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Review

Campylobacter as a cause of gastroenteritis in humans and animals

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Campylobacter is a zoonotic microorganism in human and animal population which causes gastroenteritis; it is widespread in nature. Under the present systematics, Campylobacter (Campylobacteriaceae) family consist of 16 species and 6 subspecies. Illness can occur in various animals. It causes dysentery in swine, abortion and infertility in cows, abortions in sheep and goats, avian hepatitis in poultry, and enteritis in many species including primates and pets. Campylobacter do not produce toxins in food and it is known that *Campylobacter jejuni* produce endotoxin (unstable at higher temperature), enterotoxin and citotoxin. Humans can be infected if they have a direct contact with animals and contaminated food. Campylobacteriosis in humans caused by *C. jejuni* is also known as *Campylobacter enteritis*. Infective dose of less than 500 cells can cause infection.

Key words: *Campylobacter*, gastroenteritis, humans, animals, food.

INTRODUCTION

Campylobacter helveticus

Campylobacter, especially *Campylobacter jejuni*, is a major cause of gastroenteritis worldwide in humans and animals (Anonymous, 2003; Ivanović, 2008a). This microorganism was first described by McFadeyan and Stockman in England (1913), marking it as the cause of abortion in sheep. Several years later in the United States, it was found that this microorganism causes abortion in cows. Beside this, some other micro-organisms are isolated from aborted fetus named *Vibrio fetus*. In 1962, King brought in epidemiological connection presence of *Vibrio jejuni* and *Vibrio coli* in chicks with human gastrointestinal infection. In 1963, Sebald and Veron separated these microaerophylic microorganisms as a new race called *Campylobacter*.

Division of *Campylobacter*

Under the systematics given by Garrity (2005), Campylobacter family consists of 16 species and 6 subspecies. The author gave the following overview of *Campylobacter* species.

Campylobacter fetus

C. fetus has two subspecies: *C. fetus* subsp. *fetus* and *C. fetus* subsp. venerealis. *C. fetus* subsp. *fetus* can be found in cattle and sheep intestinal area and can affect aborts in both animals. In humans, it can cause diarrhea, aborts, bacteriemia, endocarditis and meningitis. *C. fetus* subsp. venerealis is widespread in nature. It was isolated from human blood. It cannot survive in cattle intestinal tract, but can be adapted in genital tract of cattle, sheep, contaminate the sperm, and after insemination, it causes females' infertility.

Campylobacter coli

C. coli were isolated from pigs that had gotten dysentery, and from cattle, poultry, ostriches and dogs. In humans, *C. coli* are the second cause of gastrointestinal illness. Although it has the closest genetic relationship with *C. jejuni*, it causes less severe symptoms of *C. jejuni* from feces of pigs that had suffered diarrhea; another type of *Campylobacter* was isolated, which is similar to *C. jejuni* and *C. coli* but there are genetic differences between these two species. Marked as *Campylobacter hyoilei*, but has not found a place in the taxonomy. It was assumed that *C. hyoilei* could be *C. coli* subsp. hyoilei (Duim et al., 2001).

Campylobacter concisus

C. concisus was first isolated from the mouth of humans during peridontal disease. Animals do not represent a reservoir for this species. The role of *C. concisus* in peridontal disease is not yet clear, because the microorganism can be found in healthy and sick persons. The ability of *C. concisus* to cause gastrointestinal illness is slight, although these bacteria can be isolated from feces of diarrhea sick people and from the feces of healthy people. Cytotoxic activity of *C. concisus* has not yet been described, although the cytotoxic effect on CHO cells was confirmed (Macuch and Tanner, 2000).

Campylobacter curvus

C. curvus is also located in the mouth of people. It does not penetrate into cells and does not cause gingival gum disease. It is sometimes isolated from diarrhea, but does not produce significant clinical symptoms (Lindblom et al., 1995).

