

CAMPYLOBACTER

HISTORY

Awareness of the Public Health implications of Campylobacter infections have evolved over more than a century. In 1886 Escherich observed organisms resembling Campylobacter in stool samples from children with diarrhoea. In 1913 McFaydean and Stockman identified Campylobacter in foetal tissue of aborted sheep. In 1957 King described the isolation of Campylobacter from blood samples of children with diarrhoea and in 1972 clinical microbiologists in Belgium first isolated Campylobacter from stool samples of patients with diarrhoea. The development of selective growth media in the 1970's permitted more laboratories to test stool specimens for Campylobacter and soon Campylobacter species were established as common human pathogens. Campylobacter jejuni infections are now the leading cause of bacterial gastro-enteritis in the United States and in Europe. In 1996 46% of laboratory confirmed cases of bacterial gastro-enteritis reported in the United States were caused by Campylobacter species, with a similar proportion in the United Kingdom and the rest of Europe.

DISEASE PREVALENCE

In the United States an estimated 2.4 million cases of human Campylobacter occur each year. In England and Wales in 2001 over 55,000 cases of Campylobacter infections were detected from stool specimen examination, which due to under reporting/testing could be anything between 10% to 30% of the actual number of cases. Similar levels of cases can be detected in other parts of Europe. Commonly reported symptoms of patients with laboratory confirmed infections include diarrhoea, fever, abdominal pain. In one study approximately half of the patients with laboratory confirmed Campylobacteriosis reported a history of bloody diarrhoea. Less frequently Campylobacter infections produce bacteraemia, septic arthritis and other extra intestinal symptoms. The instance of Campylobacteriosis in HIV infected patients is higher than in the general population. For example, in Los Angeles County between 1983 and 1987, the reported incidents of Campylobacteriosis in patients with AIDS was 509 cases per 100 thousand population some 39 times higher than the rate for the general population. Deaths from Campylobacter infection are rare and occur primarily in infants, the elderly and patients with underlying illnesses.

SEQUELAE TO INFECTION

Guillain Barre syndrome, a disorder resulting in acute neuro muscular paralysis, is a serious sequelae a Campylobacter infection. An estimated one case of GBS occurs from every 1,000 cases of Campylobacteriosis. Up to 40% of patients with the syndrome have evidence of recent Campylobacter infection. Approximately 20% of patients with GBS are left with some disability and approximately 5% die. Campylobacteriosis is also associated with reiter syndrome a reactive arthropathy. In approximately 1% of patients

with Campylobacteriosis the sterile post infection process occurs 7 to 10 days after the onset of diarrhoea. Multiple joints can be affected, particularly the knee joint. Pain and incapacitation can last for months or become chronic.

TREATMENT OF CAMPYLOBACTER INFECTIONS

Supportive measures, particularly fluid and electrolyte replacement, are the principle therapies for most patients with Campylobacteriosis. Severely dehydrated patients should receive rapid volume expansion with intravenous fluids. For most of the patients, oral re-hydration is indicated. Although Campylobacter infections are usually self limiting, antibiotic therapy may be prudent for patients who have a high fever, bloody diarrhoea or more than eight stools in 24 hours; immuno-suppressed patients, patients with bloodstream infections, and those whose symptoms worsen or persist for more than one week from the time of diagnosis.

ANTI MICROBIAL RESISTANCE

The increasing rate of human infections caused by anti microbial resistance strains of Campylobacter makes clinical management of cases of Campylobacteriosis more difficult. Anti-microbial resistance can prolong the illness and compromise treatment of patients with bacteraemia the rate of anti microbial resistant enteric infections is highest in the developing world, where the use of anti microbial drugs in humans and animals is largely unrestricted. A 1994 study found that most clinical isolates of Campylobacter jejuni from US troops in Thailand resistant to ciprofloxacin. In the industrialised world the emergence of fluoroquinolone resistant strains of Campylobacter jejuni illustrates the need for prudent antimicrobial use in food animal production. After fluoroquinolone use in poultry was approved in Europe resistant Campylobacter jejuni strains emerged rapidly in humans during the early 1990's. Similarly within 2 years of the 1995 approval of fluoroquinolone use for poultry in the United States the number of domestically acquired human cases of ciprofloxacin resistant Campylobacteriosis doubled in Minnesota. In a 1997 study conducted in Minnesota 20% of Campylobacter jejuni isolates obtained from chicken purchased in grocery stores was ciprofloxacin resistant.

