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Campylobacter: An Emerging Pathogen*

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Abstract: During the past decade *Campylobacter* has been shown to be responsible for enteritis in human and animal. The natural habitats of most *Campylobacter* species are the intestines of birds and other warm-blooded animals. These organisms may enter the environment, including drinking water, through the feces of animals, birds or infected humans. *Campylobacter* survive in aqueous environment for several weeks at temperature around 4°C and may enter the human food chain at slaughter of the animals. Although, many methods and media have been developed for detection of the *Campylobacter* from various samples, universally accepted methods and media are not available yet. Milk, mushrooms, hamburger, pork, shellfish and eggs are vehicles of *Campylobacter* however, most *Campylobacter* enteritis acquired by the consumption and handling of poultry. The sensitivity of *Campylobacter* to heat, acidic pH, food preservatives and irradiation must be considered as plus points to prevent of *Campylobacter* transmission to human beings. On the other hand, the antibiotic resistant character of *Campylobacter* is a negative point for control of *Campylobacter* infection in developed and developing countries. *Campylobacter* is usually causes a self-limited illness, but in more severe cases of gastroenteritis, antibiotics are usually begun before culture results are known. Nowadays, antibiotics such as erythromycin and ciprofloxacin have been recommended for treatment of campylobacteriosis however, resistance to these antibiotics has been rising.

Key words: *Campylobacter*, sources, isolation, infections

BIOLOGY OF *CAMPYLOBACTER*

In the beginning of the 20th century *Vibrio fetus* was found to be responsible for spontaneous abortions in cattle and sheep (Allos 2001). This organism unlike *Vibrio* species didn't grow well under atmospheric oxygen tension and did not ferment sugars. In 1963 Sebald and Veron suggested the name *Campylobacter* for this organism to differentiate it from *Vibrio* species (Catteau, 1995). Campylobacters are microaerophilic, nonproteolytic, nonlipolytic and nonsaccharolytic, so they neither ferment nor oxidize carbohydrates. Hence, they obtain energy from oxidation of amino acids or tricarboxylic acids (Grau, 1991).

The genus *Campylobacter* contains 16 species and 6 subspecies (On, 2001), Many of these species have been implicated in human enteritis. But the most common cause of *Campylobacter* enteritis is 'thermophilic' campylobacters viz., *Camp. jejuni*, *Camp. coli*, *Camp. lari* and *Camp. upsaliensis*. The term 'thermophilic' for some species of *Campylobacter* is proposed not because of their ability to grow at high temperatures but to emphasize their inability to grow below 30°C. The

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optimum temperature and microaerophilic nature of thermophilic *Campylobacter* indicates that they arenot likely to be able grow outside of the mammalian gut. Thermophilic *Campylobacter* cannot grow in food or in water and therefore they should be considered 'food-borne' rather than 'food-poisoning' organisms (Stanley *et al*., 2003).

Camp. jejuni is the most commonly identified species (Nachamkin *et al*., 1992) and was formerly named *Camp. fetus* subsp. *jejuni*. It differs from the other species in that it is hippurate positive (Catteau, 1995). The other species of thermophilic *Campylobacter* such as *Camp. coli* and *Camp. lari* have also been associated with enteric disease in human (Barros-Velázequez *et al*., 1999) Nowadays *Camp. hyointestinalis* also identified as cause of enteric disease in cattle (Trachoo, 2003).

Gasteroenteritis caused by *Campylobacter* spp. has been recognized as one of the important public health problems in the developed countries (Chaveerach *et al*., 2002). *Camp. jejuni*, *Camp. coli* and *Camp. lari* are the most important pathogenic species of thermophilic campylobacters (Skirrow, 1994). Food is the most common vehicle for transmission of *Campylobacter* and some studies indicate that up to 70% of sporadic cases of campylobacteriosis were associated with eating chicken (Fang *et al*., 1991; Scott *et al*., 1998). Other identified food vehicles of campylobacters in developed countries include unpasteurized milk, undercooked meats, mushrooms, hamburger, cheese, pork, shellfish and eggs (Alterkruse *et al*., 1999). Several studies have revealed that 30-100% of poultry, 40% of cattle and 60-80% of swine carry campylobacters in their intestinal tract; hence, campylobacters are associated with foods of animal origin (Doyle, 1984). Household pets with diarrhea have often been shown to be the source of infection for man (Frost, 2001). Few hundred cells of *Campylobacter* can produce illness in babies; young children and debilitated people. Symptoms of the infection vary from mild (watery diaarea) to severe (bloody diarrhoea). Other symptoms are fever, nausea, abdominal cramps and (seldom) vomiting (Ketley, 1997).

