

EPIDEMIOLOGY

Verocytotoxin-producing *Escherichia coli* (VTEC) O157 and other VTEC from human infections in England and Wales: 1995–1998

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A total of 3429 isolations of verocytotoxin-producing *Escherichia coli* O157 (VTEC O157) was confirmed from human sources in England and Wales during the period 1995–1998. The largest annual total was 1087 in 1997. Most infections occurred in the third quarter of each year. The overall rate of infection ranged from 1.28 to 2.10/100 000 population and showed regional variation. The highest incidence was in children aged 1–4 years. Annually, between 5% and 11% of strains were from patients who had travelled abroad. There were 67 general outbreaks of infection represented by 407 (11.9%) VTEC O157 isolates. Outbreaks involved transmission by contaminated food or water, person-to-person spread and direct or indirect animal contact, and five were associated with foreign travel. The majority (76%) of strains carried verocytotoxin (VT) 2 genes and 23.3% were VT1+VT2. Most strains had the flagellar antigen H7, but *c.* 14% were non-motile. Approximately 20% of isolates were resistant to antimicrobial agents, predominantly streptomycin, sulphonamides and tetracycline. In addition to VTEC O157, strains of serogroup O157 that did not possess VT genes were identified. These were either derivatives of VTEC O157 that had lost VT genes or were strains with H antigens other than H7 that have never been associated with VT production. Strains of VTEC other than O157 were characterised. Most were associated with diarrhoea, bloody diarrhoea or haemolytic uraemic syndrome and had virulence markers in addition to VT.

Introduction

Verocytotoxin-producing *Escherichia coli* O157 (VTEC O157) are associated with human disease including mild diarrhoea, severe bloody diarrhoea with abdominal pain and, in up to 10% of cases, haemolytic uraemic syndrome (HUS) [1, 2]. The organism causes sporadic and epidemic infections. The largest outbreak in the UK occurred in Central Scotland in 1996, resulting in >500 cases and 21 deaths [3]; a series of related outbreaks in Japan in 1996 involved >10 000 cases [4, 5]. Although O157 is the most important VTEC serogroup, >100 serotypes of *E. coli* have been shown to produce verocytotoxin(s) (VT). Some non-O157 VTEC have been associated with outbreaks of infection [6–9].

Transmission of VTEC O157 to man occurs mainly by

consumption of contaminated food, including undercooked beef and meat products, unpasteurised milk and ready-to-eat products including cooked meats and vegetables that have been contaminated [2]. Healthy cattle are the main reservoir of the organism, but it is also carried by sheep and other animals [10, 11]. Direct or indirect contact with animals provides an alternative route by which infection is acquired [12–16] and person-to-person transmission of the organism occurs in families and institutional settings such as day nurseries and nursing homes [17–20].

The importance of disease due to VTEC O157 and other VTEC has been increasingly recognised [21–23]. Surveillance of VTEC-associated infection is now undertaken by several countries in the European Community and elsewhere, although the criteria and methods applied vary. Since the last detailed report of laboratory surveillance in England and Wales [18] there has been a rise in the number of infections caused by VTEC O157 and in the number of outbreaks. This study reports the continued laboratory surveillance of VTEC O157 in England and Wales from 1995 to 1998.

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Materials and methods

Isolation of E. coli O157 in diagnostic laboratories

Stool samples from patients with diarrhoea, bloody diarrhoea and haemolytic uraemic syndrome were examined for *E. coli* O157 by National Health Service (NHS), Public Health Laboratory Service (PHLS) and private laboratories in England. In Wales, testing of all first-time acute stool samples for *E. coli* O157 has been performed since 1990 [24]. During investigation of sporadic infections and outbreaks, stools from asymptomatic contacts were sometimes tested for carriage of *E. coli* O157. Faecal samples were plated on Sorbitol MacConkey agar (SMAC), or SMAC containing cefixime 0.05 mg/L and potassium tellurite 2.5 mg/L (CT-SMAC) and putative *E. coli* O157 were identified as sorbitol non-fermenting colonies after incubation for 18 h at 37°C [25, 26]. Colonies were tested for agglutination with anti-O157 serum or anti-O157 latex agglutination kits and identified biochemically as presumptive *E. coli* with commercially available tests before referral to the Laboratory of Enteric Pathogens (LEP).

