highest infection rate, approaching nearly 10%. Even in high-income countries, reported infection rates for episiotomy wounds are 0.8%–11%, while in resource-limited settings they can be as high as 25%.[20] The clinical consequences are manifold: wound infections not only increase maternal pain and discomfort but also significantly reduce quality of life and increase healthcare burden, such as requiring additional antibiotic therapy, more outpatient visits, and even hospitalization.[21] Beyond perineal wounds, postpartum complications such as cesarean section incision infection, endometritis, and urinary tract infection can also cause moderate-to-severe pelvic or abdominal pain. It is important to emphasize that infections during the puerperium remain a significant cause of maternal morbidity and mortality worldwide. In low-resource regions, severe infections (puerperal fever) can lead to sepsis and death; even in high-income countries, postpartum sepsis remains the second leading cause of peripartum mortality.[22] Therefore, timely identification and management of infections are crucial for alleviating postpartum pain and reducing the disease burden.

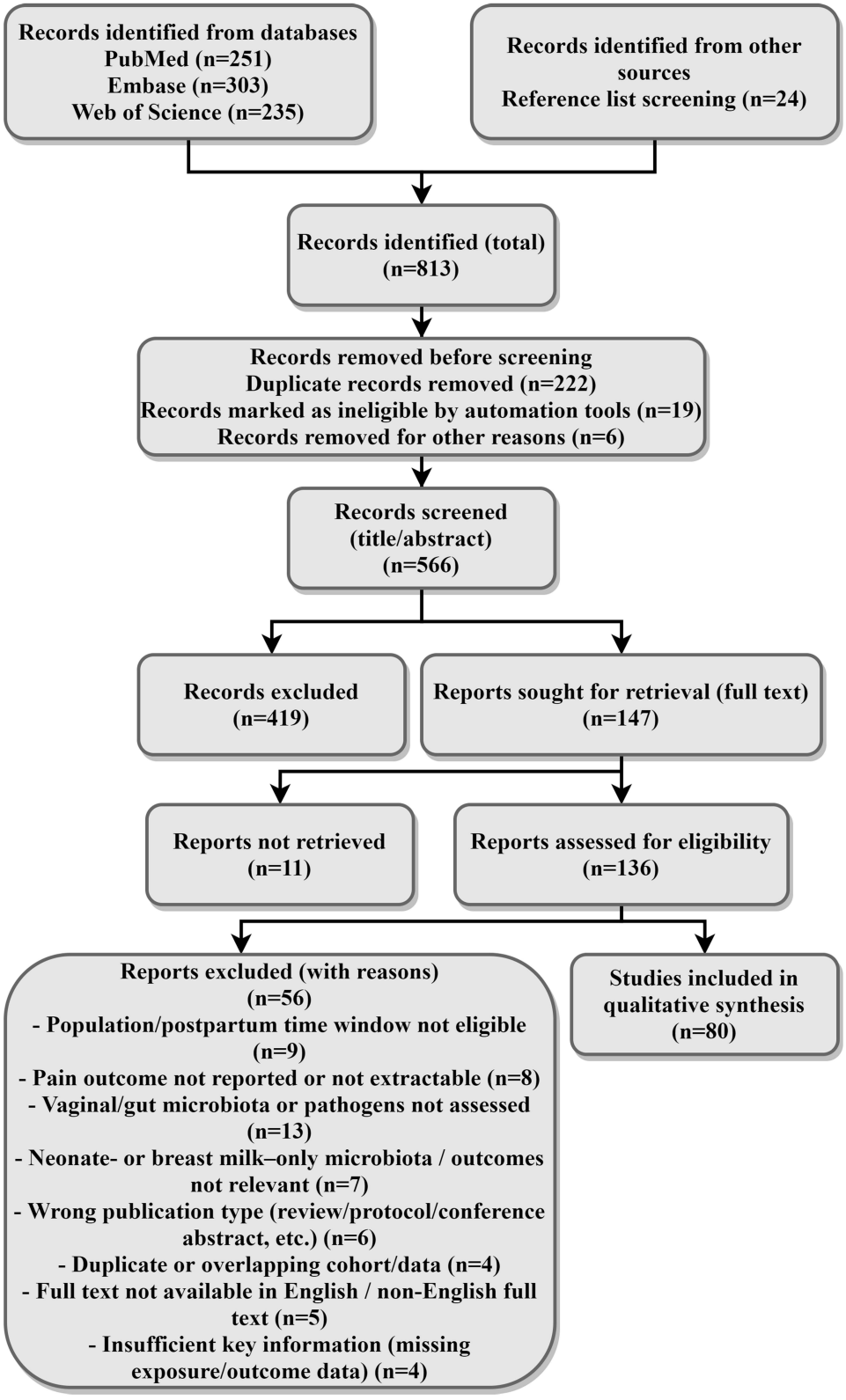

Figure 1 Flow diagram of study selection.

