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EDITORIAL

Relationship between *Helicobacter pylori* infection and colorectal polyp/colorectal cancer

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Abstract

Helicobacter pylori (*H. pylori*) plays an important role in the development of gastric cancer, although its association to colorectal polyp (CP) or colorectal cancer (CRC) is unknown. In this issue of *World Journal of Gastrointestinal Surgery*, Zhang *et al* investigated the risk factors for *H. pylori* infection after colon polyp resection. Importantly, the researchers used R software to create a prediction model for *H. pylori* infection based on their findings. This editorial gives an overview of the association between *H. pylori* and CP/CRC, including the clinical significance of *H. pylori* as an independent risk factor for CP/CRC, the underlying processes of *H. pylori*-associated carcinogenesis, and the possible risk factors and identification of *H. pylori*.

Key Words: *Helicobacter pylori*; Colorectal polyp; Colorectal cancer; Risk factor

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Core Tip: *Helicobacter pylori* (*H. pylori*) infection is a significant risk factor for colorectal adenomatous polyp (CAP) or colorectal cancer (CRC), and it may contribute to the development of CRC *via* an inflammatory response, gastrin stimulation, intestinal flora modulation, and virulence factor invasion. In contrast, the diagnosis of CAP may independently predict the probability of *H. pylori* infection after colon polyp removal.

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INTRODUCTION

Helicobacter pylori (*H. pylori*) is a microaerophilic, gram-negative bacteria that lives mostly in the stomach and duodenum of humans. It spreads to individuals by a variety of routes, including oral-oral, fecal-oral, and gastro-oral pathways[1]. Because to *H. pylori*'s various transmission channels, growing medication resistance, poor eradication rate, and insufficient treatment duration, 43.1 percent of the world population was infected from 2011 to 2022[2]. In impoverished nations, the *H. pylori* infection rate approached 70%, while infection rates in affluent countries varied from 25% to 50%[3]. Although most people with *H. pylori* infection are asymptomatic, long-term infection may cause chronic gastritis and is linked to a variety of non-gastric ailments, including atherosclerosis[4], type 2 diabetes[5], anemia[6], osteoporosis[7], and some immune-related disorders[8]. Furthermore, 1%-3% of those infected with *H. pylori* develop gastric cancer[9], and *H. pylori* infection is also strongly related with mucosa-associated lymphoid tissue lymphoma[10] and colorectal cancer (CRC)[11]. As a result, the World Health Organization's International Cancer Research Centre designated *H. pylori* as a Class I biological carcinogen[12]. In conclusion, screening and eradication of *H. pylori* is an important primary preventative method for tumorigenesis[13].

In recent decades, the connection between *H. pylori* and colorectal polyp (CP) or CRC has sparked attention. And, there is accumulating clinical and basic experiment evidence indicating that *H. pylori* infection is a risk factor for CP/CRC and promotes the development of CRC. In this paper, two researchers (DQ-Y and Y-L) conducted independent literature searches using PubMed, Embase, and Cochrane Library databases to obtain more comprehensive literature data. The search was conducted from inception to December 2023, with search keywords including "*Helicobacter pylori*", "*H. pylori*", "*Helicobacter pylori* infection", "*H. pylori* infection", "colorectal polyp", "CP", "colorectal adenomatous polyp", "CAP", "colorectal adenoma", "colorectal neoplasm", "colorectal neoplasia", "colorectal cancer", "colorectal carcinoma", "CRC", and "colorectal tumor". Additionally, manual searches of the reference lists of the obtained articles were conducted to avoid any omission of studies. Further, we investigate the link between *H. pylori* and CP/CRC, as well as the possible pathogenic processes involved.

