ISSN 1007-9327 (print)
ISSN 2219-2840 (online)

World Journal of *Gastroenterology*

World J Gastroenterol 2024 September 28; 30(36): 4014-4082

Published by Baishideng Publishing Group Inc

World Journal of Gastroenterology

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NAME OF JOURNAL
World Journal of Gastroenterology

ISSN
ISSN 1007-9327 (print) ISSN 2219-2840 (online)

LAUNCH DATE
October 1, 1995

FREQUENCY
Weekly

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PUBLICATION DATE
September 28, 2024

PUBLISHING PARTNER
Shanghai Pancreatic Cancer Institute and Pancreatic Cancer Institute, Fudan University
Biliary Tract Disease Institute, Fudan University

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World J Gastroenterol 2024 September 28; 30(36): 4078-4082

DOI: 10.3748/wjg.v30.i36.4078

ISSN 1007-9327 (print) ISSN 2219-2840 (online)

LETTER TO THE EDITOR

Beyond bacteria: Role of non-bacterial gut microbiota species in inflammatory bowel disease and colorectal cancer progression

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Specialty type: Gastroenterology and hepatology

Provenance and peer review: Invited article; Externally peer reviewed.

Peer-review model: Single blind

Peer-review report's classification

Scientific Quality: Grade C

Novelty: Grade C

Creativity or Innovation: Grade C

Scientific Significance: Grade B

P-Reviewer: Wang J

Received: June 9, 2024

Revised: August 11, 2024

Accepted: September 6, 2024

Published online: September 28, 2024

Processing time: 102 Days and 20 Hours

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Abstract

This letter emphasizes the need to expand discussions on gut microbiome's role in inflammatory bowel disease (IBD) and colorectal cancer (CRC) by including the often-overlooked non-bacterial components of the human gut flora. It highlights how viral, fungal and archaeal inhabitants of the gut respond towards gut dysbiosis and contribute to disease progression. Viruses such as bacteriophages target certain bacterial species and modulate the immune system. Other viruses found associated include Epstein-Barr virus, human papillomavirus, John Cunningham virus, cytomegalovirus, and human herpes simplex virus type 6. Fungi such as *Candida albicans* and *Malassezia* contribute by forming tissue-invasive filaments and producing inflammatory cytokines, respectively. Archaea, mainly *methanogens* are also found altering the microbial fermentation pathways. This correspondence, thus underscores the significance of considering the pathological and physiological mechanisms of the entire spectrum of the gut microbiota to develop effective therapeutic interventions for both IBD and CRC.

Key Words: Gut microbiota; Colorectal cancer; Inflammatory bowel disease; Dysbiosis; Bacteriophages; Methanogens; Fungi

Core Tip: This letter to the editor intends to contribute to the conversation surrounding the role of gut microbiota in the progression of conditions such as inflammatory bowel disease and colorectal cancer. The letter emphasizes the importance of recognizing microbial components beyond bacteria that also play a significant role in the pathogenesis of these diseases, along with encouraging further studies in this area to better understand the role of viruses, fungi and archaea.

Citation: Haque H, Zehra SW, Shahzaib M, Abbas S, Jaffar N. Beyond bacteria: Role of non-bacterial gut microbiota species in inflammatory bowel disease and colorectal cancer progression. *World J Gastroenterol* 2024; 30(36): 4078-4082
URL: https://www.wjgnet.com/1007-9327/full/v30/i36/4078.htm
DOI: https://dx.doi.org/10.3748/wjg.v30.i36.4078

TO THE EDITOR

It was with great interest that we read the article by Quaglio *et al*[1]. The article reviewed how different types of human gut micro biomes may contribute to inflammatory bowel disease (IBD) and colorectal cancer (CRC). It also tried to find a common link between the two pathologies. However, we found that the discussion mainly focused on bacterial species, overlooking the potential involvement of other microbial components such as viruses, fungi, and archaea that also inhabit the human gut flora and have recently been implicated in these diseases when in a state of dysbiosis.

VIRUSES

Bacteriophage

Several viruses, predominantly bacteriophages, also form a major part of gut microbiota and its bacterial population[2]. According to an estimate, the human gut contains approximately 10^{15} phages, outnumbering the 10^{14} gut bacteria by around 10-fold[3]. They act as natural predators to bacteria of the gut, selectively killing some of its strains and maintaining an appropriate composition[4]. Moreover, bacteriophages, through some of their interactions in the gut, also release certain cytokines that help strengthen the immune system of the body[3].

