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Review Paper

Microbial dysbiosis and carcinogenesis: Exploring the contribution of the microbiome to major human cancers

Asma Mohammadizade*

Department of Microbiology, Faculty of Sciences, Kerman Branch, Islamic Azad University, Kerman, Iran

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ABSTRACT

Scientific progress has greatly enhanced our understanding of the complex link between cancer and the microbiome. Research suggests that the way the human body interacts with its microbial communities is a key factor in maintaining health and in the development of disease. The human microbiome has numerous benefits, including the Regulation of essential processes such as immune signaling and metabolism, which contribute to the proper functioning of the host. Microbiota imbalance is associated with the development and progression of cancer. This review article aims to investigate the microbiome's effect on the development and progression of cancer, which could lead to significant progress in the prevention, diagnosis, and treatment of this cancer. Overall, further studies are needed to provide new insights into the diagnosis, prevention, and treatment of cancer. Also, the design of clinical research related to the interaction of the microbiome genome and the human host genome could be of interest, and signaling pathways involved in disease.

Corresponding Author(s):
Asma Mohammadizade, MSc
Address: *Department of Microbiology, Faculty of Sciences, Kerman Branch, Islamic Azad University, Kerman, Iran*
Tel: +98 914 0576419
E-mail: asma.mohammadi24@gmail.com

1. Introduction

The term microbiota is all microbial species associated with the human body, and microbiome is the collection of microorganisms' genomes. The human microbiome includes bacteria, viruses, and fungi that are in various parts of the body [1,2]. It should be noted, however, that in many cases these two terms are used interchangeably; in this article, the two are considered synonymous. In recent years, metagenome sequencing (the content of all genome a microbial community) has become a very effective method for microbiome analysis, capable of identifying bacterial, fungal, and viral genes [3]. Each person's gut microbiota has a unique composition, highly variable between individuals, likely influenced by the host's genetic content [4]. The human gut contains about 100 trillion microorganisms, including bacteria, viruses, and even fungi, whose composition and abundance are influenced by age, diet, ethnicity, genetic factors, and lifestyle [5].

At birth, microbiota is transmitted to the newborn via contact with environmental microbes, colonizing and proliferating within the body. Between the ages of 3 and 5, the composition changes to resemble that of an adult [6]. Microbiota serve many roles, including aiding digestion, producing beneficial and abundant metabolites, strengthening the immune and nervous systems, and adapting to drug metabolism. The composition varies not only between individuals but also between different organs within the same person [7]. Even genetically similar people or those living together may differ in microbiota composition by as much as 90% [7].

Microbiota are highly sensitive to environmental changes; for instance, high-calorie diets, sedentary lifestyles, urban living, and smoking can all rapidly alter them [8]. With age, microbial diversity declines, making them more susceptible to disruption [8]. Fortunately, in the last decade, advances in sequencing, in particular 16S rRNA sequencing, genomics, and bioinformatics the relationship between the microbiome and human disease have been clearer. Microorganisms can cause inflammation, Changes in the immune response, activating specific proteins, and producing carcinogenic metabolites cause tumorigenesis. Therefore, studying the link between the microbiome and tumors can help Designing targeted therapies against tumors and improve diagnostics [9].

Cancer results from uncontrolled cell proliferation, associated with unregulated tumor growth, reduced sensitivity to cell cycle arrest signals, and the prevention of programmed cell death [10]. Like many complex diseases, cancer progression is influenced largely by gene-environment interactions. as yet genetic and cellular mechanisms cause carcinogenesis are increasingly well understood, particularly with the help of next-generation sequencing that identifies Frequent mutations and epigenetic changes, but our understanding of how environmental factors contribute is relatively low [11]. Until recently, the link between cancer and microorganisms living in our bodies was unclear. However, bacteria, fungi, eukaryotes (e.g., yeasts), and viruses (including bacteriophages) are environmental factors to which we are continually exposed, and recent studies show they can major impact on carcinogenesis [12]. This study aims to examine the role of the human microbiota in cancers of the stomach, intestine, breast, and lung. The major mechanisms through which microbiota cause carcinogenesis across different organs are summarized in Figure 1.

