Feidhmeannacht na Seirbhíse Sláinte
Health Service Executive

hpsc

Epidemiology of Verotoxigenic *E. coli* in Ireland 2004

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Introduction

Verotoxigenic *E. coli* (VTEC) are so-called because of their ability to produce one or both of two verotoxins (VT1 and VT2). They are an important cause of gastroenteric illness in Ireland with between 42 and 88 cases of VTEC O157 reported annually between 1999 and 2004.[1] Their relative significance lies not in their numbers but in the severity of illness they can cause and in the requirement for prompt public health action to prevent further transmission. They cause a wide range of symptoms, from mild diarrhoea to haemorrhagic colitis with severe abdominal pain and bloody diarrhoea. Illness is usually self-limiting and resolves after about eight days. A proportion of patients however (approx. 9% of symptomatic Irish cases) develop haemolytic uraemic syndrome (HUS), a life-threatening complication.

E. coli O157 was the first *E. coli* serogroup to be associated with this distinctive illness but several other verotoxin-producing *E. coli* serogroups have been reported, including O26, O111, O103 and O145. The primary reservoir is believed to be cattle, although VTEC have been isolated from a variety of healthy animal carriers including sheep, horses, goats and wild birds, and while this organism was first recognized as a foodborne pathogen (the 'burger bug'), it is now known that it can also be transmitted through contaminated water, the environment and by direct contact with animal carriers. Person-to-person spread is an important mode of transmission in households, child-care facilities and institutions.

Methods

Since 1999 HPSC, in co-operation with Directors of Public Health in each health board region, have operated an epidemiological surveillance system for VTEC O157; the data reported under this system has formed the basis of the HPSC annual reports on *E. coli* O157 for the last 5 years. Details on how this system operates, and the case definition used, have been outlined in previous reports. [1]

In 2004, changes to the infectious disease legislation resulted in all VTEC becoming notifiable (S.I. 707 of 2003), and this annual report is the first that aims to describe disease caused in Ireland by VTEC of all serogroups. Enhanced information was supplied as in the previous years by health board personnel, and in addition, typing data was provided by the HSE-SWA Public Health Laboratory at Cherry Orchard Hospital which offers specialist diagnostic and typing services for VTEC.

Data from the enhanced surveillance system in 2003 was also compared with data from the HSE-SWA PHL database retrospectively. A composite list of cases from 2003 showed that 88 cases of VTEC O157 and 7 cases of VTEC O26 occurred. The data quoted in this report for 2003 has been updated to reflect the combined data.

Results

Sixty-one confirmed cases of VTEC were notified to HPSC during 2004, an incidence rate of 1.56 per 100,000. This compares with 95 cases reported in 2003 (2.4/100,000), a reduction of 36% (Figure 1 and Table 1). Among these 61 cases were 52 cases of VTEC O157 (1.3/100,000), four VTEC O26, two VTEC O111, and one each of VTEC O145, O146 and O Ungroupable.

Table 1. Number and crude incidence rates (CIR) VTEC and VTEC O157 infection, Ireland 1999-2004

Year	No. VTEC O157	CIR VTEC O157* (95% CI)	No. VTEC ‡	CIR VTEC* (95% CI)
1999	51	1.4 (1.0-1.8)	N/A	N/A
2000	37(42)§	0.9 (0.6-1.3)	N/A	N/A
2001	50 (52)§	1.3 (0.9-1.6)	N/A	N/A
2002	68 (70)§	1.7 (1.3-2.2)	N/A	N/A
2003†	88	2.2 (1.8-2.7)	95	2.4 (1.9-2.9)
2004	52	1.3 (1.0-1.7)	61	1.6 (1.2-2.0)

* Data from 1996 census was used to calculate the rate in 1999 while the 2002 census were used to calculate rates from 2000-2004, rates exclude non-residents.
† Composite data from 2003 –see methods section
§ Brackets include non-resident cases
‡ Includes serogroup O157

