

A point source outbreak of campylobacter infection related to bird-pecked milk

T. RIORDAN¹, T. J. HUMPHREY² AND A. FOWLES³

¹Public Health Laboratory, Church Lane, Exeter EX2 5AD

²The Food Unit, Public Health Laboratory, Church Lane, Exeter EX2 5AD

³Department of Control of Infection, Royal Devon & Exeter Hospital (Wonford),
Barrack Road, Exeter EX2 5DW

(Accepted 9 November 1992)

SUMMARY

A point source outbreak of *Campylobacter jejuni* affected 11 children in a day nursery. Milk consumed by the children was known to have been pecked by magpies on occasions. Illness was significantly associated with consumption of milk on a single morning. Examination of milk from a bottle pecked after the outbreak yielded campylobacters. The level of contamination was approximately six cells of *C. jejuni* per 500 ml of milk.

INTRODUCTION

In England and Wales in 1991 there were over 32000 reported cases of campylobacter enteritis (Cowden, personal communication) caused principally by *Campylobacter jejuni*. Most cases are sporadic and a variety of vehicles is thought to be responsible for infection. The most important is probably contaminated chicken meat [1]. Relatively few outbreaks are reported and, where vehicles have been identified, contaminated water and unpasteurized milk [2–5] are often implicated.

A survey of raw unpasteurized milk on sale to the public revealed that approximately 6% of samples were contaminated with *C. jejuni* [6] although, in general, the levels of contamination were low [6, 7]. *Campylobacter jejuni* is heat sensitive and is killed, easily, by the proper pasteurization of milk. Where outbreaks related to pasteurized milk have occurred [8] faults in pasteurization are thought to have been responsible. Such occurrences are rare and pasteurized milk has an enviable safety record. Recent reports [9–11] have indicated that consumption of milk from bottles pecked by corvid birds, principally magpies (*Pica pica*) and jackdaws (*Corvus monedula*) is significantly associated with sporadic cases of campylobacter enteritis.

It is well established that sporadic campylobacter infections show a predictable annual peak in late spring. Evidence from the studies outlined above has suggested that consumption of bird-pecked milk may be a significant factor in this seasonal increase. We present a point source outbreak in which bird-pecked milk was implicated.

MATERIALS AND METHODS

The outbreak

At the end of May 1991, a telephone report was received of an outbreak of diarrhoeal illness at a day nursery. The initial report was of 15 children being affected over a period of 4 weeks. Stool samples had been submitted from two of the more severely affected children and had yielded *Campylobacter* sp. An investigation was instigated.

The nursery had a total of 69 children on register although the maximum attendance per session was 42. The children were divided into four units by age group. Each unit functioned independently having its own main room, toilets and washing facilities. Paper hand towels were provided. A rigorous environmental cleaning programme was in operation for all areas including the toilets.

The following meals were served each day: mid morning milk and fruit, cooked lunch, afternoon milk with sandwiches.

Each day 20 pints of pasteurized milk in glass, one-pint bottles with silver caps were delivered before 08.00 when staff arrived. The nursery was surrounded by mature trees and magpies were known to be prevalent in the area. A system of covering the bottles to prevent pecking was in operation but apparently the milkman sometimes forgot to use it. Questioning of the cook revealed that, on occasions, pecked milk was given to children to drink without further treatment. Milk was distributed to the four units in jugs, the allocation being around 1½–2 pints per unit for the older age groups.

*Microbiological investigations**Clinical samples*

Stool samples were requested from all those children noted to have had symptoms, together with an equal number of children who had not had any illness. All samples were cultured for salmonellas, shigellas and campylobacters using standard methods. Samples from children with diarrhoea were examined for ova and cysts and stained by modified Ziehl Neelsen for *Cryptosporidium* sp. Specimens obtained within 48 h of onset of symptoms were examined for viruses by electron microscopy using a double spin concentration technique.

Examination of milk samples

The nursery staff were instructed that milk for consumption must be protected from pecking, however, over a period of 3 weeks, three bottles of milk not for consumption were deliberately left uncovered each day. The milk in two bottles where the tops had been pecked was examined for the presence and numbers of campylobacters using a most probable numbers (MPN) technique adapted from methods shown to maximize the detection of *C. jejuni* in naturally contaminated milk samples. [6, 7].

One 250 ml sample, five 50 ml samples and five 5 ml samples of milk from each bottle were mixed with equal volumes of double strength 'Exeter' medium [12] and incubated and cultured as described previously [6, 7, 12]. Isolates were confirmed as *C. jejuni* using standard methods.

Table 1. Correlation of campylobacter results with illness

	Campylobacter culture	
	+	-
No illness	0	15
Ill outside period 20-22 May	0	9
Illness with onset 20-22 May	6	0

Table 2. Correlation between date of onset of illness and severity of symptoms

	Illness resulting in absence from nursery	
	+	-
Onset 20-22 May	11	0
Onset at other period	4	6

Epidemiology

The nursery records were scrutinized for details of attendance. Information was obtained on unexpected absences due to illness and the nature of illnesses experienced by children. Details were also requested of milk consumption for all the children in the nursery. The children in the nursery were regarded as a cohort. Associations between exposures and illness were compared using Fisher's exact test.

