

Role of campylobacter spp. in human and animal disease: a review¹

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Although well recognized as a pathogen by veterinary surgeons (under its earlier name of vibrio), the campylobacter organism was largely unknown in the field of human medicine until the early 1970s. At that time, the difficulties in isolating campylobacters from faeces were surmounted by a joint veterinary/medical approach in Belgium, when a method employed for the diagnosis of 'vibrionic abortion' in animals was applied to human patients (Dekeyser *et al.* 1972).

Initial work (Butzler *et al.* 1973) suggested that campylobacters were a cause of diarrhoea in children, and campylobacter enteritis (*C. jejuni*/*C. coli*) was shown to be a common disease in Britain by Skirrow (1977) who also mentioned a zoonotic aetiology for several human cases.

Symptoms of campylobacter infection in man

In man, thermophillic campylobacters produce an enterocolitis (Butzler & Skirrow 1979) leading to the symptoms of profuse diarrhoea (\pm blood, pus or mucus) with abdominal pain. The disease may last from a few days to several weeks and fever and general malaise may be present. Rarely, campylobacter bacteraemia has been recorded and patients show an increase in complement fixating, agglutinating and bactericidal antibody titres. For example, Watson *et al.* (1979) found a significant increase in agglutinating titres (> 320) in 77.2% of affected patients. Compared to other enteric pathogens such as salmonella, relatively few campylobacters are needed to produce human disease: 500 organisms taken by mouth in 200 ml of milk (in which the organism does not multiply) were sufficient to produce symptoms four days later, followed by antibody responses in a research worker (D A Robinson, personal communication)!

The disease is commoner in children, amongst whom the infection spreads readily, and has even occurred perinatally (Mawer & Smith 1979). Campylobacter infections may be present concomitantly with other diseases. For example, of 59 patients with apparent flare-ups of inflammatory bowel disease (chronic ulcerative colitis and Crohn's disease), *C. jejuni* was isolated from 4 (Newman & Lambert 1980). Further examples were given by Goodman *et al.* (1980). Obviously, misinterpretation of a campylobacter infection as an exacerbation of inflammatory bowel disease would lead to needless corticosteroid or immunosuppressive medications. Newman and Lambert therefore suggested that isolation of campylobacter (as well as salmonella, shigella and *Clostridium difficile*) should be attempted in suspected flare-ups of inflammatory bowel disease.

Evidence of human infection

The number of human campylobacter infections reported to the Communicable Disease Surveillance Centre in 1979 was 8577. These were mostly symptomatic cases and suggested the importance of campylobacters compared to other enteric pathogens such as salmonella, where isolations from 12 251 symptomatic cases and asymptomatic excretors were made during the same period. Further evidence that campylobacters produce human enteric disease is presented in Table 1 and a more extensive review has been given by Butzler & Skirrow (1979).

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Table 1. Prevalence of human campylobacter infections

Source	% positive faecal samples			Reference
	Patients with GI symptoms	In-contact patients	Asymptomatic patients	
Children	5.1	—	1.3	Butzler <i>et al.</i> 1973
All ages	7.1	—	0	Skirrow 1977
All ages	13.9	1.7	0.6	Bruce <i>et al.</i> 1977

Note: Age predisposition – e.g. in Bruce *et al.* (1977) 38% of patients were children under 10 years of age

Table 2. Diseases of animals associated with campylobacter infections

Pigs:	Proliferative haemorrhagic enteropathy/Porcine intestinal adenomatosis Abortion
Sheep:	Abortion
Cattle:	Infertility and abortion. ? Enteritis ? Mastitis
Horses:	Pyrexia, colic and acute diarrhoea in foals
Dogs and cats:	? Enteritis
Birds:	? Symptomless
Simian primates:	Enteritis Abortion

Diseases of animals associated with campylobacter infections

These are summarized in Table 2. In animal species most work has probably been done on the role of *C. spurotum* subsp. *mucosalis* in enteric disease in pigs (for review see Yates *et al.* 1979). In cattle, campylobacters have been shown to produce mastitis experimentally (Lander & Gill 1979) but have not been isolated from naturally-occurring cases. They may also cause bovine enteritis (A1-Mashat & Taylor 1980). Similarly, *C. jejuni/C. coli* were isolated from five foals with pyrexia, colic and acute diarrhoea but 15 other diarrhoeic animals were negative (Atherton & Ricketts 1980). Campylobacters may produce infertility and abortion in pigs, sheep and cattle (MacLaren & Wright 1977), possibly by reducing the oxygen tension *in utero* (Ware 1980).