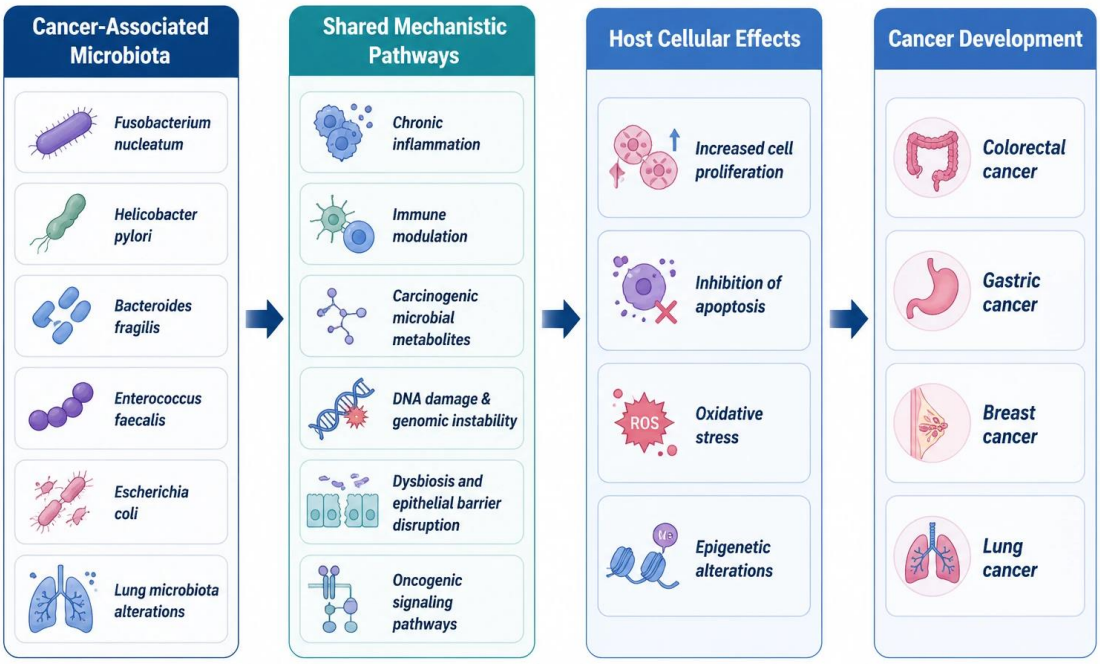

Figure 1. Shared mechanistic pathways linking microbiota dysbiosis to cancer development.

2. Colorectal cancer

Colorectal cancer ranks as the third most common cancer throughout the world and the fourth leading cause of cancer death. In Iran, it ranks fourth in men after stomach cancer, bladder cancer, and prostate cancer, and second in women after breast cancer [13]. While incidence is lower than in developed countries, rates are decreasing by 2% annually in developed countries but increasing in developing Asian nations. Early-onset CRC (<40 years) accounts for 2-8% of cases in developed nations but 15-35% in the Middle East; in Iran, early-onset colorectal cancer is reported in 20% of cases. This issue may be due to lifestyle changes and increased consumption of meat and fat, and decreased consumption of grains and fiber in the Iranian diet [14]. Because the cause of colorectal cancer is mainly related to environmental and epigenetic factors, and genetic factors play a smaller role in its creation. As a result, it is very important to investigate environmental factors and how they affect this cancer. between environmental factors, Recently, the microbiota plays an important role, as well as its enzymes and metabolites, and the production of oxygen-free radicals [15].

