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The Role of the Microbiome in the Occurrence, Progression, and Treatment of Ovarian Cancer: Mechanistic Insights and Clinical Translation

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Abstract. Ovarian cancer (OC) is the most lethal tumor of the female reproductive system, mainly because most cases are diagnosed at an advanced stage and show high recurrence and resistance to therapy. Advances in microbiome research have shown that microbial communities are closely linked to cancer development, progression, and treatment response. Microbiota in the gut and reproductive tract influence ovarian cancer through several mechanisms, including regulation of systemic immunity, maintenance or induction of chronic inflammation, hormonal effects, and metabolic changes. Microbial imbalance can enhance oncogenic signaling, weaken immune surveillance, and alter estrogen metabolism, which together promote tumor growth and contribute to chemoresistance. Microbial metabolites such as short-chain fatty acids and indole derivatives act as bioactive regulators of the tumor microenvironment, while microbial components like lipopolysaccharides can activate inflammatory pathways that support tumor survival. This review summarizes current evidence on the diverse roles of the microbiome in ovarian cancer biology and explains key links between microbial alterations and tumor pathophysiology. It also examines how gut and reproductive tract microbiota shape treatment outcomes, particularly responses to chemotherapy and immune checkpoint inhibitors. Emerging microbiome-based strategies, including probiotic interventions, fecal microbiota transplantation (FMT), and modulation of microbial metabolites, are highlighted for their potential to improve patient prognosis. With the integration of multi-omics technologies, artificial intelligence, and systems biology, the microbiome may become an important tool for early detection, personalized treatment, and precision oncology in ovarian cancer.

Keywords: Ovarian cancer, Microbiome, Inflammation, Immune modulation, Immune checkpoint inhibitors

1. Introduction

OC is one of the most lethal malignancies of the female reproductive system, characterized by high mortality and limited therapeutic success. Due to the absence of specific early symptoms, more than 70% of patients are diagnosed at an advanced stage (FIGO III–IV), resulting in a five-year survival rate below 40% [1]. The disease exhibits remarkable heterogeneity, driven by a complex interplay

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among genetic, hormonal, inflammatory, and metabolic factors. Understanding its underlying mechanisms remains essential for developing improved diagnostic and therapeutic strategies.

With the rapid evolution of microbiology and multi-omics technologies, the human microbiome has emerged as a key regulator of health and disease, including cancer. Both gut and reproductive tract microbiota influence tumorigenesis through immune modulation, inflammation, metabolic reprogramming, and DNA repair regulation. Microbial dysbiosis has been linked to tumor progression, chemoresistance, and prognosis in multiple malignancies. Accordingly, the microbiome is increasingly recognized as a potential source of diagnostic biomarkers and therapeutic targets in ovarian cancer.

Despite increasing interest, research on the ovarian cancer microbiome remains limited compared with studies on gastrointestinal malignancies. A comprehensive synthesis of current evidence is therefore essential to clarify how microbial communities contribute to ovarian oncogenesis and to assess their translational potential in clinical settings.

This review systematically summarizes current knowledge regarding the role of the microbiome in the initiation, progression, and treatment of ovarian cancer. It first outlines the epidemiological and pathological features of OC, followed by a discussion of microbial mechanisms involved in carcinogenesis, including inflammation, immune modulation, metabolic alteration, and genotoxic effects. Subsequent sections examine how the gut and reproductive tract microbiota specifically influence ovarian tumor biology and therapeutic response, with additional consideration of possible contributions from oral and skin microbiota. The review concludes by evaluating microbiome-based biomarkers, emerging therapeutic strategies such as probiotics and FMT, and future prospects for clinical translation within precision oncology frameworks.

2. Epidemiology, pathology, and traditional mechanisms

2.1. Epidemiology

OC represents about 3% of all female malignancies yet remains the leading cause of death among gynecologic cancers [2]. Data from the World Health Organization and the International Agency for Research on Cancer show marked global differences in incidence and mortality: rates tend to be higher in developed countries and lower in many regions of Asia and Africa, likely reflecting variations in genetic background, lifestyle, and reproductive patterns [3].

