
SHORT PAPER

A longitudinal study of *Escherichia coli* O157 in fourteen cattle herds

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SUMMARY

Escherichia coli O157 shedding in 14 cattle herds was determined by faecal culture at intervals of approximately 1 month for up to 13 months. The overall prevalence was 1·0% (113/10832 faecal samples) and 9 of the 14 herds were detected as positive. Herds positive 2 years previously ($n = 5$) had a higher prevalence of positive cattle (median = 1·9%) than herds which had been negative on a previous sampling ($n = 8$, median = 0·2%). Weaned heifers had a higher prevalence (1·8%) than did unweaned calves (0·9%) or adults (0·4%). For all herds the highest prevalence occurred in the summer months, which resulted in most of the positive faecal samples being collected on a minority of sampling visits.

Human illness associated with infection by *Escherichia coli* O157 has been reported with increasing frequency since first reported in 1982 [1–3]. Considerable evidence points to a bovine reservoir for many of the known food-borne *E. coli* O157 infections in humans [4, 5]. Thus, many recent studies have focused on the epidemiology of this organism in cattle herds [6–9].

In order to provide a minimum estimate of the herd prevalence of *E. coli* O157 in the Northwestern USA, faecal samples were collected from animals of all ages in 60 dairy herds during 1991–2 [6]. *E. coli* O157 was found in 5 (8·3%) of these 60 herds.

The purpose of this study was to use longitudinal sampling in selected cattle herds to improve the estimate of herd prevalence of *E. coli* O157 (proportion of herds in which the agent exists), and to provide additional evidence on seasonal variation in the prevalence of *E. coli* O157 shedding in cattle [6, 7]. An additional purpose of this study was to determine

the prevalence of *E. coli* O157 over time in a contract calf raising operation which received calves from many dairy herds.

Thirteen dairy herds and one contract dairy calf raising operation in the states of Washington, Oregon, and Idaho were visited in intervals of approximately 1 month. Herds that had been negative for *E. coli* O157 in a 1991–2 survey (7 herds) [6] or 1993 (1 herd) were designated ‘initially negative’ (IN). Herds which had tested positive for *E. coli* O157 in 1991–2 (4 herds) [6] or 1993 (1 herd) were designated ‘initially positive’ (IP). The contract calf raising operation included in the study received each week 25–60 calves aged < 1 week from approximately 25 dairy herds.

Where possible in both IN and IP herds, 60 or more faecal samples were collected on monthly visits to each farm distributed as follows; rectal swabs from 10 unweaned calves, and 20, 10 and 20 fresh faecal pat swabs from weaned heifers, non-lactating cows and

Table 1. Summary of results obtained from sampling for *E. coli* O157 in 13 dairy herds and 1 calf raising operation

Herd status*	Date range during which herd was sampled monthly		Number of visits	Number sampled	Number (%) + for <i>E. coli</i> O157
IP	JUN93	JUN94	13	744	15 (2.0%)
IP	AUG93	JUL94	10	603	18 (3.0%)
IP	JUN93	JUN94	14	1836	23 (1.3%)
IP	JUN93	JUN94	12	1232	24 (1.9%)
IP	JUN93	MAY94	12	982	0 (0%)
Total				5397	80 (1.5%) Median = 1.9%
IN	MAR94	MAY94	3	165	0 (0.0%)
IN	JUN93	NOV93	6	330	1 (0.3%)
IN	JUN93	NOV93	6	349	2 (0.6%)
IN	MAR94	MAY94	3	180	0 (0%)
IN	MAR94	MAY94	3	174	0 (0%)
IN	JUN93	JUL94	17	1331	5 (0.4%)
IN	JUN93	JUN94	12	696	3 (0.4%)
IN	JUL93	JUN94	12	695	0 (0%)
Total				3920	11 (0.3%) Median = 0.2%
Calf raiser	MAY93	JUN94	15	1515	22 (1.5%)
Grand total				10832	113 (1.0%)

* *E. coli* O157 status based on single prior sampling of 60 animals; IP, initially positive; IN, initially negative herds.

lactating cows. Consecutive visits to IP herds ranged from 10–14, and for IN herds 3–17.

In the calf raising operation four groups of 25–60 calves were sampled monthly by rectal swab for the time they were on the premises (approximately 6 months). In addition, 60 fresh faecal pats from weaned heifers in other cohorts were also collected each month.

All samples were cultured [10] and *E. coli* O157 isolates were assayed for the Shiga-like toxin gene types I and II by PCR [11] or by using DNA probes [12] in non-radioactive colony hybridizations [13].

A total of 10832 faecal samples were collected and 113 (1.0%) yielded *E. coli* O157 (Table 1). This is somewhat higher than previously reported in the same region [6], possibly due to improvements in detection methods [10]. Consistent with previous findings, a higher prevalence was observed in weaned heifers (58/3483 = 1.7%) than in unweaned calves (13/1040 = 1.3%) or adults (20/4762 = 0.4%).

Despite receiving 25–60 new calves per week from numerous farms, the prevalence of *E. coli* O157 in the calf raising operation (22/1,515 = 1.5%) was similar to that observed among heifers maintained in dairy

herds. This indicates that *E. coli* O157 prevalence on a farm is probably not a function of the number of animal movements into that farm.

When 60 faecal samples were collected on a single visit, *E. coli* O157 was previously found in only 5 of 60 (8.3%) herds [6]. Using monthly sampling, however, *E. coli* O157 was identified on 1 or more occasions in 4 of 8 IN herds. This suggests that a survey involving a single sampling of a herd will underestimate the prevalence of infected herds. The prevalence observed in IN herds was much lower than that in IP herds (11/3920 = 0.3% vs. 80/5397 = 1.5%) suggesting that higher *E. coli* O157 prevalence is a temporally stable condition in some herds.

