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Similarities and Differences between Helicobacter Pylori and Campylobacter Jejuni and Measures to Combat Them

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Annotation: Epsilonproteobacteria bacteria such as Campylobacter jejuni and Helicobacter pylori cause foodborne infections that cause human campylobacteriosis, which is the leading cause of bacterial gastroenteritis worldwide. Infected people develop abdominal pain and diarrhea after eating infected poultry meat, which is the main source of transmission of pathogens to humans. After acute enteritis, postinfectious disorders affecting the nervous system, joints or intestines may occur. Immunodeficiency concomitant diseases of infected patients cause bacteremia, which causes septicemia and vascular inflammation. Prevention of human infection is achieved through hygienic measures aimed at reducing pathogenic contamination of food products. Molecular targets for the treatment and prevention of campylobacteriosis and Helicobacteriosis include pathogenicity and virulence factors of bacteria involved in motility, adhesion, invasion, oxygen detoxification, acid resistance and biofilm formation. Drugs that suppress pro-inflammatory immune reactions caused by Campylobacter and Helicobacter endotoxin lipooligosaccharide have recently added to this list of treatment methods. To reduce the risk of both antimicrobial resistance and the post-infectious effects of acute enteritis, new pharmaceutical approaches will combine anti-pathogenic and anti-inflammatory effects. This review presents the latest methods and trends in the fight against Campylobacter and Helicobacter infections, as well as molecular targets for prevention and treatment.

Keywords: Epsilonproteobacteria, Arcobacter, peptic ulcer disease, gram-negative bacteria, human gastrointestinal pathogen.

Annotation. *Epsilonproteobacteria* types, including *Arcobacter, Campylobacter,* and *Helicobacter* are commonly associated with vertebrate hosts, and some are considered significant pathogens. *Epsilonproteobacteria* associated with vertebrates are generally considered to be restricted to endothermic mammals and birds. Recent studies have shown that ectothermal reptiles represent a clearly and largely exclusive community of *Ellipsopterobacteria*. These taxa include taxa that potentially cause diseases in humans. *Arcobacter* has several taxa that are widespread among reptiles and often have a wide range of hosts. The reptiles contain a wide range of completely new *Helicobacter taxa* that appear to have originated from an ectothermal host. Some species, such as *Campylobacter fetus*, have a distinct intraspecific host dichotomy with genetically similar traits found in mammals and reptiles. These taxa can provide important information about host adaptation and co-evolution of the host symbiont [1-6]. Pathogens of the gastrointestinal tract of *Campylobacter spp.* and *Helicobacter spp.* They are the main causes of acute gastroenteritis and gastric diseases. It is currently believed that *Campylobacter spp.* It is the main bacterial cause of gastroenteritis in humans. Lack of resources leads to the spread of *Campylobacter* infection in young children, which leads to stunted growth and lifelong physical and cognitive disabilities. 1 in 100 people a year develops *Campylobacter*-related disease in high-resource regions. It is possible that *Helicobacter spp.* colonizes the human stomach, which increases the likelihood of developing ulcers and stomach cancer [7, 8, 9]. The most common type is *Helicobacter pylori* and some studies show that up to half of the population is infected. A large number of factors of survival and virulence of both *Helicobacter spp.* and *Campylobacter spp.* allows them to successfully survive and persist. Food contamination contributes to the spread of microorganisms *Campylobacter spp.* and *Helicobacter spp.* to a person [10, 11, 12].

The main purpose of the presented manuscript is a brief analysis of the similarities and differences between *Helicobacter pylori* and *Campylobacter jejuni* infections that are considered relevant today and cause very serious diseases, as well as measures to combat them.

Helicobacter pylori* and *Campylobacter jejuni. Formerly known as *Epsilonproteobacteria*, a group of metabolically and physiologically diverse prokaryotes, today called the *Campylobacterota* type, includes many aptogenic agents. They, in turn, include pathogenic and commensal heterotrophic genera such as *Helicobacter, Campylobacter*, *Wolinella* and *Arcobacter*, as well as free-living populations of sulfide ecosystems, which are considered important primary producers [13, 14, 15]. Among the pathogenic representatives of *Campylobacterota,* some species are etiological agents of the corresponding infections and serious concomitant diseases in humans. *Campylobacter jejuni* is the leading cause of bacterial foodborne gastroenteritis worldwide; it is responsible for 80-90 percent of all diagnosed cases of campylobacteriosis. The bacterium is a component of the commensal microbiota, which is present in many birds, other wild animals and domestic animals. Humans are infected mainly when they eat infected meat and other foods. Very young children, the elderly, and people with weakened immune systems may experience serious complications and even death, although infection usually causes only mild and self-canceling bloody diarrhea [16, 17, 18]. More than half of the world's people have *Helicobacter pylori* in their stomach, which leads to prolonged inflammation of the gastric epithelium. In some cases, this can lead to atrophic gastritis, peptic ulcer disease, gastric adenocarcinoma and lymphoma associated with the mucous membrane. This microorganism is the only bacterial pathogen belonging to class I carcinogens, and it is associated with 90% of cases of non-cardiac gastric cancer worldwide [19, 20, 21].

