



Gut microbiotas attributed to disorders and diseases of the gastrointestinal tract, colorectal cancer, bladder cancer: Geographical factors, inflammation, metabolic toxic

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Abstract

Recently, there were several publications that attributed gut microbiota (GM) to various gastrointestinal tract functional disorders and diseases, including inflammatory bowel diseases, colon cancer, pancreatic cancer, and diverticulosis. GM is attributed to the initiation of urinary tract diseases and bladder carcinoma (BCa). The concern is whether GM is dysbiotic or protective. We explored the studies on GM contribution to colorectal cancer and BCa. Selected studies from different geographical regions on tissue samples or faecal samples from patients with colorectal cancer and controls. The results showed diverging results of microbiota abundance, genus, class, and phylum. These data indicated that other factors of environmental, diet, ethnic, and personal factors are contributors to GM in the initiation of inflammation and tumors. GM are not inhabitants in the urinary tract; it is postulated that GM attributes to BCa *via* the circulating metabolic toxins in the initiation of tumorigenesis and BCa.

Key Words: Gut microbiota; Colorectal cancer; Bladder cancer; Dysbiosis; Metabolic toxins; Geographical cohort; Tissue microbiota; Faecal microbiota; Metagenomics; Next-generation sequencing

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Core Tip: Several studies on gut microbiota (GM) and its attribute to colorectal cancer and bladder carcinoma (BCa) from different geographical regions on tissue or faecal samples from patients with colorectal cancer and controls have shown diverging non-unified results of microbiota abundance, genus, class, and phylum. These data indicated that the impact of environmental factors, diet, and ethnic factors, in addition to GM, contributes to carcinoma. GM are not inhabitants in the urinary tract; the postulation that GM attributes to BCa assumes that the circulating metabolic toxins are tumorigenic and would initiate BCa.

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INTRODUCTION

Research has been rapidly done over the past two decades on human microbiomes. Huge works have conducted on microbiota to investigate the probabilities of using it in the treatment of multiple diseases. Different human conditions have been shown to correlate with alterations in microbiota composition; this situation is referred to as dysbiosis[1]. This often fails to account for confounders such as age, body mass index, sex, and medication. Recently, Zhu *et al*[1], published an elegant study on gut microbiota(GM) in functional constipation. Factors such as microbial community, interactions that occur because of immunological, metabolic, or other functional changes in the host, rather than being directly causal. Attempts to define which gut microbiota (GM) composition definitively influences disease progression have so far failed to conclude a definite consensus due to a lack of consistency between different studies.

Changes in GM have been associated with a range of diseases in humans. Microbiota Enterobacteriaceae is commonly found in different situations. The increased levels of inflammation are at both local and systemic levels. Inflammation would deplete the GM and allow facultative anaerobes such as Enterobacteria ceae to proliferate. This will have an impact on the metabolic output of the microbiota and its interactions with the host. Multiple host factors would influence GM-related diseases, among these factors are medications, age, immune response, and environmental factors[2]. Sequence-based methods have been applied for microbiome research. Biases would exist at every step, ranging from sample collection, storage, DNA extraction, chosen bioinformatic pipelines, and reference databases for analyses of the data[3].

GM AND COLORECTAL CANCER: DIVERSITY IN RESULTS FROM GEOGRAPHICAL COHORT, TISSUE SAMPLES, AND FAECAL SAMPLES

The human microbiota includes bacteria, archaea, viruses, and fungi. They are emerging as an important feature of human health and disease. Currently, access to the genomic data of human cells and of microbiota is more affordable and accessible. A major challenge is to integrate microbiome data into precision medicine approaches for the prevention, diagnosis, and treatment of diseases such as cancer. Colorectal cancer (CRC) is the third most prevalent cancer worldwide [1]. Specific dietary factors and eating patterns have shown to affect GM. It has been demonstrated that the GM becomes dysregulated and is an active participant in the tumorigenesis of CRC[2]. Investigations have shown the roles of GM in all aspects of CRC, including the roles of *Fusobacterium nucleatum* and *Peptostreptococcus anaerobius* in the initiation and progression of CRC. Besides, GM can modify responses to treatment, including chemotherapy and immune checkpoint inhibitors (ICIs)[4].