Campylobacter gracilis

C. gracilis occurs in the presence of peridontal disease, although its role is not completely clarified. It can occur during pleuropneumonia (Tanner et al., 1981).

Campylobacter helveticu

C. helveticu belongs to the group that was isolated from feces of healthy cats and dogs (Stanley et al., 1992), and those who had diarrhoea. It commonly occurs in cats than in dogs. It does not cause diarrhoea in humans.

Campylobacter hominis

C. hominis was first isolated from the feces of humans. The basis of 16S rRNA gene sequences was determined, because it was not isolated on plates. It may be significant for people with diarrhoea (Lawson et al., 2001).

Campylobacter hyointestinalis

C. hyointestinalis also has two subspecies: *C. hyointestinalis* subsp. hyointestinalis and *C. hyointestinalis* subsp. lawsonii. *C. hyointestinalis* subsp. hyointestinalis was isolated from diarrheal of the feces of pig (Hänninen et al., 2002). It can also occur in healthy pigs, cattle, monkeys, hamsters, reindeer and elephants (Gebhart et al., 1985). In humans it occurs periodically. *C. hyointestinalis* subsp. lawsonia was isolated for the first time along with *C. hyointestinalis* subsp. hyointestinalis in the intestinal tract of pigs (Gebhart et al., 1983). Their roles were not fully determined.

Campylobacter jejuni

C. jejuni also contains two subspecies: *C. jejuni* subsp. jejuni and *C. jejuni* subsp. doylei. The first was isolated from intestinal contents of cattle. In 1970, it has been found to cause gastroenteritis in humans. In addition to gastritis, *jejuni* subsp. *jejuni* in humans can lead to septicemia and to neurological disorders. It is also a saprophyte in animals: chickens, cattle, pigs, sheep, dogs and ostriches (Dingle et al., 2001). *Jejuni* subsp. *doylei* is significantly different from subsp. *jejuni* in the way of transmission, distribution and characteristics. There is no evidence that animals are hosts. It was isolated from people who had enteritis, gastritis, and septicemia (Lastovica and Skirrow, 2000).

Campylobacter lanienae

C. lanienae was isolated from workers who have worked on farms but did not show any symptoms. These organisms were subsequently isolated from cattle's and pigs. Finding the description of this is controversial and still show that its pathogenicity is yet to be clear (Inglis and Kalischuk, 2003).

Campylobacter lari

C. lari was first isolated from the seagulls and differed ecologically, genetically and phenotypically similar to species of *C. jejuni* and *C. coli*. It was isolated from the digestive tract of wild birds, poultry, beef, and shellfish and from wastewaters (Lastovica and Skirrow, 2000).

Campylobacter mucosalis

C. mucosalis colonized the intestines of pigs. It causes proliferative enteritis and is important for the development of disease. Its occurrence in other animals and humans is yet to be found (Lawson and Gebhart, 2000).

Campylobacter rectus

C. rectus usually occurs as a periodontal pathogen in humans (Macuch and Tanner, 2000).

Campylobacter showae

C. showae was isolated from human gingival wounds; it was described as a primary pathogen in periodontal disease of humans. However, as many species occur in this disease, this is only an hypothesis that causes disease (Macuch and Tanner, 2000).

Campylobacter sputorum

C. sputorum appears as saprophyte in feces of cattle; it is unclear whether it causes diarrhea and abscesses in humans (On et al., 1998a).

Campylobacter upsaliensis

C. upsaliensis was first isolated from the feces of dogs. It was Isolated in sporadic cases of gastroenteritis in humans; it naturally inhabits the digestive tract of pets, dogs and cats when it may cause diarrhea (Bourke et al., 1998).

Bacteroides urealyticus

B. urealyticus species is currently being tested and based on some characteristics, which could be taxonomic ranks of *Campylobacter* species. The species that were tested by their biochemical and genetic characteristics becomes *Campylobacter* species, such as Lawsonia, Arcobacter, Helicobacter, and Sulfurospirillum Sutterella (Vandamme et al., 1995).