Genetic predisposition plays a major role. Germline mutations in BRCA1 or BRCA2 substantially increase lifetime risk, and approximately 10–15% of epithelial ovarian cancers harbor these mutations [4]. Lynch syndrome, caused by pathogenic variants in mismatch-repair genes (MLH1, MSH2, MSH6, PMS2), also elevates risk for ovarian malignancies [5].

Reproductive and hormonal factors further modify susceptibility. Early menarche, late menopause, nulliparity, and infertility are associated with increased risk, whereas pregnancy, breastfeeding, and prolonged use of oral contraceptives confer protection [6–8]. Postmenopausal hormone replacement therapy, particularly estrogen-only regimens, increases the risk of ovarian cancer, whereas combined estrogen–progestin therapy appears to mitigate it [9]. Chronic inflammatory conditions—such as endometriosis or pelvic inflammatory disease—additionally contribute to oncogenic risk by promoting oxidative stress and DNA damage [10].

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2.2. Pathology and molecular subtypes

Epithelial ovarian cancer constitutes over 90% of all OC cases and is subdivided into distinct histotypes with specific molecular features. High-grade serous carcinoma (HGSOC)—the predominant subtype—accounts for roughly 70% of EOCs and is defined by TP53 mutations and BRCA1/2-related homologous recombination deficiency. Low-grade serous carcinoma (LGSOC) is less frequent, characterized by KRAS or BRAF mutations and slower progression. Other subtypes include mucinous carcinoma (MOC), clear cell carcinoma (CCOC), and endometrioid carcinoma (EnOC), each with unique genomic profiles and variable chemoresistance patterns.

Non-epithelial ovarian cancers, comprising germ-cell and sex-cord stromal tumors, are less common but clinically distinct. These variations underscore the need for personalized approaches to diagnosis and therapy.

2.3. Conventional pathogenesis

The pathogenesis of ovarian cancer involves a network of genetic, hormonal, and inflammatory events. BRCA1/2 mutations compromise DNA repair, leading to genomic instability, while TP53 mutations—present in nearly all HGSOCs—disable cell-cycle control and apoptosis. Other key mutations (KRAS, PTEN, PIK3CA, ARID1A) activate proliferative and survival pathways.

Hormonal influences are substantial in ovarian cancer. Estrogen receptor signaling supports cellular proliferation and enhances angiogenesis. Chronic inflammation further contributes to tumor development through activation of NF-κB, IL-6, and TNF-α pathways, which generate an immunosuppressive environment that favors tumor growth and resistance to therapy. Together, these factors illustrate how genetic instability, hormonal imbalance, and persistent inflammation collectively drive ovarian oncogenesis.

3. Microbiome and cancer: general mechanisms

3.1. The human microbiome

The human microbiome consists of diverse microbial populations residing in the gut, reproductive tract, oral cavity, skin, and other anatomical sites. Under physiological conditions, these microorganisms support host homeostasis by regulating immune responses, coordinating metabolic functions, and strengthening epithelial barriers. Dysbiosis—an alteration in microbial composition or function—has been associated with inflammatory disorders, metabolic abnormalities, and cancer development.

The gut microbiota plays a particularly central role, influencing immune system maturation, hormone metabolism, and systemic inflammatory signaling. In the female reproductive tract, the microbiota is typically dominated by Lactobacillus species, which preserve mucosal stability by producing lactic acid and maintaining an acidic environment. Disruption of this balance may increase susceptibility to infection, promote chronic inflammation, and facilitate malignant transformation.

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3.2. Mechanistic pathways linking microbiota and cancer

3.2.1. Chronic inflammation

Persistent infection and inflammation are recognized as key drivers of carcinogenesis. Pathogens such as Helicobacter pylori (gastric cancer) and Fusobacterium nucleatum (colorectal cancer) promote pro-inflammatory signaling through NF-κB and cytokine cascades, fostering mutagenesis and tumor proliferation. Similar inflammatory pathways may operate in the ovary, where chronic pelvic inflammation enhances oncogenic potential [11].