Pain itself is also an important clinical indicator of infection. For example, infection of the postpartum perineal wound often initially presents as severe localized wound pain, accompanied by swelling, foul-smelling discharge, or wound dehiscence.[23] In current obstetric practice, the diagnosis of perineal wound infection primarily relies on these local manifestations, including marked perineal pain, purulent discharge, and wound dehiscence. For newly delivered women, a certain degree of wound pain and discomfort is expected during normal postpartum recovery, which may mask early signs of infection. Therefore, healthcare providers need to remain vigilant about "normal" versus "abnormal" pain. If the severity of wound pain does not match the objective healing status or continues to worsen, a potential infection should be suspected.[24] The association between postpartum pain and infection is also reflected in their impact on maternal psychological status and functional recovery. Persistent severe pain can hinder mothers from ambulating and caring for the newborn, increasing the risk of anxiety and depression. Studies have shown that puerperal infections exacerbate maternal anxiety and significantly increase the incidence of postpartum depression, interfering with mother-infant bonding and breastfeeding.[24,25] In summary, infections and wound issues play essential roles in the occurrence and persistence of postpartum pain, increasing the burden of postpartum recovery. Reducing infection rates and promptly controlling infections are therefore of critical clinical significance for alleviating postpartum pain and improving maternal outcomes.

Postpartum Vaginal Microecological Remodeling and the Transition to Pathogen Enrichment

The female vaginal microecology undergoes dramatic changes during pregnancy and childbirth. In a healthy pregnancy, the vaginal environment is usually dominated by Lactobacillus with low microbial diversity, a state that helps suppress the proliferation of pathogenic bacteria and protects pregnant women from ascending infections.[26] However, after delivery, the sharp decline in hormonal levels during the puerperium (especially reduced estrogen), together with physiological changes in the vaginal environment after placental delivery, leads to rapid reassembly of the vaginal microbiota. Longitudinal studies from pregnancy through the postpartum period consistently show that the vaginal microbiome of postpartum women becomes significantly more diverse, with prior Lactobacillus dominance disrupted.[27,28] In a longitudinal observation of 48 women, Nunn et al found that postpartum vaginal microbiota α-diversity increased significantly, the abundance of Lactobacillus was markedly lower than in late pregnancy, and the relative proportions of streptococci (such as Streptococcus agalactiae [Group B Streptococcus]) and anaerobes (such as Prevotella bivia) increased significantly.[29] Another metagenomics-based study comparing the vaginal microbiota of women at 6 weeks postpartum with those of reproductive-age and postmenopausal women likewise showed that Lactobacillus was no longer dominant in the postpartum group, which exhibited the highest vaginal microbiota diversity, accompanied by enrichment of multiple potential pathogens.[30]

Specifically, typical beneficial commensals in the vagina of puerperal women (such as Lactobacillus crispatus and *L. iners*) were far less abundant than in women of reproductive age. In contrast, specific opportunistic pathogens increased significantly, including Atopobium vaginae, Gardnerella vaginalis, Prevotella species, and Escherichia coli from the gut.[31] Pathogens such as Chlamydia trachomatis could even be detected in vaginal samples of some women. These changes make the postpartum vaginal community configuration closer to the so-called "community state type IV (CST-IV)"—a state of low Lactobacillus with a multitude of mixed taxa. Nevertheless, the extent and clinical implications of these microbiota changes vary across studies, and some findings remain inconsistent due to differences in sampling time points and analytical methods.[32–34] Specifically, while many studies emphasize a shift toward "pathogen-enriched" states, this is not a universal trajectory. Some longitudinal cohorts have identified a subset of women who maintain Lactobacillus dominance or achieve rapid microbial stabilization within weeks of delivery.[27,33,35] These discrepancies highlight a significant methodological limitation in the current literature: the lack of standardized sampling intervals and the high inter-individual variability in "baseline" microbial signatures, which complicate comparisons across diverse global populations.

In a study that collected vaginal samples at multiple time points from 5 days to 19 months postpartum, fewer than one-third of women still had Lactobacillus-dominated communities, while most of the remainder exhibited highly heterogeneous communities composed of various anaerobes.[36] In the early postpartum period, decreased estrogen levels

lead to reduced glycogen content in the vaginal epithelium, thereby affecting the survival of Lactobacillus; meanwhile, postpartum vaginal discharge (lochia) increases vaginal pH, creating conditions for the growth of facultative anaerobes.[37] Another study examined compositional changes in postpartum vaginal secretions and found that lactic acid content decreased significantly, whereas proinflammatory factors, such as heat shock protein 70 (Hsp70) and hyaluronic acid, increased.[38] These changes indicate that delivery-induced vaginal mucosal injury and dramatic hormonal shifts "remodel" the vaginal microenvironment, weakening Lactobacillus's competitive advantage and favoring colonization by diverse microbial communities.[33] It is noteworthy that dysregulation of the postpartum vaginal microecology may render women more susceptible to pathogenic invasion. The study by Li et al found that the female reproductive tract's susceptibility to pathogenic microorganisms increases significantly postpartum and warrants clinical attention. This also explains why the postpartum period is a high-incidence stage for infections such as bacterial vaginosis (BV): BV-associated taxa, such as Gardnerella and Atopobium, are common postpartum, and pathogens previously suppressed during pregnancy have the opportunity to proliferate.[34] When such microbiota disturbances coincide with postpartum wounds or tissue injury, clinical infections are easily triggered and lead to pain. In summary, during the puerperium, the vaginal microbiota shifts from "Lactobacillus protection" to "coexistence of diverse consortia and opportunistic pathogens." This change is both part of the normal physiological resolution after childbirth and a breeding ground for pathogenic microorganisms, forming a critical microecological basis for postpartum infection and pain.