SOURCES OF *CAMPYLOBACTER*

Most of the campylobacters grow at 37 and 42°C under microaerophilic conditions. These characters help pathogenic *Campylobacter* to grow in their natural habitat (intestine of warm-blooded birds and mammals). Poultry is the primary food vehicle for *Campylobacter*. Some studies indicate that up to 70% of sporadic cases of campylobacteriosis are associated with eating chicken. "Surveys by the USDA demonstrated that up to 88% of broiler chicken carcasses in the USA are contaminated with *Campylobacter*" (Anonymous, 1998). Inglis *et al*. (2004) reported that animal faeces are sources of a large number of *Camp. lanienae* and *Camp. jejuni* cells and suggested that *Camp. lanienae* may be pathogenic to cattle and that novel species of *Campylobacter* may occur within their GI tracts.

In general food, particularly the surface of meat could be contaminated with *Camp. jejuni* though contact with feces, but it should be noted that the *Camp. jejuni* do not multiply in food the temperature is lower than 30°C Although, *Campylobacter* survive at 4°C, these bacteria are sensitive to heat (Uradzinski *et al*., 1993). Therefore, pasteurization of milk and adequate cooking of meat will destroy this organism (Doyle, 1998).

SURVIVAL OF CAMPYLOBACTERS IN ENVIRONMENT

The morphology of *Campylobacter* spp. changes with age in culture media and varies from a spiral (predominant in young cultures) to coccoidal form (predominant in older cultures). The formation of these coccoidal forms may represent a physiological response of these bacteria to

environmental stress including heat. The natural habitat of the campylobacters has been identified as the gut of human and animals. Therefore, this form of the campylobacters may provide a protective means to survive under adverse environmental conditions outside the intestinal tract of the animals (Smibert, 1978). Konkel *et al*. (1998) have reported the thermal stress response in *Camp. jejuni* due to the presence of 24 heat-shock proteins. Moore and Madden (2000) stated that presence of the heat-shock proteins in *Camp. jejuni* enable them to survive sublethal thermal shock, such as mild thermal processes or pasteurization.

Despite the susceptibility of *Camp. jejuni* to atmospheric oxygen and its inability to grow at ambient temperature, this bacterium has been isolated from natural aquatic environments including river water, ground water, coastal water and lake water (Hanninen *et al*., 1998). Stelzer *et al*. (1988) stated that wastewater could be considered as a source of *Camp. jejuni*. Bolton *et al*. (1987) observed that the river waters contain a variety of *Campylobacter* spp., which could be considered as a potential source of *Camp. jejuni*. Buswell *et al*. (1998) explained that *Camp. jejuni* collected from various aquatic systems and at various temperatures (4, 10, 22 and 37°C) could not grow on conventional media. Kusters *et al*. (1997) and Federighi *et al*. (1998) have reported that the exposure of *Campylobacter* isolates to an aquatic environment resulted in loss of their culturability.

Pickert and Botzenhart (1985) tested the survival of *Camp. jejuni* in drinking water, river water and sewage and reported that *Camp. jejuni* could only survive for a few days. This study showed that the concentration of oxygen or nutrients in the water did not affect the survival of *Camp. jejuni*. Maximal survival of *Campylobacter* spp. has been reported at low temperatures for four months (Rollins *et al*., 1986).

ISOLATION TECHNIQUES AND MEDIA

Isolation of campylobacters was not successful until 1972. In 1972 Dekeyser *et al*. isolated *Campylobacter jejuni* from faeces by culturing filtrate on a blood-thioglycolate agar medium containing bacitracin, polymyxin B sulfate, novobiocin and actidione.