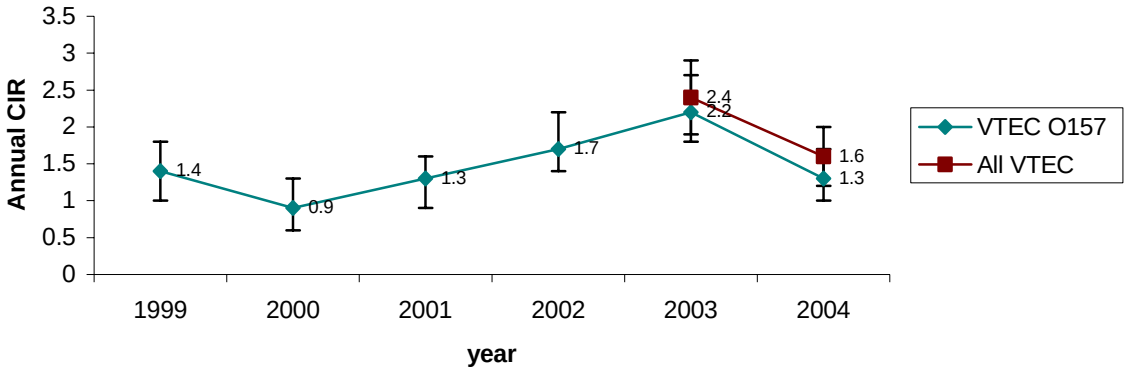

Figure 1. Crude incidence rate with 95% confidence intervals in Ireland, 1999-2004

Regional distribution

Regional variation was noted in the numbers of cases reported (Table 2), with the highest incidence rates this year in the North-Eastern and Western Health Boards, however the number of cases per region is small, and differences unlikely to be significant.

Table 2. Number, crude incidence rate (CIR) and age-standardised incidence rate (ASIR) with 95% confidence intervals of confirmed cases of VTEC by health board of residence, Ireland, 2004

Health Board	No. cases all VTEC	No. cases VTEC O157	CIR VTEC (95% CI) per 100,000	ASIR VTEC (95% CI) per 100,000
ERHA	12	11	0.9 (0.4-1.4)	0.9 (0.4-1.4)
MHB	2	2	0.9 (-0.3-2.1)	0.9 (-0.4-2.3)
MWHB	6	4	1.8 (0.4-3.2)	1.8 (0.4-3.2)
NEHB	11	10	3.2 (1.3-5.1)	3.1 (1.3-5.0)
NWHB	3	2	1.4 (-0.2-2.9)	1.3 (-0.2-2.8)
SEHB	6	5	1.4 (0.3-2.5)	1.4 (0.3-2.5)
SHB	11	8	1.9 (0.8-3.0)	1.9 (0.8-3.1)
WHB	10	10	2.6 (1.0-4.2)	2.6 (1.0-4.2)
Total	**61**	**52**	**1.6 (1.2-2.0)**	

Age-sex distribution

The highest incidence was recorded in young children (Figure 2), which is consistent with previous years. This was particularly pronounced among male cases. It was also notable that all non-O157 VTEC infections reported were in persons less than 15 years or over 65 years (Figure 3). Non-O157 serogroups comprised 30% of VTEC cases in children less that 5 years in 2004.

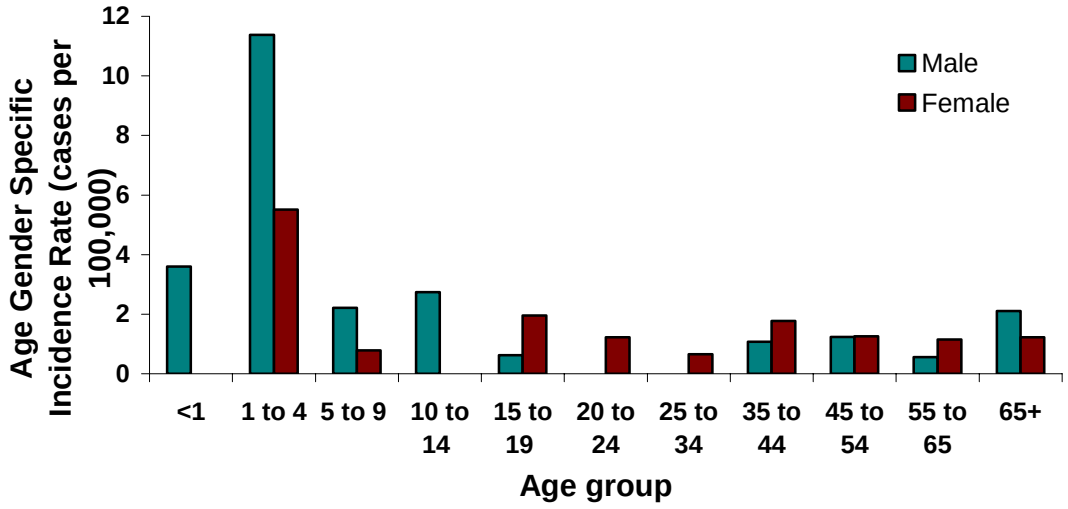

Figure 2. Age-specific incidence rate (per 100,000 population) of confirmed cases of VTEC, Ireland 2004