Confirmation of VTEC O157

Isolates received by LEP were confirmed biochemically as *E. coli* and serotyped [27]. Resistance to antimicrobial agents was determined by an agar dilution method [28]. The final plate concentrations of the drugs were (mg/L): ampicillin, 8; chloramphenicol, 8; gentamicin, 4; kanamycin, 16; sulphonamide, 64; tetracycline, 8; trimethoprim, 2; furazolidone, 8; nalidixic acid, 16; ciprofloxacin, 0.125 and 1.0. Strains were tested by colony hybridisation for the presence of VT1 and VT2 genes with digoxigenin-labelled DNA probes [29] and with the fluorescein-labelled *eae*O157 probe directed against the C-terminal end of the *eae* gene of VTEC O157 [30]. Some strains confirmed as *E. coli* O157 did not hybridise with VT probes and were investigated further by PCR.

Further examination of faecal samples

Stools from some patients with bloody diarrhoea or HUS did not yield *E. coli* O157 on primary screening. These faecal samples were sent to LEP to test for low levels of *E. coli* O157 not detected by direct plating and for the presence of VTEC belonging to other O serogroups. Enrichment culture for *E. coli* O157 was in trypticase-soy broth containing bile salts and novobiocin for 6 h [25, 31]. Immunomagnetic separation (IMS) with anti-O157 beads (Dynal) was performed as described by the manufacturer and beads were plated on CT-SMAC [32]. Sorbitol-non-fermenting colonies were picked, their identity was confirmed and they were tested for the presence of VT genes.

Preliminary evidence of the presence of other VTEC in stools was obtained by PCR. A sweep of growth from

SMAC agar was grown for 2–3 h in nutrient broth; the culture was boiled and 2 µl were added to a multiplex PCR reaction (see below). Specimens with evidence of amplification of VT sequences were re-plated on to MacConkey agar to yield separated single colonies that were replica-plated on to nylon membrane. VT-positive colonies were identified by hybridisation of the filter with a mixture of VT1 and VT2 probes and then picked from the retained master plate [18]. They were confirmed as *E. coli*, serotyped and tested with individual VT probes.

PCR tests

A multiplex PCR tested coliform growth from faecal samples for the presence of VT genes and the C-terminal end of the *eae* gene of *E. coli* O157. The sequences of the primers for detection of VT were: primer P6, 5' GGCAGATACAGAGGGAATTTTCG 3'; primer P7, 5' TGATGATGGCAATTCAGTAT 3'. The product size was 217 bp. This PCR detects but does not differentiate VT1 and VT2 and amplifies the VT2, VT2c and VTe gene subtypes. The *eae* primers that amplified a fragment of 410 bp have been described previously [30]. Although this reaction is not totally specific for O157 VTEC, very few other VTEC serotypes give this product [30]. In the PCR reactions, annealing was at 55°C and extension at 72°C. Products were separated on agarose 2% gels [33].

Tests on VTEC of serogroups other than O157

Confirmed VTEC strains that belonged to serogroups other than O157 were tested by DNA probing for the presence of the *eae* gene [34] and for sequences associated with enterohaemolysin production [35].

Analysis of data

VTEC O157 strains were received from laboratories in England and Wales and confirmed in LEP as *E. coli* O157 that encoded a verocytotoxin. Isolates that were negative for VT genes by probe and PCR were analysed separately. Duplicate cultures from the same individual were discounted. Within family and general outbreaks, all isolates from different patients were included, as were cultures from contacts, even if they were asymptomatic. For seasonality studies, isolates were included by date of receipt in LEP, as the date of onset of infection was not usually known. Laboratories usually sent cultures within 1–3 days of isolation. If a delay was known to have occurred, the date of isolation was obtained and used in the data set. Analysis of geographical distribution was based on the health region of the laboratory sending the culture. Data associated with foreign travel and general outbreaks were based on information received with the cultures sent to LEP and also from reports to CDSC. Differences in the age-specific incidence rates by gender were analysed by a χ^2 test.

