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# Campylobacter enteritis associated with the consumption of free school milk

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## SUMMARY

A large outbreak of campylobacter enteritis associated with the consumption of free school milk is described. The outbreak had an abrupt onset, and lasted for about 3 weeks; it involved mainly school children in the 2-4 and 5-7 year old age groups. During this period it was established from epidemiological and microbiological data that some 2500 children were infected. The source of the epidemic was almost certainly contaminated milk, although bacteriological proof could not be obtained. Biotyping of isolates was of considerable epidemiological value and showed the involvement of two distinct strains, one of which was dominant. Epidemiological evidence of limited person to person transmission of the infection was obtained; febrile convulsions as a prodromal sign of the illness was recognized for the first time. Strains of *Campylobacter jejuni*, and samples of patients' serum collected during this outbreak have enabled subsequent studies to be initiated on the serotyping of the responsible organism, on the serological response of patients infected with the organism, and on experimental infection of the bovine udder which demonstrated its potential as a source of *C. jejuni* in raw milk. A careful search of the literature suggests that this is the largest documented outbreak of campylobacter enteritis.

## INTRODUCTION

From time to time milk has been associated with outbreaks of campylobacter enteritis (Cook & McKay, 1977; Robinson *et al.* 1979; Porter & Reid, 1980). The present report describes the largest milk-associated outbreak of this condition yet recorded.

During the third week of March 1979 a sharp increase in the number of routine

isolates of *Campylobacter* spp. was noted in the laboratory, mainly from children of primary school age. Enquiries revealed that many school children in the district were affected with a diarrhoeal illness, and that the overall absentee rate from schools was unusually high.

## METHODS

### *Epidemiological studies*

Initially, some of the affected schools were visited, and head teachers of infant and junior schools were asked to supply attendance records for the period under review. In addition, all general practitioners in the health district were asked to investigate and report to the District Community Physician all cases of diarrhoea. As many afflicted families as possible were visited by Environmental Health Officers, who collected data and faecal specimens. The information obtained from these families included such details as family size and composition, incidence and nature of symptoms among family members, type and source of milk consumed. At an early stage in the outbreak the milk derived from one particular dairy appeared to be implicated, so that its milk processing plant and workers were subjected to a detailed public health and microbiological enquiry. Specialist epidemiological support was provided by the Centre for Disease Surveillance and Control, Colindale.

### *Bacteriological studies*

All primary faecal specimens from patients with symptoms were examined for salmonellas and shigellas as well as for *Campylobacter* spp.; specimens from contacts, and second and subsequent faecal samples from patients were examined for campylobacters only. The method used for campylobacters was direct plating onto Skirrow's medium (1977), with incubation at 43 °C, in an atmosphere of 5% oxygen plus 7% $CO_2$; cultures were examined daily for 3 days.

Milk, milk socks, and milk bottles from the suspect dairy were examined culturally by a number of different enrichment and filtration procedures.

Most of the campylobacter isolates were biotyped (Skirrow & Benjamin, 1980) and 26 serum samples, together with their 'homologous' isolates were investigated for serological response (Jones, Eldridge & Dale, 1980), and for serotype by Abbot *et al.* (1980).

## THE OUTBREAK

The course of the outbreak, which had a moderately explosive onset, is illustrated in Fig. 1. This increase in symptomatic cases was mirrored by the number of isolations which rose correspondingly from the 'normal' rate of 2 per week, to 12 per day at the height of the episode; during this period there were 406 isolates from 334 patients. After 3 weeks the number of new cases began to fall, and were soon back to the 'normal' rate for the district; there were no secondary peaks.

The age and sex distribution of 324 proven cases is summarized in Table 1. The preponderance of cases in children of age groups 2-4 years and 5-7 years is also reflected in Fig. 2. which plots school absentees for this period.

The *excess* of absentees among school children over the 3-week period commencing 12th March was 4700 compared with the preceding week, and the apparent