However, when the equilibrium is disturbed, bacteriophages can contribute to the development of many gastric conditions including IBD, CRC, and IBD-associated CRC through several mechanisms. An increased abundance of certain phage species such as *Caudovirales*, *Escherichia*, and *Enterobacteria* has been seen in patients with IBD[3]. Moreover, virulent phages capable of targeting host bacteria and causing changes in the abundance of species are more commonly found in patients with IBD. For instance, these phages promote the growth of pathogenic proteobacteria such as *Escherichia coli* (*E. coli*) and *Fusobacteria* that drive inflammation and reduce the populations of potentially protective Firmicutes[3]. Additionally, certain phages cause lysis of bacterial species releasing cellular debris in the gut microenvironment, eventually inducing inflammation and increasing the risk of CRC[5].

Other viruses

Viruses contribute to the oncogenesis of CRC through direct viral infection of cells and indirect modulation of bacterial community composition[6]. Epstein-Barr virus (EBV), human papillomavirus (HPV), and John Cunningham virus (JCV) are the three known human carcinogenic viruses linked with the disease pathogenesis[7]. In fact, HPV infection considerably increases the relative risk of CRC (risk ratio = 2.97), while the presence of JCV substantiates the risk by 4.7 times[8,9]. Moreover, through the inactivation of p21 and mdm2 expression, the HPV16 E6 oncoprotein may decrease the transcriptional activity of p53 and further contribute to CRC[10]. In the case of IBD, EBV, cytomegalovirus (CMV), and human herpes simplex virus type 6 (HHV-6) are more commonly found in the mucous membrane of the colon and affect both the course of the disease and the effectiveness of treatment[11]. EBV and CMV are more prevalent in IBD patients compared to healthy controls, suggesting their role in the onset of the disease rather than its severity and clinical evolution[12]. CMV reactivation under intestinal barrier disruption, inflammation, or immunosuppressive therapy affect the prognosis of IBD by causing additional mucosal damage[13,14].

FUNGI

Candida albicans

Other important residents of the human gut are fungal species, such as *Candida albicans* (*C. albicans*)[15]. However, even a slight imbalance can provide them an opportunity to cause pathologies such as IBD, CRC along with others, in the same gut.

In the case of IBD, *C. albicans* form tissue-invasive filaments that secrete Candidalysin toxin to disrupt epithelial barriers. Recent research isolated the species from both healthy and IBD patients and on comparison, found IBD-derived isolates more readily able to form filaments[16]. They also showed altered cell wall composition and modulated

expression of adhesion-associated genes such as *IHD1*[16]. Additionally, when exposed to Candida strains from IBD patients, NETosis induction and increased swarming behavior were observed with neutrophils from healthy donors and monocytes releasing greater amounts of pro-inflammatory interleukin (IL)-1b[16]. This highlights the role of Candida in promoting inflammation in IBD. Besides, through examination of rectal swabs obtained from fifty-two patients with adenoma/CRC, significant overexpression of *C. albicans* was seen showing a potential association between the two[17]. From another experiment, it was found that deletion of the Dectin-3 gene could be a possible reason behind the onset and/or progression of CRC as it increases the abundance of *C. albicans*[18]. Dectin-3 is a type of pattern receptor mainly expressed on the surface of immune cells and plays an important role in the recognition and binding of pathogens like *C. albicans*[18]. A positive link was also seen between the frequency of IL-22 in tumor tissues of colon cancer patients and the presence of *C. albicans*[18].

Malassezia

Malassezia is a genus of fungi found naturally on the skin surfaces of animals and serves as a pathobiont for most skin diseases in humans such as dandruff and dermatitis. With growing developments in the field of gut microbiota, recent mycobiome analysis has also indicated *Malassezia* species as a core taxon in the intestinal microbiota[19]. Further research is being carried out to investigate the role of *Malassezia* species in the progression of various diseases, especially CRC and IBD.

IBDs evolve from genetic and environmental factors that ultimately activate the host's immune response against the gut microbiota. When dealing with IBDs, especially ulcerative colitis and Crohn's disease, the species *Malassezia restricta* (*M. restricta*) plays a significant role in exacerbating the inflammatory response. With regards to various types of research, high levels of *Malassezia* species in IBDs and various cancers serve as circumstantial evidence of the role of commensal fungi in the prognosis of both diseases. According to Limon *et al*[20], higher levels of *M. restricta* were found to be associated with the Risk Allele CARD9S12N, specific for Crohn's disease. The increased production of inflammatory cytokines and mediators due to excess amounts of *Malassezia* species caused severe intestinal inflammation along with shortening of the colon, mucosal erosion, infiltration, and a worsening prognosis was observed. *M. restricta* was further observed to exacerbate colitis in mouse models by activating the intestinal immune system[21].

