Review

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Gut Microbiome Dynamics and Their Emerging Role in Breast and Colorectal Cancer: Implications for Diagnosis and Therapy

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Abstract

The gut microbiome, a complex community of microorganisms residing in the gastrointestinal tract, is increasingly recognized as a critical player in cancer pathophysiology. Its influence extends beyond local gastrointestinal disorders to systemic diseases, including breast and colorectal cancers. This review examines current evidence on the interplay between gut microbial dysbiosis and the development, progression, and treatment response in both malignancies. In colorectal cancer (CRC), pathogenic bacteria such as *Fusobacterium nucleatum*, *Escherichia coli*, and *Bacteroides fragilis* have been implicated in tumor initiation and progression through mechanisms involving chronic inflammation, DNA damage, and immune modulation. Meanwhile, in breast cancer, the gut microbiome may contribute indirectly via regulation of estrogen metabolism, immune signaling, and systemic inflammation. Furthermore, the gut microbiota is increasingly being explored as a source of non-invasive biomarkers for early detection and disease monitoring, as well as a modifiable factor that could enhance therapeutic efficacy. Interventions such as probiotics, prebiotics, dietary modulation, and fecal microbiota transplantation (FMT) hold promise in reprogramming the microbiome to support antitumor responses and reduce treatment-related toxicity. Despite the growing body of research, translating these findings into clinical application requires deeper mechanistic understanding and standardized methodologies. This review emphasizes the need for longitudinal and multi-omics studies to unravel causal relationships and identify therapeutic targets. Understanding gut microbiome dynamics offers exciting opportunities for developing microbiota-informed strategies for cancer prevention, diagnosis, and therapy—particularly in breast and colorectal cancers.

Keywords: gut microbiome; breast cancer; colorectal cancer; microbial dysbiosis; microbiota-targeted therapy

1. Introduction

The human gut microbiome is a dynamic and diverse population of microbes that coexist in a complex ecosystem, comprising bacteria, viruses, fungi, and archaea. Numerous physiological functions, including digestion, metabolism, and immune system modulation, depend on these microbes. Dysbiosis, or disruptions in the gut microbiota, has been linked to several illnesses, such as diabetes, obesity, inflammatory bowel disease, and cancer (1).

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Breast and colon cancers represent the malignancies with the highest occurrence worldwide. The WHO claimed that breast cancer constituted 2.3 million new cases in 2020, rendering it the most prevalent cancer worldwide, whilst colorectal cancer ranked as the third most frequent, with 1.93 million instances documented (2). Although genetic and environmental factors are widely recognised as key drivers of cancer development, growing research underscores the substantial impact of the gut microbiota on modulating cancer risk and affecting its progression.

Recent research indicates that the gut microbiota significantly influences cancer development and therapy (3).

Investigating the role of the gut microbiome in breast and colon cancers may provide new insights into their pathogenesis and pave the way for treatments that target the microbiota. Thus, this review aims to summarize current findings on the gut microbiome's involvement in breast and colon cancer development, identify the underlying mechanisms linking microbiome alterations to carcinogenesis and highlight the therapeutic implications of modulating the gut microbiome in cancer prevention and treatment. By delving into these aspects, we hope to shed light on the potential of microbiome-based interventions in combating these prevalent cancers.

2. Overview of the Gut Microbiome

2.1. Composition of the Gut Microbiome

The human gastrointestinal system contains a varied and active population of bacteria known as the gut microbiota. This intricate ecosystem comprises bacteria, viruses, fungi, and archaea, with bacteria forming the majority. The microbiota is primarily classified into four principal bacterial phyla: Firmicutes, Bacteroidetes, Actinobacteria, and Proteobacteria. These microbial groups play a pivotal role in maintaining overall health by regulating metabolic processes, modulating immune responses, and providing protection against pathogens (4).

Firmicutes: The fermentation of dietary fibres into short-chain fatty acids (SCFAs) is a metabolic activity performed by this phylum, which constitutes the predominant component of the human gut microbiota. Genera including *Lactobacillus, Bacillus, Clostridium, Enterococcus*, and *Ruminococcus* are members of the Firmicutes. According to the Human Microbiome Project Consortium (5), these bacteria are essential for the digestion of complex carbohydrates and the production of SCFAs, which are good for gut health and systemic metabolism.