Postpartum Gut Microecological Changes and Their Association with Emotion/Pain Processing

Similar to the vaginal microenvironment, the female gut microbiota undergoes dynamic changes during pregnancy and postpartum. In late pregnancy, due to maternal metabolic and immune regulatory needs, the gut microbiota often exhibits a distinct pattern, including increased α-diversity, an altered Firmicutes-to-Bacteroidetes ratio, and increased levels of specific opportunistic pathogens, mimicking a metabolic syndrome-like state to store energy.[39] After delivery, with the sharp decline in pregnancy hormones and dramatic lifestyle changes (diet, sleep), the maternal gut microbiota begins to transition from the pregnancy state. However, current understanding of the maternal gut microbiota postpartum remains relatively limited. Some studies suggest that the postpartum gut microbiota may partially revert to the pre-pregnancy state in the early puerperium but is still influenced by residual effects of pregnancy and postpartum-specific factors.[9] For example, reductions in postpartum estrogen and progesterone levels may alter the composition of the gut microbiota by affecting the activity of bacterial hormone-metabolizing enzymes (such as β-glucuronidase). A low-estrogen environment may lead to decreased levels of certain hormone-dependent commensals, while other taxa increase.[40] Additionally, maternal dietary patterns, sleep deprivation, psychological stress, and lactation behaviors (which affect hormone and metabolic levels) may all shape postpartum changes in the gut microbiota. These dynamic changes have potential associations with postpartum mood and pain states. Recent studies have focused on the role of the gut microbiota in regulating central nervous system function and pain perception via the gut–brain axis.[41,42] Substantial evidence indicates that gut microbiota dysbiosis is associated with mood disorders such as depression and anxiety, as well as chronic pain syndromes.[43,44] For postpartum women, postpartum depression (PPD) is a common complication of childbirth, and its pathogenesis involves multiple disturbances, including neuroendocrine, immune, and microbiota factors.[45]

Accumulating evidence indicates that the gut microbiota of patients with PPD differs from that of healthy postpartum women, with depletion of beneficial genera such as Bifidobacterium and Bacteroides and enrichment of taxa associated with aberrant metabolic potential.[46] A Mendelian randomization study identified five gut bacterial species with causal associations with PPD; for instance, higher abundances of Prevotellaceae and Bifidobacterium may reduce depression risk by increasing xanthine and lysophosphatidic acid.[47,48] These findings suggest that gut microbiota disturbances may be associated with adverse postpartum mood and pain; however, causal relationships remain to be fully established.[46] Moreover, several studies have failed to replicate specific bacterial "signatures" for postpartum depression or pain, suggesting that the gut microbiota's influence may be secondary to more dominant clinical factors such as acute sleep deprivation or the abrupt withdrawal of pregnancy hormones.[49,50] Most current research remains cross-sectional, leaving it unclear whether microbial dysbiosis is a primary driver or a secondary consequence of the physiological and

psychological stress associated with the puerperium.[51–53] Maternal psychological status is closely related to pain perception. Slomian et al showed that women with higher postpartum pain scores were more likely to develop depressive symptoms in the early postpartum period.[54] Women with postpartum depression reported more pain-related complaints, more frequently required additional analgesics, and had poorly controlled postpartum pain.

Thus, postpartum pain and low mood often co-occur and may influence each other. Shared mechanisms include chronic low-grade inflammation and neuroendocrine imbalance induced by gut microbiota dysbiosis.[55] Infections during the puerperium can exacerbate maternal stress and depression risk, highlighting the role of microbiota-related immune responses in maternal mood and pain. Although direct studies on postpartum gut microbiota and pain are limited, evidence suggests that postpartum changes in gut microbiota can modulate central nervous system excitability and pain regulation by influencing inflammatory mediators and neurotransmitters.[56] For example, short-chain fatty acids and tryptophan metabolites produced by the gut microbiota act on the vagus nerve and blood–brain barrier to exert analgesic or pronociceptive effects, while endotoxin (LPS) entering the bloodstream increases nociceptive sensitivity.[57,58] These mechanisms may explain co-occurring postpartum pain and mood disorders. Interventions targeting the gut microbiota may offer new approaches to alleviating postpartum chronic pain and psychological problems. A summary of key microbial shifts in the postpartum vagina and gut, along with their clinical implications, is provided in Table 2.

