J. Hyg., Camb. (1983), **91**, 227–233
Printed in Great Britain

Milk-borne campylobacter enteritis in a rural area

By E. P. WRIGHT*, H. E. TILLETT
Communicable Disease Surveillance Centre, Public Health Laboratory Service, London NW9 5EQ

J. T. HAGUE
Environmental Health Department, Derbyshire District Council, Bakewell DE4 1BY

F. G. CLEGG
Veterinary Investigation Centre, State Veterinary Service, Sutton Bonington, Loughborough LE12 5RB

R. DARNELL
Department of Microbiology, Derbyshire Royal Infirmary, Derby DE1 2QY

J. A. CULSHAW
The New Surgery, Tideswell, Near Buxton, Derbyshire SK17 8NF

J. A. SORRELL†
Derbyshire Area Health Authority, Boden House, Derby DE1 2PH

(*Received* 21 *March* 1983; *accepted* 5 *April* 1983)

SUMMARY

During November and December 1981 more than 50 residents in a village in Derbyshire had an acute gastrointestinal illness. One month later a second outbreak occurred affecting another 22 people. *Campylobacter jejuni* was isolated from 12 patients; no other gastrointestinal pathogens were identified. A case-control study showed an association with the consumption of unpasteurized milk from one particular farm. No new cases were identified for 6 months following the application of a Pasteurization Order from 26 January to 23 February 1982.

INTRODUCTION

Campylobacter jejuni is now well recognized as a common cause of acute gastroenteritis. Most infections are sporadic and no cause is identified, but outbreaks have been associated with the consumption of unpasteurized milk

* Present address: Department of Pathology, Royal East Sussex Hospital, Cambridge Road, Hastings, TN34 1ER.

† Present address: South East Staffordshire Health Authority, Robert Bewick House, Burton District Hospital Centre, Belvedere Road, Burton-on-Trent, DE13 0RB.

(Robinson & Jones, 1981), water (Mentzing, 1981) and food (Skirrow, Fidoe & Jones, 1981). Between November 1981 and January 1982 two outbreaks took place in a village situated in the Peak District of Derbyshire, which are reported here.

METHODS

Epidemiological and Bacteriological studies

Between 24 November and 21 December 1981, 46 people with acute gastrointestinal symptoms were identified among about 1800 residents of a Derbyshire village and interviews carried out among the earlier known cases suggested that milk from one particular farm was the vehicle of infection. This outbreak ceased spontaneously and between 22 December 1981 and 13 January 1982, no new cases were identified. However a second explosive outbreak took place between 14–26 January 1982, when a further 22 residents in the same village with a similar clinical illness to the previous outbreak were identified.

All faecal specimens from patients with symptoms were examined initially for salmonellas, shigellas and campylobacters; later as the nature of the outbreak became apparent they were examined for campylobacters only. Routine campylobacter isolation techniques were used (Skirrow, 1977). Regrettably the early campylobacter isolates were lost, but biotyping and serotyping was undertaken on two isolates.

When the second outbreak began in January the Communicable Disease Surveillance Centre was invited to assist in the investigation. A case-control study was undertaken in the town to identify the sources of infection. A case was defined as a patient with an acute gastrointestinal illness with diarrhoea or severe abdominal pain occurring at the time of the first outbreak. A control was found for each case by visiting their neighbours using a pre-determined plan (Wood, 1967). Families of cases and controls were interviewed and the information obtained including family size, symptoms, recent food histories particularly consumption of poultry, milk and water, contact with animals and pets, and recent travel abroad.

The milk processing plant, workers and milking herd of the suspect farm were subjected to a detailed microbiological inquiry by the Veterinary Investigation Centre at Sutton Bonington. On 21 December, 4 weeks after the start of the first outbreak, 50 faeces and 60 milk samples from individual cows of the milking herd of this farm were examined for campylobacters using a selective medium containing vancomycin, polymyxin B, trimethoprim, bacitracin and actidione. Milk filters were not collected on this visit. Further sampling at the farm took place on 22 January; 59 bovine rectal swabs, three individual milk samples, one milk filter and a rectal swab from a farm dog were collected. Later additional milk filters were collected.