Bacteroidetes: Bacteroidetes, the second most prevalent group in the human gut, is mostly represented by genera such as *Prevotella* and *Bacteroides*. Complex proteins, lipids, and carbohydrates are broken down by these microorganisms. Their existence has been connected to immune response control and the breakdown of dietary polysaccharides (4).

Actinobacteria: Even though they are less common, actinobacteria are a significant phylum that supports gut health maintenance. To support the immune system, maintain gut integrity, and break down fibre and produce SCFAs, genera such as *Bifidobacterium* are essential (6). This group has been linked to early microbiome formation and is frequently seen in infants' stomachs (7).

Proteobacteria: They make up a small portion of the human gut microbiome, but they are involved in many metabolic processes and have been linked to inflammatory diseases when their numbers rise. *Escherichia coli* and *Helicobacter pylori* are two members of this phylum that can cause dysbiosis and disease pathogenesis, especially in people with weakened immune systems (5).

2.2. Factors Influencing Microbiome Diversity

According to new research, lifestyle factors like stress and exercise may also have an effect on the gut microbiota's makeup, which may have an effect on general health and disease risk. Although the gut microbiota's makeup varies from person to person, it is impacted by environmental variables, genetics, and food. It is essential for preserving health and identifying the host's vulnerability to illness.

Diet: Diet is one of the factors influencing the composition and diversity of the gut microbiome. A diet rich in plant-based foods, such as fruits, vegetables, and whole grains, contains a variety of fibres and polyphenols that support the development of beneficial bacteria like *Bifidobacterium* and *Lactobacillus*. On the other hand, bacteria like Firmicutes, which are connected to obesity and

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metabolic disorders, can proliferate when a diet rich in fats and carbohydrates is consumed (8). Diets high in fibre increase the synthesis of SCFAs, which maintain immune system balance and reinforce the integrity of the barrier of the gut (6).

***Genetics*:** In addition to nutrition and environment, genetics also affects the microbiome's makeup. Although host genetic traits have an impact on microbial diversity and the abundance of certain microbial species, these effects are negligible in contrast to those of the environment (9). The way the microbiome interacts with the host and reacts to different stimuli can be influenced by genetic differences in the host's immune system and metabolism.

***Antibiotics*:** These are potent substances that attack a variety of microorganisms, upsetting the gut microbiota. Antibiotic abuse or overuse might reduce microbial diversity, which could facilitate the spread of opportunistic infections. Repeated or prolonged use of antibiotics can alter the gut microbiota more permanently, raising the risk of infections and metabolic diseases (10, 11). Additionally, antibiotics may cause inflammatory illnesses such as inflammatory bowel disease (IBD) by changing the microbial balance in a way that impacts immune responses (7).

***Lifestyle*:** The gut microbiota is also impacted by lifestyle choices like stress levels, sleep habits, and physical activity. Frequent exercise increases the diversity of the microbiome and improves gut health by encouraging the growth of good bacteria. However, long-term stress can cause intestinal inflammation and the expansion of dangerous bacteria (12). Stress can alter the gut-brain axis, changing the composition of the microbiota and perhaps contributing to systemic inflammation and gastrointestinal illnesses (13).

***Environmental Factors*:** The microbiome can be affected by several variables, including living circumstances, geographic location, and exposure to chemicals or contaminants in the environment. Compared to people who live in urban settings, those who live in rural areas with less exposure to industrialized environments typically have a more diversified microbiota (14,15). Additionally, early-life exposures, such as breastfeeding and delivery method (Vaginal vs. C-section), have a lasting effect on the formation of the microbiome (7).

2.3. The Microbiome's Function in Immune System Control and Metabolism

The gut microbiota is essential to preserving general health because of its impact on the immunological response, metabolism, and even behavior. Numerous factors, such as lifestyle, antibiotics, genetics, and food, affect its makeup. Understanding the microbiome's role in the development of cancer, especially breast and colon cancer, holds enormous potential for future treatment approaches as we continue to unearth the intricate relationships between the microbiome and human health.