A proinflammatory state, which can be activated by changes in the GM, these alterations may affect the mucosal integrity, resulting in the induction of systemic inflammation and carcinogens produced by *Escherichia coli* and gram-negative bacteria, among these released products are extracellular vesicles and lipopolysaccharide that can pass to the circulation and produce metabolic and immunological reactions. Persistent inflammation contributes to cancer initiation through the induction of epithelial-mesenchymal transition produced by *Fusobacterium nucleatum*, which is abundant in tissues of CRC, *Escherichia coli* produces cytotoxic necrotizing factor 1[4].

Chinese study-tissue microbiota

A research study on the correlation of microbiota and CRC in Chinese patients with CRC was done by Wang *et al*[5] on 36 CRC tissue samples and their corresponding nearby normally appearing colon tissues. Tissue samples were retrieved during surgery. The study revealed that CRC tissues are different in the abundance of microbiota taxa, butyrate colonic metabolites, and methylation gene expression compared with the nearby normal tissues. There was an increased abundance of genus *Fusobacterium*, a decreased level of 4-hydroxybutyric acid, and expression of immune-related peptidase inhibitor 16, Fc receptor-like A, and lymphocyte-specific protein 1. The study showed that *Prevotella 2* was correlated with the down-regulated expression of metallothionein 1M[5]. Genus *Paenibacillus* and genus *Enterococcus* were decreased and correlated with increased lactic acid level, and correlated with changes in expression of pho-

spholipase C beta 1, and immunoglobulin superfamily member 9. The study identified associations between microbial taxa, butyrate-related metabolites, and DNA methylation-associated gene expression in CRC tissues compared to normal tissues, and normal colonic mucosa tissues from CRC patients. The study showed the role of microbiota in the carcinogenesis of CRC. These findings offer insight into the contribution of protein toxins in causing CRC, concomitant with other factors, such as microbial metabolites, mutations, and inflammatory environment[5].

Chinese study of fecal microbiota

Investigation of the microbial diversity and communities in healthy individuals and patients suffering from CRC was conducted by He *et al*[6]. They studied the fecal microbiota in 61 patients with CRC and 72 normal individuals. The excrement of the two individual groups was analyzed by 16S rDNA sequencing, which showed that the abundance of the Firmicutes phylum was reduced in CRC tissues. The Firmicutes phylum was down-regulated, while the Bacteroidetes phylum was up-regulated compared with normal tissues. The species of *Faecalibacterium prausnitzii*, *Bacteroides vulgatus*, and *Fusicatenibacter saccharivorans* were lower in CRC compared to normal. At the species level, *Prevotella copri*, *Bacteroides uniformis*, and *Escherichia coli* were higher in CRC compared to normal. The results showed that beneficial microbiota were decreased in CRC, while dysbiotic microbiota had increased in the CRC group[6].

Cuba study-tissue microbiota

A study analyzing 34 samples, corresponding to 17 individuals with CRC in Cuba, was conducted by García *et al*[7]. The demographic distribution of age and sex of CRC was insignificant. The study provides an understanding of microbial influences on CRC. Performing analyses of alpha diversity, taxonomic composition, biomarkers, and metabolic pathways, it showed a correlation between GM and CRC. The study showed that the abundance of Fusobacteria was related to the CRC side and microsatellite instability[7].

Indian study-faecal microbiota

Investigation of microbiota in Indian individuals was conducted by Bamola *et al*[8]. Stool samples were collected from healthy adults ($n = 12$) and patients with colon cancer ($n = 12$). Subjects in both groups were "Indian nonvegetarian"; the samples from colon cancer patients were obtained before chemotherapy, radiotherapy, and surgical intervention; the diagnosis of CRC had been histopathologically confirmed. Analysis was done using 16S ribosomal RNA amplicon sequencing. Operational taxonomic units were calculated for different bacterial taxon including phylum, class, order, family, and genus level. The observed results indicated a considerable difference in the bacterial diversity between the groups[8]. Phylum Firmicutes was significantly dominant in both groups, followed by Bacteroidetes, Actinobacteria, and Proteobacteria, which clearly indicates the dominance of Phylum Firmicutes in the Indian population. Phylum Firmicutes and Actinobacteria were significantly abundant in the healthy group, while phylum Bacteroidetes in the colon cancer group. Bacterial genera *Megamonas*, *Megasphaera*, *Mitsuokella*, and *Streptococcus* were significantly abundant in the healthy group, and *Veillonella*, *Prevotella*, and *Eubacterium* in the CRC group. Bacterial genus *Bradyrhizobium* was present in the healthy group, and *Alistipes*, *Coproccoccus*, *Dorea*, and *Rhodococcus* were present in the CRC group but absent in the healthy group. They showed that GM plays an important role in the development of CRC. The geography, lifestyle, and dietary habits of Indians are different from those of the Western world; thus, microbiota studies of the Western population could not be extrapolated to their Indian counterparts[8].

