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SHORT PAPER

An outbreak of *E. coli* O157 infection with evidence of spread from animals to man through contamination of a private water supply

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SUMMARY

An outbreak of *E. coli* O157 infection occurred in the Highland Region of Scotland in the summer of 1999. The source of the outbreak was traced to an untreated private water supply. All six cases identified arose in visitors to the area, and most had very limited exposure to the contaminated water. Permanent residents on the same supply were unaffected. The *E. coli* O157 isolates from the water, sheep faeces collected from around the source and the human stool samples were indistinguishable using pulsed field gel electrophoresis. Previously reported outbreaks of *E. coli* O157 linked to potable water supplies have resulted from structural or treatment failures, which allowed faecal contamination of source water. Here, contamination of the water supply and subsequent human infection was due to the use of an untreated, unprotected private water source in a rural area where animals grazed freely.

Escherichia coli O157 was first reported to have been isolated from water in 1989 [1]. Drinking water has been implicated in human *E. coli* O157 infection, but proven or strongly suspected outbreaks of the infection linked to potable water supplies remain infrequent, although such outbreaks have caused significant morbidity and mortality [2–8]. Contamination of water supplies with *E. coli* O157 is presumed to arise from faecal wastes from the main animal reservoirs (cattle, sheep and man). Due to the difficulty of isolating organisms from environmental samples however, confirmation of the likely source and route of contamination is rarely discovered. We are aware of only two previous outbreaks in the United Kingdom where there has been strong microbiological evidence of a connection between contamination of potable water supplies by animal faeces and human illness (personal communication) [4]. Here, we report on an outbreak of *E. coli* O157 infection in which strains of the organism, indistinguishable using pulsed field gel electrophoresis, were isolated from animals, a private water supply and the human cases.

This outbreak occurred in the summer of 1999 in Applecross, a small village on the west coast of Scotland. Over a 7-week period, six cases of *E. coli* O157 infection in tourists visiting Applecross were investigated by the Public Health Department of Highland Health Board (Table 1).

All of the cases were linked to a campsite, which was provided with untreated drinking water from a private supply that originated in a 'spring' in an area of land grazed by sheep and deer. Surveillance of the water was carried out by Protective Services (Environmental Health) officers from the Highland Council in accordance with current regulations. A sample taken 1 week prior to the first case of illness had failed bacteriological tests (total coliforms of 11 c.f.u./100 ml and *E. coli* of 15 c.f.u./100 ml), resulting in the issue of a 'Boil Water' notice. Further samples taken during the outbreak repeatedly showed levels of indicator organisms above the relevant Significant

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Table 1. *Summary of cases and microbiological results*

Date of onset (1999)	Age (years)	Direct culture	IMS	Serology	Phage type	Toxin type	Comments
26 May	3	No stool specimen	NA	−	NA	NA	Ate at campsite café
1 June	1	+	+	−	21/28	VT_1^-, VT_2^+	Sibling of first case. Likely secondary spread
21 July	8	−	−	+	NA	NA	HUS. Using unboiled for brushing teeth
21 July	4	+	+	−	21/28	VT_1^-, VT_2^+	Single exposure to contaminated water
29 July	5	+	+	−	21/28	VT_1^-, VT_2^+	Drinking water over 2–3 h only
29 July	Adult	−	+	−	21/28	VT_1^-, VT_2^+	As above

Medical Risk Values. Analysis of water samples for *E. coli* O157 was carried out using routine methods at the microbiology laboratory of the local District General Hospital and was negative. Samples sent to the Scottish *E. coli* O157 Reference Laboratory in Aberdeen for analysis using immunomagnetic separation did, however, show the presence of *E. coli* O157 in the water samples.

Water samples were also taken from five houses on the same private supply as the campsite. *E. coli* O157 was isolated from one sample, although none of the permanent residents of these houses reported any gastrointestinal symptoms during this outbreak.

Samples of sheep and deer faeces were collected from the campsite and the area around the private water supply source. These were tested by immunomagnetic separation at the Scottish Agricultural College in Inverness using the method described by Synge et al. [9]. *E. coli* O157 was isolated from two samples of sheep faeces taken from near the water source.