CHARACTERISTICS OF *CAMPYLOBACTER*

Morphological characteristics

Bacteria of the genus of Campylobacter, according to their morphological characteristics are described as slender, spiral winding sticks, with one or two coils, 0.2 to 0.5 µm wide and 0.5 to 5 µm long, although the cell up to 8 µm in length can occur. Sticks genus *Campylobacter* can take the form of commas, letter S or seagull's wings. They are Gram negative; they do not form spores and may have a spherical shape of the body in a stationary phase culture. Oxidase and catalase are positive, and has many types of reduced nitrate. *Campylobacter* species are highly mobile and their motion is as a corkscrew (Ivanović, 2008a).

One or more flagella located at both ends of the cell enabled them to move, and consist of a fiber diameter of 20 µm, hook, basal body, as well as a large disk that is associated with the end part where there is a hook, and who turns the basal body. Mobility next to movement is necessary for their penetration into intestinal mucosa cells. The surface structure of bacteria allows the pathogen to successfully avoid and overcome the host defense mechanism. Components of bacterial cell surface are proteins outside the cell membrane (OMP), lipopolysaccharide (LPS), glycocalics, as well as product like toxins that cross the surface. They are the ones that cause damage to the host (Ivanovic, 2008a). *C. fetus* produces a high M-protein antigen, which is able to take the form of the surface layer and, in fact, to make the microcapsule. This layer is antifagocitic, and its role may be important in the pathogenesis of *C. fetus* infection. The flagella occur because of its protein nature and represent an important surface antigen of *C. jejuni*, but in other *Campylobacter* species, flagella present adherence factors.

Growth conditions

Optimal growth temperature was at 42℃, although it can grow at lower and higher temperatures than optimal (30.5 to 45℃). Campylobacter grows best at the pH value from 6.5 to 7.5. It usually requires a reduction of oxygen level - optimal growth in the presence of 3 to 5% oxygen and 2 to 10% of carbon-dioxide. Optimal water activity for growth is aw = 0.997 (0.5% NaCl) (NACMCF, 1995).

Survival

Campylobacter had a better survival in foods at refrigeration temperature than at room temperature. Thus, the survival time is up to 15 times longer at 2℃ than at 20℃. *Campylobacter* can survive up to one hour on hands and in wet surfaces. At below zero temperatures, after an initial decrease, the number slowly decreases. Therefore, freezing do not immediately inactivate bacteria in food. It survives in a modified environment and vacuum packaging. Usually, it survives less in the atmosphere where the oxygen concentration is high (NACMCF, 1995).

Inactivation

It is inactivated when heated at 55℃ and above this temperature. Inactivation in foods in which the pH<4.9 and pH>9.0 especially when the temperature is above the cooling temperature. It is believed that Campylobacter is sensitive to drying. It is sensitive to NaCl concentrations higher than 1%, and death comes slowly at 2% (D time is 5 to 10 h). Ascorbic acid and several spices can inhibit there. It is sensitive to gamma irradiation. It is estimated that, if exposed to 2 kGy, there is a reduction of 6 D, and 2.5 kGy - to 10 D reduction. 2 to 3 kGy doses is sufficient to carry out decontamination of meat (NACMCF, 1995). Bacteriostatic effect of some spices and additives is possible for *C. jejuni* (Ivanovic et al., 2007).

Pathogenesis

The pathogenesis of Campylobacter infection is very complex and consists of various factors. Virulence factors are adhesion, invasive ability and production of toxins. Adhesion to the surface of epithelial cells is probably the deciding factor for colonization, but the number of bacteria can increase the local concentration and secretion of bacterial products, which results in citopatogenius effect. It is believed that this is the primary mechanism for mucosal damage of colon and causes inflammation (Deepika et al., 2008). *Campylobacter* species invade the gastrointestinal tract and damaged epithelial cells are shed into the lumen. Invaded cells become swollen and surrounded, and changes that they showed in the regulation of ion transport are likely due to the produced cytotoxin and its impact.