3.2.2. Immune modulation

The microbiome profoundly shapes host immunity. Balanced microbial communities enhance antigen presentation and cytotoxic T-cell responses, whereas dysbiosis impairs immune surveillance, facilitating tumor immune escape. Certain bacterial taxa modulate dendritic cell and macrophage function, influencing responsiveness to immune checkpoint inhibitors such as PD-1/PD-L1 blockers.

3.2.3. Metabolic reprogramming

Microbial metabolites—including short-chain fatty acids (SCFAs), bile acids, and indole derivatives—act as signaling molecules that influence tumor metabolism and immunity. SCFAs, such as butyrate, exert anti-inflammatory and anti-proliferative effects [12]. Conversely, secondary bile acids can activate oncogenic Wnt/β-catenin signaling. Microbial regulation of estrogen metabolism, mediated by β-glucuronidase enzymes (the "estrobolome"), may also alter hormonal exposure, potentially affecting ovarian tumor growth [13,14].

3.2.4. Genotoxicity

Certain bacteria produce genotoxins that directly damage host DNA. For example, Escherichia coli strains producing colibactin induce double-strand breaks, while H. pylori CagA protein perturbs DNA repair [15]. In ovarian cancer, similar genotoxic microbes may contribute to early neoplastic transformation within the reproductive tract epithelium.

4. Microbiome influences on ovarian cancer development and progression

4.1. Gut microbiota and ovarian cancer

The gut microbiota is the largest and most metabolically active microbial community in the human body, critically involved in immune regulation, hormone metabolism, and systemic inflammation. Emerging evidence suggests that alterations in gut microbial composition may influence ovarian cancer initiation and progression through several interconnected pathways, including estrogen metabolism, immune modulation, and intestinal barrier integrity.

4.1.1. Estrogen metabolism and the "estrobolome"

Estrogen plays a major role in ovarian tumorigenesis. The gut microbiota modulates systemic estrogen levels via the "estrobolome," a collection of bacterial genes encoding β-glucuronidase enzymes that deconjugate estrogens, restoring them to active forms [13,14]. Increased β-glucuronidase activity enhances circulating estrogen, which may promote ovarian cancer cell

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proliferation via estrogen receptor (ER) signaling. In contrast, microbial imbalance reducing these functions may disrupt hormonal homeostasis, further driving oncogenic transformation.

4.1.2. The gut–ovary axis

The "gut–ovary axis" concept posits that gut microbiota influence ovarian physiology through systemic immune and metabolic pathways. Microbial metabolites such as short-chain fatty acids (SCFAs) and bile acids can reach ovarian tissue via circulation, regulating inflammation, oxidative stress, and immune infiltration. Dysbiosis can increase intestinal permeability ("leaky gut"), allowing microbial components such as lipopolysaccharide (LPS) to enter circulation, triggering systemic inflammation and activating oncogenic NF-κB and IL-6 pathways [15].

4.1.3. Immune homeostasis

Gut dysbiosis can impair the balance of immune cells, including T regulatory (Treg) and cytotoxic T lymphocytes (CTLs). Reduced microbial diversity or loss of beneficial species (Bifidobacterium, Akkermansia) correlates with weaker anti-tumor immune surveillance. Conversely, restoration of eubiosis enhances immune-mediated tumor suppression. These findings highlight that maintaining gut microbial equilibrium may help modulate systemic immunity against ovarian tumor development.

4.1.4. Reproductive tract microbiota and ovarian cancer

The female reproductive tract microbiome, particularly that of the vagina, cervix, and uterus, plays a critical role in maintaining mucosal immunity and epithelial integrity. A healthy reproductive tract is dominated by Lactobacillus species, which produce lactic acid, lowering pH and preventing colonization by pathogens. Dysbiosis, characterized by a reduction in Lactobacilli and an increase in anaerobes such as Prevotella, Fusobacterium, and Escherichia coli, has been observed in ovarian cancer patients [16,17].