Microbiota-Related Inflammation and Peripheral/Central Neural Sensitization Mechanisms

The microbiota-gut-brain axis is increasingly recognized as a critical mediator of host immune responses and neural signaling.[11] Alterations in the gut microbiota can influence both peripheral and central inflammation, affecting pain perception and sensitivity. These interactions highlight how microbial communities may contribute to the development and persistence of postpartum pain. Whether the infection involves a perineal wound, the endometrium, or other sites, invasion by pathogenic microorganisms initially elicits a robust local immune-inflammatory response.[64] Pathogens and their associated molecules (such as lipopolysaccharide [LPS] from Gram-negative bacteria) are recognized by host pattern recognition receptors (such as Toll-like receptor TLR4), thereby activating innate immune cells including macrophages and neutrophils, and subsequently triggering a cascade of inflammatory mediator release.[65] For example, after LPS binds to TLR4, immune cells are stimulated to secrete high levels of proinflammatory cytokines (such as

Table 2 Characteristics of Microbial Shifts and Clinical Implications

Microbial Site	Postpartum Changes	Key Taxa Affected	Clinical Implications*	Evidence Strength†
Vagina	Increased diversity; loss of *Lactobacillus* dominance	↓ *Lactobacillus crispatus, L. iners*; ↑ *Gardnerella vaginalis, Atopobium vaginae, Prevotella* spp., *Escherichia coli*	Associated with increased risk of puerperal infections, perineal wound complications, and bacterial vaginosis; may contribute to heightened pain perception through local inflammation	Postpartum-specific human (multiple studies)[27–30,36–39]
Gut	Dynamic remodeling; partial reversion to pre-pregnancy state; influenced by diet, sleep, hormones	↓ *Bifidobacterium, Bacteroides*; ↑ *Prevotellaceae*, other opportunistic taxa	Modulates systemic inflammation and gut–brain axis signaling, which may influence mood and pain sensitivity; linked to postpartum depression in some studies	Postpartum-specific human (limited);[9,45–48] extrapolated from non-postpartum populations[41–44]
Vagina–Gut Interaction	Microbial migration from gut to vagina; convergence during peripartum	Overlapping taxa: *Enterococcus* spp., *E. coli*, anaerobes	Cross-organ microbial dysbiosis is associated with pelvic inflammation and may contribute to persistent pain; systemic immune activation may amplify symptoms	Emerging; primarily associative[59–63]

Notes: *Clinical implications are derived from postpartum-specific studies where available; supporting references are provided in the table †Evidence strength categories: Postpartum-specific human = direct evidence from human postpartum cohorts; Emerging = limited human data or consistent animal data awaiting replication; Extrapolated = derived from non-postpartum populations.

interleukin-6 and tumor necrosis factor-α), chemokines (such as CXCL1 and CCL20), and lipid mediators (such as prostaglandin E2).[66] These mediators induce vasodilation, tissue edema, and heightened sensitivity of nociceptive nerve endings at the site of infection. Substances such as prostaglandin E2 reduce the excitability threshold of nociceptive neurons, inducing peripheral sensitization so that even minor stimuli can evoke pain signals.[67]

Meanwhile, bacterial products can also directly activate sensory neuron receptors. For instance, lipopolysaccharide (LPS) binds to TLR4 receptors on sensory neurons, leading to sensitization of pain fibers expressing TRPA1. This process increases neuronal excitability and contributes to acute pain and hypersensitivity, highlighting a direct mechanism by which bacteria induce nociception independent of classical inflammatory pathways.[68]

Under sustained peripheral inflammatory stimulation, nociceptive signals are transmitted to the central nervous system via the dorsal horn of the spinal cord. Within the central nervous system, spinal glial cells (particularly microglia) are highly responsive to peripheral inflammatory and injury signals. Microglia, as intrinsic immune effector cells of the central nervous system, play key roles in the initiation and maintenance of chronic pain. When large volumes of peripheral nociceptive signals continue to influx, microglia are activated from a resting to a reactive state, undergoing morphological and phenotypic changes and releasing increased amounts of cytokines and neuroactive substances.[69] These activated microglia secrete proinflammatory mediators such as tumor necrosis factor-α (TNF-α) and interleukin-1β (IL-1β), thereby inducing central sensitization at the spinal level. Specifically, TNF-α and IL-1β enhance the responsiveness of dorsal horn neurons to afferent signals, inhibit the function of inhibitory neural pathways, and amplify the transmission of pain signals.[70] Consequently, even after the original peripheral injury has resolved, the nervous system remains in a heightened pain-sensitive "alert" state, contributing to the chronification of pain perception. In this context, microbiota-driven inflammation functions as both an "initiator" and an "amplifier": on one hand, pathogenic microorganisms trigger the initial inflammatory response, inducing peripheral sensitization;[71] on the other hand, sustained stimulation by microbial products (such as endotoxins entering the circulation through damaged mucosa) can maintain central glial cells in a proinflammatory state, collectively promoting the transition from acute to chronic pain.[72] It must be emphasized, however, that while these pathways, particularly the roles of TLR4 and spinal glial activation are well-supported in rodent models of chronic pain, their direct role in human postpartum populations remains partially hypothetical.[73] Most human data rely on systemic cytokine correlations, which lack the anatomical specificity to confirm central nervous system involvement.[74,75] Therefore, while these mechanisms provide a robust conceptual framework, they require rigorous validation through longitudinal human studies utilizing advanced neuroimaging or cerebrospinal fluid analysis to distinguish speculative theory from established clinical fact.

