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EDITORIAL

CAMPYLOBACTER: A SYNOPSIS

In 1957 King reported culturing thermophilic, microaerophilic bacilli from the blood of patients with diarrhoea (1). This discovery was followed a few years later by two reports from the laboratory of Butzler and colleagues who had isolated a vibrio-like organism from the stools of patients with diarrhoea (2,3). These "related vibrios" were shown to be similar to *Vibrio fetus*, an organism which had been recognised as a pathogen of animals for more than 70 years. Neither organisms fermented glucose and they differred from other species of the genus Vibrio in their DNA composition (4). For these reasons a new genus was created and given the morphologically apposite name *Campylobacter*: "kampylos" meaning curved in Greek and "bakterion" meaning "little rod". The next development came in 1977 when Skirrow described a disease which he called Campylobacter enteritis and, in a major breakthrough, developed a highly selective culture medium to grow the organisms (5). These reports led to a surge of interest and research into the taxonomy and pathogenesis of the genus as well as studies on clinical and epidemiological aspects of what was rapidly discovered to be a disease of variable severity.

Research during the last 15 years has shown that *Campylobacter* spp. have been found wherever they have been specifically sought and appear to occur world-wide. *Campylobacter* spp. have been found to be responsible for between 5% and 14% of acute episodes of diarrhoea and may also be involved in bacteraemia, gastritis, duodenitis, gastro-duodenal ulcers, the Guillain-Barre' syndrome and neonatal meningitis (6, 7). The principal organisms in the genus *Campylobacter* which cause enteritis are *C. jejuni, C. coli* and *C laridis* (8). Of these three *C. jejuni* is the most common, with children more often infected than adults. Two new species in the enteritis group, *C. cinaedi* and *C. fennelliae,* have recently been isolated from rectal swabs taken from homosexual men with diarrhoea (9). Another important group recently identified are the gastric *Campylobacter*-like organisms (GCLO) of which one, GCLO-1, has been named *Campylobacter pylori*. Research has shown a strong association between *C. pylori* and both gastritis and gastroduodenal ulcers (10), giving rise to hopes for a simple curative treatment for these diseases in some cases (11).

The diagnosis of *Campylobacter* enteritis is based on identifying bacteria in faecal samples by microbiological culture using selective agar media. A less common method is to use non-selective media after the faeces has been filtered through a membrane with a pore size of 0.65 μm, which acts to exclude most other bacteria as *Campylobacter* spp. are relatively small organisms. The agar media are incubated at a temperature of 42°C in 4-6% oxygen with added carbon dioxide, and a result can be expected after 72 to 96 hours. The techniques are relatively lengthy and expensive compared with methods to isolate other enteric pathogens and by the time the results of the culture tests are available, symptoms have usually subsided. Diagnostic tests involving microscopy have been developed using stained faecal smears, dark field microscopy or direct immunofluorescence, but these rely on large numbers of organisms being present in the small sample of faeces being examined and have not found favour in routine laboratory diagnosis. A better prospect for a simple and quick routine diagnostic test would be one based on a radioactively labelled gene probe (12) but this would depend on identifying stable and species specific regions of the bacterial genome. Diagnostic tests to detect antigens could also offer a rapid means of diagnosis once the reagents had been prepared. However, species-specific and stable antigens must be present on the surface of the organism, and monoclonal antibodies to these antigens are almost a prerequisite if a diagnostic test is to be senstive and specific enough.

Campylobacter is a well recognised pathogen in developed countries but its epidemiology is poorly understood in developing countries,

where a considerable number of healthy people may excrete the organism in their faeces with no signs of illness (13, 14). Whether this is a result of acquired immunity or because of differences in strains of the organism will require further research. Whatever the reason, reservoirs of infections in humans in addition to those in animals, soil and water, all contribute to a complicated tangle of transmission and infection which needs to be unravelled.

Serotyping, biotyping and phage typing have all been developed to identify strains and to study the source of epidemics and the spread of infections. These methods are of limited clinical usefulness while some specific techniques have not yet been fully evaluated or are too complicated for routine use in the laboratory. A comprehensive and practical scheme of classifying species and strains will undoubtedly help to solve many perplexing questions of variable virulence and the sources of infections.

Campylobacter enteritis is usually a self-limiting disease although relapses may occur in untreated cases. Several antimicrobials are effective as treatment, and erythromycin or sulfamethoxazole-trimethoprim are the drugs most commonly used.

Campylobacter spp. are capable of causing both a watery or secretory diarrhoea in humans, as well as dysentery, but the pathogenesis is still uncertain. The main barrier to our understanding of the processes of disease has been the lack of an experimental animal which is susceptible to the infection and which shows symptoms similar to those in man. Enterotoxins have not been detected by testing routinely grown *C. jejuni* using Y1 adrenal cells, Chinese hamster ovary (CHO) cells or suckling mice. Neither has the Sereny test shown *Campylobacter* to have any ability to invade tissues under these circumstances. But adherence, invasion, cytotoxin and enterotoxin production is now being reported (15-17). It appears that under certain culture conditions some strains of *C jejuni* produce a cholera toxin-like enterotoxin which binds to GM1-ganglioside and brings about a secretion of fluid from the gut. This toxin causes CHO cells to elongate and is neutralised by cholera antitoxin (15). It may be that *C. jejuni* for example, the most common member of the genus, may resemble *Escherichia coli* in that some strains are enterotoxigenic, some strains are enteroinvasive while others are non-pathogenic. Whatever the case, a clear understanding of the mechanisms of pathogenesis is likely to depend on experiments using animals.

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