4.1.4.1. Inflammation and oncogenesis

Pathogenic bacteria induce chronic inflammation by stimulating cytokines such as IL-1β, IL-6, and TNF-α, which can foster tumorigenic conditions through angiogenesis and immune suppression. Reactive oxygen and nitrogen species generated during inflammation cause oxidative DNA damage and mutations, thereby promoting malignant transformation.

4.1.4.2. Viral infections

Although human papillomavirus (HPV) is most strongly linked to cervical cancer, several studies have suggested potential associations between viral infections (HPV, Epstein–Barr virus, herpes simplex virus) and ovarian carcinogenesis [18,19]. These viruses may alter gene expression or induce DNA instability within ovarian epithelium, but further mechanistic studies are required.

4.1.4.3. Microbial composition as diagnostic marker

Changes in the reproductive tract microbiome may serve as early indicators of oncogenic transformation. Reduced Lactobacillus abundance and enrichment of anaerobic bacteria correlate

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with poor prognosis and chemotherapy resistance [16]. Therefore, microbiome-based profiling could complement traditional biomarkers such as CA125 or HE4 in early detection.

4.2. Other microbiome niches

Beyond the gut and reproductive tract, oral and skin microbiota may also indirectly influence ovarian cancer.

The oral microbiome—particularly pathogens such as Porphyromonas gingivalis and Fusobacterium nucleatum—has been associated with systemic inflammation and cancer progression, possibly through hematogenous dissemination of microbial metabolites or immune-modulating factors [20]. F. nucleatum has been identified in several extra-oral tumors and may facilitate metastasis by altering interactions between tumor cells and the immune system.

Similarly, the skin microbiota can influence systemic immune regulation. Staphylococcus aureus produces superantigenic toxins that activate inflammatory pathways, whereas the commensal species Staphylococcus epidermidis demonstrates anti-inflammatory properties [21]. Although their involvement in ovarian cancer is not well established, these microbes may contribute to systemic inflammatory balance and immune responsiveness, thereby exerting indirect effects on tumor behavior.

5. Microbiome and therapeutic response

5.1. Chemotherapy sensitivity and resistance

Standard treatment for ovarian cancer involves cytoreductive surgery followed by platinum-based chemotherapy (e.g., cisplatin, carboplatin). Although initial responses are often favorable, recurrence and platinum resistance occur in up to 70% of advanced cases. Recent studies indicate that gut microbiota can influence both chemotherapy metabolism and tumor chemosensitivity [22–24].

Certain bacterial species can metabolize or inactivate platinum drugs, reducing their cytotoxic efficacy. Dysbiosis may also alter the redox environment of the tumor microenvironment, thereby modulating drug uptake. Conversely, beneficial microbial metabolites, particularly SCFAs, have been shown to inhibit efflux transporters like P-glycoprotein, enhancing intracellular drug retention.

Microbial components such as LPS can activate Toll-like receptors (TLRs), inducing NF-κB and STAT3 signaling, which promotes survival and DNA repair in tumor cells, contributing to drug resistance [23]. Moreover, patients with reduced microbial diversity exhibit higher resistance to platinum agents [24]. Therefore, microbial modulation—via probiotics, antibiotics, or FMT—may represent a promising adjunct to overcome chemoresistance.

5.2. Immunotherapy and microbial modulation

Although immune checkpoint inhibitors (ICIs) targeting PD-1/PD-L1 pathways have revolutionized cancer therapy, their efficacy in ovarian cancer remains limited. Gut microbiota composition has emerged as a critical determinant of immunotherapy response [25–27].

Beneficial taxa such as Akkermansia muciniphila and Bacteroides species enhance anti-tumor immunity by promoting dendritic cell activation and cytotoxic T-cell infiltration. In contrast, excessive Firmicutes or Bacteroidetes may suppress T-cell activation. Antibiotic use, by disrupting beneficial microbial populations, is associated with reduced ICI efficacy [25].