Results

Properties of VTEC O157 strains

From 1995 to 1998, LEP received 3429 VTEC O157 isolates from laboratories in England and Wales (Table 1). The annual totals were greater than those previously reported [17, 18]. The majority of strains (76%) carried VT2 genes only; 23.3% encoded both VT1 and VT2 and VT1-only strains were very rare. All confirmed VTEC O157 failed to ferment sorbitol in 24 h on solid medium. They possessed either the flagellar antigen H7 or were non-motile (Table 1); the proportion of non-motile (H) isolates over the 4-year period was *c.* 14%. DNA hybridisation showed that all the strains possessed the *eae* gene of VTEC O157.

The total included 39 strains of VTEC O157 isolated in LEP by the IMS technique from 400 stool samples that were negative on direct culture in the sending laboratory. These were from patients with bloody diarrhoea or HUS or were contacts of known cases.

Over the 4-year period, an average of 20% of VTEC O157 strains (range 17.4–22.8%) were resistant to one or more antimicrobial agents tested. Drug resistance was a useful epidemiological marker for some VTEC O157 strains [17, 18], but had no relevance to clinical infections. The proportion of resistant strains had not increased since 1994, but was higher than that seen in 1992 (10.2%) and 1993 (14.5%) [18]. The resistance profiles (R-types) were limited, with 87% of strains resistant to SSu, SSuT or SuT (S, streptomycin; Su, sulphonamide; T, tetracycline). Strains resistant to four or more drugs accounted for 1.3% (range 0.5–2.4%) of strains; no more than two strains per year had the same R-type. Resistance to both nalidixic acid and ciprofloxacin was observed for the first time in 1998 in two epidemiologically related cases who had travelled to the Canary Islands.

Epidemiology of VTEC O157 infections

There was a peak of infection each year in the months of July, August and September (Fig. 1). The incidence of infection was highest in children aged 1–4 years

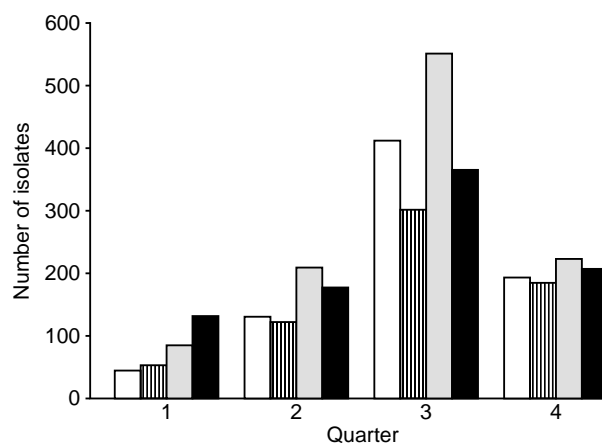


Fig. 1. Quarterly incidence of VTEC O157 in England and Wales, 1995–1998: □, 1995; ▨, 1996; ▩, 1997; ■, 1998.

(Fig. 2). Females > 15 years old were significantly ($p < 0.00001$) more likely than males to be diagnosed as suffering from VTEC O157 infection. This difference was most marked in the 25–34-year-old and 45–59-year-old age groups. VTEC O157 were reported from all regions of England and Wales (Table 2), although the North and South Thames regions had consistently lower incidences. A regional rate exceeded 3/100 000 for the first time in 1997 (Trent region). The regional variation can, in some part, be attributed to outbreaks of infection (see below). A proportion of VTEC O157 infections was linked to foreign travel (Table 3), and in 1997 and 1998 there were general outbreaks of infection associated with travel abroad (see below) especially to Spain, Turkey and Greece.