PATHOGENESIS

The pathogenesis of Campylobacter infection involves both host and pathogen specific factors. The health and age of the host and Campylobacter specific humoral immunity from previous exposure influence clinical outcome after infection. In a volunteer study, Campylobacter jejuni infection occurred after the ingestion of as few as 800 organisms. Rates of infection increased with the ingested dose. Rates of illness appeared to increase when inocula were ingested in a suspension buffered to reduce gastric acidity.

SURVIVAL IN THE ENVIRONMENT

The survival of *Campylobacter* outside the gut is poor and replication does not readily occur. *Campylobacter* grows best at 37 degrees to 42 degrees Celsius. It grows best in a low oxygen or microaerophilic environment such as an atmosphere of 5% oxygen 10% carbon dioxide and 85% nitrogen. The organism is sensitive to freezing, drying, acidic conditions (pH of less than 5), and salinity.

TRANSMISSION TO HUMANS

Most cases of human *Campylobacteriosis* are sporadic, outbreaks have different epidemiological characteristics from sporadic infections. Many outbreaks occur during the Spring and Autumn. Consumption of raw milk was implicated as the source of infection in 30 of 80 outbreaks of human *Campylobacteriosis* reported in the US between 1973 and 1992. Outbreaks caused by consumption of raw milk, often involve farm visits during the temperate seasons. In contrast sporadic *Campylobacter* isolates peak during the summer months. A series of case controlled studies identified some risk factors for sporadic *Campylobacteriosis*, particularly handling raw poultry and eating undercooked poultry. Other risk factors accounting for a smaller proportion of sporadic illnesses include drinking untreated water; travelling abroad; eating barbecued pork or sausage; drinking raw milk or milk from bird-pecked bottles; contact with dogs and cats, juvenile pets or pets with diarrhoea. Person to person transmission is uncommon. Overlap is reported between sero-types of *Campylobacter* found in humans, poultry and cattle indicating that foods of animal origin may play a major role in transmitting *Campylobacter* to humans.

Infants have the highest age specific *Campylobacter* isolation rate, approximately 14 per 100,000 person years. As children get older the isolation rates decline to approximately 4 per 100,000 for young adolescence. A notable feature of the epidemiology of human *Campylobacteriosis* is the high isolation rate among young adults, approximately 8 per 100,000. Among middle aged and older adults the isolation rate is less than 3 per 100,000. The peak isolation rate in young children and infants is attributed in part to susceptibility on first exposure and to the low threshold for seeking medical care for infants. The high rate of infection during early adulthood, which is pronounced among young men is thought to reflect poor food handling practices in a population that until recently had relied on others to prepare meals.

RESERVOIRS

The ecology of *Campylobacter* involves wildlife reservoirs, particularly wild birds. Species that carry *Campylobacter* include migratory birds, ducks, geese and seagulls. The organism is also found in other wild and domestic bird species as well as in rodent. Insects can carry the organism on their exoskeleton.

The intestines of poultry are easily colonised with *Campylobacter*. Day old chicks can be colonised with as few as 35 organisms. Most chickens in commercial operations are colonised by 4 weeks. Vertical transmission (ie from breeder flocks to progeny) has been suggested in one study that it is not widely accepted. Reservoirs in the poultry environment include beetles, unchlorinated drinking water, farm workers, birds and pests. Feed is an unlikely source of *Campylobacter* since they are dry and *Campylobacters* are sensitive to drying.

Campylobacter jejuni is a common organism of the intestinal tract of cattle. Young animals are more often colonised than older animals, as are cattle which are fed concentrates as opposed to grazing animals. *Campylobacters* are found in natural water sources throughout the year. The presence of *Campylobacters* is not clearly correlated with indicated organisms of foetal contamination (eg *e coli*). In temperate regions, organism recovery rates are highest during the cold season. Survival in cold water is important in the life cycle or is thought to be important in the life cycle of *Campylobacters*.