Formation of toxins

The Campylobacter toxins are not produced in food. It is well known that *C. jejuni* produces endotoxin unstable at higher temperatures, enterotoxin and cytotoxin (Wassenaar, 1997). Enterotoxin is a large protein and unstable at higher temperatures and becomes completely inactive at 56℃ for one hour or within 10 min at 96℃. A lower pH value partially inactivated it and Toxic activity of crude toxins decreases during storage at 4℃ in a month, or within one week at -20℃. Enterotoxin induces adenilate-cyclase activity in the intestinal mucosa and disturbs normal ion transfer in enterocites, which results to diarrhea (Wassenaar, 1997).

C. jejuni secreted cytotoxin (CDT), which acts in or through the pores of cells. Mechanisms of action of toxicity appears in two prevailing ways. The first way would be the inhibition of cell protein and others - inhibition of actinic filaments of muscle cells. It has a toxic effect on a number of mammalian cells. It is thermolabile and destroyed at temperatures of 100℃ for 30 min, however, it is stable at 60℃ for 30 min (AbuOun et al., 2005). It is trypsin sensitive. Production of CTD toxins determinated three genes: cdtA, cdtB and cdtC. These genes are responsible for its activity. For now, it is known that cdtB encode activity (toxic components of toxin), while cdtA and cdtC are responsible for action or entry into the host cell. The role of CDT in human campylobacteriosis is unclear (AbuOun et al., 2005).

METHODS AND MEDIA FOR DETECTING *CAMPYLOBACTER*

For testing the presence of *Campylolobacter* in foodstuffs, feed and water, methods ISO 10272-1:2006, ISO/TS 10272-2:2006 and ISO 17995:2005 were applied respectively. However, none of these ISO methods can be applicable for the isolation of *Campylobacter* from live animal samples. Above these methods, there are a variety of molecular methods (PCR) used for their detection, although enzymatic-immunological tests apply only in testing samples of human feces.

Media

The selected media are used in ISO standardized methods, but the number of microorganisms may be very small, media for the enrichment of the substrate depending on the sample can be selective, are necessary. To isolate *Campylobacter* from fecal or intestinal/caecal samples preconcentration is not necessary (Martin et al., 2002).

Incubation

The microaerophilic atmosphere (5% O2, 10% CO2 and 85% N) is necessary. Incubation temperature is 42℃, because it is optimal for *C. jejuni/coli* growth. It usually grows on a solid media in 24 to 48 h at 42℃ (Vandamme, 2000).

Confirmation

The identification of colonies on solid media is based on the color and their appearance. In the media from Skirrow or other media that contain blood, characteristic of *Campylobacter* colonies are pink, round, convex, smooth and shiny with regular edges. On media that contain carbon, characteristic colonies were grayish, flat and wet, with a tendency to spread, and can be metallic. Microscopic examination of the morphology and motility show us typical spiral or curved thin sticks S form, which is moving in a spiral form. Older cultures show less mobility and have cocoidal forms. For the confirmation, commercial tests are also used.

EPIZOOTIOLOGY

Poultry

Poultry is the primary host, and thus the reservoir of termophilic *Campylobacter* species. In poultry, it is often colonized gut with *C. jejuni* (65 to 95%), rarely with *C. coli* and other Campylobacter species (Newell and Wagenaar, 2000). *Campylobacter* is usually not present in the intestinal tract of poultry younger than two weeks. When a colonization occurs, it quickly spread the farm through coprophagi, so that up to 100% of poultry in the flock could be infected within 72 h (Ivanovic et al., 2004). Campylobacter can be isolated from fresh feces, ceckal content or cloacal swab. Depending on the method of cultivation, the percentage of findings of the pathogen ranged from 83.33% in chickens from the farm to 89.65% in chickens from rural households (Ivanovic et al., 2004). Processing of poultry carcass technology, mechanical or manual evisceration, plucking feathers (Ivanovic, 2003a) and cooling in speenchilleru allow easy transfer of jejuni/coli from the contents of the digestive tract or from the feathers to surface of the hulls (Ivanovic et al., 2008b). Contamination of poultry meat with jejuni can contribute to the cooking utensils and hands of workers who carry out food preparation.