In 1977 Skirrow isolate *Campylobacter* from faeces on a selective medium, this medium was blood agar supplemented with trimethoprim, polymyxin B and vancomycin. Blaser *et al*. (1979) developed the Campy-BAP medium. Another selective medium is Campylosel (Biomerieux), which comprises cefoperazone vancomycin and amphotericin B as selective agents. Bolton *et al*. (1982) developed the Preston medium, useful to isolate *Campylobacter* spp. from environmental samples. The efficiency of this medium for the isolation of *Campylobacter* was checked on various samples such as faeces and water. The results obtained indicated that some strains of *Camp. coli* were sensitive to polymyxin B (Barros-Velázquez *et al*., 1999).

Since blood is an expensive component and its quality is variable, Bolton *et al*. (1984) replaced it with charcoal. These authors described the Charcoal Cefoperazone Deoxycholate Agar medium (CCDA). Walmsley *et al*. (1989) isolated *Camp. upsaliensis* on both media CSM (Charcol-based selective medium) and SKM (Skirrow medium) without preliminary enrichment or filtration steps. In addition, Aspinall *et al*. (1993) formulated another blood-free selective medium viz., CAT (Cefoperazone, Amphotericin B, Teichoplanin) for the isolation of *Campylobacter* at 37°C. This medium contained cefoperazone, amphotericin B and teichoplanin as selective agents. CAT medium selected *Camp. upsaliensis* better than the CCDA medium (Byrne *et al*., 2001).

A most probable number method was developed for the enumeration of *Campylobcters* from environment samples based on Preston enrichment broth by Bolton *et al*. (1982). It was stated, "It is sometimes advisable to use an enrichment step for the isolation of *Campylobacter* spp. The enrichment

step may increase the isolation rate of campylobacters, especially when the microbial load in the sample is low". Tran (1998) devised a new blood-free enrichment broth (BFEB), which enabled *Camp. jejuni* strains to be isolated under aerobic conditions.

At first membrane-filtration techniques were designed for the isolation of *Vibrio fetus* (*Camp. fetus*) from cattle and later from human beings. Unlike many bacteria, campylobacters usually pass through 0.45 μm pore size filters. To perform this technique, 10% suspension of faeces was passed through the membrane-filter and the filtrate inoculated onto the plate containing selective medium (Barros-Velázquez *et al*., 1999).

Ribeiro *et al.* (1984) reported that the filters in Preston enrichment medium increased the isolation rate of *Campylobacter*. Another method that recommended by them was centrifugation of sample and further seeding of the bacterial sediment in selective medium.

In general, all conventional selective media for isolation of *Campylobacter* spp. incorporate different antibiotics as selective agents in order to achieve maximum isolation rates. However, unfortunately some strains of *Campylobacter* are sensitive to some of the antibiotics in the media (Corry *et al*., 1995). Thus for isolation of these strains a membrane filter method, which relies on the ability of *Campylobacter* and other bacteria, e.g., *Helicobacter,* to pass through a 0.45 or 0.65 μm pore size membrane filter, was recommended. Non-selective media may then be inoculated with the filtrate. However, variation in the different brands of membrane filters may lead to inconsistencies. In the results it has been considered that only 10% of these organisms to pass through the filter (Steele *et al*., 1984). It should also be noted that the membrane filter method depends on the ability of the motile bacteria small enough to pass through the pore of filter onto the non-selective growth medium, while some species with cell bodies too large or with specific growth requirements would not be isolated by this method (Engberg *et al*., 2000). On the other hand, Kulkarni *et al.* (2002) expressed that the combination of the current methods (filter technique and conventional technique) would not detect the unusual species of campylobacters.

Recently Baserisalehi *et al.* (2004a) recommended Kapadnis-Baseri device (KB device) and sample processing-Kapadnis Baseri medium (preT-KB method) (Baserisalehi *et al*., 2004b) for isolation of *Campylobacter* spp. from environmental samples without using antibiotics. The KB device was designed based on motility and activity of campylobactes at low temperature and enables to isolate and enumerate *Campylobacter* spp. from the environmental samples. The preT-KB method was recommended for selective isolation of *Campylobacter* spp. from environmental samples based on elimination of competing bacteria at the sample and culture levels. The preT minimizes most of the competing bacteria at the sample level and the KB medium without blood and antibiotic is selective and differential eliminate rest of the competing bacteria and differentiate *Campylobacter* from the other Gram negative bacteria at the culture level.