Based on studies, the population of human gut microbiota has five main anaerobic bacterial phyla: *Firmicutes*, *Bacteroidetes*, *Actinobacteria*, *Proteobacteria*, and *Verrucomicrobia*. *Firmicutes* and *Bacteroidetes* are the most abundant [5]. The maintenance of gut microbiota homeostasis is critical for intestinal tissue integrity and overall host health. The gut microbiota performs numerous physiological functions, including modulation of immune signaling and inflammatory responses, participation in drug metabolism, degradation of dietary carcinogens, synthesis of essential vitamins, fermentation of indigestible substrates, facilitation of electrolyte absorption, promotion of intestinal epithelial cell growth and differentiation, and support of immune system development. Furthermore, it inhibits the colonization and proliferation of pathogenic microorganisms, such as *Escherichia coli* and *Clostridium* spp., contributes to hormonal regulation, and reduces susceptibility to allergic disorders. Collectively, these functions highlight the indispensable role of the gut microbiota in human health and survival [16]. Among environmental factors in the development of colon cancer, the role of microorganisms has received more attention than ever before, so that about 20% of the world's cancers are attributed to microbial microorganisms within the intestinal tract. The large intestine contains 70% of the body's total microbiota [17]. The highest population density of microbes colonizing the human body is in the large intestine, which is called the intestinal microbial flora. This microbial population includes trillions of bacteria, which have a biomass of 1.5 kg, and their total size is as large as the liver, which is the largest organ in the body [7].

The main function of the gut microbiome is related to its metabolic activity, which leads to efficient extraction of energy from food consumption, in particular by the fermentation of complex indigestible carbohydrates and producing microbial metabolites, which are known as short-chain fatty acids (SCFAs) [18]. It also contributes to producing vitamin K, B vitamins, hydrogen, carbon dioxide, methane, and converting urea into ammonia [18]. Gut microbiota through metabolite production can be effective in the process of developing colon cancer. For example, acetaldehyde-producing bacteria and sulfate-reducing bacteria can be a cause of colon cancer through the Production of metabolites, causing inflammation in the intestines and producing tumor-inducing toxicity such as acetaldehyde, hydrogen sulfide, and Secondary bile acids in the intestine [19]. On the other hand, some bacterial metabolites, by producing SCFAs such as butyrate and conjugated linoleic acid, may reduce the risk of colon cancer. These bacteria are called so-called good bacteria and have a wide distribution in different phylogenetic groups [20].

Many factors, including taking antibiotics, psychological or physical stress, radiation, changing gut peristaltic movements, and diet, can change the digestive system ecosystem and consequently change the compositional structure of its bacteria [21]. On the other hand, some anaerobic intestinal bacteria such as *Clostridium* and *Bacteroides*, as the dominant population constituting the intestinal microbiota, by producing bacterial metabolites (bile acids, fatty acids, etc.) and enzymes like β-galactosidase, β-glucosidase, sulfatase, reductase, decarboxylase, protease can also increase the risk of colorectal cancer [22].

Part of the microbial population protects the human body against colon cancer by induction of immune tolerance and neutralization of fungal pathogenic factors. But it seems that another part of the microbiota in the development and progression of cancer, through creating carcinogenic substances and metabolites or toxins, and affecting the immune system, plays a major role. For example, some bacteria, such as *Bacteroides fragilis* and *Enterococcus faecalis* by produce enterotoxins that damage the DNA of human cells [23]. Sulfide-producing bacteria such as *Fusobacterium*, *Bilophila wadsworthia* generate hydrogen sulfide, which disrupts the process of repairing genetic material, DNA, and at the same time, accelerates cell division. The FadA antigen in *Fusobacterium nucleatum* activates Wnt/β-catenin signaling, which causes DNA damage, chromosomal instability, cellular proliferation, and stimulation of tumor growth [23]. Rubinstein and colleagues showed that *F. nucleatum* through the FadA antigen attaches to E-cadherin. As a result, it activates the β-catenin signaling pathway, which induces oncogenic and inflammatory responses. Binding of FadA to E-cadherin prevents with its subsequent tumor-suppressive activity [24]. The level of Sulfidogenic bacteria in people with diets high in fat and animal protein compared to people with a diet rich in

vegetables and fruits is about 30 percent more. Therefore, a high-fat diet is an important risk factor for colon cancer [25].