Notably, dysbiosis of the gut microbiota may also influence pain modulation at distant sites via similar mechanisms. For instance, severe gut microbiota imbalance can disrupt the intestinal mucosal barrier, leading to a "leaky gut" and allowing bacterial products to enter the bloodstream, thereby eliciting systemic low-grade inflammation.[76] Under such conditions, even if the wound has healed, elevated systemic levels of inflammatory mediators and the sensitized state of central immune cells may lower the pain threshold, resulting in heightened pain responses to normally innocuous stimuli and explaining generalized pain sensitivity or persistent pain in some postpartum women.[77]

Most of the aforementioned mechanisms have been derived from animal models and chronic pain research, but are also reflected in human postpartum pain. Therefore, while these mechanisms provide important biological insights, their direct applicability to postpartum populations remains partially hypothetical and requires further validation in human studies. For example, studies have reported significant abnormalities in the diversity and abundance of the gut microbiota in patients with chronic pelvic pain and fibromyalgia, and interventions targeting the gut microbiota (such as probiotic supplementation or fecal microbiota transplantation) have been shown to reduce inflammation and improve pain symptoms.[78] This suggests that modulating the microbiota to alleviate neuroinflammation may be a viable strategy for managing chronic pain. The main pathways linking alterations in the microbiota to peripheral and central pain sensitization are summarized in Table 3. For postpartum pain, if initial tissue injury or infection establishes a pathological microbiota–immune–neural interaction chain, then targeted interventions at any link in this chain (such as timely and effective control of the infectious source, inhibition of excessive inflammatory responses, and protection or restoration of intestinal barrier function) may help interrupt the vicious cycle of pain.[79] In summary, a complex network of microbiota-driven inflammatory and neural sensitization mechanisms underlies postpartum pain, amplifying pain

Table 3 Mechanistic Pathways of Microbiota-Induced Pain Sensitization

Mechanism	Pathway	Microbial Mediators	Pain Effect	Evidence Basis*
Peripheral Sensitization	Local infection or dysbiosis → immune activation → nociceptor sensitization	LPS, bacterial toxins, pro-inflammatory cytokines (IL-6, TNF-α), prostaglandins	Associated with lowered pain threshold and heightened local wound pain	Postpartum human data (indirect);[18,20,24] established in non-postpartum inflammatory pain models[66–68]
Central Sensitization	Persistent nociceptive input → glial activation in spinal cord	TNF-α, IL-1β released by activated microglia	May amplify pain signal transmission and contribute to pain chronicity	Animal models;[69,70] emerging human data in chronic pain;[72] limited postpartum-specific evidence
Gut–Brain Axis	Gut dysbiosis → systemic inflammation → CNS modulation	SCFAs, tryptophan metabolites, LPS, circulating cytokines	Linked to altered mood and pain perception; may be associated with postpartum depression risk	Non-postpartum human data;[42–44,56,57] emerging postpartum associative studies[45–47]
Cross-Organ Microecology	Gut–vagina–pelvis microbial exchange → immune cell trafficking	Gut-derived pathogens in vagina; systemic inflammatory mediators	May contribute to persistent pelvic pain through shared inflammatory pathways	Speculative; derived from mechanistic plausibility and indirect evidence[33,59,63,78]

Notes: *Evidence basis definitions: Postpartum human data: Direct evidence from human postpartum cohorts. Non-postpartum human data: Evidence from general chronic pain, depression, or microbiome research. Animal models: Mechanistic insights from preclinical studies. Speculative: Proposed pathways without direct empirical support in postpartum populations. Footnote: LPS (lipopolysaccharide) and pro-inflammatory cytokines predominantly mediate peripheral sensitization through local immune activation. SCFAs (short-chain fatty acids) and tryptophan metabolites primarily act via the gut–brain axis, influencing central pain processing. Central glial activation amplifies signals from both peripheral and gut-derived inflammatory pathways. Mechanisms are presented within the framework of postpartum physiology, with evidence basis indicated for each pathway.

signals from peripheral injury sites to the central nervous system. Elucidating these mechanisms in greater depth may facilitate the development of more effective multi-target analgesic and anti-inflammatory strategies.

Gut–Vagina–Pelvis Interactions and Cross-Organ Microecology–Immune Integration

The microbiota in different anatomical regions of the human body are not isolated; in particular, there exists a close bidirectional relationship between the gut and the reproductive tract microbiota in women. A substantial body of evidence indicates that many bacteria colonizing the female vagina originate from the gut: the rectum serves as an important "seed bank" for the vaginal microbiota, providing a continuous bacterial input.[59] Under healthy conditions, a considerable proportion of the vaginal and gut microbiota overlap. Evidence from synthetic community and metagenomic studies indicates that healthy vaginal microbiota is consistently dominated by Lactobacillus species, particularly Lactobacillus crispatus, coexisting with Limosilactobacillus spp. and Lactobacillus jensenii.[60] These co-occurrence patterns appear robust and largely independent of host factors or strain variability. Mechanistic insights from genome-scale metabolic modeling suggest that amino acid and vitamin cross-feeding supports community stability. Together, these findings provide a conceptual framework for designing rational microbiome-based interventions targeting vaginal health. Pregnancy and labor accentuate this microbial migration: rupture of membranes and dilation of the birth canal provide pathways for gut-derived microbiota to enter the uterus and vagina. Studies have reported that during late pregnancy and the early puerperium, the compositions of the vaginal and rectal microbiota gradually converge, a phenomenon termed peripartum "microbiota convergence".[61] This cross-site microbial exchange is highly relevant to the understanding of postpartum infections and pain. It explains why many pathogens implicated in puerperal infections (such as Escherichia coli, Enterococcus, and anaerobes) resemble gut commensals. These pathogens likely originate in the woman's gut, colonize the perineum, and ascend to cause vaginitis, endometritis, or urinary tract infection.[10] Consequently, the postpartum pelvic region can be considered an intersection of gut–vagina microecological interactions: the state of the gut microbiota influences the microbial balance of the vagina and uterus, while pelvic infections can reciprocally affect the gut microbiota.