***Immune System Regulation*:** The gut-associated lymphoid tissue (GALT) is home to the bulk of the body's immune system, which interacts with the gut microbiota. Immune tolerance development and immunological balance are supported by this relationship. A healthy microbiome is necessary for the development of both innate and adaptive immune responses. Immunological dysfunction can result from dysbiosis, or abnormalities in the microbiome, which is connected to autoimmune diseases, colon cancer, and inflammatory bowel disease (IBD) (16). Dysbiosis can change the gut's response to infections, cause excessive inflammation, and compromise mucosal immunity (17).

***Metabolism*:** Energy metabolism is regulated in large part by the gut microbiota. The fermentation of complex dietary fibres and carbohydrates into SCFAs like acetate, propionate, and butyrate is one of the main roles of gut bacteria. These SCFAs give colonocytes energy, preserve the integrity of the gut barrier, and control systemic metabolic functions like insulin sensitivity, glucose metabolism, and fat storage. Additionally, SCFAs have anti-inflammatory properties that affect metabolic syndrome, type 2 diabetes, and obesity (18). Furthermore, vital vitamins including vitamin K and B vitamins, which are vital for host metabolic processes, are synthesized in large part by the microbiota (6).

***Gut-Brain Axis*:** According to a new study, this axis is a pathway via which the gut microbiota interacts with the central nervous system to affect behaviour, emotional well-being, and brain function. This two-way communication channel affects mood, stress reactions, and even cognitive

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function. It involves microbial metabolites like SCFAs and neurotransmitters. Mental health conditions such as anxiety and sadness have been connected to dysbiosis (19).

3. Gut Microbiome and Colon Cancer Development

Dysbiosis, which is broadly defined as an imbalance or alteration in the makeup of the gut microbiota, is a significant contributing factor to the development of colorectal cancer (CRC). There is solid proof that microbial dysbiosis can influence colorectal carcinogenesis through a number of mechanisms, including immune modulation, chronic inflammation, and direct interactions with epithelial cells. Gagnière et al. (20), in their study, showed that the gut microbiota is crucial for maintaining gut homeostasis, and its alteration can foster an environment that is favourable for the development of cancer.

3.1. Inflammation and Tumorigenesis

The gut's pro-inflammatory and anti-inflammatory bacteria populations become unbalanced as a result of dysbiosis. An elevated inflammatory response brought on by this imbalance may result in DNA damage, the accumulation of mutations, and the encouragement of carcinogenesis (21). Beneficial gut bacteria normally produce metabolites like short-chain fatty acids (SCFAs) that have anti-inflammatory properties, but when pathogenic bacteria are present, their production may decline. An inflammatory cascade may be sustained by this decrease in conjunction with the proliferation of pro-inflammatory bacteria, creating an atmosphere that can support the formation of malignant cells (6).

Additionally, by modifying the activity of T-cells, macrophages, and dendritic cells, gut bacteria can affect the immune system and cause either immunological tolerance or immune activation, all of which can have an impact on the development of cancer (22). Tumour immune evasion may be facilitated by the microbiome's influence on immune cells' capacity to identify and eliminate tumour cells.

3.2. Inflammatory Pathways in Colon Cancer Development

Microbiota-Induced Immune Modulation

The gut microbiota is crucial in regulating immunological responses, which can either inhibit or encourage the development of colorectal cancer. Chronic inflammation can be exacerbated by dysbiosis, which can result in an overabundance of immune cells such as T-helper (Th17) cells and macrophages (22). It has been demonstrated that some microbial species, such as Fusobacterium nucleatum, improve immune evasion by inhibiting T-cell responses, which permits tumour growth (23).

3.3. Microbial Metabolites and Carcinogenesis

Short-Chain Fatty Acids (SCFAs): Dual Roles in Colon Cancer

SCFAs, including butyrate, acetate, and propionate, are key microbial metabolites derived from the fermentation of dietary fibres. They play dual roles in CRC, acting as both protective agents and potential tumour promoters under dysbiotic conditions.