Incubation Conditions

The thermotolerant campylobacters viz., *Camp. jejuni, Camp. coli, Camp. lari* and *Camp. upsaliensis* grow well at 42-43°C. Although the optimum temperature for the growth thermophilic *Campylobacter* is 42-43°C, they can grow at 37°C but not below 30°C or above 47°C (Barros-Velázquez *et al*., 1999). All species of *Campylobacter* are strictly microaerophilic; i.e. they exhibit growth at low oxygen tension and oxygen acts as final electron acceptor during respiration process, but they do not tolerate the atmospheric oxygen concentration (21% v/v). *Campylobacter* spp. requires oxygen concentrations ranging from 5-10%. They also require 1-15% carbon dioxide concentration to grow (Barros-Velázquez *et al*., 1999). Several methods and commercial systems such as the Gas Generating Kits and the Campy-Pak system have been developed provide microaerophilic atmosphere anaerobic jar for growth of the organisms. Gas mixture containing (v/v)

15% carbon dioxide and 80% nitrogen and 5% oxygen is another technique to provide microaerophilic conditions for growth of the campylobacters during incubation. To provide microaerophilic conditions in anaerobic jar, air must be removed by vacuum technique and replaced it by gas mixture (Barros-Velázequez *et al.*, 1999).

EFFECT OF TEMPERATURE, FOOD PRESERVATIVES AND IRRADIATION ON *CAMPYLOBACTER*

Campylobacters are more sensitive to heat than other Gram-negative pathogens (ICMSF, 1996). The effect of spraying chicken carcasses with water at different temperatures (20, 55 and 60°C) on survival of *Camp. jejuni* was investigated by Li *et al.* (2002). Their results indicated that the 55 and 60°C water spray treatments significantly reduced *Campylobacter* by more than 0.78 log cfu/carcass compared with the 20°C water spray treatment. However, with 50 ppm chlorine spray treatment at three different temperatures the reduction of *Campylobacter* population was not significantly different. The chilling process with 50 ppm chlorinated ice water at 4°C reduced more *Camp. jejuni* (approximately 1 log CFU/carcass) among the water spray treatments but did not result in greater reduction of *Camp. jejuni* among the chlorine spray treatments. In general, campylobacters are rapidly inactive by heating at 55°C and above (Anonymous, 2001).

Effect of food preservatives on survival of five strains of *Camp. jejuni* (Pen 2, 3, 6, 10 and 20) was studied by Uradzinski *et al.* (1993). Their results indicated that the chemical preservatives added to meat sample at concentrations usually used in meat processing affected in differential way the survival of different strains of *Camp. jejuni. Campylobacter jejuni* Pen 2 was resistant to all preservatives. Pen 3 and Pen 10 were sensitive to sodium nitrate and Pen 10 was also sensitive to sodium chloride. *Camp. jejuni* Pen 20 was sensitive to sodium chloride, but potassium nitrate, sodium ascorbate and Hamine stimulated growth of this strain. Kelana *et al.* (2003a) studied the effects of storage temperature (4, 22 and 30 °C), pH (4.0 to 8.5) and sodium chloride concentration (0.25 to 7.5% w/v) on the survival of *Campylobacter jejuni* ATCC 35921 in Mueller-Hinton broth under aerobic conditions. At comparable pH, *Camp. jejuni* cells die most rapidly at 30°C and most slowly at 4°C. At 4°C *Camp. jejuni* was sensitive to 2.5% NaCl concentration. However, the level of inactivation at this storage temperature was also significantly lower than that observed at 20 and 30°C. In an additional experiment they reported that the minimum, optimum and maximum temperatures for the growth of *Camp. jejuni* ATCC 35921 in solid media were found to be 30, 40 and 45°C, respectively. At optimum growth temperature, *Camp. jejuni* ATCC 35921 was able to grow well at pH 5.5 to 8.0 and in the presence of 1.70 to 1.75% NaCl. At its minimum growth temperature, however, *Camp. jejuni* ATCC 35921 could grow only at pHs 6.5 to 8.0 and the presence of 0.5% NaCl. L-Fucose, D-fucose and sodium desoxycholate were shown to inhibit the growth of *Camp. jejuni* (Kelana *et al*., 2003b). *Campylobacter* growth is inhibited in foods at less than pH 4.9 and above pH 9. They are rapidly inactivated in foods at pH <4.0 especially at above refrigeration temperatures. Campylobacters are sensitive to low water activity (dryness) but under certain refrigeration conditions can remain viable for several weeks (Anonyomus, 2001).