METAGENOMIC DATASETS ANALYSIS TO IDENTIFY LINKS BETWEEN THE GUT MICROBIOME AND CRC, WORLDWIDE STUDY-FEACAL MICROBIOTA

To identify the relationships between the GM and CRC, shotgun metagenomic sequencing was conducted on the stool microbiome of 140 CRC patients and controls recruited in two cohorts, and these were analyzed in the context of 624 additional samples from five publicly available and geographically diverse metagenomic studies. The results validated the results on two novel datasets of 60 CRC and 65 controls, and 40 CRC and 40 controls. In total, the study considered 413 samples from CRC patients, 143 from subjects with adenoma, and 413 control samples. The large-scale meta-analysis of sequencing data from 768 faecal metagenomes and 969 faecal metagenomes from geographically distinct cohorts of patients with CRC from France, China, Austria, the United States, Canada, and Italy has unraveled several core pathogenic species that are enriched in these patients. Most notable is the enrichment of oral pathogens, including *Fusobacterium nucleatum*, *Parvimonas micra*, *Peptostreptococcus stomatis*, *Peptostreptococcus anaerobius*, *Porphyromonas asaccharolytica*, *Solobacterium moorei*, and *Prevotella intermedia*. Besides CRC, elevated levels of oral pathogens can be found in the GM of patients with colorectal adenomas, including *Fusobacterium nucleatum*, *Solobacterium moorei*, and *Lachnospirillum*, suggesting that gut dysbiosis is an early event in colorectal tumorigenesis. Specific colon-resident bacterial strains have also been shown to be enriched in patients with CRC in other studies, including enterotoxigenic *Bacteroides fragilis*, pks+ *Escherichia coli*, *Streptococcus gallolyticus*, and *Morganella morganii*[9].

Chinese study-tissue microbiota

The study examined the colonic mucosa obtained by colonoscopy: CRC ($n = 52$), colorectal polyps ($n = 47$), and tumor-free colon individuals ($n = 61$). The study showed that Proteobacteria and Firmicutes were persistent in the mucosa adjacent to adenomas and in the mucosa adjacent to CRC. These findings showed that dysbiosis in the adjacent mucosa had occurred in the colonic micro-environment prior to tumor development. The study showed taxonomic differences at

the phylum level in tumors and adjacent mucosa. The study postulated that dietary carbohydrates could be associated with incidences of CRC[10]. Inflammation or colitis may also favor the growth of specific bacterial populations that could elicit oncogenesis. The study concluded that GM enhances intestinal cell permeability leading to modulation of inflammation in a process of tumorigenesis *via* activities of co-occurring dysbiosis microbiota[10].

Iranian study-tissue microbiota

Dadgar-Zankbar *et al*[11] investigated microbiota in 30 Iranian patients with colon cancer and their paired adjacent healthy tissues, by the quantitative polymerase chain reaction method. They found that *Bacteroides fragilis* was present in 30 tumors. There was a correlation between *Bacteroides fragilis* and the expression of signaling pathway genes, including *CCND1*, *TP53*, *BCL2*, *BAX*, *WNT*, *TCF*, *AXIN*, *APC*, and *CTNNB1*. The study showed that *Bacteroides fragilis* was detected in 100% of tumor samples and 86% of healthy tissues[11]. The high level of *Bacteroides fragilis* had a direct relationship with the high expression of *AXIN*, *CTNNB1*, and *BCL2* genes. They concluded that *Bacteroides fragilis* had a higher abundance in the CRC samples compared to healthy tissues. They postulated that *Bacteroides fragilis* leads to CRC by changing cellular signaling pathways[11].