THE ASSOCIATION BETWEEN *H. PYLORI* AND CP/CRC

Several case-control and retrospective cohort studies have shown a substantial link between *H. pylori* infection and the prevalence of CP/CRC[14-17]. Similarly, cross-sectional studies conducted in various areas and nations consistently shown that *H. pylori* infection was an independent risk factor for CP, especially colorectal adenomatous polyp (CAP)[18-21]. Boustany *et al*[22] reported the first findings from a large population-based analysis of 47714750 people, establishing an independent link between a history of *H. pylori* infection and the risk of CRC. A large-scale meta-analysis of 48 studies found that both CAP and CRC were linked to *H. pylori* infection[23]. Yang *et al*[24] used a fixed-effects model to analyze 27 studies and discovered that different study methods, including case-control studies [odds ratio (OR) = 1.26, 95% confidence interval (95%CI): 1.16-1.36], cross-sectional studies (OR = 1.92, 95%CI: 1.17-3.16), and multiple detection methods (OR = 2.63, 95%CI: 1.09-6.31), all supported the strong link between *H. pylori* infection and an increased risk of CRC. A prospective European multi-center investigation found that serum antibodies to *H. pylori* protein C and cytotoxin-associated gene A were linked to an elevated risk of CRC[25]. *H. pylori* infection in patients with atrophic gastritis [26], intestinal metaplasia[27], diabetes[28], and metabolic syndrome[29] may enhance the risk of CAP. Further-more, as demonstrated in Table 1, *H. pylori* infection is linked to pathological clinical characteristics such as the size, number, location, and pathological type of CAP.

Individuals with present or past *H. pylori* infection have a 30% to 50% higher risk of developing CRC[30]. *H. pylori* infection was discovered as a predictive factor for the construction of the CAP-risk model, which has shown excellent clinical relevance in prospective cohort studies[31]. Triple treatment coupled with long-term use of proton pump inhibitors may lower the incidence of CAP caused by *H. pylori* infection[32], and *H. pylori* eradication before to surgery may become a novel immunotherapy method for CRC[33]. In contrast, if *H. pylori* is not adequately treated and continues, it might considerably increase the recurrence risk of CAP[34-36].

However, several clinical research did not find a substantial link between *H. pylori* and CP/CRC[37-39]. We take into account the criteria for positive *H. pylori* infection, *H. pylori* diagnostic methods, the interference of *H. pylori*-related diseases[26], incomplete colonoscopy, sample size and age[40,41], as well as ethnic[30,41] and regional environmental differences, all of which may lead to inconsistent results.

Zhang *et al*[42] performed thorough scientific research in this issue of *World Journal of Gastrointestinal Surgery* and proposed a novel route for researching the association between *H. pylori* and CP. The research found that age, body mass index (BMI), and CAP were independent predictors of *H. pylori* infection after post-colon polyp resection. In addition, the research used R software to create a column-line graph prediction model. The model has been demonstrated to have strong calibration, discrimination, and prediction abilities in predicting *H. pylori* risk by calculating calibration curves,

Table 1 The correlation between *Helicobacter pylori* infection and the clinicopathologic characteristics of colorectal polyp

Characteristics	Variables related to *H. pylori* infection		Ref.
	Variable	Correlation with *H. pylori* infection	
Polyp size (diameter)	> 1.0 cm	Positive	[14,93,95,96]
Polyp/adenomatous polyp number	> 1.0	Negative	[93]
		Positive	[17,95-99]
Pathological type	Adenoma (all)	Positive	[19-21,23,26,27,35,97,98,100-106]
	Tubular adenoma	Positive	[34,107]
	Villous adenoma	Positive	[14,93,96,107]
	Tubular villous adenoma	Positive	[14,34]
	Hyperplastic polyp	Positive	[36,96,103]
	Serrated polyp	Positive	[108]
Adenomatous polyp stage	Advanced adenoma	Positive	[17,23,93,96,103,105]
Distribution of adenomatous polyp	Proximal	Positive	[17,19]

H. pylori: *Helicobacter pylori*.

receiver operating characteristic curves, and decision curve analysis curves, as well as validating the model validation cohort. This prediction approach might aid in the early detection of *H. pylori* infection.