There is conflicting evidence as to whether campylobacters cause canine or feline enteritis. Whilst several studies show no statistical differences in prevalence rates between normal and diarrhoeic animals (see Tables 3 & 4), a mild colitis was produced experimentally in gnotobiotic puppies without invasion of the mucosa by the organism (Prescott & Barker 1980) and death in a dog with haemorrhagic enteritis was associated with campylobacter bacteraemia (Slee 1979).

The symptoms in simian primates are similar to those in man (Tribe *et al.* 1979) but abortions associated with campylobacter infections have also been recorded (Tribe & Frank 1980).

Campylobacters can be isolated from a high percentage of healthy chickens (Bruce *et al.* 1977).

Prevalence of animal infection

As mentioned, campylobacters are a common isolate from chickens, and in one study (Bruce *et al.* 1977) *C. jejuni/C. coli* were present in 62% of chicken carcasses and 68% of chicken caecal contents. The poultry preparation process has little effect: *C. jejuni* has been found in 72% of chicken carcasses at the start and end of the processing procedure and was still present in 48% after refrigerated delivery to a simulated point of sale (Simmons & Gibbs 1979).

In cattle, campylobacters were isolated from the intestinal mucosa of 16 of 47 animals examined post mortem and it was suggested that they may be a cause of bovine enteritis (A1-Mashat & Taylor 1980).

Table 3. Prevalence of canine campylobacter infections

Source	% positive faecal samples		Reference
	Normal	Diarrhoeic	
Kennels/veterinary practice:			Bruce <i>et al.</i> 1980
Adults	49●	—	
Puppies	39●	38●	
Kennels/veterinary practice:			Fleming 1980
Adults	0	4.8	
Puppies	0	28.4	
Veterinary practice: all ages	6	8	Holt 1980
Veterinary practice: all ages	11.1	10.4	Hosie <i>et al.</i> 1979

● enrichment cultures used, increasing isolation rates by 46%

Table 4. Prevalence of feline campylobacter infections

Source	% positive faecal sample	Reference
Adult cats, RSPCA kennels	45●	Bruce <i>et al.</i> 1980
Veterinary practice:		Gruffydd-Jones <i>et al.</i> 1980
Diarrhoeic animals	2.2	
Normal animals	0	

● enrichment cultures used, increasing isolation rates by 46%

That campylobacters cause enteric disease in simian primates is suggested by the isolation prevalence rates of 17.8% from the faeces of normal animals compared with 61.4% from those of diarrhoeic primates (Tribe *et al.* 1979).

Recently, interest has been shown in the prevalence rates in cats and dogs. A useful summary is given by Skirrow (1981) but the data relating to Britain are summarized in Tables 3 and 4. It should be noted that the percentages are those given by the authors but the statistical significance of some surveys is limited by the number of animals sampled.

Epidemiology of human campylobacter infections

It must be emphasized that campylobacter infection spreads readily among people, particularly children, and that other infected humans are the commonest source of the disease for man. As mentioned previously, however, most animal species are capable of harbouring campylobacters, some of which are identical serotypes to those causing disease in man (Lauwers *et al.* 1981), and human outbreaks associated with contact with animals or animal products have been reported. In this context, most zoonotic outbreaks appear to be associated with milk or poultry.

Cattle can be symptomless excretors and individual cows have been shown to excrete the same campylobacter serotype in their faeces (but not milk) for at least four months (Robinson & Jones 1981). Human outbreaks of campylobacter enterocolitis associated with ingestion of unpasteurized milk where the same campylobacter was isolated from milk filters on the farm are not uncommon (Robinson *et al.* 1979, Robinson & Jones 1981). The organisms are probably present because of faecal contamination rather than primarily-infected milk.

Human illness has followed ingestion (e.g. Hayck & Cruikshank 1977) or handling of infected poultry (Skirrow 1977) and poultry workers would seem to be at risk; 49% of poultry and duck workers reacted positively to a compliment fixation test compared with 5% of controls (D A Robinson, personal communication). Cruikshank (1979) noted, however, that broiler factory workers were not absent from work because of gastrointestinal disease more frequently than other groups.

It has been estimated that only approximately 5% of human cases originate from dogs

(Skirrow 1981). Examples are given by Skirrow (1977, 1981) and Blaser *et al.* (1978), and are usually associated with poor hygiene. Only 4 cases of campylobacter infection in man originating from cats have been recorded (Svedhem & Norkrans 1980, Skirrow *et al.* 1980).

Other species which may rarely act as a source of human infection are sheep (Duffell & Skirrow 1978) and simian primates (Tribe *et al.* 1979).

