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F.U. Vet. J. Health Sci.
2026; 40 (3): 301 - 306
<http://www.fusabil.org>

The Role of Gut Microbiota in Cancer: Inflammation, Tumor Microenvironment, and Carcinogenesis

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The aim of this review is to evaluate the role of the gut microbiota in cancer development, with particular emphasis on inflammation, the tumor microenvironment, and carcinogenesis, in light of current evidence. The gut microbiota represents a dynamic ecosystem that plays a critical role in regulating metabolism, immune system functions, and epithelial barrier integrity. Diet is one of the major factors shaping microbiota composition and influences various physiological processes by modulating microbial diversity and metabolite production.

Disruption of microbiota homeostasis may contribute to the development of various diseases through alterations in immune responses, increased inflammation, and impairment of tissue homeostasis. Emerging evidence suggests that the gut microbiota exerts significant regulatory effects on tumor initiation and progression, particularly in gastrointestinal malignancies. Furthermore, findings from veterinary medicine indicate that similar mechanisms may occur across animal species and may provide valuable insights for comparative oncology approaches. In conclusion, a better understanding of the relationship between gut microbiota and cancer may facilitate the development of novel diagnostic tools and targeted therapeutic strategies in both human and veterinary medicine.

Key Words: Cancer, dysbiosis, gut microbiota, inflammation, tumor microenvironment

Kanserde Bağırsak Mikrobiyotasının Rolü: İnflamasyon, Tümör Mikroçevresi ve Kanser Gelişimi

Bu derlemenin amacı, bağırsak mikrobiyotasının kanser gelişimindeki rolünü; özellikle inflamasyon, tümör mikroçevresi ve karsinogenez açısından güncel literatür doğrultusunda değerlendirmektir. Bağırsak mikrobiyotası; metabolizma, bağışıklık sistemi fonksiyonları ve epitel bariyer bütünlüğünün düzenlenmesinde önemli rol oynayan dinamik bir ekosistemdir. Diyet, mikrobiyota kompozisyonunu şekillendiren temel faktörlerden biri olup, mikrobiyal çeşitlilik ve metabolit üretimini etkileyerek çeşitli fizyolojik süreçleri düzenlemektedir.

Mikrobiyota dengesinin bozulması; immün yanıt değişiklikleri, artmış inflamasyon ve doku homeostazının bozulması yoluyla çeşitli hastalıkların gelişimine katkıda bulunabilmektedir. Güncel çalışmalar, bağırsak mikrobiyotasının özellikle gastrointestinal sistem malignitelerinde tümör gelişimi ve progresyonunda önemli düzenleyici etkilere sahip olduğunu göstermektedir. Ayrıca veteriner hekimlik alanındaki bulgular, benzer mekanizmaların hayvan türlerinde de görülebileceğini ve karşılaştırmalı onkoloji yaklaşımına katkı sağlayabileceğini ortaya koymaktadır. Sonuç olarak, bağırsak mikrobiyotası ile kanser arasındaki ilişkinin daha iyi anlaşılması, hem insan hem de veteriner hekimlikte yeni tanısal ve hedefe yönelik tedavi stratejilerinin geliştirilmesine katkı sağlayabilir.

Anahtar Kelimeler: Kanser, disbiyozis, bağırsak mikrobiyotası, yangı, tümör mikroçevresi

Introduction

Microorganisms are among the earliest forms of life, having emerged approximately 3.5 billion years ago, and have played a fundamental role in shaping the biological structure of the planet (1). Today, the effects of these organisms on host health and disease development have been clearly demonstrated (2). The microbiota refers to the entire community of microorganisms residing in different regions of the body, particularly in the gastrointestinal tract, and the gut microbiota represents a complex ecosystem that exists in a symbiotic relationship with the host (3). One of the most important regulators of this system is diet (4).

Changes in lifestyle and dietary habits are associated with an increase in intestinal diseases, and there is growing evidence that diet plays a critical role in the pathogenesis of various conditions, including diabetes, obesity, and colon cancer (5–7). Different types of diets have been shown to rapidly and markedly alter the composition of the gut microbiota as well as the structure of the intestinal epithelium (8). These changes are thought to modulate inflammation through microbial composition and immune responses (4, 9, 10)

The gut microbiota is a fundamental biological factor that plays a critical role in digestion, metabolism, and immune system functions, and contributes to the maintenance of epithelial integrity (11). Disruption of this balance leads to dysbiosis and an increase in inflammatory processes (12, 13).