POTENTIAL CARCINOGENIC EFFECT OF *H. PYLORI* ON CRC

Impact of H. pylori-related inflammation on CRC

H. pylori is a major immunostimulant. Ralser *et al*[11] discovered that in the Apc mutant mouse model, *H. pylori* infection recruited CD3+ T cells within the colon tissue epithelium, upregulated CD8+ T cells, and caused Treg cell loss and differentiation into potentially pathogenic Foxp3+ IL-17A+ T cells[43], resulting in a specific pro-inflammatory immune response. Furthermore, the research found that *H. pylori* might activate the STAT3 cancer pathway in colon cells, further blocking Treg cell infiltration[44], which was followed by elevated Ki67 expression and reduced intestinal barrier marker PAS[11]. This research also provides the first direct evidence of a causal link between *H. pylori* infection and CRC. Furthermore, *H. pylori* may produce chronic gastritis, which exacerbates the systemic inflammatory response[45]. Inflammation has been linked to an increased risk of CRC, and long-term usage of aspirin as an anti-inflammatory medication may help prevent CRC[46]. Future research in CRC will concentrate on the inflammatory stimulation and immunological modulation related with *H. pylori* infection.

Gastrin and CRC

Gastric atrophy produced by *H. pylori*-related gastritis results in hypergastrinemia following hypochlorhydria[47], and serum gastrin and *H. pylori* IgG are linked with an increased risk of CRC[48]. As a result, more research has focused on the relationship between gastrin and CRC, and it has been discovered that cholecystokinin B/gastrin receptors are overexpressed in CAP and CRC[49], and the gastrin binding capacity of CRC is ten times greater than that of normal colonic mucosa, promoting cell growth[50,51]. Furthermore, gastrin and progastrin have been shown to be released by CRC cells [52], and they operate as autocrine/paracrine growth factors to stimulate growth/anti-apoptosis in normal colonic mucosal cells, influencing gland development and shape[53,54]. Surprisingly, certain CRC cell proliferation may rely more on autocrine progastrin[55].

The role of gastrin in facilitating the advancement of the adenoma-carcinoma sequence in the colorectum has also received attention. Autocrine progastrin may activate Src kinase in CRC cells, hence mediating the proliferation/anti-apoptotic actions of endocrine/autocrine progastrin[56-58]. Furthermore, *in vitro* and *in vivo* research have revealed that the progastrin-related pathway may disturb normal stem cell populations by upregulating CD133, CD44, DCLK1, and Lgr5 stem cell markers, increasing cancer cells' oncogenic and metastatic potential[56,59-61]. Progastrin also binds to the G protein-coupled receptor 56 in colon stem cells, conferring growth potential[62]. Furthermore, gastrin has been shown to upregulate many colon cancer target genes, including cyclin D1, vascular endothelial growth factor, vascular endothelial-cadherin, and matrix metalloproteinase-2, promoting tumor development[63-65]. Although different data point to the oncogenic qualities of gastrin and its derivatives, further study is required to determine the particular involvement of gastrin in the development of CRC.

Changes in intestinal flora associated with H. pylori infection

Environmental variables, such as food structure and intestinal flora, account for 80%-85% of CRC, and changes in intestinal microbial balance may function as environmental triggers for CRC[66]. The quantity of intestinal flora varies with the origin and evolution of CRC[67]. Simultaneously, increased abundance of pro-tumor bacteria such as polyketide synthase-positive (pks+) *Escherichia coli* (*E. coli*), enterotoxigenic Bacteroides fragilis, and Fusobacterium nucleatum can promote intestinal inflammation *via* toxins or metabolites, facilitate tumor cell proliferation and migration, and create an immunosuppressive microenvironment that limits anti-tumor immunity[68,69]. The preceding data indicate that the intestinal flora plays an important role in the development of CRC and influences tumor chemotherapy and immunotherapy[70].