The presence of the bacterium *Streptococcus gallolyticus*, which is a subspecies of *Streptococcus bovis*, has been reported in 71% Intestinal cancer patients. Unlike *S. gallolyticus*, other *Streptococcus bovis* subspecies were observed in 17% of these patients, which suggests a link between this bacterium and colon cancer [26].

3. Gastric cancer

In spite of the evidence that suggests an imbalance between the gastric microbiome and the host can help to cause gastric cancer, no specific mechanism has been found to prove it. The composition and function of the gastric microbiome can help in the prognosis and diagnosis of gastric cancer [27]. Microbiota with a heightened risk of tumor development contribute to the three stages of primary, secondary, and tertiary interactions. Primary interactions refer to the direct contact between microbiota and the tumor microenvironment. For example, the link between *Helicobacter pylori* and gastrointestinal cancers can be considered a primary interaction. *H. pylori* causes several types of digestive cancers, especially gastric cancer. Secondary interactions refer to the communication between a set of microbes in tumor tissue. This level of interaction helps to identify biomarkers for diagnosing different types of cancer. But it should be noted that these diagnostic processes are often complex. Tertiary interactions between microbiota and tumors are related to the effect of microbial communities on the location of tumors in different parts of the body [27]. Studying this level of interactions is performed to determine the relationship between microbial species and tumor formation. Which can ultimately be effective in treating cancer patients and reducing the toxicity of chemotherapy. Also, by understanding these interactions more, they can improve the body's immune system. As a result, tertiary interactions, like secondary interactions, can help in the diagnosis of various cancers [27].

The gastric microbiota can vary in different gastric microenvironments; for example, *Prevotella copri*, *H. pylori*, and *Bacteroides uniformis* are observed significantly less frequently, while *Prevotella melaninogenica*, *Streptococcus anginosus*, and *Propionibacterium* increase in tumor microenvironments [27]. Numerous studies have shown that the microbiome contributes to the main stages of carcinogenesis, including tumor-associated inflammation, tumor growth, angiogenesis, DNA damage, and Induction of genomic instability [27, 28]. *H. pylori* is one of the main factors which causes gastrointestinal diseases such as gastric ulcers, gastritis, and stomach cancer. This bacterium through producing toxins and stimulating inflammatory responses causes serious damage to the gastric mucosa. As time goes by, these damages can cause genetic and epigenetic changes that lead to the development of gastric cancer. *H. pylori* activates inflammatory responses through stimulating immune reactions in gastric epithelial cells and absorbing immune cells to the site of infection (such as neutrophils, macrophages, and lymphocytes) [29].

Moreover, humans are exposed to nitrosating agents through internal processes and consumption of external resources like processed meats, smoked fish, and certain vegetables [30]. Foods contain low levels of nitrite, which is mainly formed when oral bacteria in saliva reduce nitrate to nitrite. After nitrite enters the stomach, it is transformed into nitrosating agents, which can be prevented by ascorbic acid. Bacteria like *Clostridium*, *Veillonella*, *Haemophilus*, *Staphylococcus*, *Neisseria*, *Lactobacillus*, and *Nitrospirae* play a role to gastric cancer by stimulating the production of nitrosating compounds [30].

4. Breast cancer

Despite extensive strategies in the diagnosis and treatment of cancer, breast cancer remains the most common cause of cancer death among women, and it is estimated that about 2.3 million women around the world are affected by it each year. It is predicted that by 2030, the number of people with this cancer will be 2.7 million per year [31]. Various factors such as race, age, family history, BRCA1/2 mutations, diet, physical inactivity, alcohol consumption, and estrogen levels affect the likelihood of developing the disease, its progression, and mortality [31].