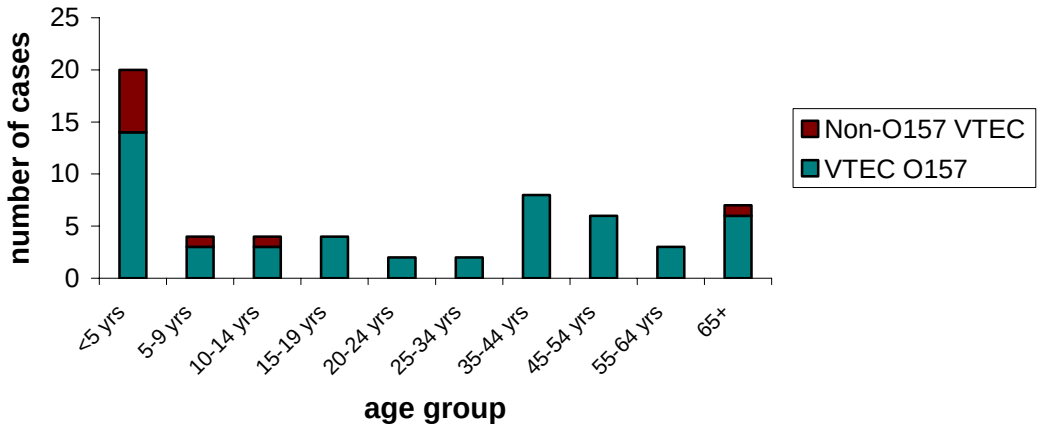

Figure 3.Confirmed cases of VTEC infection stratified by age group, Ireland 2004

Clinical Features

Information on symptoms was available for 56 cases, of whom 47 (84%) were reported as symptomatic. Reported symptoms included: bloody diarrhoea in 25 cases (53%), and HUS in four cases (8.5%). All four cases of HUS occurred in children under 10 years of age. Significantly, two of these HUS cases were caused by non-O157 VTEC.

Seasonality of VTEC cases

The largest number of cases in 2004 occurred in the third quarter (46%), very similar to the trend observed in previous years (Figure 4), although the peak in the number of cases was not as pronounced. Specifically, all non-O157 cases were reported in quarter 2 and 3.

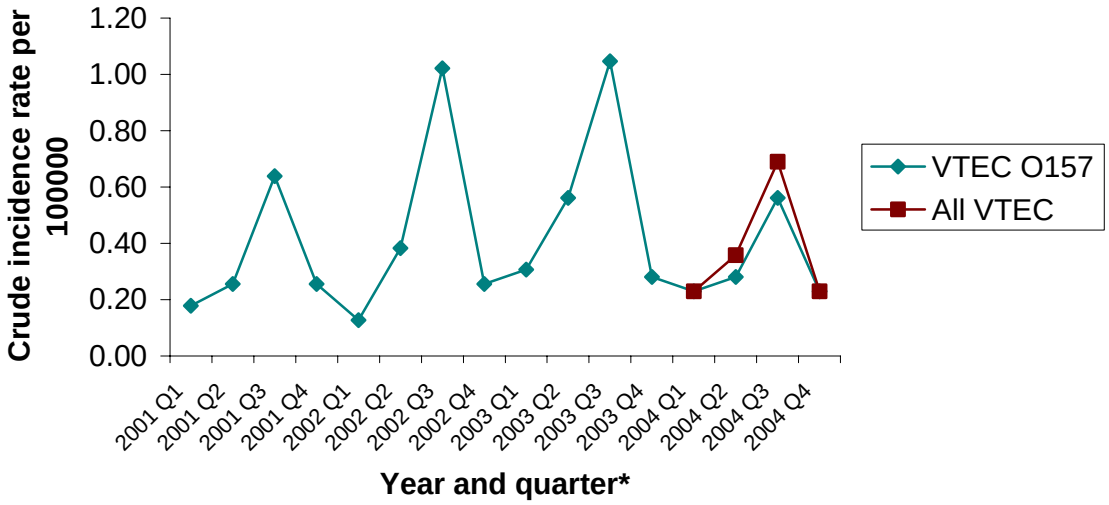

Figure 4. Confirmed VTEC cases by quarter of onset of symptoms, Ireland, 2001-2004