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Received : 22.05.2026
Accepted : 20.07.2026

Microbiota and Microbiome

The microbiota refers to the community of microorganisms residing in the host and includes bacteria, fungi, viruses, and other microorganisms located in various anatomical regions, particularly the gastrointestinal tract, as well as the oral cavity, skin, and urogenital system (14). The microbiome, on the other hand, describes the total genetic material and functional capacity of these microorganisms. Although these two terms are sometimes used interchangeably in the literature, microbiota refers to the microbial community, whereas microbiome denotes the genetic and functional characteristics of this community (15–17). Therefore, the microbiota is considered a functional system that influences host physiology (18).

The gut microbiota exists in a symbiotic balance with the host and plays a role in many essential functions, including vitamin synthesis (K and B group), the breakdown of complex carbohydrates, production of short-chain fatty acids, amino acid synthesis, and bile acid metabolism (19). In addition, microbiota-derived metabolites influence organs such as the liver and cardiovascular system through circulation, thereby modulating metabolic processes and contributing to the regulation of the immune system (20). In healthy individuals, the majority of the gut microbiota is composed of the phyla Firmicutes, Bacteroidetes, and Actinobacteria (21). Microbial density is highest in the colon, and the mucus structure regulates the localization of microorganisms (22, 23).

The microbiota begins to develop after birth and acquires an individual-specific composition in early life under the influence of environmental and genetic factors (24). It is a dynamic ecosystem that exhibits a unique composition in each individual. Current estimates indicate that the human body harbors approximately 38 trillion bacterial cells, a number comparable to the approximately 30 trillion human cells; moreover, advanced molecular analysis techniques have revealed extensive microbial diversity, ranging from approximately 15,000 to 36,000 species (25–27). The composition of the gut microbiota is significantly influenced by physiological factors such as intestinal motility, pH, redox balance, dietary content, and gastrointestinal secretions, as well as environmental factors including antibiotic use, age, and lifestyle (28).

Microbiota and Macronutrients

One of the most important factors influencing the microbiota is dietary macronutrients. Carbohydrates constitute the primary energy source for the gut microbiota, and indigestible polysaccharides are converted via fermentation into short-chain fatty acids (SCFAs) such as acetate, propionate, and butyrate. These metabolites contribute to host energy metabolism and provide approximately 10% of the daily energy requirement. SCFAs activate GPR41 and GPR43 receptors in the intestinal epithelium, thereby stimulating peptide YY secretion; they also modulate energy balance and adipogenesis through glucagon-like peptide-1 (GLP-1) and leptin. Through these mechanisms, the microbiota

can influence nutrient absorption and energy storage, thereby contributing to the development of metabolic diseases (29).

High-fat diets disrupt intestinal barrier integrity, increase permeability, and elevate circulating lipopolysaccharide (LPS) levels. Increased LPS triggers low-grade inflammation and activates pathways involved in the development of metabolic diseases (30). Microbiota-derived metabolites have also been reported to influence host gene expression. In this context, the microbiota may suppress fasting-induced adipose factor (FIAF), increase LPS activity, and inhibit PGC-1, which plays a critical role in the regulation of energy metabolism. These processes have been shown to be associated with metabolic diseases such as obesity and diabetes (31).

Protein-rich diets may influence cardiovascular risk through microbiota-mediated mechanisms. Dietary choline and phosphatidylcholine are converted by the gut microbiota into trimethylamine (TMA), which is subsequently oxidized in the liver to trimethylamine N-oxide (TMAO). Increased TMAO levels have been associated with the development of atherosclerosis (32). Similarly, elevated plasma TMAO levels have been shown to significantly increase the risk of cardiovascular disease (33).

Microbiota and Gut Health

The increasing consumption of processed foods has made nutrient composition and food safety important determinants of health and has been associated with numerous conditions, including obesity, diabetes, cardiovascular diseases, immune system disorders, and cancer (34). In this context, the use of probiotics and prebiotics in nutrition has attracted considerable attention. Probiotics are defined as live microorganisms that, when administered in adequate amounts, confer health benefits to the host (35, 36). They are required to possess characteristics such as resistance to gastrointestinal conditions, adherence to the epithelium, and the ability to produce antimicrobial compounds (35, 37).