When stressed *Campylobacters* enter a 'viable but non-culturable state' characterised by uptake of amino acids and maintenance of an intact outer membrane, but inability to grow on selective media; such organisms, however, can be transmitted to animals.

CAMPYLOBACTER IN THE FOOD SUPPLY

Campylobacters are found in many foods of animal origin. Surveys of raw agricultural products support epidemiological evidence implicating poultry, meat and raw milk as sources of human infection. Most retail chicken is contaminated with *Campylobacter*; one study in the USA reported an isolation rate of 98% from retail chicken meat. Levels of between 60% and 80% have been found in various European studies. *Campylobacter* counts often exceed 10^3 per 100 grams. Skin and giblets have particularly high levels of contamination. In one study, 12% of raw milk samples from dairy farms in Eastern Tennessee were contaminated with *Campylobacter*. Raw milk is presumed to be contaminated by bovine faeces; however direct contamination of the milk as a consequence of mastitis also occurs. *Campylobacters* are also found in raw milk in one study *Campylobacters* were present in 5% of raw ground beef and 40% veal specimens.

CONTROL OF CAMPYLOBACTER INFECTION

On the Farm

Control of *Campylobacter* contamination on the farm may reduce contamination of carcasses, poultry, and red meat products at retail level. Epidemiological studies indicate that strict hygiene reduces intestinal carriage in food producing animals. In field studies, poultry flocks that drank chlorinated water had lower intestinal colonisation rates than poultry that drank unchlorinated water. Experimentally, treatment of chicks with common bacterial and immunisation of older birds reduced *Campylobacter*

colonisation. Because intestinal colonisation with *Campylobacter* readily occurs in poultry flocks, even strict measures may not eliminate intestinal carriage by food producing animals. It is also possible that the organic poultry could be at greater risk of *Campylobacter* colonisation than are intensively reared birds because of the greater risk of exposure to environmental sources of infection. One study currently being undertaken in the North of Ireland, has indicated that levels of *Campylobacter* at poultry arriving at slaughter, are actually reduced by feeding the birds prior to transport as opposed to the normal practice where food is withheld immediately prior to transportation to slaughter.

AT PROCESSING

Slaughter and processing provide opportunities for reducing *Campylobacter* counts on food animal carcasses. Bacterial counts on carcasses can increase during slaughter and processing steps. In one study up to a 1,000 fold increase in bacterial counts on carcasses was reported during transportation to slaughter. In studies in chickens and turkeys at slaughter, bacterial counts increased approximately 10 to 100 fold during de-feathering and reached the highest level after evisceration. However, bacterial counts on carcasses decline during other slaughtering and processing steps. In one study, forced air chilling of pig carcasses caused 100 fold reduction in carcass contamination. In other field studies scalding reduced carcass counts to near or below detectable levels. Adding sodium chlorate or trisodium phosphate to the chiller water in the presence of an electrical current reduced *Campylobacter* contamination of chiller water by 2 log units. In a slaughter plant in the UK use of chlorinated sprays and the maintenance of clean working surfaces resulted in a 10 to 100 fold decrease in carcass contamination. In another study lactic acids spraying of pig carcasses reduced counts by at least 50% to often undetectable levels.

CONCLUSIONS

Campylobacter must be anticipated as transient contaminants on all kitchen surfaces and food processing equipment used for raw food, especially raw poultry. Although, at the moment, knowledge is incomplete as to how *Campylobacter*s are transmitted, however it must be assumed that there is transmission of *Campylobacter* from food handlers, food equipment and food surfaces and hence the thorough disinfection of these surfaces between different food products is essential.

Normal good hygienic practises, will be effective against *Campylobacter* infections, special concerns in relation to *Campylobacter* will include the following:-

1. Pets
2. Un-pasteurised dairy products
3. Un-pasteurised milk or anything made with un-pasteurised milk
4. Un-treated water supplies

5. Anything that may have been contaminated by birds (for example packed milk bottles)
6. Poultry