Pigs

Unlike birds, where is predominant jejuni (Ivanovic et al., 2007a), in swine there is predominant coli. In pigs, these pathogens causes disease that is characterized by diarrhea, or the appearance of mucus and/or blood in the faeces. The main place where the infections are naturally small intestine, the intestinal villi at the beginning of the ileum, where there is infiltration of the lamina propria with polymorphonuclear cells and monocytes. In pathoanatomical findings in animals, where coli was isolated, major changes were found in the small intestine and their content. Intestinal mucosa was inflammatory through entire length and filled with blood. Intestinal villi were stunted in jejunum and ileum. Histological examination reduced intestinal villi and crypts were found, which contained inflammatory cells, infiltrated lamina propria with polymorphonuclear leukocytes and eosinophils. Lymphoid follicules are prominated in the submucosis.

The primary contamination of pig skin with jejuni/coli occurs more in pens due to rolling pigs in a crate or as a result of contact with animal feces, which is the holder of a large number of saprophytic and pathogenic microorganisms (Ivanovic et al., 2007a). There is a possibility that due to the stress that occurs in animals during transport, stunning and slaughter may reach the bacteremia by portal bloodstream, or this microorganism is found in the liver. If this happens, then it is very possible that the pathogen enters the bile ducts, and through them in the bile. Ivanovic et al. (2007b), in their testing, they isolated coli from the cecum (49.51% of samples), the peritoneum (20.38% of samples) and from bile (31.06% of samples). In production of pork, especially on the slaughter line, there are great opportunities for contamination of carcasses by this pathogen (Ivanovic et al., 2007c).

Cattle, sheep and goats

Intestinal tract of sheep and goats mainly colonize *C. jejuni, C. coli, C. hyointestinalis* and *C. fetus*. These types are labeled as the cause of abortion in sheep and goats. It is assumed that infection occurs orally, perhaps even before the occurrence of pregnancy. Later in pregnancy, these agents are transmitted through the placenta to the fetal digestive tract. The sheep and goats - a mother, and lambs and kids, in addition, general bacteremia are excreted with the feces of mucoidal content and traces of blood. In the intestinal tract of lambs and kids, there was a decrease in epithelial cells in the intestinal wall with the appearance of large amounts of mucous fluid (Ivanovic et al., 2008).

The intestines appeared as necrotic intestinal villi in places that were filled with polymorphonuclear cells. In some cells the mitotic division was noticed. In young animals, the number of microorganisms is higher than in the elderly. In older animals, microorganisms are occasionally detected in the feces, probably due to the small number, because there were occasional cause of diarrhea which was knocked out (Goelz, 2003). The same author pointed out that people who assist in lambing are infected via the placenta during birth or during rehabilitation retention of abortion. In addition, he stated that the infection is common in people who consume insufficiently cooked or roasted lamb and with shepherds who are not protected when abortion in ewes and removing the infected fetus.

Ivanovic et al. (2004b, 2005), found the presence of the pathogen in the liver (69.23%) and the mucosa of the small intestine (76.92%). In 2008, the same authors (Ivanovic et al., 2008), have found the presence of C. jejuni/coli in clinically healthy lambs (40% of the small intestine, liver 25%) and goats (33.68% small bowel, liver 23%). jejuni/coli can also be isolated from feces of infected of healthy cows (Wesley et al., 2000). In natural caused infections, large areas of ileum was histologically changed, including the intestinal villi. Abscesses are formed on the crypts of intestinal villi and leads to inflammation of the lamina propria with hyperplasia of submucosic lymphoid tissue. jejuni/coli in percentage is more present in cattle younger than one year, than in cows aged two and over. The authors also pointed out that raw milk may be a carrier of Campylobacter to humans. It is also important to point out that this microorganism can be transmitted from the udder of one cow to another using machine milking.