Outbreaks of infection with VTEC O157

A minority of VTEC O157 infections was associated with 67 general outbreaks of infection (Table 3), these represented 407 isolates [15, 20, 36–40]. Individual outbreaks were small and only six involved ≥ 10 patients; the largest outbreak was in 1995 in North Wales [20]. There was a marked geographical variation in the outbreaks, 28 of which were in two health regions (Table 2). Most outbreaks were in the

Table 1. VTEC O157 from human infections in England and Wales 1995–1998

Year	Total VTEC O157 isolates*	Percent of VTEC O157 isolates that were			
		VT1	VT2	VT1+2	Non-motile (H ⁻) [†]
1995	792	0.6	80.2	19.2	15.8
1996	660	0.6	76.1	23.3	8.0
1997	1087	1.1	72.8	26.1	13.7
1998	890	0.7	74.7	24.6	19.3
Total/overall %	3429	0.7	76.0	23.3	14.4

*Isolates received from laboratories in England and Wales that were confirmed by LEP as VTEC O157. The presence of VT genes was determined by DNA probe [29]. Annual totals for previous years were: 1981 (1); 1982 (1); 1983 (6); 1984 (9); 1985 (30); 1986 (76); 1987 (89); 1988 (49); 1989 (119); 1990 (250); 1991 (361); 1992 (470); 1993 (385); 1994 (411) [17, 18].

[†]Strains hybridising with at least one VT probe that did not express motility [27]. All other VTEC O157 had the flagellar antigen H7.

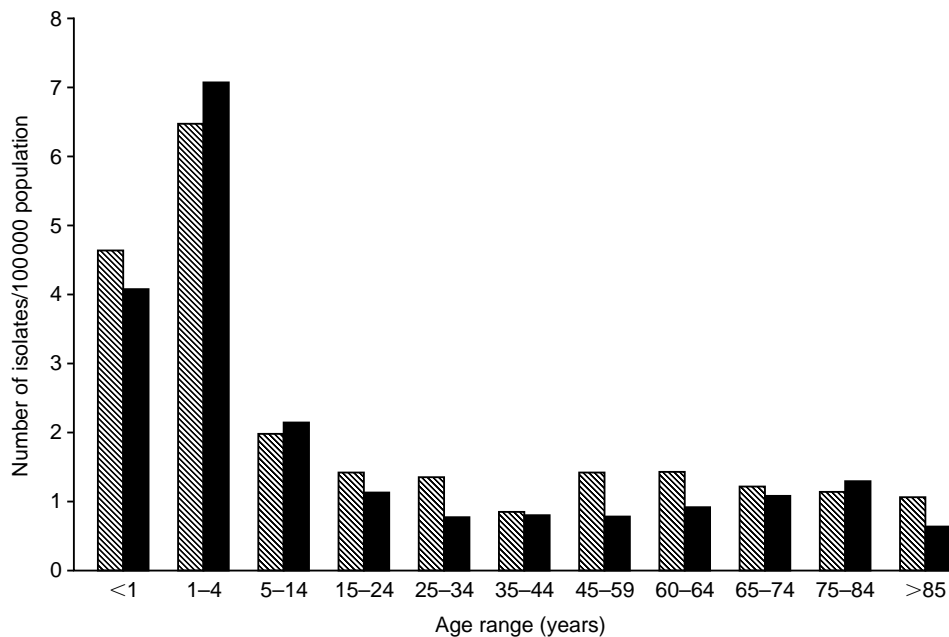


Fig. 2. Rate (/100 000) of isolation of VTEC O157 in England and Wales, 1995–1998, analysed by the age and sex (▨, female; ■, male) of the patient. For each year, data on patient age and sex were available for between 89% and 93% of the total number of strains submitted to LEP.

Table 2. Geographical distribution of VTEC O157 in England and Wales 1995–1998

Region	1995		1996		1997		1998		1995–98 Outbreaks*
	N	Rate	N	Rate	N	Rate	N	Rate	
Northern & Yorkshire	126	1.90	109	1.64	150	2.26	162	2.45	16
Trent	65	1.36	105	2.19	156	3.25	78	1.63	12
Anglia & Oxford	63	1.19	62	1.07	113	2.13	82	1.55	5
North Thames	43	0.63	50	0.73	94	1.37	89	1.30	3
South Thames	55	0.81	46	0.68	71	1.05	87	1.29	2
South & West	165	2.51	110	1.68	162	2.47	138	2.10	9
West Midlands	106	2.00	57	1.07	137	2.58	76	1.43	6
North West	88	1.33	85	1.29	161	2.43	115	1.74	7
Wales	81	2.78	36	1.23	43	1.47	63	2.15	1
Annual total/rate	792	1.54	660	1.28	1087	2.10	890	1.71	61†

N, number of isolates. Rates are calculated /100 000 using Office of National Statistics population figures for 1995 and are not directly comparable to previously published data for 1992–1994 because of boundary changes.