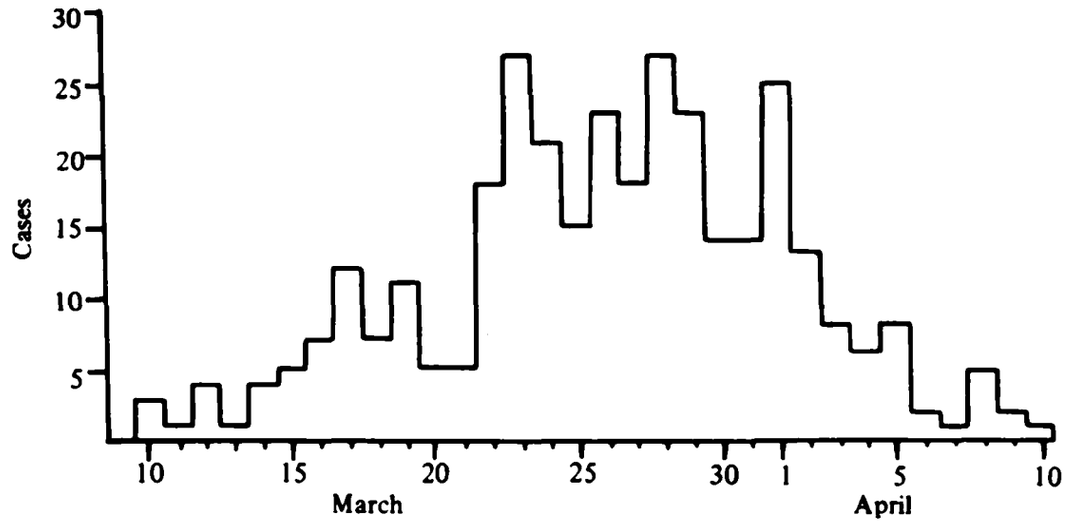


Fig. 1. Symptomatic cases of campylobacter enteritis by date of onset.

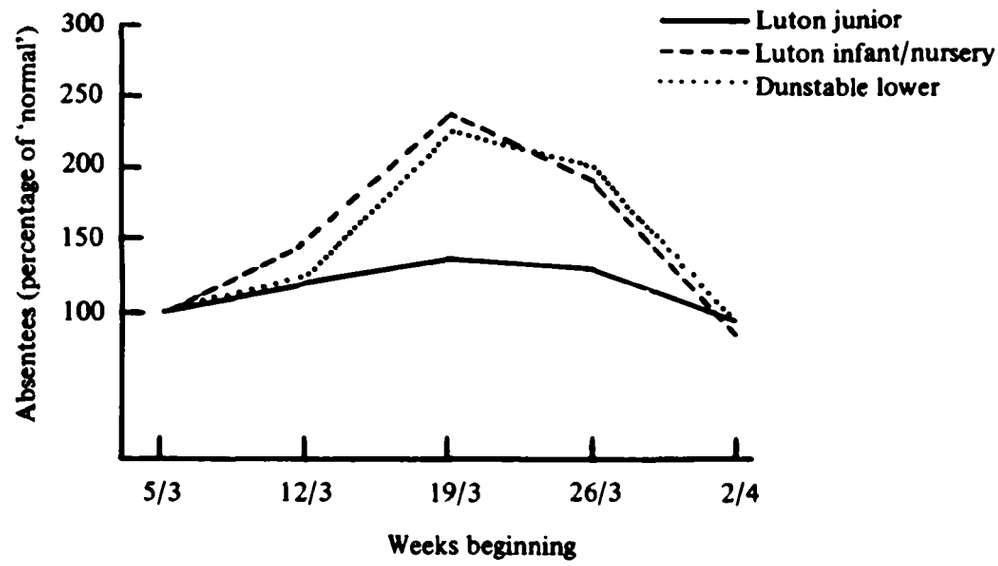


Fig. 2. Number of children absent from school each week as a percentage of the 'normal' absentee rate (i.e. that of the week preceding the outbreak).

excess cases of diarrhoeal disease among these absentees during the same period was 2550. Most of this excess of absentees occurred in the Luton Infant/Nursery groups and the Dunstable Lower School groups (all below 7 years of age), the incidence of absenteeism among older age groups who did not receive free milk (Luton Junior) barely deviated from normal.

It soon became clear that the only relevant factor common to afflicted children was their consumption of *free* school milk, which was supplied by a single milk processing plant to the whole district. One private primary/secondary school was of special interest for although all age groups in this school received milk, only those who drank the free school milk (5-7 year olds) became infected; the other children whose milk was not free and came from a different dairy, were little affected. In this school the attack rates among 5-7 year olds and over-7 year-olds were 15 of 90 (18%) and 4 of 100 (4%) respectively.