Probiotic supplementation, particularly with Lactobacillus and Bifidobacterium, can enhance PD-1 blockade responses by modulating cytokine profiles (e.g., IL-12, IFN-γ) and restoring immune

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balance [26]. Moreover, fecal microbiota transplantation (FMT) from ICI-responsive donors has been shown to restore sensitivity in resistant patients [27]. These findings highlight the therapeutic potential of manipulating gut microbiota to optimize immunotherapy outcomes.

5.3. Probiotics, FMT, and microbial-derived metabolites

Probiotics—especially Lactobacillus species—support reproductive tract homeostasis and exert anti-tumor effects through the production of metabolites such as indole-3-lactic acid (ILA), which inhibits tumor cell proliferation and promotes apoptosis [28]. In models of gastrointestinal toxicity, Bifidobacterium supplementation has been shown to reduce chemotherapy-induced adverse effects [29].

FMT, initially developed for the treatment of Clostridioides difficile infections, is now being explored in oncology to enhance immunotherapy efficacy and reduce chemotherapy toxicity [27]. Clinical studies indicate that transferring gut microbiota from ICI responders can improve immune activation and tumor control in non-responders. Moreover, FMT may help restore microbial equilibrium in the gut, alleviating treatment-related gastrointestinal complications.

Microbial metabolites, including SCFAs, bile acid derivatives, and indole compounds, modulate tumor microenvironment signaling and systemic inflammation. Targeting these metabolic pathways could offer novel therapeutic avenues.

6. Microbiome-based biomarkers and clinical applications

6.1. Microbiome as a diagnostic and prognostic biomarker

With the advent of high-throughput sequencing and integrated multi-omics technologies, microbiome analysis has become a powerful tool in cancer biomarker discovery. The microbial composition of ovarian cancer patients has been shown to differ significantly from that of healthy controls, suggesting its potential utility in early diagnosis and disease monitoring [29–31].

Alterations such as reduced Lactobacillus abundance in the reproductive tract and enrichment of anaerobic genera (Prevotella, Fusobacterium) have been consistently reported in ovarian cancer [16,17]. These microbial signatures may reflect local immune dysregulation and inflammation within the tumor microenvironment. Similarly, gut microbiome diversity is often decreased in ovarian cancer patients and correlates with disease stage and treatment response [24].

Advanced metagenomic and metabolomic profiling have identified specific microbial species and metabolites that serve as potential non-invasive biomarkers, detectable via blood, vaginal fluid, or fecal samples. The application of liquid biopsy techniques now enables detection of microbial DNA fragments, exosomes, or metabolites in circulation, offering a promising complement to traditional biomarkers such as CA125 and HE4 [30-32].

Machine learning algorithms have further enhanced diagnostic accuracy by integrating multi-omics microbial data. Models trained on microbial and metabolic profiles have successfully distinguished ovarian cancer patients from controls, achieving high sensitivity and specificity [33]. These findings underscore the feasibility of microbiome-based screening as an adjunct to existing diagnostic frameworks.

6.2. Microbiome-targeted therapeutic strategies

Targeting the microbiome has emerged as a new direction in precision oncology. Growing evidence indicates that microbial composition influences treatment efficacy, prompting the development of

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microbiome-focused interventions aimed at enhancing immune responses, overcoming therapeutic resistance, and reducing treatment-related toxicity [34,35].

Supplementation with probiotics (e.g., Lactobacillus, Bifidobacterium) and prebiotics that promote beneficial bacterial growth can restore microbial balance, improve mucosal immunity, and reduce inflammation. In ovarian cancer, probiotics have shown potential to mitigate chemotherapy-induced gastrointestinal toxicity and modulate immune checkpoint inhibitor responses [29,35].

While antibiotics can transiently modify microbiota composition, indiscriminate use may eliminate beneficial taxa and impair therapeutic response—especially immunotherapy [25]. Future strategies should focus on selective antimicrobial approaches that target tumor-promoting bacteria while preserving commensal populations.

FMT offers a promising approach for restoring healthy microbial communities. In clinical studies, transferring microbiota from ICI responders improved therapeutic efficacy in non-responders, highlighting its potential for immunotherapy sensitization [27,34]. Controlled trials are now underway to evaluate FMT safety and efficacy in gynecologic malignancies.