All *E. coli* O157 isolates from the cases, water samples and sheep faeces were subjected to phage typing and pulsed field gel electrophoresis at the *E. coli* O157 Reference Laboratory in Aberdeen. All were confirmed as phage type 21/28, VT_1 negative, VT_2 positive. The isolates were indistinguishable on pulsed field gel electrophoresis. The combination of the microbiological evidence and the absence of any other common factor linking the cases strongly supports the hypothesis that *E. coli* O157 was spread from animals to man via contamination of the private water supply [10].

The restriction of symptomatic infection to tourists in this outbreak is consistent with previous research highlighting varying patterns of susceptibility to *E. coli* O157 and an increased risk of infection associated with travel and transient populations [11, 12], although the mechanism for this variable susceptibility is debated [13, 14]. Four of the cases appear to have had very limited exposure to the contaminated water, consistent with the known low infective dose for *E. coli* O157 [15]. These findings raise questions over the acceptability of allowing the use of untreated private water supplies, especially by transient populations in guest-houses, holiday cottages and on campsites. At the very least, it would seem appropriate to inform visitors where water supplies are untreated, and advise them of the potential risks.

The Private Water Supplies (Scotland) Regulations, 1992 prescribe sampling frequencies and standards for private supplies [16]. These regulations do not however, make specific reference to testing for the presence of *E. coli* O157 when waters fail to meet indicator organism standards, and practices vary between laboratories. Due to the very low infective dose of *E. coli* O157, significant risk of infection with this organism may arise in waters that only just meet standards for indicator organisms [17]. If *E. coli* O157 is detected, there is currently no national protocol for action by public and environmental health departments.

In the period July to December 1999, 39% of category two private water supplies tested in England and Wales (which includes supplies to hospitals, residential homes, holiday sites and food preparation premises) were positive for *E. coli* [18]. These statistics reveal the extent of the potential risk from private water supplies in areas where *E. coli* O157 is likely to be a contaminant.

E. coli O157 is often not isolated from water samples even when there is good circumstantial evidence that water is the source of the infection [19]. This may be due to inappropriate technique or insensitive culture methods, but also to the likely sporadic nature of such contamination, which makes

multiple samples and rigorous analytical procedures essential.

Since the *E. coli* O157 present in the water may be 'stressed' they may only grow on first incubation at 37 °C and not at 44 °C. Hence they may be dismissed as coliforms if subculturing and further tests for O157 are not performed. Equally, since the levels of *E. coli* O157 in the water may be low direct plating of a routine 100 ml filtered sample may fail to demonstrate the organism. It is recommended that when there is a high index of suspicion a 5 l sample of water is filtered and tested by immunomagnetic separation thereby increasing the sensitivity and specificity of the isolation method.

Carriage of *E. coli* O157 in sheep has been demonstrated, although the rate of carriage may not be as high as in cattle [20]. This makes the isolation of the organism from environmental samples in this outbreak even more unusual. Lambing ewes were associated with human cases of *E. coli* O157 infection [21], but never before have sheep on a hillside without direct contact with man been implicated.

A Scotland-wide survey of carriage rates in cattle is currently underway, and it may be valuable to extend this to other farm species, and possibly even to wild mammals such as deer and to birds, which have also been proposed as reservoirs of infection.