EXPERIMENTAL ANIMAL MODEL OF INDUCTION OF CRC WITH HUMAN GM: THE ROLE OF INFLAMMATION AND GM

Experiment 1

The germ-free interleukin-10-deficient (IL-10^{-/-}) mice are administered with azoxymethane (AOM) with either the commensal murine adherent-invasive *Escherichia coli* NC101 or the human commensal *Enterococcus faecalis* OG1RF; both bacteria cause aggressive colitis in IL-10^{-/-} mice. Consequently, the AOM-treated IL-10^{-/-} mice developed severe colitis. 80% of *Escherichia coli*-associated mice developed invasive mucinous adenocarcinoma, while *Enterococcus faecalis*-associated mice rarely developed tumors. Cytokines expressed in both arms in inflammation and colon cancer, including IL-6, tumor necrosis factor- α , interferon- γ , IL-1 β , IL-18, IL-17, and IL-23, were expressed in both groups. Infiltrating CD3⁺ T cells, F4/80⁺ macrophages, and Ly6B.2⁺ monocytes and neutrophils were similar in both groups. The experiment clearly demonstrates that microbes, including *Escherichia coli*, influence CRC in mice. The carcinogenic effect of *Escherichia coli* NC101 initiated genotoxic microorganisms promote CRC in the presence of the carcinogen AOM in IL-10^{-/-} mice[12].

Experiment 2

A study explored the pathogenesis of colonic carcinogenesis by *Bacteroides fragilis* (ETBF) that secretes *Bacteroides fragilis* toxin that causes human inflammatory diarrhea[13]. The study indicates that ETBF is colonizing mice colon. ETBF causes colitis and induces colonic tumors[13]. ETBF induces selective colonic signal transducer and activator of transcription-3 and is characterized by a selective T helper type 17 response distributed between CD4⁺ T cell receptor- $\alpha\beta$ ⁺ and CD48-TCR $\gamma\delta$ ⁺ T cells. Antibody-mediated blockade of IL-17 and IL-23 receptors that amplify T helper type 17 responses inhibits ETBF-induced colitis, colonic hyperplasia, and colonic tumors[13].

GM AND BLADDER CARCINOMA: CIRCULATING METABOLIC BIOMARKERS, MYCOBIOMES, GUT-BLADDER AXIS

GM are not inhabitants in the urinary tract; the postulation that GM attributes to bladder carcinoma (BCa) assumes that the circulating metabolic toxins are tumorigenic and would initiate BCa. Observational studies and clinical trials on the link between GM and BCa have shown that the association is not completely elucidated due to various confounding factors. Circulating metabolic biomarkers (CMBs): The interaction between GM and BCa is unclear. It was postulated that CMBs have been the causative factors. Fifteen GMs and 12 CMBs were found to be associated with BCa[14]. *Dorea*, which is a gram-positive bacterial genus from the Lachnospiraceae family, is present in human stools. Was found to significantly increase the risk of developing BCa. Additionally, it was postulated that the total cholesterol levels in small high-density lipoprotein and its cholesterol esters are a causative factor for BCa[14].

GM plays a pathogenic role in the initiation and recurrence of BCa[15,16]. A genome-wide association study on 233 CMBs from 136016 participants in 33 cohorts was conducted by Karjalainen *et al*[17] to investigate the metabolic pathways in the disease pathogenesis. The role of CMBs as an etiological factor between GM and BCa is still not completely elucidated[17]. Previous studies to define the connection between GM and BCa have been considered. Most of the conclusions were primarily based on observational studies of GM abundance in stool or in urine[18-20].

Lipid metabolism

Expanded evidence has revealed the contribution of lipid metabolism in the initiation of BCa, recurrence, and progression [21]. GM may play a role in modulating immune responses, and prognosis of BCa, CMBs is a component of these events that play crucial roles in the initiation of BCa[22,23].

Specific GM taxa and BCa

Yin *et al*[24], identified GM by applying a genome-wide association study on the MiBioGen consortium and data for 5 urological cancers from the United Kingdom biobank and FinnGen consortium to examine the causality to BCa. It was revealed that the family Rikenellaceae, genus *Allisonella*, genus Lachnospiraceae Unclassified Genus Group 001, genus *Oscillibacter*, genus *Eubacterium coprostanoligenes* group, genus *Eubacterium ruminantium* group, genus Ruminococcaceae Unclassified Genus Group 013, and genus *Senegalimassilia* were related to BCa. Their study confirms the role of specific GM taxa on BCa[24]. Surber *et al*[25] showed that in BCa patients abundance of specific GM genera was abundant, they are: *Rubrobacter* and *Geobacillus*, which were more prevalent in BCa patients, others GM, like *Roseomonas*, *Streptococcus*, and *Lactobacillus*, were more prevalent in controls[25].