*Assigned on the basis of the location of the sending laboratory. One outbreak in the North West region was included in a report on surveillance of VTEC O157 among Welsh residents [47].

†There were six additional outbreaks during the 4-year period that involved patients from more than one health region.

Table 3. Association of isolates of VTEC O157 from England and Wales with general outbreaks or foreign travel: 1995–1998

Year	Total VTEC O157	Number (%) associated with general outbreaks	Number (%) associated with recent foreign travel
1995	792	113 (14.3)	41 (5.2)
1996	660	69 (10.5)	60 (9.1)
1997	1087	153* (14.1)	117* (10.8)
1998	890	72† (8.1)	90† (10.1)
Total	3429	407 (11.9)	308 (8.9)

*Includes six isolates from patients included in an outbreak in the Canary Islands [39].

†Includes eight isolates from patients included in three separate outbreaks of infection acquired abroad.

community (Table 4) and some were linked to specific restaurants and catering facilities or to consumption of meat from butchers' shops. In at least 27 community-based outbreaks, transmission was probably through

contaminated food or beverages, and in most instances the association was epidemiological. Fourteen outbreaks were in institutional settings including children's day nurseries and residential homes for the elderly

Table 4. General outbreaks of infection with VTEC O157 in England and Wales: 1995–1998

Outbreak setting	1995	1996	1997	1998	Total
Community					
General	2	6	7	8	23
Restaurant/catering	3	4	5	0	12
Butcher's shop	2	2	0	0	4
Institution					
Hospital/residential	2	0	3	3	8
Nursery/school	2	1	2	1	6
Farm/open farm or animal contact	0	1	6	2	9
Foreign travel	0	0	2	3	5
Total	11	14	25	17	67

and involved person-to-person spread (Table 4). Outbreaks linked to 'open' or other farms involved transmission of infection by direct or indirect animal contact. There were five outbreaks in which the VTEC O157 strain was acquired abroad. One of these occurred in Fuerteventura in 1997 and was probably caused by consumption of water from a contaminated well [39]; the others involved travel to Greece (two outbreaks), Majorca (one outbreak) and Lanzarote (one outbreak), but the sources of infection were unknown.

VT-negative isolates of E. coli O157

From 1995 to 1998, 94 strains were confirmed as *E. coli* O157 but were negative for the presence of VT genes by DNA hybridisation and PCR. Thirty-seven of these were sorbitol non-fermenting strains of serotypes O157:H7 or O157:H⁻ and possessed the *eae* gene of VTEC O157. Thus, they had the recognised properties of 'VTEC O157' but lacked VT sequences. Strains of this type made up *c.* 1% of the total (3466) sorbitol non-fermenting *eae*O157-positive strains (both VT⁺ and VT⁻) over the 4-year period. About 62% of these strains that appeared to have lost VT genes were from patients with diarrhoea, but none was associated with bloody diarrhoea or HUS. The remaining isolates were from asymptomatic individuals or those whose clinical details were not stated.

The remaining VT-negative strains (57) were negative for the *eae*O157 gene and expressed H antigens other than H7 or were non-motile or H untypable. H antigens identified were 1, 3, 8, 9, 10, 11, 12, 16, 25, 31, 34, 39, 40, 42, 43, 45 and 49, but only the O157:H45 and O157:H⁻ serotypes occurred among VT-negative, *eae*O157-negative strains in each of the 4 years. About 77% of individuals from whom these strains were isolated had diarrhoea and there were four cases of bloody diarrhoea.