RESULTS

Forty-six residents were identified with an acute gastrointestinal illness during November and December 1981. Of these, 22 (48%) were children aged less than 10 years. The clinical features were diarrhoea in 40 (87%) and severe abdominal pain in 36 (78%). Other symptoms included vomiting, nausea, fever, headache,

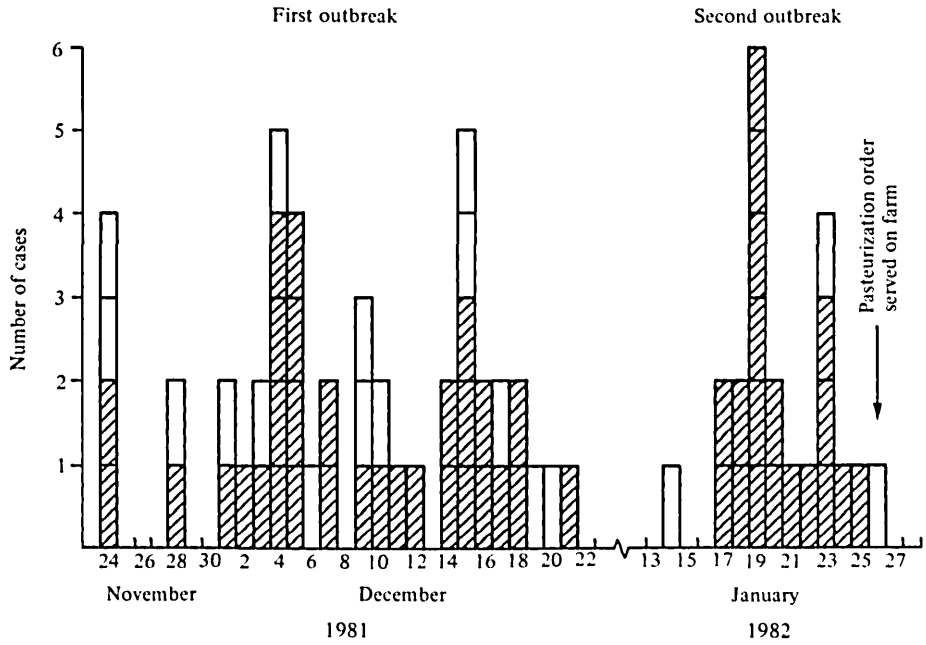

Fig. 1. Cases by date of onset of symptoms. ⊡, Illness associated with raw milk.

shivers and myalgia; only five (11%) noticed blood in the stools. Symptoms were often severe and three children together with two adults were admitted to hospital. During the second outbreak a further 22 residents had a similar illness, 10 (45%) were children aged less than 10 years. The course of the outbreaks is shown in the figure.

During the first outbreak, of 25 specimens of faeces from cases, 12 yielded *C. jejuni* and no other bacterial gastrointestinal pathogens were identified. However of 20 specimens of faeces examined from cases during the second outbreak, none yielded campylobacters.

Case-control studies

Altogether 139 completed questionnaires were analysed, 46 from cases and 93 from controls (Table 1): six of the controls were rejected because of symptoms which fulfilled the criteria of a case definition; four of these are known to have consumed unpasteurized milk from the suspect farm. An analysis of the foods consumed showed significant higher illness rates for those who had eaten frozen poultry and drunk unpasteurized milk (Table 2) and a lower rate among those who drank pasteurized milk. It is possible that people who drank unpasteurized milk from the suspect farm also tended to eat frozen poultry and not drink pasteurized milk. To test this hypothesis the food preferences were compared by consumption of pairs of items (Table 3). The higher illness rates for those who had had unpasteurized milk remain regardless of whether or not frozen poultry had been eaten. Within the two groups – those who drank unpasteurized milk and those who did not – the attack rates were still slightly higher for those who had had frozen poultry than for those who had not. Individual χ^2 tests on the two tables are not

Table 1. *Age and sex distribution of cases and controls in the first outbreak*

	Ill			Not ill		
Age (years)	Male	Female	Total	Male	Female	Total
0–9	14	8	22	3	5	8
10–19	3	0	3	6	6	12
20–29	5	5	10	4	5	9
30–39	2	4	6	9	8	17
40–49	2	0	2	6	5	11
50–59	0	0	0	5	7	12
60–69	0	0	0	6	7	13
70–79	1	2	3	1	2	3
80	0	0	0	0	2	2
Total	27	19	46	40	47	87