Growth and survival of *Camp. jejuni* and *Camp. coli* at different temperatures (-20, 4 and 25°C) on pieces of raw chicken and pork were investigated by Solow *et al.* (2003). In their experiment *Campylobacter* sp. (10^4 CFU cm^{-2}) were inoculated on pieces of raw, irradiated chicken or pork skin and exposed to temperatures ranging from -20, 4 and 25°C under either microaerobic or aerobic conditions. Their results indicated that viable count over 48 h declined 2 or 3 log CFU cm^{-2} at -20°C and 1 to 2 log CFU cm^{-2} at 25°C regardless of skin type, species of *Campylobacter,* or level of oxygen. At 4°C there was no significant change in the number of *Campylobacter* over 48 h. At both 37 and

42°C, the number of viable *Camylobacter* increased significantly (2 to 3 CFU cm^{-2}) under microaerophilic conditions but decreased significantly (2 to 3 log CFU cm^{-2}) under aerobic conditions.

Chaveerach *et al.* (2002) evaluated the effect of organic acids on survival of *Campylobacter* spp. Ten strains of *Campylobacter* spp. were mixed with water of broiler feed separately and pH of the mixtures was adjusted to 4.0, 4.5, 5.0 and 5.5 by four acids viz., formic, acetic, propionic and hydrochloric acids. A combination of three organic acids was used in two different formulations ratios viz., formic: acetic: propionic in 1:2:3 and 1: 2: 5 proportion, at pH 4.0, 4.5, 5.0 and 5.5. The individual acids and their mixtures showed the strongest bactericidal effect on *Campylobacter* at pH 4.0 (the lowest pH tested). In contrast; at pH 5.0 and 5.5, the bactericidal activity of the four acids was low. The combination of organic acids showed a synergistic bactericidal activity at pH 4.5. The effect of the combined organic acids was stronger than the commercial products. Morphological cell changes were also studied by transmission electron microscope to determine the effect of the organic acids on the cell structure of *Campylobacter*. Some loss of outer membranes of the bacteria was found in treated groups. Therefore, based on their study, we can conclude that organic acids, individually or in combination, have a strong bactericidal effect on *Campylobacter* spp., their routine application to the water supply or in the feed of poultry farms could prevent or reduce *Campylobacter* transmission (Chaveerach *et al.*, 2002).

Diker *et al.* (1987) investigated effect of three disinfectants on survival of *Campylobacter jejuni* isolated from poultry. In this study the bacteria were suspended in two-fold dilutions of ethyl alcohol, formalin and benzalkonium chloride and viable count determined at different time intervals (1 to 60 min). The results indicated that seventy percent ethyl alcohol and 2.5% of formalin killed all bacteria within one minute. Benzalkonium chloride (1:50.000) was effective against all bacteria within 5 min. Based on these results they concluded the recommended standard concentrations of disinfectants studied are adequate to destroy *Camp. jejuni*.

Campylobacters are more sensitive to gamma radiation than most vegetative Gram-negative bacteria including: *Salmonella* and *E. coli* with D values of about 0.12-0.32 KGy in chilled meat (Collins *et al.*, 1996). Bhavsar *et al.* (2004) evaluated the effect of gamma radiation on survival of two strains of *Camp. sputorum* and *Camp. coli* in different food products. According to their report the effect of gamma radiation was dependent upon the medium in which the campylobacters were treated, e.g., the D_{10} value of *Camp. sputorum* was 2 KGy when it was in milk while 0.5-0.6 KGy when in meat. The D_{10} value of *Camp. coli* was 0.2-0.4 KGy when in meat, while 1.17 KGy when in milk. Based on these data they concluded that the effect of gamma radiation on the *Campylobacter* spp. varied with the type of food. These results were in accordance with those by Patterson (1995) who checked the sensitivity of *Campylobacter* spp. to gamma radiation, in poultry meat. These isolates were sensitive to 2.4 KGy, which is within the FDA limit of 3 KGy. Higher sensitivity of *Camp. jejuni* to UV light than other Gram-negative bacteria such as *E. coli* or *Y. enterocolitica* was also reported by Butler *et al.* (1987).