Isolation of VTEC other than serogroup O157

Table 5 shows the properties of 11 strains of VTEC belonging to serogroups other than O157 that were either referred as possible causes of sporadic VTEC-associated illness or were isolated in the LEP from stools of patients or contacts. Nine patients were symptomatic, including one with HUS and one individual who also carried VTEC O157. An additional isolate was from an asymptomatic child whose parents were infected with VTEC O157 and in one further case clinical details were not stated. Five strains, associated with diarrhoea or HUS were serotype O26:H11.

The strains were examined by DNA probing for virulence markers associated with VTEC (Table 5). Eight strains hybridised with the probe from the

Table 5. VTEC of serogroups other than O157 from human sources in England and Wales, 1995–1998*

Year	Serotype [†]	Infection [‡]	VT type	<i>eae</i> probe	<i>eae</i> O157 probe	CVD419 probe	Comments
1995	O26:H11	D	VT1	+	-	+	
	O26:H11	D	VT1	+	-	+	
	O26:H11	D	VT1	+	-	+	
	O76:H7	BD	VT2	+	- [§]	+	
1996	O?:H ⁻	BD	VT1	+	-	+	
1997	O26:H11	D	VT1	+	-	-	
	O146:H5	D	VT1	-	-	-	Patient also had VTEC O157
	Orough:H12	NK	VT2	+	-	-	
1998	O26:H11	HUS	VT2	+	-	+	
	O118:H12	None	VT2	-	-	-	Family contact of VTEC O157
	O128ab:H2	BD	VT1+2	-	-	+	

*Includes cultures sent to LEP as putative pathogens from cases of infection; strains isolated in LEP from faecal samples from patients with symptoms consistent with a VTEC infection but from whom an isolate of *E. coli* O157 had not been made; strains from contacts of O157 VTEC infection.

[†]O? indicates that O antigen could not be identified with currently available antisera. O rough indicates that smooth colonies could not be obtained for serotype determination.

[‡]D, diarrhoea; BD, bloody diarrhoea; HUS, haemolytic uraemic syndrome; NK, not known.

[§]Weak reaction in both probe and PCR tests.

conserved region of the *eae* gene associated with the formation of attaching and effacing lesions. One of these (O76:H7) also hybridised weakly with the *eae*O157 probe, but it did not cross-react with anti-O157 serum; it differed phenotypically from VTEC O157 in that it fermented sorbitol and produced β -glucuronidase. The majority of the non-O157 VTEC strains hybridised with the CVD419 probe that includes sequences encoding enterohaemolysin.

Discussion

Isolations of VTEC O157 by laboratories in England and Wales increased from 1468 during 1992–1994 [18] to 3429 during 1995–1998. While laboratory methods for the isolation of *E. coli* O157 have improved, particularly with the use of CT-SMAC agar to detect low levels of putative VTEC O157 amongst heavy growth of faecal flora, this was not an attributable cause, as this method is not routinely used in clinical laboratories. However, it did enable the reference laboratory to identify additional VTEC O157 representing *c.* 1% of the total. Testing for *E. coli* O157 has been broadened with the recommendation that all diarrhoeal stools should be screened [21]. There is an increased awareness of VTEC O157 and pressure to perform tests, particularly since the large outbreak of infection in Central Scotland in late 1996 [3]. The data may reflect a higher disease burden, which is supported by a steady rise in the number of outbreaks [17, 18, 41] and in reports of VTEC O157 in food animals [11, 42].

As reported previously, young children were most likely to be infected, although the high incidence also reflects the greater likelihood that this group will be taken to a general practitioner for diarrhoeal symptoms. Young children may also have specific risk factors including inadequate personal hygiene, habits such as nail-biting and thumb-sucking and close contact with domestic animals and their food. All these factors have been identified as significant in outbreaks or incidents of infection of young children that have involved transmission by animal contact and person-to person spread [15, 16, 20, 43]. Asymptomatic carriage of VTEC O157 has been noted in young children who have been screened as contacts of cases [15], LEP unpublished data]. The marked gender difference for VTEC O157 infection between adult females and males may be because women are more likely than their male counterparts to be involved in food preparation or caring for sick children or other relatives. An important risk factor identified for the development of VTEC O157 infection was care of an existing case [44].