Chronic *H. pylori* infection is a major cause of diminished microbial diversity in the stomach[71]. It also boosts the number of microorganisms in stomach cancer, including nitrate-reducing bacteria, nitrosylobacteria, and *E. coli*, which promotes nitrate metabolism. The resultant N-nitroso compounds function as carcinogens and promote tumorigenesis [72]. An increasing body of evidence suggests that *H. pylori* infection also impacts the intestinal flora. *H. pylori* invasion of the intestinal mucosa may lead to reduced intestinal permeability[72] and inhibit *E. coli* DNA[73]. *H. pylori* may also trigger host immune responses, thereby altering the intestinal flora[74,75]. Furthermore, long-term *H. pylori* infection can alter the pH in the stomach, enabling more microorganisms to overcome the acid barrier and enter the distal intestinal tract[75]. CagA also can stimulate the overproliferation of intestinal stem cells and alter the host microbiota[76]. Recent study has revealed that *H. pylori* promotes the enrichment of *Akkermansia* spp. and *Ruminococcus* spp., which breakdown intestinal mucus, in the colon tissue of *H. pylori*-infected mice, resulting in a pro-inflammatory and pro-carcinogenic microbiota signature[11]. It is possible that *H. pylori* infection weakens the intestinal barrier. Furthermore, Luo *et al*[77] discovered that in the early stages of CRC development, *H. pylori* infection promotes the amplification of temperate phages to disrupt intestinal virome homeostasis and interacts with bacterial communities to target tumor-associated bacteria such as *Lactobacillus*[78] and *Enterococcus faecalis*[79], promoting the development of CRC in mice. To summarize, *H. pylori*-induced intestinal flora dysbiosis is a significant risk factor for the development of CRC.

The virulence factors of H. pylori

H. pylori's virulence components, including as CagA, vacuolating cytotoxin A (VacA), and high-temperature requirement A, play a crucial role in the process by which *H. pylori* causes stomach cancer. These virulence factors may increase the onset and development of gastric cancer by altering cell structure and shape, as well as impairing cell proliferation and apoptosis[80]. Some investigations have shown that CagA expression[81-83] and the serological response to *H. pylori* VacA, particularly in African Americans[30], are related with an elevated risk of CRC formation. Furthermore, infection with robust strains of CagA-positive *H. pylori* may contribute to the development of CRC by eliciting increased inflammatory responses[84]. There is some indication that *H. pylori* may be present in CPs[85,86], however further data is required to show the direct influence of *H. pylori* bacterial components on the development of CRC. It cannot be ruled out that virulence factors like VacA may easily spread outside the stomach wall[30,87], thereby causing cancer.

RISK FACTORS FOR *H. PYLORI* INFECTION

A variety of variables impact *H. pylori* infection rates, including economic position, living circumstances, cleanliness, lifestyle, occupation, and drinking water quality[88]. It is also difficult for *H. pylori* infections to resolve spontaneously [89]. According to current research, *H. pylori* infection rates are increased in obesity[90], portal hypertensive gastropathy [91], periodontal disease[92], and colon polyps[93]. In addition, untreated dental caries may have an impact on systemic *H. pylori* infection[94]. Zhang *et al*[42] hypothesized that age, BMI, and CAP were risk factors for *H. pylori* infection after post-colon polyp surgery. Perhaps particular disorders can be utilized to identify those who are at high risk for *H. pylori* infection.

CONCLUSION

In conclusion, Zhang *et al*[42] performed a retrospective clinical study and discovered that age, BMI, and CAP were independent predictors of *H. pylori* infection after postcolon polyp surgery. They also created a column-line graph prediction model with high calibration and predictive power for predicting the probability of *H. pylori* infection. However, the *H. pylori* prediction model may still be improved. Future studies should control for major confounding variables such as age, metabolic parameters, smoking, alcohol intake, physical activity, food, racial variations, socioeconomic level, and antibiotic usage. The investigation should also look into the association between the period of *H. pylori* infection or recurrence following post-colon polyp surgery and CAP. Simultaneously, multi-center, large-scale prospective research should be carried out to create a scientifically rigorous *H. pylori*-CRC screening program. More emphasis should be placed on the simultaneous diagnosis of CAP and *H. pylori*-related disorders[4-8,92,94] or specific diseases[91]. This technique will not only increase the screening rate of those at high risk of *H. pylori* infection and the diagnostic rate of infected individuals, but will also allow for a better understanding of the mechanism behind the link between *H. pylori* infection and the development of CRC.

FOOTNOTES

Author contributions: Liu Y contributed to the writing, editing of the manuscript and table, and literature search; Yang DQ contributed to the discussion, design of the manuscript and literature search; Jiang JN contributed to the discussion and design of the manuscript; and Jiao Y designed the overall concept and outline of the manuscript.

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