Prebiotics, on the other hand, are non-digestible components that are fermented by beneficial microorganisms in the intestine, thereby increasing the production of short-chain fatty acids (SCFAs) (38). The resulting metabolites—acetate, propionate, and butyrate—reduce intestinal pH, provide energy to epithelial cells, and support the immune response; they also contribute to the reduction of inflammation and the improvement of metabolic balance (39, 40). Plant-based foods rich in dietary fiber exhibit prebiotic effects (41, 42). Synbiotics are products that combine probiotics and prebiotics to produce a synergistic effect, enhancing the colonization and functional activity of probiotics (43). Postbiotics are metabolic products of probiotics, including components such as short-chain fatty acids, organic acids, and bacteriocins, which exert immunomodulatory and metabolic effects (39). In recent years, there has been a growing number of studies investigating the protective and therapeutic potential of these components (44).

Microbiota and Cancer

The relationship between the gastrointestinal microbiota and cancer has long been recognized. In particular, the association of *Helicobacter pylori* with gastric cancer and *Fusobacterium nucleatum* with colorectal cancer (CRC) represents some of the most well-known examples of this link (45, 46). However, it has been demonstrated that the effects of the microbiota are not limited to the local level; rather, they play a significant role in cancer development and progression by modulating systemic immune responses (47). Accordingly, the microbiota is considered among the key determinants of cancer (48).

The tumor microenvironment (TME) is a complex structure involving dynamic interactions between tumor cells and stromal and immune cells. Hypoxia and metabolic reprogramming within this environment facilitate the colonization of facultative and anaerobic microorganisms, thereby promoting the formation of an intratumoral microbiome (49). Intratumoral microorganisms may play an important role in shaping tumor development, progression, and response to therapy. Recent evidence further suggests that the gastrointestinal microbiota exhibits marked regional specialization and interacts closely with resident immune cell populations at the mucosal level. Yerlikaya et al. demonstrated that distinct microbial communities coexist with region-specific distributions of T lymphocytes, macrophages, eosinophils, and other mucosal immune cells throughout the equine large intestine, emphasizing the role of microbiota-driven immune modulation in shaping local tissue microenvironments (50). Moreover, the authors reported that fecal microbiota composition may not accurately reflect mucosa-associated microbial populations, highlighting the importance of evaluating host-microbiota interactions directly at the mucosal interface. These findings support the concept that spatial organization of the microbiota and local immune responses may contribute to chronic inflammation and microenvironmental alterations associated with carcinogenesis.

Microorganisms can increase genomic instability by inducing DNA damage through the production of

genotoxins and reactive oxygen species. In particular, certain strains of *Escherichia coli* and *Fusobacterium nucleatum* have been highlighted in this process (11). In addition, disruption of DNA repair mechanisms and alterations in epigenetic regulation are among the important components of microorganism-mediated carcinogenesis.

Furthermore, specific microorganisms can promote cancer development by establishing a tumor-supportive inflammatory microenvironment. Enterotoxigenic *Bacteroides fragilis* and *Fusobacterium nucleatum* activate inflammatory signaling pathways, increase cytokine production, and facilitate the infiltration of tumor-promoting immune cells (51). Similarly, virulence factors such as CagA of *Helicobacter pylori* and FadA of *Fusobacterium nucleatum* contribute to carcinogenesis by enhancing cell proliferation and inflammation (52).

Microbial components and metabolites modulate tumor progression through Toll-like receptor-mediated inflammation, epithelial-mesenchymal transition, and cellular proliferation. In addition to bacteria, fungal and viral components also play a role in carcinogenesis, and species of *Malassezia* have been shown to contribute to pancreatic cancer through complement activation (53, 54). The global distribution of major cancer types according to incidence and mortality rates is summarized in Table 1 (55).