Location, similarities and differences of Helicobacter pylori and Campylobacter jejuni. These are gram-negative microaerophilic bacteria that are pathogens of the human gastrointestinal tract. *Helicobacter pylori* inhabits the gastroduodenal region, and *Campylobacter jejuni* inhabits the intestinal mucosa. Endotoxin molecules of both bacterial species contribute to their pathogenesis and autoimmune effects in different ways. On the other hand, *H. pylori* causes chronic gastric infection, which leads to gastritis, peptic ulcers and finally stomach cancer. Based on insufficient phosphorylation and acylation of the lipid component A, which interacts with immune receptors, *H. pylori* endotoxin has significantly lower endotoxic and immunoactivity compared with enterobacteriaceae endotoxin [22, 23, 24]. If the immune response to endotoxins is reduced, this can lead to the prolongation of *H. pylori* infection and, consequently, to the continuation of infection.

Table 1. Similarities and differences between *Helicobacter pylori* and *Campylobacter jejuni.*

№	Similarities and differences	Types of bacteria	
		Helicobacter pylori	***Campylobacter jejuni***
1.	**Genus**	**gram-negative bacteria**	**gram-negative bacteria**
2.	**Pathogenicity**	**human gastrointestinal pathogen**	**human gastrointestinal pathogen**
3.	**Colonization**	**the gastroduodenal compartment**	**the intestinal mucosa**
4.	**Diseases**	**gastritis, peptic ulcers and eventually gastric cancer while**	**diarrhoeal disease,**
5.	**Wand shape**	**helical shaped rods and have multiple sheathed flagella**	**curved and have a single polar flagellum**

- similarities - differences

On the other hand, this contrasts with acute infection causing *C. jejuni,* where obvious inflammation leads to pathology and diarrhea, and the endotoxin of this infection is endotoxically and

immunologically active. In addition, both *H. pylori* and *C. jejuni* exhibit molecular mimicry in the saccharide components of their endotoxins, which can cause autoreactive antibodies. *H. pylori* exhibits mimicry against Lewis antigens and some ABO blood groups, and *C. jejuni* exhibits mimicry against gangliosides. The former contributed to the development of inflammation and atrophy of the stomach, which is a precursor to stomach cancer, and the latter plays an important role in the occurrence of the neurological disorder Guillain-Barre syndrome. Both diseases cause serious autoimmunity problems [25, 26, 27].

Epidemiology. Up to 14 percent of diarrhea patients who seek medical care are infected with *C. jejuni,* which is the leading cause of diarrhea in developed countries. Continuous asymptomatic carriage is rare. Diseases are highest in children under 1 year of age and gradually decrease during childhood. Young people show the second peak. *C. jejuni* enteritis causes discharge in summer, as in other intestinal pathogens. On the contrary, up to 40 percent of healthy children in developing countries may be carriers of this microorganism at any time [28, 29, 30]. Infection *H. pylori* is more common among the population of developing areas than among the population of more developed countries. In addition, infections are more common among people living in places with poor sanitation, which may indicate that fecal-oral transmission occurs there. Seropositive infection persists for many years and possibly for a lifetime. In developed countries, the annual incidence is approximately 1% of the adult population. Sometimes infections can be transmitted from person to person through infected endoscopes. Other gastric organisms such as *Helicobacter* have been found in many animals such as rodents, primates, pigs and ferrets, but with the exception of primates and possibly cats, isolates are very different from human ones. Human contact with animals is not frequent enough to explain the widespread spread of *H. pylori* infection among humans [31, 32, 33].

Diagnosis. It is difficult to distinguish enteritis caused by *Campylobacter* from enteritis caused by other pathogens. The presence of neutrophils or blood in the feces of patients with acute diarrheal diseases is an important sign of *Campylobacter* infection. A presumptive diagnosis can be achieved by detecting noticeable mobility in a fresh stool sample using dark-field or phase-contrast microscopy or certain forms of vibrions detected after Gram staining. The microorganism can be extracted from a culture of faeces or, less often, blood to confirm the diagnosis. Due to the fact that in order to isolate *C. jejuni*, it must grow in a microaerobic atmosphere, special laboratory methods are required. In order to prevent the spread of competing microorganisms in the fecal flora, seeding methods must be selective. Once upon a time, media containing antibiotics were used to isolate *C. jejuni*, to which *C. jejuni* is resistant, but to which most common flora are susceptible. However, due to their mobility and small diameter, *Campylobacter* organisms have been isolated by filtration methods that do not use an antibiotic-containing medium. The use of filters in combination with a non-selective medium improves the yields of fecal culture of both *C. jejuni* and atypical intestinal *Campylobacter*. Polymerase chain reaction (PCR)-based methods have been developed for rapid detection, culture confirmation and typing of *C. jejuni* strains.When using selective methods, suspicious colonies can be easily identified by their distribution pattern, mucous appearance and grayish color. A series of biochemical reactions can differentiate *Campylobacter* species. Serological diagnostic methods are currently only research tools. A non-radioactive gene probe is available for the rapid identification of *C. jejuni* and *C. coli* from isolated colonies [11, 14, 18, 19, 21].