Butyrate: As a histone deacetylase (HDAC) inhibitor that controls gene expression and encourages death in colorectal cancer cells, butyrate is well-known for its anti-inflammatory and anti-tumorigenic qualities (24). Butyrate metabolism is changed in the presence of dysbiosis and a high-fat diet, which may promote the energy metabolism of cancer cells and lead to tumour growth (21).

Acetate and Propionate: Propionate and acetate have been linked to immune response modulation and gut barrier integrity maintenance. Their functions in CRC are still context-dependent, though, as some data indicates that they may accelerate tumour growth under pro-inflammatory environments (25).

3.4. Bile Acid Metabolism and Genotoxic Effects

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Gut bacteria convert bile acids, which are mostly produced in the liver, into secondary bile acids including lithocholic acid (LCA) and deoxycholic acid (DCA). Because of their capacity to cause oxidative stress, DNA damage, and apoptosis resistance, these metabolites have been linked to the development of colorectal cancer (CRC) (26). By encouraging colonic inflammation and epithelium damage, high-fat diets exacerbate the carcinogenic potential of bile acids by increasing their output.

3.5. Production of Secondary Metabolites (Toxins, Reactive Oxygen Species)

Certain gut bacteria produce toxins and reactive oxygen species (ROS) that contribute to mutagenesis and carcinogenesis.

Colibactin: Colibactin is a genotoxin produced by *Escherichia coli* that causes DNA double-strand breaks in colonic epithelial cells, resulting in mutations and genomic instability (27).

Bacteroides fragilis toxin (BFT): BFT, which is secreted by enterotoxigenic *Bacteroides fragilis*, increases the chance of developing colorectal cancer by causing tight junctions between epithelial cells to break down and promoting inflammatory signalling (28).

Reactive Oxygen Species (ROS): According to Jones et al. (29), certain harmful bacteria cause the stomach to produce more ROS, which damages DNA oxidatively and increases the chance of malignant transformation.

3.6. Key Bacteria Implicated in Colon Cancer

Several bacterial species have been identified as key players in colon cancer development and dysbiosis is linked to immune system regulation, chronic inflammation, and direct microbial interactions with epithelial cells. These bacteria not only contribute to the initiation and progression of CRC but also alter the local environment, influencing both tumorigenesis and metastasis.

Fusobacterium nucleatum

The development of colorectal cancer has been closely linked to *Fusobacterium nucleatum*. Compared to normal colon tissues, this bacterium is frequently more abundant in CRC tissues. Research has demonstrated that by influencing immunological responses, namely by increasing the recruitment of pro-inflammatory macrophages to tumour locations, *F. nucleatum* can stimulate the growth of tumours (23). Furthermore, *F. nucleatum* can initiate signalling pathways that support the migration and proliferation of cancer cells by adhering to and invading epithelial cells. This bacterium is thought to be a possible biomarker for colorectal cancer (CRC) and has been linked to a bad prognosis (30).

Escherichia coli

Both colon cancer and inflammatory bowel disease (IBD) have been linked to *Escherichia coli*, especially the adherent-invasive strain *E. coli* (AIEC). By producing genotoxic substances such as colibactin, AIEC can cause DNA damage in colon cells (31). Genetic alterations and the subsequent emergence of malignant tumours are caused in part by this DNA damage. Furthermore, *E. coli* can promote an environment that is favourable to cancer by activating some oncogenic signalling pathways, such as those involved in cell cycle regulation and apoptosis resistance (32).

Bacteroides fragilis

Another bacterium that has been connected to colorectal cancer is *Bacteroides fragilis*. *Bacteroides fragilis* toxin, or BFT, is a toxin produced by some strains of *B. fragilis* that has been demonstrated to damage DNA and increase intestinal inflammation. Tight connections between epithelial cells may be broken by this toxin, increasing intestinal permeability and allowing pro-inflammatory immune cells to infiltrate. Colon cancer can develop as a result of the inflammatory milieu that results (33). *B. fragilis* has also been demonstrated to stimulate signalling pathways that control cell survival and proliferation, which lends greater credence to its involvement in the genesis of cancer.