Initially, breast tissue was thought to be a sterile place. Until 1996, researchers noticed the presence of unique bacteria in breast tissue. They were different from other organs in the body. Since the breast has fatty tissue and extensive blood vessels, it is a suitable place for the growth and activity of various types of bacteria. Theories about bacterial presence include migration from skin through nipple ducts, oral contact, or translocation from the gut [31]. Theories about bacterial presence include migration from skin through nipple ducts, oral contact, or translocation from the gut [31].

In breast cancer, Changes in the gut microbiota can affect the incidence of this disease by affecting metabolism and estrogen-related or estrogen-independent mechanisms, Such as the effect on the immune system and production of metabolites [32]. Estrogen levels are very important in breast cancer. One of the main regulators of estrogen is the gut microbiota. Gut bacterial genes called estrobolome encode the enzyme β-glucuronidase, which deconjugates estrogen and converts it into its active form, so that it can bind to its receptors and activate intracellular cascades. Bacteria that produce β-glucuronidase include *Escherichia*, *Citrobacter*, *Bifidobacterium*, *Bacteroides*, and *Clostridium* [32]. Some gut microbes can also convert inactive steroid hormones into active steroids by

producing sulfatase. In addition, the gut microbiota can convert dietary polyphenols into estrogen [32]. Therefore, changes in the amount and type of gut microbiota cause changes in estrogen levels and, as a result, affect the growth of breast cancer. In other words, estrogen levels are significantly dependent on the gut microbiota [32].

Lithocholic acid is a secondary bile acid. It is produced only by the gut microbiota from primary bile acids. Lithocholic acid reduces breast cancer cell proliferation by 10-20%, inhibits angiogenesis, and inhibits epithelial-to-mesenchymal transition, while increasing p53 expression and enhancing the antitumor immune response. It has been found that its levels are reduced in the early stages of breast cancer compared to the control group, which indicates a decrease in its synthesis by gut bacteria [33].

Since breast tissue is rich in lymphatic vessels, nutrients, and fat, a wide range of bacteria are also present in breast tissue, which enter the ducts directly through the nipple [34]. Researchers identified the 16S rRNA microbiomes of healthy breast tissue by sequencing and culturing. The most abundant phyla of bacteria in normal breast tissue include *Proteobacteria*, *Firmicutes*, and *Actinobacteria*, whereas these bacteria are present in the minority in other tissues [34].

Filippou and colleagues found in 2024 that in cancer tissue samples there is a significant reduction in bacterial communities compared to healthy breast tissue. A decrease in the amount of bacteria in a healthy person can increase the risk of breast cancer. It is predicted that bacteria stimulating host immune responses help maintain the healthy condition of breast tissue [35]. Urbaniak et al. in 2016 found that in women with breast cancer there is a higher prevalence of Staphylococcus aureus and *Enterobacteriaceae* than in healthy women. In addition, during their studies, they found that bacteria such as *Lactococcus*, *Streptococcus*, and *Prevotella* are found more in the breast tissue of healthy people than in cancerous tissue. *Streptococcus*, especially *Streptococcus thermophilus*, has the ability to protect the host's DNA, giving it anti-cancer properties by producing antioxidant metabolites that neutralize reactive oxygen species. *Prevotella* also provides anti-inflammatory properties by producing propionate [36].

The bacteria *Gluconacetobacter*, *Fusobacterium*, *Atopobium*, *Lactobacillus*, and *E. coli* have also been observed to be more abundant in malignant than benign breast tissue [37]. Thompson et al. in 2017 found that in breast tumor tissue *Proteobacteria* are abundant, while *Actinobacteria* are prominently seen in surrounding non-tumorous tissue. They also reported that the amount of *Streptococcus pyogenes* increases in tumor tissue, which is a bacterium associated with increased systemic estrogen via β-glucuronidase activity, and it increases the risk of breast cancer [38].