6.3. Integration of multi-omics and systems biology

Given the complexity of host–microbe interactions, multi-omics integration—encompassing metagenomics, transcriptomics, metabolomics, and single-cell sequencing—has become essential for elucidating the microbiome's role in ovarian cancer [36-37]. Techniques such as spatial transcriptomics and single-cell RNA sequencing allow mapping of microbial influences within the tumor microenvironment, revealing interactions between bacteria and immune or stromal cell populations at single-cell resolution.

In parallel, systems biology and AI-driven computational modeling are being used to predict causal relationships between microbial alterations and oncogenic pathways. For instance, artificial intelligence models trained on microbiome datasets have demonstrated predictive power for ovarian cancer risk and prognosis [33]. These integrative approaches are expected to accelerate biomarker validation and therapeutic discovery.

6.4. Large-scale and longitudinal studies

Despite growing evidence, most microbiome–ovarian cancer studies remain small-scale or cross-sectional. To confirm causality and temporal dynamics, longitudinal cohort studies are urgently needed [37,38]. Such studies can elucidate how microbial changes precede or follow tumor development and how they interact with host genetics, diet, and environmental exposures.

Future research should aim to include diverse populations across age, ethnicity, and geographic regions, to ensure the reproducibility and universality of findings. Establishing large, standardized datasets will also enable meta-analyses and data harmonization, facilitating global collaboration in microbiome–oncology research.

7. Future directions and clinical translation

7.1. Toward precision microbiome medicine

The future of ovarian cancer therapy lies in microbiome-informed precision medicine, which tailors treatment according to individual microbial and host profiles. Integration of microbiome diagnostics with genomic and immunologic data can identify patients who may benefit from microbial interventions alongside conventional therapies.

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Emerging concepts such as “microbiome fingerprinting” and personalized microbial therapeutics could guide treatment decisions, such as determining which patients are likely to respond to immunotherapy or benefit from probiotic supplementation [35]. Ultimately, combining microbial modulation with chemotherapy, immunotherapy, or targeted therapy may improve response rates and prolong survival.

7.2. Challenges and ethical considerations

While promising, microbiome-based oncology faces several challenges: 1) Causality and Mechanism: Most current studies are correlational; establishing causative links requires experimental and longitudinal validation. 2) Standardization: Variations in sample collection, sequencing, and analysis pipelines hinder reproducibility. 3) Safety: Interventions such as FMT carry risks of pathogen transmission or immune disturbance. 4) Ethical Issues: Microbiome data involve personal health information and require careful governance. 5) Addressing these challenges through standardized protocols, ethical guidelines, and interdisciplinary collaboration will be essential for successful clinical translation.

7.3. Interdisciplinary collaboration

Microbiome–cancer research is inherently interdisciplinary, bridging microbiology, oncology, immunology, and computational biology. Collaborative efforts between clinicians, biotechnologists, and data scientists are vital to accelerate translational applications. Large-scale consortia integrating multi-omics datasets and clinical outcomes will help uncover microbial networks driving ovarian cancer, advancing the field toward microbiome-guided therapeutic design [38].

8. Conclusion

Over the past decade, the human microbiome has emerged as a critical component in the pathophysiology of ovarian cancer. Gut and reproductive tract microbiota influence disease onset and progression through hormonal regulation, immune modulation, inflammation, and metabolic signaling. Dysbiosis not only contributes to tumorigenesis but also affects treatment responses, including chemotherapy resistance and immunotherapy efficacy.

Microbiome profiling holds promise for early detection, therapeutic optimization, and personalized medicine in ovarian cancer. Future studies integrating multi-omics, artificial intelligence, and longitudinal designs are essential to unravel causal mechanisms and validate microbiome-based biomarkers. Moreover, targeted microbial interventions—such as probiotics, FMT, and metabolite supplementation—offer new opportunities for improving therapeutic outcomes.

Ultimately, by bridging molecular oncology and microbial ecology, microbiome research provides a transformative framework for reimagining ovarian cancer prevention, diagnosis, and treatment in the era of precision medicine.

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