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REFERENCES

1. McGowan KL, Wickersham E, Strockbine NA. *Escherichia coli* O157:H7 from water. Lancet 1989; i: 967–8.
2. Jones IG, Roworth M. An outbreak of *Escherichia coli* O157 and campylobacteriosis associated with contamination of a drinking water supply. Public Health 1996; **110**: 277–82.
3. Dev VJ, Main M, Gould I. Waterborne outbreak of *E. coli* O157. Lancet 1991; **337**: 1412.
4. Communicable Disease Surveillance Centre. Outbreak of verocytotoxin producing *Escherichia coli* O157 infection in Dorset. CDR 1998; **8**: 186.
5. Swerdlow DL, Woodruff BA, Brady RC, et al. A waterborne outbreak in Missouri of *Escherichia coli* O157:H7 associated with bloody diarrhoea and death. Ann Int Med 1992; **117**: 812–9.
6. Jackson SG, Goodbrand RB, Johnson RP, et al. *Escherichia coli* O157:H7 diarrhoea associated with well water and infected cattle on an Ontario farm. Epidemiol Infect 1998; **120**: 17–20.
7. Centres for Communicable Disease Control. Outbreak of *Escherichia coli* O157:H7 and campylobacter among attendees of the Washington county fair – New York, 1999. MMWR 1999; **48**: 803–4.
8. Pebody RG, Furtado C, Rojas A, et al. An international outbreak of verocytotoxin-producing *Escherichia coli* O157 infection amongst tourists: a challenge for the European infectious disease surveillance network. Epidemiol Infect 1999; **123**: 217–23.
9. Synge BA, Ternent H, Hopkins GF, et al. A comparison of buffered peptone water with and without antibiotics for the isolation of *E. coli* O157 from bovine faeces using immunomagnetic separation. In: Duffy G, Garvey P, Coia J, Wasteson Y, McDowell DA, eds. Proceedings of Concerted Action CT98-3935-verocytotoxigenic *E. coli* in Europe. 1. Methods for verocytotoxigenic *E. coli*. Dublin: Teagasc, 1998: 171.
10. Communicable Disease Surveillance Centre. Strength of association between human illness and water: revised definitions for use in outbreak investigations. CDR 1996; **6**: 65.
11. Benton C, Forbes GI, Paterson GM, Sharp JCM, Wilson TS. The incidence of waterborne and water-associated disease in Scotland from 1945 to 1987. Water Science Technol 1989; **21**: 125–9.
12. Adak GK, O'Brien SJ, Gilliam C, Smith HR. The PHLS case-control study of *Escherichia coli* O157 infection in England. In: Third SCIEH verocytotoxigenic *E. coli* update. Glasgow: Scottish Centre for Infection and Environmental Health, 1999: 11.
13. Heuvelink AE, Van de Kar NC, Van Der Velden TJ, Chart H, Monnens LA. Verocytotoxin-producing *Escherichia coli* infection in household members of children with haemolytic-uraemic syndrome in the Netherlands. Pediatr Infect Dis J 1999; **18**: 709–14.
14. Currie CG. Mucosal and systemic immune responses to the lipopolysaccharide of *E. coli* O157. In: Third SCIEH verocytotoxigenic *E. coli* update. Glasgow: Scottish Centre for Infection and Environmental Health, 1999: 35.
15. Griffin PM, Tauxe RV. The epidemiology of infections caused by *Escherichia coli* O157:H7, other enterohaemorrhagic *E. coli*, and the associated haemolytic uraemic syndrome. Epidemiol Rev 1991; **13**: 60–98.
16. Guidance Notes on the private water supplies (Scotland) regulations. Edinburgh: Scottish Office Environment Department, 1992.

17. Stanfield G, Gale P, Carrington E. Scoping study on *E. coli* O157 in water. Edinburgh: Scottish Office, 1997.
18. Communicable Disease Surveillance Centre. Surveillance of waterborne diseases and water quality: July to December 1999. CDR 2000; **10**: 65–7.
19. Chalmers RM, Aird H, Bolton FJ. Waterborne *Escherichia coli* O157. J Appl Microbiol Symposium Supplement 2000; **88**: 124S–32S.
20. Chapman PA, Siddons CA, Harkin MA. Sheep as a potential source of verocytotoxin-producing *Escherichia coli* O157. Vet Rec 1996; **138**: 23–4.
21. Allison L, Thomson-Carter F, Gray D, Rusbridge S, MacLennan M. Human cases of *E. coli* O157:H7 infection associated with exposure to lambing ewes. In: Association of Veterinary Teachers and Research Workers, 51st Scientific Meeting, 1998: 28.