Cats and dogs

Jejuni in cats and dogs induces diarrhea, but in spite of that some healthy dogs are noticed. These animals are spotted as significant source for humans. In dogs beside jejuni, upsaliensis was also isolated. Cats younger than six months have some blood in their stools, but it could pass without it. Infectious in cats can pass unnoticed, (asimptomatic) (Hackett and Lappin, 2003).

Other species of animals

It is described that *Campylobacter* can cause gastrointestinal disease in skunks, canadian martens, primates - monkeys, hamsters, rats and guinea-pig. However, in these animals, intensity and clinical signs of disease was different. The most common infections passed on their own, especially where clinical symptoms are milder. Skunks are among the small number of animals that developed illness with diarrhea, like in humans. In primates, Campylobacter was isolated in healthy and in animals that had diarrhea (Dyer and Stoltenow, 2000).

EPIDEMIOLOGY

In humans jejuni also produce campylobacteriosis which is known as *Campylobacter enteritis*. Infectious dose of *Campylobacter* can be less than 500 cells (Hocking, 2003). Simptoms which cause Campylobacter infection are shown between first and eleventh day (oftenly between second and fifth day) after intoxication. It usually starts with pain in the muscles, headache and fever. Diarrhea often occurs following blood and mucus in stool. Signs of nausea and vomiting are rarely seen. The symptoms may take between 1 to 7, usually five days. Infection is usually self limiting. C. enteritis affect small children (younger then 5 years old) and young population; death is very rare. Campylobacter complications are rare, like artritis – Reiter's syndrome. Very strong abdominal pain could be present, and usually mistreated as appendicitis. 1% of infected can occur in reactive artritis, and 0.1% Guillain-Barré syndrom (Hocking, 2003). This syndrom can occur between 1 to 3 weeks after illness (Hocking, 2003). Nearly 15% of people who have got Guillain-Barré syndrom incure, 3 to 8% dyed, and the rest of them suffers from some level of invalidity. Bacteriosis may also occur, especially in eldely people (Curtis, 2007), and in immuno compromised persons, particularly HIV-infected. Neonatal sepsis caused by C. jejuni in babies between 2 to 3 weeks old may occur (Wolfs et al., 2001).

Campylobacter is usually transmitted to humans in direct contact with animals and through food. People who are in direct contact with animals, veterinarians who assist in partus or removing aborted fetus and the placenta, are particularly at risk. It was pointed out in many studies that dogs and cats can be a source of this bacteria, especially in children Ivanović (2008a).

Laboratory animals may be a reservoir of *C. jejuni* and through them people who perform experiments can be infected. Foods are important factors in the transmission of this organism to humans. *Campylobacter* is commonly found in fresh chicken meat and its products. The presence of this organism was found in other types of meat such as beef, mutton, pork, but also in offal of these species. *Campylobacter* was isolated from fresh milk, fish, seafood and salads (Curtis, 2007).

In addition to food, this microorganism may be found in drinking water. In rural areas where surface water is used for drinking, is more likely to occur in *Campylobacter enteritis*. There is a possibility that people, especially young people became infected during swimming in river backwaters where the birds have their nests (Kuusi et al., 2004).

Sexual transmission from man to woman is not described. It is also not described that *C. jejuni* causes vaginal infection. However, in gay population, there was a transfer from man to man. In infected men, nine days after the infection occurred, blood was seen in the diarrheal stool, and pain and drainage of pus from the rectum were observed (Allos and Blaser, 1995).

Authors suggested that *C. jejuni* might lead to immunity in people who were already infected with this pathogen and that as a result had all associated clinical symptoms, as well as in those that were longer in touch with this zoonotic microorganism, but had no symptoms (Glass et al., 1994; Kagnoff and Eckmann, 2001).

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