3.7. Evidence from Human and Animal Studies

Both human cohort studies and animal models have provided compelling evidence supporting the role of the gut microbiome in CRC development.

3.7.1. Human Studies

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According to research, *Fusobacterium nucleatum* is more prevalent in CRC tissues and is linked to both treatment resistance and a poor prognosis (34).

Different microbial signatures that distinguish healthy people from those with precancerous adenomas and tumours have been identified through microbiome sequencing of CRC patients (35).

3.7.2. Animal Models

Increased rates of DNA damage and tumour growth were seen in germ-free mice colonized with *Escherichia coli* strains that produce colibactin (36).

The carcinogenic effects of secondary bile acids were demonstrated in mice models subjected to high-fat diets and settings enriched in bile acids, which resulted in increased intestinal inflammation and colorectal carcinogenesis (37).

4. Gut Microbiome and Breast Cancer Development

4.1. Gut–Breast Axis: Systemic Effects of the Microbiome

The systemic interactions between the gut microbiota and breast tissue, especially through immunological and hormonal regulation, are referred to as the gut–breast axis. The estrobolome, a collection of genes from gut microbes that metabolize estrogen, is an essential part of this axis (38). By controlling the enterohepatic circulation of estrogens, the estrobolome affects estrogen homeostasis. According to Hu et al. (39), some gut bacteria, especially Clostridia species, express β-glucuronidase enzymes that deconjugate estrogens, enhancing their re-absorption and raising the levels of estrogen in the blood.

Hormone-driven malignancies, especially estrogen receptor (ER)-positive breast cancer, have been closely associated with elevated systemic estrogen levels. According to Kwa et al. (40), increased estrogen exposure encourages DNA damage, cell division, and tumour growth. On the other hand, dysbiosis, which is defined by a decreased variety of good bacteria, can result in decreased metabolism of estrogen, changing the hormonal equilibrium and fostering an environment that is favourable to the growth of tumours (41).

According to new research, gut dysbiosis may also impact breast tissue homeostasis through systemically circulated microbial metabolites. Low-grade inflammation and oxidative stress are linked to breast carcinogenesis and can be triggered by specific lipopolysaccharides (LPS) and microbial toxins (42).

4.2. Microbial Metabolites and Breast Cancer Risk

The gut microbiome produces a variety of metabolites that influence breast cancer risk, either protectively or detrimentally.

4.3. Short-Chain Fatty Acids (SCFAs) and Their Dual Roles

The gut microbiota ferments dietary fibre to produce SCFAs, including butyrate, acetate, and propionate. These substances are known to have anti-inflammatory and anti-cancer effects. As a histone deacetylase (HDAC) inhibitor, butyrate prevents DNA damage and encourages cancer cells to undergo apoptosis (43). According to Gopalakrishnan et al. (44), SCFAs maintain the integrity of the intestinal barrier and stop systemic inflammation, which can lead to breast cancer. However, SCFA synthesis may decrease in dysbiotic settings, resulting in heightened inflammation and a pro-tumorigenic environment (45).

4.4. Bile Acid Metabolism and Genotoxic Effects

Bile acid metabolism is impacted by dysbiosis, which causes secondary bile acids such as lithocholic acid (LCA) and deoxycholic acid (DCA) to build up. These substances have been connected to elevated oxidative stress and DNA damage, both of which accelerate the development of cancer (26). It has been demonstrated that DCA activates pro-inflammatory pathways (NF-κB, IL-6, and TNF-α), which aids in the growth of tumours in breast cancer caused by obesity (41).

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Epidemiological studies have linked a higher prevalence of breast cancer to high-fat diets and changes in the microbiome that prefer bacteria that metabolize bile acids (38).

4.5. Obesity and Lipid Metabolism

The gut microbiota is important for energy balance and lipid metabolism, and obesity is a major risk factor for postmenopausal breast cancer (44). According to Fernández et al. (43), dysbiosis increases insulin resistance, chronic inflammation, adipokine imbalance, and metabolic endotoxemia, all of which are factors that contribute to breast cancer. Gut microbes affect estrogen production, tumor-promoting pathways, and cholesterol metabolism (26).