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Table 1. *Age and sex distribution of* 324 *proven cases of campylobacter enteritis*

| Age | Male | Female | Total (%) |
|---|---|---|---|
| 0–1 | 5 | 7 | 12 (4) |
| 2–4 | 28 | 14 | 42 (13) |
| 5–7 | 81 | 64 | 145 (45) |
| 8–9 | 12 | 12 | 24 (7) |
| 10–14 | 13 | 4 | 17 (5) |
| 15+ | 44 | 40 | 84 (26) |
| Total | 183 | 141 | 324 (100) |

The commonest clinical presentation was severe abdominal pain and diarrhoea, often with blood and mucus in the stool, and pyrexia. Exceptionally, much more severe illness was encountered; 4 adults were admitted to a surgical unit with a diagnosis of 'acute abdomen' but in all of them the early development of bloody diarrhoea forestalled surgical intervention. In addition, 14 children were admitted to hospital, 9 of whom presented with grand-mal convulsion and pyrexia in excess of 38 °C, but without gastrointestinal symptoms or signs; in these patients also, bloody diarrhoea soon developed. Indeed, during the outbreak, febrile convulsion, occurring in older children, became recognized as prodromal to the onset of severe campylobacter enteritis (Havalad *et al.* 1980).

### *Bacteriological results*

*C. jejuni* was isolated and recognized without difficulty in primary cultures of faecal samples. Altogether, 325 isolates from 316 sources were examined 'blind' for their phenotypic characters by one of us (M.B.S.). Of these, 271 were indistinguishable from one another (the 'epidemic strain'), and a further 29 formed a second biotype subgroup – the 'sub-epidemic strain'). The remaining 25 isolates comprised a miscellaneous group of organisms, easily distinguishable from the two 'epidemic' groups, and usually from one another. Nine patients who were tested twice yielded pairs of strains with identical characteristics; no patient had more than one biotype. Eight families had 2 different biotypes among its members.

Investigations of pets kept at schools did not reveal any campylobacters, but 2 of 12 samples of dog faeces, collected from school playing fields, yielded campylobacters. These, however, were different from both the 'epidemic' and 'sub-epidemic' strains.

### *Source of the outbreak*

As already noted the only factor that formed a common link between primary cases was milk supply. The suspect dairy supplied free milk to all of the schools in the district, but its output accounted for only 10-15% of the *total* milk distributed to the district as a whole – domestic and other.

Further evidence for the incrimination of this dairy was shown by the distribution of biotypes in infected children (Tables 2, 3). Thus, among 144 children infected with the epidemic strain, 100 (69%) drank the suspected milk; 30 (21%) had not drunk the milk but had brothers or sisters who had done so, suggesting intrafamilial spread of the infection. The remaining 14 patients probably became infected either from non-familial school contacts or by consuming suspect milk at home. These general conclusions are further supported by the figures relating to the home milk

Table 2. *Distribution of campylobacter biotypes among* 163 *symptomatic children in relation to free school milk consumption* (*suspect dairy*)

| School milk consumed by | Number of patients yielding campylobacters | | |
|---|---|---|---|
| | Epidemic strain | Sub-epidemic strain | Miscellaneous |
| Patient himself | 100 | 10 | 2 |
| Sibling only | 30 | 3 | 0 |
| Neither | 14 | 3 | 1 |
| Total | 144 | 16 | 3 |

Table 3. *Distribution of campylobacter biotypes among* 163 *symptomatic children in relation to home milk supply*

| Home milk supply from | Number of patients yielding campylobacters | | |
|---|---|---|---|
| | Epidemic strain | Sub-epidemic strain | Miscellaneous |
| Suspect dairy | 16 | 6 | 0 |
| Other dairy | 121 | 9 | 3 |
| Not known | 7 | 1 | 0 |
| Total | 144 | 16 | 3 |

Table 4. *Distribution of campylobacter biotypes among* 55 *adults in relation to home milk supply*

| Home milk supply from | Number of adults yielding campylobacters | | |
|---|---|---|---|
| | Epidemic strain | Sub-epidemic strain | Miscellaneous |
| Suspect dairy | 7 | 0 | 0 |
| Other dairy | 36 | 2 | 8 |
| Not known | 1 | 1 | 0 |
| Total | 44 | 3 | 8 |

Table 5. *Distribution of campylobacter biotypes among* 48 *adults whose home milk supply was not suspect in relation to their children's consumption of free* (*suspect*) *school milk*

| Consumption of free school milk by children | Number of adults yielding campylobacters | | |
|---|---|---|---|
| | Epidemic strain | Sub-epidemic strain | Miscellaneous |
| Drank school milk | 34 | 1 | 0 |
| Did not drink school milk | 3 | 2 | 8 |
| Total | 37 | 3 | 8 |

supply of the same patients (Table 3). Thus, of the 121 patients infected with the epidemic strain who consumed 'safe' milk at home, 21 of these can be explained only by person to person spread within or outside the home. The relatively high incidence of the 'epidemic strain' among adults (Tables 4, 5), whose home supply was *not* from the suspect dairy probably reflects person to person (child to parent) transfer of the organism, rather than primary infection from another source.