Table 2. *Exposure of cases and controls during the first outbreak*

	Ill				
	Positive response	(%)	Total		
Fresh poultry	5	(11)	46		
Frozen poultry	21	(46)	46		
Pasteurized milk	28	(61)	46		
Unpasteurized milk	35	(76)	46		
Cold water	18	(39)	46		
Animals/pets	23	(50)	46		
Travel abroad	0	(0)	46		
	Not ill				
	Positive response	(%)	Total	χ^2	P
Fresh poultry	12	(14)	87	NS	
Frozen poultry	17	(20)	87	8·8	0·003
Pasteurized milk	70	(80)	87	5·0	0·03
Unpasteurized milk	28	(32)	87	21·5	< 0·00001
Cold water	30	(34)	87	NS	
Animals/pets	49	(56)	87	NS	
Travel abroad	1	(1)	87	NA	

NA, not applicable; NS, not significant.

significant, but Cochran's test to combine the two is significant ($P = 0{\cdot}03$) but at a reduced level compared with the raw data in Table 2 ($P = 0{\cdot}003$). Thus the apparent association with frozen poultry is only partly due to unpasteurized milk drinkers tending to eat frozen poultry and at this stage some suspicion remained. When the source of these foods were studied it was found that 31 of the 35 who were ill and reported drinking unpasteurized milk, had had milk from a single farm. In comparison there were seven sources of frozen poultry but eight of those ill had had frozen chicken from a single shop compared with none of those who were not ill. These eight came from two families – both families using unpasteurized milk. The significance of unpasteurized milk (Table 2) was entirely accounted for by the tendency for those who drank unpasteurized milk not to buy pasteurized milk.

Table 3. *Cochran's test to compare attack rates according to consumption of frozen poultry, having first allowed for the effects of consuming unpasteurized milk*

	Drank unpasteurized milk			No unpasteurized milk		
	Ill	Not ill	Total	Ill	Not ill	Total
Frozen poultry	18 (72%)	7	25 (100%)	3 (23%)	10	13 (100%)
No frozen poultry	17 (45%)	21	38 (100%)	8 (14%)	49	57 (100%)
Total	35	28	63	11	59	70

Table 4. *History of unpasteurized milk and frozen poultry consumption among case-control pairs*

	Case				Case		
Control	Milk	No milk	Total	Control	Poultry	No poultry	Total
Milk	8	4	12	Poultry	5	4	9
No milk	17	6	23	No poultry	9	17	26
Total	25	10	35	Total	14	21	35

Because whole families were interviewed the age distribution of cases and controls was dissimilar. A more rigid analysis was applied to 35 case-control pairs where good age-group, neighbourhood and sex matching were made (Table 4). There is a significant association with raw milk ($P < 0{\cdot}01$) but not for frozen poultry (McNemars test: binomial probability for discrepant pairs).

Of 12 cases from whom *C. jejuni* was isolated, 10 were known to have drunk unpasteurized milk from the suspect farm. Unpasteurized milk from the same farm was known to have been drunk by 19 of the 22 cases identified during the second outbreak.

Farm studies

The suspect farm supplied 240 pints of unpasteurized milk daily to about 30 families in the community, including the primary school for children whose parents had requested they have unpasteurized milk. The cows were kept under very wet conditions. The herd was divided so that half the cows lived 12 h in the cowshed while the other half fed for 12 h on a silage face in another building. During the particularly cold weather the floor of this building was covered with a mixture of silage liquor and fluid faeces. The udders and sides of the cows were wet and covered with faeces and precipitation within both buildings was severe. *Campylobacter jejuni* was isolated from 2 of 50 cows sampled on 21 December, however further samples from the herd collected on 22 January 1982 did not yield campylobacters. All samples of milk and the milk-filters were negative for campylobacters. The canine faeces was also negative. A single human strain and a bovine strain were both identified as *C. jejuni* biotype 1 with slight differences between them in sensitivity to metronidazole. The human strain was serotype 2; the bovine strain was non-serotypable.

All four dairy workers had a gastrointestinal illness during the 2 months before the outbreak; bacteriological studies were not done however, and all four workers

had negative faecal cultures for campylobacters during investigation of the outbreak. All families in the study received water direct from the main supply and the local Water Authority reported no problems with the supply during the time of the outbreak.