4.6. Inflammation and Immune Dysregulation

Persistent inflammation is a defining feature of breast cancer development, with the gut microbiota playing a crucial role in regulating the immune system.

An imbalance in the gut microbiota, or dysbiosis, can result in elevated levels of pro-inflammatory cytokines, including Tumor Necrosis Factor-alpha (TNF-α)-which promotes tumour cell survival and angiogenesis, Interleukin-6 (IL-6)- which activates STAT3, driving cancer cell proliferation and immune evasion, and Nuclear Factor-kappa B (NF-κB) - involved in promoting chronic inflammation and facilitating tumour development (46).

The gut microbiome also affects immune surveillance mechanisms, which are essential in controlling cancer progression. Regulatory T cells (Tregs) are modulated by gut bacteria, potentially leading to immune tolerance of tumour cells (42).

Also, certain bacterial metabolites can suppress Natural Killer (NK) cell activity, reducing the body's ability to target cancer cells (41).

4.7. Evidence from Human and Animal Studies

The gut–breast axis highlights how systemic effects of microbiome dysbiosis influence hormonal regulation, inflammation, and metabolism, ultimately contributing to breast cancer (BC) risk.

4.8. Human Studies

The Role of the Estrobolome in Estrogen Metabolism

The group of gut microorganisms that metabolize circulating estrogens is known as the estrobolome. Some bacteria produce β-glucuronidase enzymes, which raise systemic estrogen levels by reactivating estrogen metabolites in the gut (39). Particularly in post-menopausal women, dysbiosis can result in increased estrogen reabsorption, extended estrogen exposure, and an increased risk of breast cancer (38).

4.9. Microbiome Signatures in Breast Cancer Patients

Pro-inflammatory bacteria like *Escherichia, Staphylococcus*, and *Enterococcus* were significantly more abundant in BC patients' breast tissue microbiomes when comparing samples from malignant and non-cancerous tissue (47). Conversely, there was a decrease in helpful bacteria such as *Lactobacillus*, indicating that a protective microbial community could aid in preventing carcinogenic processes (48).

4.10. Faecal Microbiota and Breast Cancer Risk

Goedert et al. (38) conducted a study examining faecal microbiota composition in post-menopausal women, finding that women with higher breast cancer risk had a greater proportion of Firmicutes and Proteobacteria, alongside lower Bacteroidetes diversity. These findings suggest a systemic link between gut microbiota composition and hormone-driven cancers.

4.11. Animal Models

Germ-Free Mice and Hormone-Driven Tumors

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In mouse models, germ-free (GF) conditions were shown to significantly reduce mammary tumour incidence, reinforcing the idea that microbial metabolites contribute to tumour development (49). However, when GF mice were recolonized with microbiota from obese mice, they exhibited higher systemic estrogen levels, increased inflammation, and accelerated tumour growth (49-55).

4.12. Microbial Modulation of Immune Responses in Breast Cancer

Kwa et al. (40) demonstrated that colonization with pro-inflammatory gut microbiota enhances immune suppression in breast tissue, reducing CD8+ T-cell infiltration and increasing the expression of T-regulatory (Treg) cells, which promotes tumour immune evasion.

4.13. Obesity, Microbiota, and Breast Cancer Risk

Liu et al. (56) conducted experiments where microbiota from obese monkeys was transplanted into germ-free mice, leading to increased adiposity, chronic inflammation, and mammary tumour development. This highlights the role of the gut microbiome in obesity-driven breast cancer.

5. Therapeutic and Preventive Strategies Targeting the Microbiome

5.1. Probiotics and Prebiotics

Modifying the gut microbiome through dietary approaches, such as the use of probiotics and prebiotics, has shown promising results. Probiotic strains like *Lactobacillus rhamnosus* and *Bifidobacterium longum* have exhibited anti-inflammatory and anti-cancer properties in preclinical studies. Similarly, prebiotics, such as dietary fibres and oligosaccharides, promote the selective growth of beneficial bacteria, thereby amplifying their protective effects.