RESULTS

Examination of stool samples

A total of 30 stool specimens was received, 15 being from children with symptoms. *Campylobacter* sp. were isolated from six stool specimens from symptomatic children. All other investigations were negative.

Isolation of campylobacters from milk

Campylobacter jejuni, at a level of six cells per 500 ml of milk, was isolated from one of the two bottles of pecked milk examined.

Epidemiology

The epidemic curve for all cases of gastrointestinal illness shows the spread of gastrointestinal illness over a period of 5 weeks. However there was an obvious peak of cases in the period 20-22 May 1991. All the cases with stools positive for campylobacter occurred during this period (Table 1). This association was statistically significant ($P = 0.0002$, Fisher's exact test). Table 2 indicates that illness during this period was more likely to result in absence from the nursery ($P = 0.004$, Fisher's exact test). On this basis it was concluded that the cases occurring in the week beginning 20-22 May constituted a distinct entity and the case definition was constructed as follows: An illness involving acute diarrhoea

Table 3. *Correlation between cases and attendance*

	Exposed		Not exposed		
	Ill	Not ill	Ill	Not ill	
Attendance on 17 May 91 a.m.	11	29	0	29	$P < 0.001$
Attendance on 17 May 91 p.m.	10	31	1	27	$P < 0.02$

Table 4. *Correlation between cases and milk consumption*

	Regularly drank milk		Did not drink milk		
	Ill	Not ill	Ill	Not ill	
All children present on 17 May 91 a.m.	11	20	0	9	$P < 0.036$

sufficient to result in absence from the nursery for at least one day, with onset 20–22 May 1991.

Using this case definition, exposure to possible environmental sources was examined. The incubation period of campylobacter infection is typically 3–5 days so that the epidemic curve for cases was suggestive of a point source. Table 3 shows there was a strong association between cases and attendance on the morning of 17 May 1991 ($P = 0.001$, Fisher's exact test).

Not all children attending the nursery drank bottled cows' milk. A number of babies drank expressed breast milk, other children drank soya milk or skimmed milk. Table 4 shows that illness was associated with consumption of bottled cows' milk for those children present on the morning of 17 May 1991 (putative time of exposure).

DISCUSSION

It seems clear cut that amidst the background of other, presumably viral, cases of gastroenteritis, a discreet outbreak of campylobacter infection occurred as a tight cluster. Cases appear to have been exposed on a single day. There was no obvious food source but infection was associated with drinking bottled cows' milk. This, together with the history of pecking of milk by magpies and the subsequent isolation of campylobacters from pecked milk, strongly suggests that the source of infection was pecked milk. This highlights the need for public education since simple measures to prevent consumption of pecked milk would eliminate this source of infection.

ACKNOWLEDGEMENTS

We are grateful to the staff of the nursery and colleagues in Exeter City Council Environmental Health Department for their collaboration in investigating the outbreak and to Mrs P. Goodman for typing the manuscript.

REFERENCES

1. Shane SM. The significance of *Campylobacter jejuni* infection in poultry: a review. *Avian Pathol* 1992; 189–213.

2. Robinson DA, Jones DM. Milk-borne campylobacters infection. *BMJ* 1981; **282**: 1374–6.
3. Kornblatt AN, Barrett T, Morris GK, Tosh FE. Epidemiologic and laboratory investigation of an outbreak of campylobacter enteritis associated with raw milk. *Am J Epidemiol* 1985; **122**: 884–9.
4. Palmer SR, Gully PR, White JM, et al. Water-borne outbreak of campylobacter gastroenteritis. *Lancet* 1983; **i**: 287–90.
5. Report of the Committee on the Microbiological Safety of Food. The microbiological Safety of Food, Part 1, London: HMSO, 1990.
6. Humphrey TJ, Hart RJC. Campylobacter and salmonella contamination of unpasteurized cows' milk on sale to the public. *J Appl Bacteriol* 1988; **65**: 463–7.
7. Humphrey TJ, Beckett P. *Campylobacter jejuni* in dairy cows and raw milk. *Epidemiol Infect* 1987; **98**: 263–9.
8. Barrett NJ. Communicable disease associated with milk and dairy products in England and Wales. *J Infect* 1986; **12**: 265–72.
9. Hudson SJ, Sobo AO, Russell K, Lightfoot NF. Jackdaws as a potential source of milkborne *Campylobacter jejuni* infection. *Lancet* 1990; **335**: 1160.
10. Southern JP, Smith RMM, Palmer SR. Bird attack on milk bottles: possible mode of transmission to man. *Lancet* 1990; **336**: 1425–7.
11. Hudson SJ, Lightfoot NF, Coulson JC et al. Jackdaws and magpies as vectors of milkborne human campylobacter infection. *Epidemiol Infect* 1991; **107**: 363–72.
12. Humphrey TJ. Techniques for the optimum recovery of cold injured *Campylobacter jejuni* from milk or water. *J Appl Bacteriol* 1986; **61**: 125–32.