In addition, in the microbes present in the nipple fluid of women with breast cancer, the highest levels of the enzyme β-glucuronidase have been observed compared to healthy individuals. This can increase the risk of breast cancer [39]. In breast tissues of people with cancer, *Fusobacterium*, *Atopobium*, and *Bacillus* bacteria have increased. In aspiration of nipple fluids from cancer patients, an increase in *Alistipes* bacteria and a simultaneous decrease in *Sphingomonadaceae* bacteria have been observed. Also, *Methylobacterium radiotolerans* bacteria have been found in higher amounts in breast cancer tissues than in healthy tissues [40]. Furthermore, in patients with breast cancer, the amounts of *Clostridia*, *Lactobacillus*, and *Enterobacterium* are also higher than in healthy individuals [40].

5. Lung cancer

One of the factors increasingly associated with lung cancer is the human microbiota. Historically, the lungs were considered sterile organs; however, advances in culture-independent molecular techniques, particularly 16S rRNA sequencing and metagenomic analyses, have demonstrated that the respiratory tract harbors a distinct microbial community [41]. The pulmonary microbiota appears to establish shortly after birth and contributes to respiratory immune homeostasis throughout life [42]. Recent studies have highlighted that alterations in the lung microbiome are associated with chronic pulmonary diseases and may also contribute to lung carcinogenesis. In healthy individuals, the lung microbiota is commonly dominated by genera such as *Prevotella*, *Veillonella*, *Streptococcus*, *Neisseria*, and *Fusobacterium*. Dysbiosis within this microbial ecosystem may promote carcinogenesis through chronic inflammation, epithelial injury, immune dysregulation, and activation of oncogenic signaling pathways [42].

The lung microbiota plays a crucial role in regulating both innate and adaptive immune responses by modulating dendritic cells, regulatory T cells, macrophages, and cytokine production. Under physiological conditions, microbial communities help maintain immune tolerance and mucosal homeostasis [43]. However, pulmonary dysbiosis may trigger persistent inflammatory responses that facilitate tumor initiation and progression. Increased activation of M1 macrophages, Th17 cells, and pro-inflammatory mediators such as IL-17, IL-6, and TNF-α has been linked to enhanced epithelial proliferation, DNA damage, angiogenesis, and metastasis in lung cancer [43]. Furthermore, microbial-derived metabolites and pathogen-associated molecular patterns may activate oncogenic pathways including PI3K/AKT, STAT3, and NF-κB signaling, thereby promoting malignant transformation. Recent evidence also suggests that the gut-lung axis may influence lung cancer progression and responsiveness to immunotherapy through systemic immune modulation [43].

Several bacterial taxa have been reported to be enriched in patients with lung cancer. Studies have demonstrated increased abundances of *Granulicatella*,

Abiotrophia, *Streptococcus*, *Veillonella*, *Acinetobacter*, and *Acidovorax* species in tumor tissues and bronchoalveolar lavage samples from lung cancer patients [44]. In particular, *Acidovorax temporans* has been associated with smoking-related squamous cell carcinoma and TP53 mutations. Additionally, advanced-stage and metastatic lung cancers have shown increased colonization by opportunistic pathogens such as *Legionella* species [45]. Chronic pulmonary infections may further contribute to carcinogenesis; notably, *Mycobacterium tuberculosis* has been implicated in lung cancer development through sustained inflammation, fibrosis, oxidative stress, and repeated epithelial injury. Recent meta-analyses have also reported reduced microbial diversity in lung cancer patients compared with healthy controls, suggesting that microbial imbalance may serve as a potential biomarker for diagnosis, prognosis, and therapeutic response prediction [45].

6. Conclusion

Accumulating evidence from recent experimental, translational, and clinical studies strongly suggests that microbial dysbiosis plays a substantial role in the initiation, progression, and modulation of multiple human cancers, including colorectal, gastric, breast, and lung malignancies. The human microbiome contributes to carcinogenesis through several interconnected mechanisms, such as chronic inflammation, immune dysregulation, production of carcinogenic metabolites and genotoxins, induction of oxidative stress, activation of oncogenic signaling pathways, and alteration of host metabolic and hormonal homeostasis.