5.2. Fecal Microbiota Transplantation (FMT)

FMT, which involves transferring healthy microbiota to dysbiotic individuals, is being explored as a preventive and therapeutic approach for cancer. Recent studies, such as Wang et al. (57), have shown that FMT can reduce tumour progression in animal models of colorectal cancer by restoring microbial diversity and reducing inflammation. Additionally, a clinical trial by Davar et al. (58) demonstrated that FMT combined with immunotherapy improved response rates in melanoma patients, highlighting its potential applicability in cancer treatment. Further research is needed to optimize FMT protocols and evaluate long-term outcomes.

5.3. Microbiome-Based Biomarkers for Early Detection

Microbial signatures are emerging as potential biomarkers for assessing cancer risk and enabling early detection. For example, elevated levels of *Fusobacterium nucleatum* have been proposed as a diagnostic marker for colorectal cancer. Similarly, alterations in estrobolome activity may serve as indicators of breast cancer risk. Validating these biomarkers in diverse populations is essential for their clinical implementation.

5.4. Future Therapeutic Approaches

Advances in personalized medicine offer opportunities to tailor microbiome modulation strategies to individual patients, optimizing therapeutic outcomes. Combining microbiome-based therapies with existing treatments like immunotherapy and chemotherapy could enhance efficacy and reduce side effects (59 - 61).

6. Challenges, Limitations, and Future Directions

6.1. Research Gaps

Despite significant progress, gaps remain in understanding the causative links between the gut microbiome and cancer. While associations between microbial dysbiosis and tumorigenesis have been well-documented, establishing causation requires longitudinal studies and controlled experiments. The heterogeneity of microbiome composition across individuals further complicates identifying universal microbial markers of cancer. Moreover, the interplay between microbiota, host

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genetics, and environmental factors, such as diet and lifestyle, remains insufficiently explored. Addressing these gaps will require interdisciplinary approaches that integrate microbiology, oncology, and bioinformatics.

6.2. Translational Challenges

Translating microbiome research into clinical practice is hindered by variability in study methodologies and individual differences in microbiota composition. Collaborative efforts across disciplines are needed to develop standardized protocols and validate findings.

6.3. Emerging Technologies

Technologies like metagenomics and metabolomics are advancing our ability to study the microbiome. Metagenomic sequencing has facilitated the identification of microbial species and their functional contributions in colorectal cancer patients, uncovering potential biomarkers for early diagnosis. Likewise, metabolomics research has identified significant microbial metabolites, including short-chain fatty acids and secondary bile acids, which are associated with tumour formation and progression. Broadening the use of these advanced technologies to include diverse populations and longitudinal studies will be essential for future advancements.

6.4. Future Studies

Future studies should prioritize longitudinal research, include diverse populations, and incorporate multi-omics approaches to better understand the intricate relationship between the microbiome and cancer. Collaborative initiatives involving international consortia have the potential to significantly advance progress in this area.

7. Conclusions

The development of breast and colon cancer is influenced by the gut microbiota in a complex and multidimensional way, affecting immune regulation, metabolism, and inflammation. The importance of the gut microbiota in the development and spread of colorectal and breast cancers is becoming more and more clear. Studies conducted on both human and animal models consistently show that the makeup of the gut microbiota changes significantly in cancer patients, with a decrease in protective species and an increase in pro-tumorigenic bacteria. Systemic carcinogenesis is also facilitated by microbial metabolites, including oestrogens produced by the estrobolome and secondary bile acids. Animal research shows that dysbiosis promotes the establishment of tumours, despite the possibility for cancer prevention via microbiome-targeted treatments (such as probiotics, prebiotics, and dietary changes). These findings show that targeting the gut microbiota has therapeutic potential to reduce the incidence of cancer and improve treatment outcomes. Although altering the microbiota offers a potential approach to cancer treatment and prevention, more study is needed to convert these discoveries into useful therapeutic uses. The creation of novel and individualised treatment plans will be made possible by filling up the existing knowledge gaps through multidisciplinary cooperation and the application of cutting-edge technology.

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