Mycobiome

The composition of the mycobiome clustered between healthy individuals and patients with BCa is influenced by GM, which would be a factor in the initiation of BCa[25,26]. Bukavina *et al*[27] analyzed faecal samples from healthy individuals and patients with BCa. It was found that there are unique differences in mycobiome composition between BCa and healthy individuals[27]. The dominant fungus at the order level in both groups was Saccharomycetales, with a lower abundance in BCa compared to healthy individuals. In BCa, the most abundant fungal order was Hypocreales, followed by Tremellales, Sporidiobolales, and Dothideales. The mycobiome in healthy individuals was the genera *Guignardia*, *Sebacina*, and *Stylonectria*. The highest abundance differences between BCa and healthy individuals were found in Hypocreales, Tremellales, and Dothideales, which were all increased in the gut mycobiome of patients with BCa[26].

GUT-BLADDER AXIS

The interplay between GM and the urinary bladder is defined as the “gut-bladder axis”, which is still an exploratory research to investigate the correlation between dysbiotic GM as a causative factor in BCa. Specific metabolites originating from the metabolism are risk factors in BCa. N-butyl-N- (4-hydroxybutyl)-nitrosamine, Xenobiotics transformation is carried out by the GM, which is attributed to the initiation of BCa. Two-thirds of the pharmaceuticals product is excreted in urine, and one-third is excreted in stools and is exposed to GM for a longer time[20]. The gut-bladder axis is a two-directional network that connects the gastrointestinal and urinary systems, showing a connection between GM and BCa. Dysbiotic GM, microbial metabolites, and disrupted neuro-immune pathways have been postulated to be implicated in the tumorigenesis of BCa. The short-chain fatty acids, which are microbial metabolites, are shown to alter the immune microenvironment and decrease inflammatory response. Cross-organ crosstalk is mediated *via* neural pathways and common receptors, such as farnesoid X receptor, and Toll-like receptor 4[27].

GM and ICIs

Specific GMs are correlated with enhanced ICIs efficacy in the treatment of BCa. Urinary microbiome alteration has an impact on treatment outcomes of non-muscle invasive BCa with intravesical administration of bacillus Calmette-Guerin. Microbial modulation has an effect on immune response that has an influence on tumor microenvironments and response to treatment of BCa[28].

Proton pump inhibitors

Recently, it was clarified that proton pump inhibitors (PPIs) can modulate microbiota's diversity; therefore, they would alter the response to ICIs. Recent publications showed the impact of concomitant PPI treatment on outcomes of ICIs-treated patients; it was found that combined PPI and ICIs use was associated with worse overall survival for BCa patients [29].

GM and metabolites

The GM produces various metabolites that contribute to tumorigenesis. However, the correlation between integrative analysis of the GM metabolites and BCa is not fully understood. To unravel this connection, a metagenomic analysis of fecal samples of patients with BCa and healthy individuals was done. GM was significantly dysregulated in BCa patients, which included *Bifidobacterium*, *Lactobacillus*, *Streptococcus*, *Blautia*, and *Eubacterium*. BCa patients showed an increase in cholesterol sulfate. Metagenomics analysis identified the alterations of GM and metabolites in BCa[30]. A study examines the variance in the GM of patients with BCa compared with healthy adults[31]. The study reveals distinct clustering, effectively separating the BCa and healthy cohorts[31]. The evaluation of short-chain fatty acid concentration in blood and stool revealed a higher abundance of Akkermansia and Clostridia, with increased levels of fecal isobutyric acid. An increase of *Lactobacillus* and *Enterobacteriaceae* correlated with increased fecal propionic acid. Distinct clustering, effectively separating the bladder cancer and healthy cohorts[31].

CONCLUSION

The investigation of the microbiota of the gut and its connection to CRC and BCa has been extensively studied. Changes in GM have been associated with a range of diseases in humans. The GM has a wide diversity in humans due to multiple host factors. The relation of GM to CRC has been studied extensively; a point of interest is the variation of results in

relation to the location. We presented the results of the studies in different locations in France, Italy, the United States, Canada, Cuba, China, Iran, and India. The results are different. The experimental studies in animal models established the integration of human GM in the induction of inflammation and colon cancer. It is worth mentioning that data is specific and it is not to be generalized, but rather to be a guide. The study of GM and bladder cancer is still growing, and we cannot reach a definite conclusion.

FOOTNOTES

Author contributions: Wishahi M contributed to conception and design, collection and assembly of data, data analysis and interpretation, manuscript writing, revision, and final approval of the manuscript.

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