PLASMID PROFILE AND ANTIMICROBIAL SUSCEPTIBILITY OF CAMPYLOBACTERS

The probability of occurrence of plasmid in *Campylobacter jejuni* strain has been reported vary from 19to 53% (Bradbury *et al.*, 1983) and many of them are R plasmids that are transmissible among *Campylobacter* spp. (Taylor, 1983; Tenover *et al.*, 1985). Despite the importance of plasmids in virulence of numerous other pathogens, It is not confirmed that plasmids play role in the virulence of *Campylobacvter* or no. This paradigm is based on comparative study of the plasmid content and relative virulence of different *Camp. jejuni* strains in a guinea pig model of disease (Taylor *et al.*, 1984).

Yao and Guerry (unpublished data) isolated two plasmids from *Camp. jejuni* strain 81-176, one of them is R plasmid that encodes tetracycline resistance and second one encodes proteins which display strong similarity to *H. pylori* proteins. Moreover, they reported that the mutation of two of these genes affect the virulence of *Camp. jejuni* 81-176 (Bacon *et al*., 2000). However, the presence of plasmids in clinical isolates of *H. pylori* did not have any correlation with their antibiotic resistance pattern (Dharmalingam *et al*., 2003).

Antimicrobial susceptibility of *Camp. jejuni* to antibiotics was studied by Tremblay *et al*. (2003). According to them all isolates were susceptible to ampicillin, gentamicin, meropenem and imipenem, with 90% minimal inhibitory concentrations of 4, 1, 0.12 and ≤ 0.06 μg mL^{-1}, respectively. Three and two percent of the strains were, respectively, resistant and intermediate to ciprofloxacin. Thirty-four percent of the strains were resistant to tetracycline. There was a insignificant increase in resistance to ciprofloxacin and to tetracycline in recent years The percentage of intermediate and resistant MICs were, respectively, 12 and 1% for cefotaxime, 71 and 0% for erythromycin, all strains were β-lactamase negative. Lariviere *et al*. (1986) the majority of thermophilic *Campylobacter* spp. are resistant to most β-lactam antimicrobial agents. Therefore, patients suffering from acute *Campylobacter* enteritis are treated with erythromycin. Tetracycline is rarely used, because tetracycline resistance is plasmid mediated (Taylor *et al*., 1986). Resistant bacteria from animals can reach the human population by direct contact and also via food products of animal origin (Van den Bogaard *et al*., 2000). The consumption of poultry meat has been commonly associated with the development of *Campylobacter* enteritis (Deming *et al*., 1987). Resistance amongst broiler isolates of *Campylobacter* species may have implications in the treatment of poultry acquired *Campylobacter* infections.

In animals and humans, the use of antibiotics may cause an increase in the resistance of their endogenous flora. Intensive production of animals used as food sources for human such as broiler farming depends heavily on the usage of antimicrobial agents for both veterinary and growth promotion purposes (Van den Bogaard *et al*., 2000). However, Fallon *et al*. (2003), reported that the majority of *Camp. jejuni* strains isolated from poultry in Ireland were susceptible to antibiotics commonly used for human therapy, *Camp. coli* strains showed very low resistance levels and were susceptible to chloramphenicol, kanamycin, streptomycin, erythromycin, ciprofloxacin and nalidixic acid. Alfredson *et al*. (2003) have reported in southeast Queensland the incidence of thermophilic *Campylobacter* spp. resistant to erythromycin and tetracycline is low.

Regarding susceptibility of *Campylobacter* to antibiotics, there are no internationally accepted criteria of susceptibility testing break point assessment for *Campylobacter* spp. and no valid reference is available (Caprioli *et al*., 2000). Ciprofloxacin resistant strains of *Camp. jejuni* are reported isolates from United state, Finland, Spain and Thailand (Hankanen *et al*., 2003).