The gut and reproductive tract share the systemic immune milieu.[62] When a local infection occurs in the vagina or uterus, activated immune cells and inflammatory mediators are not confined to the local site. Still, they are released into

the circulation, eliciting systemic responses and thereby influencing immune homeostasis of the intestinal mucosa.[80] Similarly, systemic inflammation or immune cell "reprogramming" caused by gut microbiota dysbiosis can impact immune surveillance in the reproductive tract. For instance, short-chain fatty acids (SCFAs) produced by a healthy gut microbiota exert anti-inflammatory and barrier-maintaining effects, preventing bacterial translocation and systemic inflammation.[81] However, when analogous metabolites accumulate in the vaginal environment, divergent effects may occur: studies indicate that certain SCFAs in the reproductive tract may disrupt local microbiota and induce inflammation, resulting in adverse obstetric and gynecological outcomes.[82] This illustrates the organ-specific effects of similar microecological factors and underscores the need to consider cross-organ microbiota–immune interactions in an integrated framework. Based on current evidence, a postpartum pain–related "gut–vagina–pelvis interaction" model can be proposed: the gut and vagina, as adjacent and interconnected ecological niches, are linked through microbial migration, metabolites, and immune signals.[83] During the postpartum period, if the maternal gut microbiota is healthy (eg., enriched in anti-inflammatory commensals), the intestinal barrier remains intact and systemic inflammation is minimal; the vagina is also likely to maintain a favorable microbial balance (eg., Lactobacillus dominance), reducing the risk of infection and pain.[84] Conversely, if maternal gut microbiota is dysregulated and intestinal permeability increases, inflammatory triggers such as bacterial endotoxins can more readily enter the circulation, eliciting systemic inflammation and potentially compromising the immune barrier of the reproductive tract.[85] Concurrently, gut microbiota dysbiosis is often accompanied by overgrowth of potential pathogens in the rectal–perineal region (eg., Clostridioides difficile and Enterobacteriaceae), which can migrate to and colonize the vagina, causing microbial imbalance and infection.[63] Once the vaginal microbiota becomes dysregulated (eg., reduced Lactobacillus and elevated pH), it further promotes pathogen proliferation, triggering pelvic inflammation and pain. Within this cascade, immune cells act as "messengers" of cross-organ communication: activated lymphocytes and monocyte–macrophages circulate between the intestinal mucosa and pelvic tissues, transmitting signals across microecological environments and amplifying systemic effects of dysbiosis.[86] An integrated model can thus be described: in health, the gut–vagina microbiota and host immune system constitute a homeostatic network that maintains immune tolerance and anti-infective barriers in the birth canal and pelvis; in contrast, severe microecological imbalance at any site disrupts overall homeostasis, precipitating a local "inflammatory storm" in the pelvis and manifesting as infection-related pain.[87] This model emphasizes that addressing postpartum infections and chronic pain requires a holistic approach that considers both the local vaginal environment and the gut, with coordinated modulation of both systems being necessary for effective intervention. However, direct clinical evidence supporting these cross-organ interaction models in postpartum populations remains limited.

Effects of Obstetric Interventions on Microecology and Infection/Pain Outcomes

Various obstetric interventions, including mode of delivery, surgical procedures, and medication use can inadvertently disrupt the maternal microecological balance, thereby influencing the incidence of infection and pain outcomes. Mode of delivery directly affects microbial exposure. During vaginal delivery, the fetus traverses the birth canal and can partially "adsorb" maternal vaginal microbiota, while maternal vaginal microbiota may also be altered by trauma, bleeding, and other delivery-related factors.[88] In contrast, cesarean section reduces direct loss of vaginal microbiota but involves perioperative administration of broad-spectrum antibiotics. Routine prophylactic antibiotics typically intravenous cephalosporins effectively reduce postoperative infections, such as endometritis and wound infections, by approximately 60–70%.[89] However, such interventions can disrupt maternal vaginal and gut microbiota. Brockway et al reported that antibiotic use during pregnancy or the perinatal period markedly reduces microbial diversity, eliminating susceptible commensals such as Lactobacillus and Bifidobacterium while creating ecological niches conducive to overgrowth of drug-resistant opportunistic pathogens or fungi.[90] This may contribute to secondary infections post-cesarean section, such as Clostridioides difficile colitis or vulvovaginal candidiasis, although evidence remains heterogeneous and context-dependent. Consequently, while perioperative antibiotic prophylaxis remains essential, clinicians should weigh benefits against potential microecological disruption and consider mitigation strategies, such as using narrow-spectrum agents or timely postoperative probiotic supplementation.[91]