The gut microbiota is considered a critical regulator in the pathogenesis of colorectal cancer (CRC). Dysbiosis promotes tumor development through disruption of epithelial barrier integrity, alterations in microbial metabolite balance, and increased chronic inflammation (56). In patients with CRC, an increase in bacteria such as *Fusobacterium*, *Porphyromonas*, and *Peptostreptococcus* has been reported, whereas beneficial genera such as *Faecalibacterium* and *Bifidobacterium* are decreased (5). Experimental models have confirmed that microbiota composition plays a determining role in tumor development and the inflammatory microenvironment (57). Accordingly, microbiota-based approaches have emerged as promising strategies for the diagnosis and treatment of CRC, aiming to improve tumor control through modulation

Table 1. Global distribution of cancer types by incidence and mortality rates (55)

Rank	Cancer Type	New Cases (n)	Incidence Rate (%)	Deaths	Mortality Rate (%)
1	Breast Cancer	2.261.419	11.7	684.996	6.9
2	Lung Cancer	2.206.771	11.4	1.796.144	18.0
3	Colorectal Cancer (CRC)	1.931.590	10.0	935.173	9.4
4	Prostate Cancer	1.414.259	7.3	375.304	3.8
5	Gastric Cancer	1.089.103	5.6	768.793	7.7
6	Hepatocellular Carcinoma (HCC)	905.677	4.7	830.180	8.3
7	Cervical Cancer	604.127	3.1	341.831	3.4
8	Esophageal Cancer	604.100	3.1	544.076	5.5
9	Thyroid Cancer	586.202	3.0	43.646	0.4
10	Bladder Cancer	573.278	3.0	212.536	2.1

of microbial metabolites and regulation of the immune response (49). Therefore, it is suggested that microbiota-derived inflammatory signals may promote tumor progression by increasing the expression of angiogenic factors such as VEGF and Endoglin, and that these processes may be modulated by COX-2 inhibitors (58).

Colorectal Cancer and Microbiota

Colorectal cancer (CRC) is one of the most common malignancies worldwide and represents a major public health concern (59). The development of CRC begins with the transformation of normal epithelium into a hyperproliferative phenotype and progresses to invasive cancer through the adenoma–carcinoma sequence (60). The majority of cases are sporadic, and major risk factors include obesity, type 2 diabetes, inflammatory bowel diseases, and lifestyle-related factors (61).

Hepatocellular Carcinoma and the Gut–Liver Axis

Hepatocellular carcinoma (HCC) is an aggressive malignancy that most commonly develops on the background of chronic liver disease and cirrhosis. In recent years, the bidirectional relationship between the gut microbiota and the liver has been evaluated within the framework of the gut–liver axis, and it has been shown to play an important role in the pathogenesis of HCC (52, 62). Dysbiosis and disruption of intestinal barrier integrity lead to the translocation of microbial products to the liver via the portal circulation and to the activation of inflammatory signaling pathways mediated by Toll-like receptors. This process promotes hepatocarcinogenesis by increasing chronic inflammation, fibrosis, and cellular proliferation. In particular, the activation of pathways such as the NF- κ B signaling pathway by microbial metabolites contributes to tumor development by suppressing cell death (63).

Clinical studies have demonstrated a distinct dysbiotic microbiota profile in patients with HCC. In these patients, an increase in pathogenic bacteria such as *Bacteroides* and *Enterobacteriaceae*, along with a decrease in beneficial bacteria such as *Bifidobacterium* and *Akkermansia*, has been reported, and these

alterations are thought to be associated with disease progression (64–66).

Veterinary Perspective and Comparative Oncology

Studies in veterinary species have demonstrated that gastrointestinal microbial communities are influenced by host-related and environmental factors and may include both commensal and potentially opportunistic microorganisms (67). Comparative oncology studies have increasingly demonstrated that naturally occurring cancers in animals share important molecular, immunological, and tumor microenvironment characteristics with human malignancies (68, 69). Companion animals, particularly dogs and cats, spontaneously develop various neoplasms including mammary tumors, lymphoma, osteosarcoma, and gastrointestinal tumors, making them valuable translational models for investigating cancer biology and host–microbiota interactions (70–72). Similar to humans, alterations in gut microbial composition and immune responses have been associated with disease progression and inflammatory mechanisms in veterinary species (69, 73). Therefore, veterinary comparative oncology may provide a valuable framework for understanding microbiota-mediated mechanisms in cancer development and for identifying novel diagnostic and therapeutic approaches applicable to both human and animal health.

Conclusion and Recommendations

The gut microbiota plays an important role in cancer development through metabolic, immune, and inflammatory mechanisms and has emerged as a key regulator of host homeostasis. Increasing evidence suggests that microbiota-associated alterations contribute to carcinogenesis and tumor progression. Furthermore, similar host–microbiota interactions reported in veterinary medicine highlight the value of comparative oncology. Within the One Health framework, integrating human and veterinary evidence may improve understanding of cancer biology and support the development of novel diagnostic and therapeutic strategies.

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