Skirrow *et al*. (1995) concerning erythromycin stated that this drug should be considered as the optimal drug for treatment of *Campylobacter* infections. It is because; the rate of resistance of *Campylobacter* to erythromycin is low. Erythromycin is cheap and unlike the fluoroquinolones and tetracyclines, it may be administered safely to children and pregnant women. However, currently isolation of erythromycin resistance strains of *Campylobacter* has also been reported (Tremblay *et al*., 2003). An investigation in India on antimicrobial susceptibility of thermophilic *Campylobacter* isolate from environmental samples (Baserisalehi *et al*., 2005) demonstrated high frequency of occurrence of ampicillin resistant *Campylobacter* spp. It was therefore, concluded that ampicillin could not be a drug of choice for treatment of Campylobacteriosis. Tetracycline and gentamicin were recommended as alternative treatment for *Campylobacter* gastroenteritis. However,

ciprofloxacin was recommended as a drug of choice for treatment of Campylobacteriosis. Hence, based on foregoing evidences the authors concluded that in recommended as the geographical region of investigation, the ciprofloxacin is not yet a problem as it is in Styria, Austria.

CAMPYLOBACTER INFECTIONS

The clinical symptoms of *Campylobacter* infection vary from a mild, watery diarrhea to severe bloody diarrhea. Although, diarrhea is prominent manifestation of campylobacteriosis, typical symptoms of *Camp. jejuni* infection could be fever, nausea, vomiting, abdominal pain, headache and muscle pain.. Majority of the campylobacteriosis cases are mild and do not require hospitalization and may be self-limited. However, *Camp. jejuni* infection can be severe and life threatening under certain conditions. Death is more common when other diseases e.g., cancer, liver disease and immuno-deficiency diseases are present (Allos *et al*., 1995).

Campylobacter jejuni is the cause of diarrhea/dysentery in children and it is often related to keeping pets, chicken meat consumption and untreated drinking water (Ali, 2003). Children under the age of five and young adults aged 15-29 are the age groups most frequently affected. The incubation period (the time between exposure and onset of the first symptom) is usually two to five days, but may occur in as few as 2 days or as long as 10 days after ingestion (CDC, 1996). Long-term consequences can sometimes result from a *Campylobacter* infection. Some people may develop a disease that affects the nerves of the body following campylobacteriosis. This disease is Guillain-Barré syndrome and it is the most common cause of acute generalized paralysis in the Western world. Usually it begins several weeks after the diarrheal illness caused by *Campylobacter*. It occurs when an immune system of the infected person makes antibodies against components of the *Campylobacter* and these antibodies attach to the components of the nerve cells. It is because some chemical components of the human nerve cells are similar to some chemical component of the bacteria.

Guillain-Barré syndrome begins in the feet and spreads up the body. Weakness is most important symptom of this disease and may lead to paralysis. Two therapies viz., intravenous immunoglobulin infusions and plasma exchange, may improve the rate of recovery in patients with Guillain-Barré syndrome. Miller Fisher Syndrome is another related neurological syndrome disease that can follow campylobacteriosis and is also caused by immunological disorder. In Miller Fisher syndrome, the nerves of the head are affected. Another chronic condition that could be associated with *Campylobacter* infection is an arthritis called Reiter's syndrome. This disease is a reactive arthritis and commonly affects joints such as the knees and the lower back (Allos, 1997).

Campylobacter may also cause appendicitis or infect the abdominal cavity (peritonitis), the heart (carditis), the central nervous system (meningitis), the gallbladder (cholecystitis) the urinary tract and the blood stream (Rees *et al*., 1995, Ang *et al*., 2001). Although *Campylobacter jejuni* is one of the major causes of bacterial diarrhea worldwide (Tauxe, 1992; Taylor, 1992), the details of its molecular pathogenesis are not well understood. *Campylobacter jejuni* produces a number of compounds, which may be related to its pathogenicity. These components are cell surface molecules, hemolysins and several cytotoxins (Smith, 1996; Ketley, 1997). Although, *Campylobacter* produce several cytotoxins, only the cytolethal-distending toxin that arrests eukaryotic cells at the G2 phase of the cell cycle (Whitehouse *el al*., 1998), has been characterized in detail. Several reports relevant to invasive factors of *Campylobacter jejuni* indicted that *Camp. jejuni* strains can invade intestinal epithelial cells *in vitro* (Grant *et al*., 1993; Oelschlaeger *et al*., 1993), although levels of invasion by different strains vary considerably (Tay *et al*., 1996). *Camp. jejuni* strain 81-176, originally isolated from a diarrheal outbreak associated with raw-milk consumption (Korlath *et al*., 1985), is one of the best-characterized