Operative interventions during labor also influence maternal microbial balance. For instance, routine episiotomy, although it can facilitate wound repair, enlarges the wound area and increases susceptibility to infection and postpartum pain.[92,93] Given the proximity of the perineum to the anus, an episiotomy can provide a portal for enteric bacteria to invade deep tissues. Accordingly, current obstetric practice emphasizes restrictive episiotomy, performing it only when clinically indicated, alongside stringent aseptic technique and postoperative care.[94] Similarly, instrumental delivery (eg., forceps or vacuum extraction) is associated with greater perineal trauma and mucosal injury, leading to higher rates of wound infection and persistent pain compared to spontaneous vaginal delivery.[95] This underscores the importance of gentle birthing techniques, such as perineal warm compresses and massage, to minimize lacerations and infection-related pain. Other obstetric interventions, including urinary catheterization and repeated vaginal examinations, may also disrupt the maternal microecology. Invasive procedures, if not performed under strict aseptic conditions, can introduce exogenous pathogens or perturb vaginal microbial homeostasis; indwelling urinary catheters postpartum significantly increase the risk of urinary tract infection.[96] Minimizing unnecessary interventions and adhering to aseptic protocols are therefore crucial. Breastfeeding, although not a medical intervention, significantly affects maternal and neonatal microbiota. Breast milk contains prebiotics and probiotics that support neonatal gut health, while lactation-induced changes in prolactin and oxytocin can influence maternal stress and immune function.[97] However, lactational amenorrhea and low estrogen levels may create a vaginal environment resembling menopause—reduced Lactobacillus and slightly elevated pH—predisposing some women to recurrent dysbiosis, bacterial vaginosis, or Candida infections, often accompanied by dyspareunia.[98] Vaginal health in lactating women should therefore be closely monitored, with consideration of probiotics or topical estrogen when clinically appropriate and safe for breastfeeding.[99]

From a microecological perspective, several clinical implications emerge. First, maintaining and restoring normal microbiota is crucial. For example, a healthy Lactobacillus community, particularly L. crispatus, can prevent late-pregnancy colonization by Group B Streptococcus, thereby reducing the need for peripartum antibiotic prophylaxis.[100] Personalized antimicrobial strategies are also important: women with severe vaginal dysbiosis or recurrent bacterial vaginosis may benefit from proactive monitoring and targeted interventions, including postpartum probiotics and prevention of Clostridioides difficile infection, while avoiding unnecessary antimicrobial exposure in women with favorable microbiota profiles.[101,102] Multidisciplinary management is essential. Obstetricians should collaborate with infectious disease and microbiology specialists to ensure timely pathogen identification via culture or 16S sequencing, enabling targeted therapy rather than reliance on broad-spectrum antibiotics.[103] Finally, comprehensive pain management should incorporate anti-inflammatory and microbiota-modulating approaches for women with dysbiosis or elevated inflammatory responses; for instance, combining probiotic supplementation with anti-inflammatory nutrients, such as ω-3 fatty acids, may reduce nervous system inflammation and enhance analgesic efficacy.[104] In summary, obstetric interventions profoundly affect maternal microecology. Balancing infection prevention with preservation of the microbiota and adopting microecology-friendly strategies can reduce infection-related postpartum pain and improve maternal and neonatal outcomes. The effects of various obstetric interventions on maternal microecology and postpartum pain outcomes are summarized in Table 4.

Limitations

The conclusions of this review should be interpreted within the context of several limitations. First, the body of literature specifically addressing the relationship between postpartum pain and vaginal–gut microbiota interactions is limited. Few studies directly evaluate postpartum pain as a primary outcome; many conclusions are inferred from related fields, such as perinatal depression or chronic pain, necessitating cautious interpretation. Findings across studies are inconsistent: some report associations between specific alterations in the microbiota and postpartum pain or mood disturbances, while others do not. Such variability may reflect differences in baseline microbiota composition arising from ethnicity, geography, or lifestyle factors.

Additionally, inconsistencies in defining "postpartum pain," assessment timing, and measurement instruments precluded quantitative synthesis, necessitating a largely qualitative narrative approach and introducing a degree of subjectivity. Second, the dynamic trajectories of postpartum vaginal and gut microbiota remain insufficiently characterized. Most studies involve small sample sizes and short-term follow-up, limiting understanding of microbiota recovery beyond 6 weeks or up to 1 year

Table 4 Impact of Obstetric Interventions on Maternal Microecology

Intervention	Microecological Effect	Consequences of Infection/Pain	Clinical Notes
Cesarean Section	Reduced exposure to maternal vaginal microbiota; altered neonatal gut seeding	Increased maternal wound infection risk; moderate-to-severe incision pain	Prophylactic antibiotics recommended; microbiota restoration strategies may help
Vaginal Delivery with Episiotomy	Tissue injury → local dysbiosis	Higher risk of perineal wound infection; prolonged pain	Timely wound care and infection monitoring crucial
Antibiotic Exposure	Broad-spectrum microbial disruption	Loss of beneficial Lactobacillus and Bifidobacterium; increased pathogen colonization	Consider judicious use; potential probiotic supplementation
Breastfeeding	Modulates maternal gut and vaginal microbiota via hormonal influence	Supports beneficial microbial populations; may reduce systemic inflammation and pain	Encourage early and exclusive breastfeeding when possible