Indeed, among 277 symptomatic families studied, in no less than 188 (65%) were the first symptoms shown by children in the 2-7 year old age group.

Extensive and repeated public health inspections and bacteriological studies made at the dairy did not produce any direct evidence of its involvement; old equipment, which it is believed was improperly used on at least one occasion, does not *prove* its implication in the outbreak. Bacteriological studies at the dairy included examination of 10 milk socks, 30 milk samples, 57 bottles, and faecal samples from 6 dairy workers. Among these studies, one milk sample failed the phosphatase test on March 27th, and, significantly enough, 2 of the 6 dairy workers were symptomless carriers of the epidemic strain of *C. jejuni*. All other results were bacteriologically satisfactory, and campylobacters were not isolated.

## DISCUSSION

As a result of the pioneer studies of Skirrow (1977), a group of microaerophilic anaerobes – the campylobacters – emerged as the most frequent cause of infective diarrhoeal illness, more common, indeed, than all the other causal bacteria taken together.

The primary source of human infections with *C. jejuni* has been the cause of much speculation; poultry has seemed a likely source since these organisms are normal inhabitants of birds. The association of campylobacter infections with diarrhoea in cows was first noted by Jones, Orcutt & Little (1931), and recently there have been reports of a number of outbreaks of campylobacter enteritis in man associated with the consumption of cows' milk (Robinson *et al.* 1979; Taylor, Weinstein & Bryner 1979; Porter & Reid, 1980).

The outbreak of milk-associated campylobacter enteritis described in the present communication is of special interest, not only because of its unrivalled size (an estimated minimum number of 2500 primary cases in children alone), but also because it has shed some further light on both the epidemiology and the natural history of the disease. Until recently there was no evidence to suggest that campylobacters are excreted in milk, and earlier episodes of milk-associated infection were thought to originate from bovine faecal contamination. It is of considerable interest, therefore, that Lander & Gill (1980), working with an isolate from the Luton outbreak, were able to produce *C. jejuni* mastitis in the cow, and thus demonstrate that the bovine udder is a potential source of the organism in raw milk. It is possible that campylobacters are not infrequent inhabitants of unpasteurized cow's milk (whether derived from mastitis or from secondary faecal contamination), from which milk handlers (2 of 6 in this study) may themselves become carriers. The carriage rate of campylobacters in the normal population is low – of the order of 0·1% (personal unpublished data) but we can find no record in the literature of the faecal carriage rate among workers connected with the raw milk industry.

The source of the epidemic described in this report was almost certainly contaminated milk, although bacteriological proof was lacking. Inspection of the milk processing equipment showed that by inappropriate operation of the bypass valve system, bulk milk could be passed through the plant without being pasteurized; it is entirely possible that just such an event occurred some days prior

to declaration of the infection in the community but at a time too remote to obtain positive microbiological evidence.

Biotyping of isolates (see Skirrow & Benjamin, 1980), was of considerable epidemiological value, and showed the involvement of two distinct strains, one of which was dominant; a third small miscellaneous group of biotypically different strains was probably representative of the base line incidence in the human community. Strains of *C. jejuni* collected during the outbreak have subsequently been used in studies on the serotyping of the organisms (Abbott *et al.* 1980). Of 37 representative isolates from the present outbreak that have been examined both biotypically and serologically, D. M. Jones (1980, personal communication) found that 30 of 31 biotypically identical strains (the epidemic strain) were of the same serotype, while 2 sub-epidemic strains were identical with each other but distinct from the epidemic strain. Of the remaining 3 isolates which biotypically fell into the miscellaneous group, one was serologically similar to the epidemic strain, and one to the sub-epidemic strain. The fact that most isolates were serologically and biotypically identical is not suprising in a common source outbreak. Further study of the correlations between biotype and serotype of isolates from *different* sources is urgently required.

In the course of the study evidence for the transmission of the infection from person to person was obtained, and for the first time a febrile convulsion as a prodromal event in the illness was recognized (Havalad *et al.*, 1980).

We are greatly indebted to the many workers whose untiring efforts made this investigation possible – members of the District Community Physician's staff, neighbouring District Community Physicians, General Practitioners, Environmental Health Officers, School Head Teachers, hospital clinical colleagues and Medical Laboratory Scientific Officers at the Luton Public Health Laboratory.

We are also grateful to Dr D. M. Jones and his staff at the Public Health Laboratory, Manchester, for providing us with preliminary information on the serology of isolates from the outbreak.

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