strains of *Camp. jejuni*. This strain has been shown to cause an inflammatory diarrhea in two human feeding studies and to cause disease in experimental models using primates (Russell *et al*., 1989) and ferrets (Yao *et al*., 1997). Furthermore, *Camp. jejuni* strain 81-176 invades INT407 cells at levels higher than those of most other *Camp. jejuni* strains (Hu *et al*., 1999).

THE SEASONALITY IN HUMAN INFECTION

Rates of human infection correlate with temporal and climatic factors. In many temperate countries there is a striking spring or summer peak (Nylen, 2002), while in tropical countries there is little seasonal variation. A high level of *Campylobacter* infections in tropical countries has been found during the rainy season (Taylor, 1992). Concerning Northern Europe, bacterial infections have been observed in summer (Walder and Forsgren,1982). Interestingly, the spring is mirrored in temperate regions of the southern hemisphere including New Zealand (Brieseman, 1990), Australia (Grau, 1991) and South Africa (Franco, 1988). Tauxe (1992) reported outbreaks caused by raw milk or contaminated water have a bimodal distribution with peaks in May and October. Stanley *et al. (*2003) suggested that the seasonality of human infections might be related to poultry and bovine reservoirs of *Campylobacter*.

CAMPYLOBACTER IN DEVELOPING COUNTRIES

In developing countries, *Campylobacter* species are responsible for childhood diarrhea. They are among the most common causes of diarrhea in travelers from developed nations. Remarkably, in many studies in the United States, *Campylobacter* infections were found to cause diarrheal disease >2-7 times as frequently of infections with *Salmonella* species, *Shigella* species, or *Escherichia coli* O157:H7 (Slutsker, 1997). National surveillance programs in developed countries monitor sporadic cases as well as outbreaks of human campylobacteriosis (Altekruse *et al*., 1999). However, national surveillance programs for campylobacteriosis generally do not exist in most developing countries despite the substantial burden of disease. Most data available on campylobacteriosis in developing countries were collected as a result of support provided by WHO to many laboratories in developing countries, including grants for epidemiological studies (Coker *et al*., 2000). The epidemiology of *Campylobacter* infections is quite different in developing countries than in the industrialized world. In tropical developing countries, *Campylobacter* infections are hyperendemic among young children, especially those <2 years. Asymptomatic infections occur commonly in both children and adults, whereas, in developed countries, asymptomatic *Campylobacter* infections are unusual. In addition, in developing countries, outbreaks of infection are uncommon and the illness lacks the marked seasonal nature observed in industrialized nations. Nevertheless, in both developed and developing countries, *Campylobacter* remains one of the most common bacterial causes of diarrhea. Although, very little information is available on the prevalence of *Campylobacter* in food (Varma *et al*., 2000) and the environment India, it has been isolated from clinical samples (Bhadra *et al*., 1989; Bichile *et al*., 1992). The most intriguing finding of these studies was the occurrence of *Campylobacter* spp. in adults as well as children suffering from acute gastroenteritis.

CONCLUSIONS

Campylobacter must be considered as transient contaminant on all kitchen surfaces and equipments used for processing raw food materials, especially poultry. Our knowledge is incomplete as to how campylobacters are transmitted, but the risk of transmission through properly

heat-processed foods is probably very low. The existing reports of incidence of campylobacteriosis are based on laboratory and community based studies. Because of lack detection of the accurate information concerning *Campylobacter* infection is thought to be much higher than that of reported. Nowadays several research groups are developing rapid and high accuracy detection methods for *Camp. jejuni*. This will give new hopes for the food and animal production sectors to detect the source of infection in early stage. Although, a good detection method cannot be the only tool for elimination of the food infection, educational media can be used to increase the public awareness and understanding, which is the better tool for prevention of any foodborne illnesses.

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