Data on the distribution of VTEC O157 in the regions of England and Wales are difficult to compare with previous findings because administrative changes caused some areas to be merged. Consistently high rates were reported in some regions of relatively higher

agricultural activity, particularly involving animals, and lower population density in the North, North-West and South-West [16]. Surveillance varies by region and data may be influenced by the degree to which laboratories pursue follow-up studies and screening of contacts. The South-East of England was always the area of lowest incidence. Differences in regional distribution of VTEC O157 have been reported for Canada [45], Scotland [46] and Wales [47]. Cases associated with foreign travel were found in all regions and are probably an under-estimate of the total of VTEC O157 infections acquired abroad. Travellers may not view mild diarrhoea as unusual and may not seek medical attention unless symptoms are severe or protracted.

The number of outbreaks of VTEC O157 infection increased and involved all three major transmission routes, *i.e.*, foodborne, person-to-person spread and animal contact. The diversity of outbreak settings shows the importance of enhanced surveillance and the recognition of risk factors that may be associated with infection. Of the five outbreaks in 1997 and 1998 associated with travel abroad, four involved fewer than five patients and little epidemiological information was available. Once travellers have returned home, it may be difficult to link apparently sporadic cases from different areas of the country. The outbreak in Fuerteventura [39] affected at least 14 individuals from England, Denmark, Sweden and Finland and demonstrated the value of rapid international communication to identify patients who may be linked to an outbreak.

As in the period 1992–1994, the majority of VTEC O157 in the present study were VT2; however, the contribution of strains with both VT1 and VT2 genes increased to about one-quarter of the total. Demonstration of VT by a biological, antigenic or DNA-based method is essential for the confirmation of VTEC O157. Use of the designation O157:H7 has been increasingly used as synonymous with VTEC O157, but is unreliable. Annually, between 8% and 19% of strains from England and Wales were non-motile [18] (Table 1) and reliance on the expression of the H7 antigen without VT testing will fail to detect significant numbers of strains. A small number of strains that had the H7 antigen or were non-motile had the biochemical properties of VTEC O157 and the *eae* gene characteristic of this organism, but had no evidence of VT genes by probe or PCR tests. VT sequences are located on bacteriophages in some VTEC including O157 and loss of these elements has been reported [48]. The strains appeared to be associated with diarrhoea but not with bloody diarrhoea or HUS. Their pathogenesis may be more closely related to that of the classical enteropathogenic strains of *E. coli*, that possess the attaching and effacing property but do not produce VT [49]. VT-negative O157 derivatives have been isolated from foods and food animals [LEP, unpublished data] and are probably naturally present in the animal reservoir

from which they are transmitted to man. Serogroup O157 comprises strains with highly diverse properties [30, 50] of which VTEC and their VT-negative derivatives form one group. Among the 57 strains of *E. coli* O157 negative for both VT and *eae*O157 genes, there were 17 H antigen types but not H7. Some strains of these serotypes have been reported as associated with diarrhoea or extra-intestinal disease [30, 51, 52] and several have been isolated from cattle and meat surveys [11, 53].

There are no inexpensive simple methods for the routine detection of VTEC other than O157 available in clinical laboratories. Six of the 11 VTEC that were identified belonged to serogroups O26 and O128ab, which are known to cause serious disease [54, 55]. Overall, nine strains had genes encoding attaching and effacing lesions or enterohaemolysin production, or both, which are properties commonly present in the subclass of VTEC known as enterohaemorrhagic *E. coli* (EHEC) associated with bloody diarrhoea and HUS [1, 2]. Strains that did not have these properties were from an asymptomatic individual or from a mixed infection in which symptoms could have been caused by a VTEC O157 strain.

Infections caused by VTEC O157 have increased in importance in England and Wales, although numbers remain low compared with other gastrointestinal pathogens such as salmonellae and campylobacters [56]. However, the potential severity of disease results in high patient morbidity and significant economic cost. It is essential to maintain and enhance surveillance to identify risk factors and to obtain more evidence on the diversity of transmission routes. This information is essential to implement measures to reduce VTEC O157 infection.

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