postpartum. Heterogeneity in detection methods, including culture-based approaches, 16S rRNA sequencing, and metagenomic sequencing, further complicates comparisons across studies, as they differ in resolution and sensitivity. Third, mechanistic insights into microbiota–pain interactions are primarily derived from animal models. Rodents and other experimental models differ from humans in gestational cycle, microbial composition, and pain behavior assessment, limiting the direct applicability of these findings to postpartum populations. Fourth, this review was limited to English-language publications from 2017 onward, potentially excluding relevant earlier or non-English studies. Postpartum pain is inherently multifactorial, and the microbiota represents only one component of this complex system. Many studies inadequately controlled for confounders such as psychosocial environment, lactation status, and genetic susceptibility, complicating causal interpretation. Future research should prioritize large, prospective studies with long-term follow-up that integrate vaginal and gut microbiota profiling, pain assessments, biochemical markers, and psychological evaluations. Randomized controlled trials testing microbiota-targeted interventions, including probiotics, are warranted to assess their potential to prevent postpartum infections and chronic pain, thereby strengthening causal inference.

Future Directions and Clinical Translation

To advance the field beyond the current, largely associative evidence and toward clinically actionable insights, future research should prioritize large, prospective, multicenter cohort studies with extended follow-up (≥12 months postpartum) to characterize the temporal dynamics of vaginal and gut microbiota in relation to pain trajectories.[105] Such studies should use standardized sampling protocols, validated multidimensional pain assessment tools, and a comprehensive collection of covariates (mode of delivery, antibiotic exposure, breastfeeding, psychosocial factors, diet) to enable rigorous multivariable modeling and strengthen causal inference.[105,106] Beyond 16S rRNA sequencing, multi-omics approaches, including shotgun metagenomics, metatranscriptomics, and metabolomics, should be incorporated to identify microbial functional activity and bioactive metabolites (eg., SCFAs, bile acids, tryptophan derivatives) that mediate host–microbe interactions.[53] Concurrent assessment of host inflammatory markers, epithelial barrier function, and neurophysiological or neuroimaging parameters may help validate proposed microbiota–immune–neural mechanisms.[107] Well-designed randomized controlled trials evaluating microbiota-targeted interventions (probiotics, prebiotics, dietary modifications, and antimicrobial stewardship) are needed to move from association to causation.

Standardization of outcome measures, including consensus-driven definitions of acute, subacute, and persistent postpartum pain, validated pain assessment tools, and clear differentiation between infection-related and non-infection-related pain, is essential. Future studies should include diverse and representative populations to improve generalizability and address disparities in postpartum infection burden.[38,108] Parallel efforts should focus on developing clinically feasible tools for risk stratification and management, such as microbiota-informed screening approaches in late pregnancy or the early postpartum period. Evidence-based guidelines for microecology-informed postpartum care should balance potential benefits with cautious

interpretation to avoid overmedicalization. While promising, many proposed mechanisms and interventions remain to be validated in well-designed human studies, emphasizing the need for careful clinical translation.

Conclusion

Postpartum pain significantly impacts maternal recovery and quality of life, representing a critical focus for both clinical management and research. This review synthesizes current evidence, highlighting the central roles of vaginal and gut microecology in the onset and regulation of postpartum pain. Following delivery, the vaginal microbiota often shifts from Lactobacillus dominance to a more diverse configuration enriched with anaerobes and gut-derived pathogens, increasing susceptibility to reproductive tract infections. Concurrently, the gut microbiota undergoes remodeling during the puerperium, and dysbiosis or barrier dysfunction may influence mood and pain sensitivity via the gut-brain axis. Microbiota-driven immune inflammation spans both peripheral tissue injury and central neural sensitization, contributing to heightened pain perception.

Together, the gut and vaginal microbiota, in coordination with the immune system, form a cross-organ network that maintains pelvic homeostasis but may exacerbate inflammation and pain when disrupted. Obstetric interventions, including antibiotic use, mode of delivery, and episiotomy- can further influence maternal microecology, underscoring the importance of preserving beneficial microbiota while preventing infection. Microecology-oriented strategies show potential for clinical application, such as maintaining vaginal Lactobacillus dominance at delivery, correcting postpartum gut dysbiosis, and implementing personalized antimicrobial therapies for high-risk microbiota profiles; these approaches could reduce infection rates and mitigate chronic pain.

However, current evidence remains limited, and many proposed mechanisms are associative or hypothetical rather than conclusively causal. High-quality, longitudinal cohort studies, mechanistic investigations, and randomized controlled trials are needed to clarify the relationships between postpartum pain and vaginal–gut microecology, validate potential interventions, and optimize strategies for clinical implementation. Improved understanding of these interactions may illuminate the pathophysiology of chronic pelvic pain and postpartum depression, offering novel targets for postpartum care and enhancing long-term maternal health and quality of life.

Data Sharing Statement

Not applicable. This manuscript is a narrative review, and no new datasets were generated or analyzed during the study.

Declarations Ethics Approval, and Consent to Participate

Not applicable. This study is a narrative review and does not involve human participants or animals.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

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