At the same time, beneficial microbial communities and their metabolites may exert protective anti-inflammatory and antitumor effects, highlighting the dual role of the microbiome in cancer biology. These findings emphasize that cancer should not only be considered a genetic and cellular disease but also a disorder influenced by host-microbial interactions and environmental factors.

In colorectal cancer, specific microorganisms such as *F. nucleatum*, *B. fragilis*, and *S. gallolyticus* have been associated with DNA damage, activation of Wnt/β-catenin signaling, and promotion of inflammatory tumor microenvironments. Similarly, in gastric cancer, *Helicobacter pylori* remains one of the clearest examples of a carcinogenic microorganism capable of inducing chronic gastritis, epithelial injury, and genomic instability. Breast cancer studies have increasingly demonstrated the role of gut and breast tissue microbiota in regulating estrogen metabolism, immune responses, and tumor proliferation, while lung cancer research has revealed that pulmonary dysbiosis and the gut-lung axis may significantly influence inflammatory signaling, tumor progression, and responsiveness to immunotherapy. Collectively, these observations indicate that organ-specific microbial signatures may serve as valuable biomarkers for cancer risk assessment, early detection, prognosis, and therapeutic monitoring.

Recent advances in next-generation sequencing technologies, metagenomics, metabolomics, transcriptomics, and bioinformatics have substantially improved our understanding of the microbiome-cancer relationship. These technologies have enabled the identification of microbial communities and metabolites associated with carcinogenesis and have provided insight into the complex molecular crosstalk between microorganisms and host tissues. Furthermore, emerging evidence suggests that modulation of the microbiome through dietary interventions, probiotics, prebiotics, fecal microbiota transplantation, antibiotics, and microbiome-targeted therapeutics may represent promising strategies for cancer prevention and treatment. Importantly, the microbiome has also been shown to influence the efficacy and toxicity of chemotherapy, radiotherapy, and immune checkpoint inhibitors, suggesting a critical role in personalized oncology.

Despite these significant advances, several limitations and unanswered questions remain. Most currently available studies demonstrate associations rather than definitive causal relationships between microbial dysbiosis and cancer development. In addition, variations in sampling methods, sequencing platforms, geographic distribution, dietary habits, ethnicity, and environmental exposures contribute to inconsistencies among studies. The precise molecular pathways by which specific microbial species or metabolites promote or inhibit tumorigenesis are still incompletely understood. Therefore, future research should focus on large-scale longitudinal and mechanistic studies integrating multi-omics technologies to clarify causal interactions between the microbiome and host signaling pathways.

Overall, a deeper understanding of microbiome-associated carcinogenesis may transform future approaches to cancer prevention, diagnosis, and therapy. Identification of microbial biomarkers and therapeutic targets could pave the way for precision medicine strategies tailored to individual microbial profiles. Continued investigation into host-microbiome interactions will likely provide novel opportunities for early cancer detection, risk stratification, modulation of treatment response, and development of innovative microbiome-based anticancer therapies.

Declaration of artificial intelligence (AI) in the writing process

The author declare whether AI or AI-assisted technologies were used during the preparation of this manuscript. If used, AI tools were employed solely to improve language quality, grammar, readability, and organizational structure. The author carefully reviewed and edited all AI-generated content and take full

responsibility for the accuracy, integrity, and originality of the final manuscript. No AI tool was used to generate, analyze, or interpret scientific data or images, or to draw scientific conclusions. The use of AI-assisted technologies complies with current publication ethics recommendations and journal policies.

Authors' contributions

The author was solely responsible for literature review, methodology, initial drafting, and revised the manuscript. The author has read and approved the final version of the manuscript.

Conflict of interest

No potential conflict of interest was reported by the author.

Ethical declarations

Not applicable.

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