H. pylori can be determined using phase contrast microscopy based on the characteristic mobility of microorganisms, as well as by staining histological sections of a gastric biopsy with Gram-silver Worthin-Starry, Giemsa dyes or acridine orange. *H. pylori* can also be isolated from gastric tissue stained with hematoxylin and eosin. The presence of preformed urease can also help in the diagnosis of *H. pylori* in gastric biopsies. In addition, DNA probe and PCR methods have been developed. All of the above studies require endoscopy and biopsy. A urease breath test is a non—invasive way to detect *H. pylori* infection. Serum antibodies to *H. pylori* antigens can also help in the accurate diagnosis of infection. Methods such as these may be more sensitive than diagnostic methods involving biopsy. These non-invasive methods will greatly facilitate diagnosis in specific patients, help in the study of

the epidemiology of infection and help assess the effectiveness of antimicrobial therapy. Currently, there is a wide range of kits available for sale [12,13, 18, 21, 22, 23].

Measures to combat Helicobacter pylori and Campylobacter jejuni. Modern methods of antibiotic therapy against most clinically significant pathogens, such as *H. pylori* and *C. jejuni*, have actually declined due to the rapid emergence and spread of multidrug-resistant strains worldwide. The main obstacle in the research and development of new effective antibiotics is the identification of new molecular targets that can overcome the existing circulating resistance. The use of basic two-component regulatory systems as targets for new antimicrobial drugs and antiviral therapies against the main pathogenic Campylobacterota species may lead to new personalized combined therapies that will help to cope with the growing problem of multidrug resistance. Targeting proteins associated with two-component regulatory systems that are unique to this group of microorganisms will allow the creation of next-generation antibiotics that have highly selective antimicrobial action and have a low risk of dysbiosis [34, 35, 36, 37, 38].

Discussion. The human-pathogenic bacteria *Campylobacter jejuni* and *Helicobacter pylori* are part of *Epsilon proteobacteria*, which consists almost entirely of species with curved or spiral shapes. Although several rod-shaped forms of *Campylobacter* have been described and the rod-shaped forms of *C. jejuni* and and *H. pylori* have been isolated, the curved or spiral shape is a typical form of *C. jejuni*. The spiral shape is *C. jejuni* and *H. pylori* are necessary for colonization of bacteria during infection, for penetration through the mucous membrane of the gastrointestinal tract and for penetration into host cells using corkscrew-like mobility [1, 5, 6, 12, 18, 20]. Mobility of *C. jejuni* and *H. pylori* are crucial for host colonization and pathogenesis; strains that are immobile suffer greatly from the inability to colonize the intestines of the host. Thus, interfering with the lifestyle and virulence of bacteria can be beneficial by inhibiting the proteins responsible for the spiral shape of cells. Infection with *C. jejuni* and *H. pylori* are considered the most common cause of bacterial diarrheal diseases worldwide, causing serious complications such as inflammatory bowel disease, reactive arthritis and Guillain-Barre syndrome [21,22,23,27,28].

In the future, it is reasonable to assume that host intestinal inflammation and antibacterial resistance can be weakened by a combination of anti-inflammatory and antimicrobial drugs taken from traditional and conventional medicine. The innovative discoveries presented here support preventive agronomists and clinical studies aimed at improving the treatment and prevention of campylobacteriosis in humans [32,35,36,37,38,39].

Conclusions. Typical pathogens of the human gastrointestinal tract are Gram-negative microaerophilic bacteria *Helicobacter pylori* and *Campylobacter jejuni*. The identification of each enterotype on both farms suggests that these groupings were not random, although differences between farms may have contributed to differences in the prevalence of certain enterotypes. Various types of *Campylobacter* and *Helicobacter* have been found within these enterotypes. This may indicate microbial taxa that may increase the likelihood of colonization by these pathogens.

The control of *Campylobacter* enteritis largely depends on the fact that the microorganism does not spread from agricultural and domestic animals, animal products or contaminated water. Properly cooking and storing meat and dairy products, avoiding contaminated drinking water and unpasteurized milk, and washing hands after contact with animals or animal products are all methods that can help people avoid infection with *Campylobacter.*

The main method of treating *H. pylori* infection was antimicrobial therapy. Currently, antimicrobial therapy is one of the main methods of treating duodenal ulcers. Research is currently underway to determine the most effective combinations of antibiotics. However, data on the effectiveness of antimicrobial therapy for most cases of non-ulcerative dyspepsia caused by *H. pylori* remain unclear.

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