CRC has also been associated with microbial and fungal dysbiosis notably through higher abundance of *Malassezia* species in the gut of CRC patients. Using advanced sequencing techniques, researchers have identified specific *Malassezia* species, particularly *M. restricta* and *M. globosa*, as being enriched in CRC patients' fecal samples[22]. This observation suggests a potential association between *Malassezia* colonization and CRC pathogenesis. The increased presence of *Malassezia* species has been noted in CRC, suggesting its potential involvement in tumor formation. This involvement may occur through mechanisms such as changes in tryptophan metabolism and initiating IL-33-mediated inflammation *via* activation of the complement cascade[23].

The interactions of these fungi with gut bacteria also play a crucial role in progression and severity of both IBD and CRC. For instance, *C. albicans* can form a biofilm with bacteria like *E. coli* and *Serratia* posing increased resistance to antimicrobial treatment than a single-species biofilm and a greater inflammatory response[24,25]. Besides, the virulence of *C. albicans* is dependent on hyphae formation and the mixed biofilm can help increase the expression of hyphae leading to greater pathogenicity potentially conducive to both IBD and CRC[24,25]. Moreover, a study on 235 patients with IBD found that Malassezia is negatively correlated with beneficial bacteria such as *Bifidobacterium*, *Blautia*, *Roseburia*, and *Ruminoccocus*[26]. Reduced levels of these components would mean fewer short-chain fatty acids (SCFAs) and increased inflammation, a common feature in the development of both IBD and CRC.

ARCHAEA

Methanogens

Methane producing archaea also called *methanogens* play a significant part in the gut microbiota despite their small proportion and serve in the pathogenesis of both CRC and IBDs[27,28].

In IBD, archaeal dysbiosis can lead to shifts in the archaeome, potentially encouraging the growth of organisms like *Methanobrevibractor smithii* thus triggering the production of proinflammatory cytokines like tumor necrosis factor-α in the gut. Furthermore, the proliferation of archaea due to dysbiosis may cause intestinal biofilms to lose crucial elements like butyrate, which will facilitate the entry of possibly harmful microbes across the gut barrier, worsening inflammation and disease progression[28]. This may result in aggravation of IBD-related intestinal mucosal inflammation.

Methanogens are also responsible in the setting of CRC. The pathogenesis mainly involves butyrate, a SCFA produced by the gut bacteria that physiologically strengthens the immune system and lowers the danger of polyps. In archaeal dysbiosis, bacterial fermentation may be shifted toward methanogenesis in CRC patients. Patients with colon cancer and precancerous signs produce more methane as a result of intestinal microbiota, which when oxidized will turn into the carcinogenic form, formaldehyde[29,30]. Furthermore, methane has been proposed to reduce oxidative stress, potentially providing a favorable environment for the development of CRC.

A major role in the pathogenesis of both these conditions is the interactions of *methanogens* with bacteria. In the gut, many bacteria such as *Firmicutes* and *Bacteroides*, produce hydrogen and depend on it for their growth[31]. *Methanogens* consume this hydrogen to produce methane which in turn decreases the abundance of hydrogen-producing bacteria leading to dysbiosis in the gut. This hydrogen scavenging by *methanogens* also indirectly affects the production of SCFAs such as butyrate, acetate, and propionate that are produced by the hydrogen-producing fermentative bacteria[31]. SCFAs maintain the gut health by strengthening its lining, regulating inflammation and modulating immune cells[32].

Consequently, lower levels of SCFAs due to methanogen-induced shifts can exacerbate the risk of developing gut pathologies such as IBD and CRC.

In conclusion, although the role of bacteria in gut dysbiosis and pathogenesis of IBD and CRC is well established, recent investigations have also found a key role of other microbial components. Viruses such as bacteriophages, EBV, HPV, JCV, CMV, and HHV-6, fungal species like *C. albicans* and *Malassezia,* and archaea such as *methanogens* also contribute to the progression of these diseases along with others through various mechanisms. Understanding the physiological and pathological role of these components along with extending discoveries towards finding more of such gut constituents will eventually help in developing better targeted therapies towards both IBD and CRC.

ACKNOWLEDGEMENTS

We acknowledge all the authors whose publications we have referred to in this letter to editor.

FOOTNOTES

Author contributions: Haque H and Zehra SW analyzed the literature and wrote the letter; Shahzaib M and Abbas S conducted relevant literature search; Shahzaib M proposed the idea; Jaffar N guided us through the process; All authors have read and approved the final manuscript.

Conflict-of-interest statement: All the authors report no relevant conflicts of interest for this article.

Country of origin: Pakistan

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S-Editor: Li L
L-Editor: Webster JR
P-Editor: Cai YX

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