Pig bowel is used in Germany for making sausages. Campylobacters have been isolated from 72% of healthy pig faeces after slaughter and from 30% of the bowels after preparation for sausage-making (Sticht-Groh 1981). In countries where such pork sausages are consumed, they may represent a further source of campylobacters for human infection. In Britain, campylobacter enteritis in a human and her dog (fatal in the latter) followed the ingestion of the same luncheon meat by both (Peel & McKintosh 1978). Unfortunately, the meat was not available for bacteriological examination and it remains to be seen whether food poisoning associated with campylobacter-infected pork products is a potential hazard in the UK.

Conclusions

Campylobacters may produce a number of diseases in several species including man. The disease in man takes the form of an enterocolitis. It is transmitted readily from person to person but may, rarely, be acquired as a zoonotic infection. Cattle, poultry and dogs appear to be the commonest animal sources for human infection but their role must be kept in perspective and could be minimized by basic hygienic measures and pasteurization of milk.

References

- Al-Mashat R R & Taylor D J (1980) *Veterinary Record* **107**, 31–34
 Atherton J G & Ricketts S W (1980) *Veterinary Record* **107**, 264–265
 Blaser M, Cravens J, Powers P W & Wang W L (1978) *Lancet* **ii**, 979–981
 Bruce D, Zochowski W & Ferguson I R (1977) *British Medical Journal* **ii**, 1219
 Bruce D, Zochowski W & Fleming G A (1980) *Veterinary Record* **107**, 200–201
 Butzler J P, Dekeyser P, Detrain M & Dehaen F (1973) *Journal of Pediatrics* **82**, 493–495
 Butzler J P & Skirrow M B (1979) *Clinics in Gastroenterology* **8**, 737–765
 Cruikshank J G (1979) Lecture at 4th Meeting of Standing Conference on Food and Environmental Microbiology. Colindale Hospital
 Dekeyser P, Gossian-Detrain M, Butzler J P & Sternon J (1972) *Journal of Infectious Diseases* **125**, 390–392
 Duffell S J & Skirrow M B (1978) *Veterinary Record* **104**, 144
 Fleming M P (1980) *Veterinary Record* **107**, 202
 Goodman M J, Pearson K W, McGhie D, Dutt S & Deodhar S G (1980) *Lancet* **ii**, 1247
 Gruffydd-Jones T J, Marston M & White E (1980) *Lancet* **ii**, 366
 Hayck L J & Cruikshank J G (1977) *British Medical Journal* **ii**, 1219
 Holt P E (1980) *Veterinary Record* **107**, 254
 Hosie B D, Nichosen T B & Henderson D B (1979) *Veterinary Record* **105**, 80
 Lander K P & Gill K P W (1979) *Veterinary Record* **105**, 333
 Lauwers S, Vlaes L & Butzler J P (1981) *Lancet* **i**, 158–159
 MacLaren A P C & Wright C L (1977) *Veterinary Record* **101**, 463–464
 Mawer S L & Smith B A M (1979) *Lancet* **i**, 1041
 Newman A & Lambert J R (1980) *Lancet* **ii**, 919
 Peel R N & McKintosh A W (1978) *Lancet* **ii**, 1212
 Prescott J F & Barker I K (1980) *Veterinary Record* **107**, 314–315
 Robinson D A, Edgar W M, Gibson G L, Matchett A A & Robertson L (1979) *British Medical Journal* **i**, 1171–1173
 Robinson D A & Jones D M (1981) *British Medical Journal* (in press)
 Simmons N A & Gibbs F J (1979) *Journal of Infection* **1**, 159–162
 Skirrow M B (1977) *British Medical Journal* **ii**, 9–11
 Skirrow M B (1981) *Veterinary Research Communications* (in press)
 Skirrow M B, Turnbull G L, Walker R E & Young S E J (1980) *Lancet* **i**, 1188
 Slee A (1979) *Veterinary Record* **104**, 14–15
 Sticht-Groh V (1981) *Veterinary Record* **108**, 42
 Svedhem A & Norkrans G (1980) *Lancet* **i**, 713–714
 Tribe G W, MacKenzie P S & Fleming M P (1979) *Veterinary Record* **105**, 333
 Tribe G W & Frank A (1980) *Veterinary Record* **106**, 365–366
 Ware D A (1980) *British Veterinary Journal* **136**, 301–303
 Watson K C, Kerr E J C & McFadzean S M (1979) *Journal of Infection* **1**, 151–158
 Yates W D G, Clark E G, Osborne A D *et al.* (1979) *Canadian Veterinary Journal* **20**, 261–268