A Pasteurization Order was served on the suspect farm from 26 January to 23 February 1982. No new cases were identified during the following 6 months.

DISCUSSION

There is circumstantial evidence that unpasteurized milk may be a vehicle of transmission in human *C. jejuni* gastroenteritis in Britain (Robinson & Jones, 1981). The predominant clinical features in these outbreaks were severe abdominal pain and diarrhoea which are compatible with campylobacter enteritis. *C. jejuni* was isolated from half the patients examined during the first outbreak. Failure to isolate the organism in some cases could have been due to erythromycin being used to treat new cases of gastroenteritis as the cause of the outbreak became clear. Erythromycin shortens the faecal excretion in *C. jejuni* in adult patients (Pitkanen *et al.* 1982). It may also be possible that there was an additional unidentified agent, for example a virus, causing gastroenteritis in the community at the same time as the campylobacter outbreak.

The results of the case-control study strongly suggest that unpasteurized milk from one particular farm was the source of infection and that other known vehicles of transmission in campylobacter infection such as poultry and contaminated water could be excluded. There was also no association with contact with sick animals and pets. No new cases of gastroenteritis are known to have occurred during the 6 months following the application of a Pasteurization Order on the suspect farm, thereby providing further evidence that milk from this farm was the source of infection.

During both outbreaks 45% of cases were children under the age of 10 years. Unpasteurized milk from the same farm was delivered daily to the local junior school and two cases who were campylobacter culture-positive but not known to have definitely drunk unpasteurized milk were both children who may therefore have been exposed to the milk at school.

Although campylobacters were not isolated from any milk samples or milk filters this does not mean that the milk could not have been the source of infection. The investigation of other campylobacter outbreaks associated with unpasteurized milk have usually given similar negative results.

Galbraith, Forbes & Clifford (1982) recently reviewed communicable diseases associated with milk and dairy products and in conclusion regretted the continuing sale of untreated milk in England and Wales. *C. jejuni* does not survive efficient pasteurisation (Gill, Bates & Lander, 1981). This outbreak provides further evidence that the failure to introduce universal heat treatment of milk in Britain is detrimental to the public health.

We are greatly indebted to the many workers who contributed to this investigation including members of the Medical Officer for Environmental Health's staff, Environmental Health Officers and General Practitioners. Dr M. B. Skirrow,

Worcester Royal Infirmary, for biotyping the strains; Dr D. M. Jones, Public Health Laboratory, Manchester, for serotyping the strains; Mr K. P. Lander, Central Veterinary Laboratory, Weybridge, for further examination of the bovine faeces; Dr C. L. R. Bartlett and other staff at the PHLS Communicable Disease Surveillance Centre, London, who helped with the design of the study and preparation of this report.

REFERENCES

Galbraith, N. S., Forbes, P. & Clifford, C. (1982). Communicable disease associated with milk and dairy products in England and Wales 1951–80. *British Medical Journal* **284**, 1761–1765.

Gill, K. P. W., Bates, P. G. & Lander, K. P. (1981). The effect of pasteurisation on the survival of *Campylobacter* species in milk. *British Veterinary Journal* **137**, 578–584.

Mentzing, L-O. (1981). Waterborne outbreaks of campylobacter enteritis in central Sweden. *Lancet* ii, 352–354.

Pitkanen, T., Petterson, T., Ponka, A. & Kosunen, T. U. (1982). Effect of erythromycin on the faecal excretion of *Campylobacter fetus* subspecies *jejuni*. *Journal of Infectious Diseases* **145**, 128.

Robinson, D. A. & Jones, D. M. (1981). Milk-borne campylobacter infection. *British Medical Journal* **282**, 1374–1376.

Skirrow, M. B. (1977). Campylobacter enteritis; a 'new' disease. *British Medical Journal* i, 9–11.

Skirrow, M. B., Fidoe, R. G. & Jones, D. M. (1981). An outbreak of presumptive food-borne campylobacter enteritis. *Journal of Infection* **3**, 234–236.

Wood, P. C. (1967). Guide to shellfish hygiene. Offset Publication no. 31, pp. 73–76. W.H.O., Geneva.