A tendency for seasonal *E. coli* O157 shedding with a peak occurrence in summer was corroborated in the present study where the prevalence of *E. coli* O157 peaked at 2.6% in June and was lowest in December (0%) (Fig. 1). This seasonal pattern was consistent in all age groups tested and suggests that seasonal variation in the carriage of *E. coli* O157 by cattle may contribute to the similar seasonal pattern of human disease associated with this organism [3].

Among the seven herds followed for 10 or more

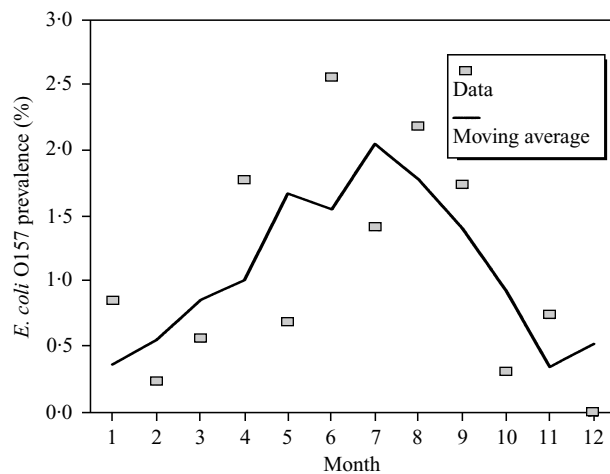


Fig. 1. Monthly prevalence of *E. coli* O157 in all herds followed for approximately 1 year ($n = 9$).

monthly sampling visits, and in which *E. coli* O157 was found, the shedding was strongly clustered temporally such that most positive samples were detected on only a few sample dates within each herd. An average of 51% (range, 30–100%) of all positive samples were detected on the single sampling visit with the greatest number of positives. For 6 of the 7 herds, the visit with the greatest number of positives occurred during the period of April–August; a seventh herd had the same prevalence for the months of April and November. No positive samples were found on 63% of all sampling visits (60% of all samples) to these 7 herds. Thus, the typical pattern of *E. coli* O157 excretion by cattle herds appears to be characterized by short periods with a relatively high prevalence of excretion separated by longer periods of reduced or undetectable shedding.

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REFERENCES

1. Neill MA, Tarr PI, Clausen CR, Christie DL, Hickman RO. *Escherichia coli* O157:H7 as the predominant pathogen associated with the hemolytic uremic syndrome: a prospective study in the Pacific Northwest. *Pediatrics* 1987; **80**: 37–40.
2. MacDonald KL, O'Leary MJ, Cohen ML, et al. *Escherichia coli* O157:H7, an emerging gastrointestinal pathogen. *J Am Med Assoc* 1988; **259**: 3567–70.
3. Ostroff SM, Kobayashi JM, Lewis JH. Infections with *Escherichia coli* O157:H7 in Washington State. The first year of statewide disease surveillance. *J Am Med Assoc* 1989; **262**: 355–9.
4. Wells JG, Shipman LD, Greene KD, et al. Isolation of *Escherichia coli* serotype O157:H7 and other Shiga-like-toxin-producing *E. coli* from dairy cattle. *J Clin Microbiol* 1991; **29**: 985–9.
5. Griffin PM, Tauxe RV. The epidemiology of infections caused by *Escherichia coli* O157:H7, other enterohemorrhagic *E. coli*, and the associated hemolytic uremic syndrome. *Epidemiol Rev* 1991; **13**: 60–98.
6. Hancock DD, Besser TE, Kinsel ML, Tarr PI, Rice DH, Paros MG. The prevalence of *Escherichia coli* O157:H7 in dairy and beef cattle in Washington State. *Epidemiol Infect* 1994; **113**: 199–207.
7. Hancock DD, Rice DH, Herriott DE, Besser TE, Ebel EE, Carpenter LV. The effects of farm manure handling practices on *Escherichia coli* O157 prevalence in cattle. *J Food Protect.* In press.
8. Chapman PA, Siddons CA, Wright DJ, Norman P, Fox J, Crick E. Cattle as a possible source of verocytotoxin-producing *Escherichia coli* O157 infections in man. *Epidemiol Infect* 1993; **111**: 139–47.
9. Garber LP, Wells SJ, Hancock DD, Doyle MP, Tuttle J, Shere JA, Zhao T. Risk factors for fecal shedding of *Escherichia coli* O157:H7 in dairy calves. *J Am Vet Med Assoc* 1995; **207**: 46–9.
10. Sanderson MW, Gay JM, Hancock DD, Gay CC, Fox LK, Besser TE. Sensitivity of bacteriologic culture for detection of *Escherichia coli* O157:H7 in bovine feces. *J Clin Microbiol* 1995; **33**: 2616–9.
11. Begum D, Strockbine NA, Sowers EG, et al. Evaluation of a technique for identification of Shiga-like toxin-producing *Escherichia coli* by using polymerase chain reaction and digoxigenin-labeled probes. *J Clin Microbiol* 1993; **31**: 3153–6.
12. Newland JW, Niell RJ. DNA probes for Shiga-like toxins I and II and for toxin-converting bacteriophages. *J Clin Microbiol* 1988; **26**: 1292–7.
13. Riely LK, Caffrey CJ. Identification of enterotoxigenic *Escherichia coli* by colony hybridization with non-radioactive digoxigenin-labeled DNA probes. *J Clin Microbiol* 1990; **28**: 1465–8.