*For asymptomatic cases, the month of onset of associated cases was used. For sporadic cases where date of onset was unknown, date of notification was used

Travel-association

Seven cases were travel-associated. The countries visited within 14 days of onset of illness were Spain (2), UK (2), Italy (1), Malaysia (1) and Turkey (1), reflecting to some extent the frequency of travel by Irish people to these destinations.

Phage Typing Results

Table 3 shows the phage types of the VTEC O157 strains isolated in 2004. As in previous years, PT32 was the commonest phage type reported, accounting for 58% of the VTEC O157 reported.

Table 3 Phage Types of VTEC O157 isolates referred to the HSE SWA Public Health Laboratory, Cherry Orchard Hospital in 2004

Phage type	Number (%)
PT32	30 (58%)
PT8	5 (10%)
PT14	5 (10%)
RDNC	3 (6%)
PT21/28	2 (4%)
PT1	1 (2%)
PT31	1 (2%)
PT34	1 (2%)
PT51	1 (2%)
N/K	3 (6%)
Total	**52 (100%)**

Verotoxin profiles of VTEC strains isolated in Ireland 2004

In 2004, 71% of VTEC O157 strains carried the genes for VT2 while a further 25% carried the genes for both VT1 and VT2 (Table 4). In contrast, 56% of non O157 VTEC isolates carried the genes for VT1 only, 11% for VT2 only and 22% VT1 and VT2. It is notable that strains for all HUS cases (both non-O157 and O157) carried the genes for VT2.

Table 4. Verotoxin typing results for the VTEC isolates referred to the HSE SWA Public Health Laboratory, Cherry Orchard Hospital in 2004

Serogroup	VT1 only	VT2 only	VT1 and VT2	N/K	Total
O157	0	37	13	2	52
O26	2	0	1	0	3
O111	1	0	1	0	2
O145	0	1	0	0	1
O146	1	0	0	0	1
O Ungroupable	1	1	0	0	2

Outbreaks
In 2004, eight clusters/outbreaks of two or more cases were reported. Microbiological and /or epidemiological evidence was obtained linking human cases in a number of these outbreaks with water and/or livestock.

The NEHB reported a general cross-health board outbreak of VTEC O157 linked to a sports club. Four confirmed cases were reported, three of whom were admitted to hospital. Drinking water used at the venue, and supplied from an untreated private well, was found positive for the outbreak strain. Epidemiological evidence was also obtained linking infection with water consumption at the venue. In total, 900 people were potentially exposed. During the investigation further non-outbreak VTEC strains were identified in environmental samples, emphasising the potential public health risk from environmental contamination.

Two additional family clusters in the NEHB were also reported as waterborne. Microbiological evidence was obtained linking water from a private well to 2 confirmed cases in one of these outbreaks.[2] For the second, water from a private well was found positive for *E. coli* and coliforms but no VTEC were isolated.

A third family cluster (WHB) was reported as being transmitted either by water or by animal contact. In this instance, both water from a group water scheme used by the family, and samples taken subsequently from sheep on the family farm, tested positive for VTEC O157 that were indistinguishable from those isolated from the human cases. While the precise route of transmission is unclear, the group water scheme had experienced problems over a protracted period of time, and was observed to be poorly maintained, with the schemes chlorinator non functional and the water source unprotected.

For the 4 remaining family/household clusters, two were suspected to be due to contact with livestock on family farms, one to person-to-person transmission and for the remaining cluster the mode of transmission was reported as unknown.

Discussion
Significant changes were made in 2004 in the reporting of cases of VTEC infection. Illness caused by enterohaemorrhagic *E. coli* (EHEC) became a notifiable disease on January 1st 2004. For the first time also in this report, typing data from the HSE-SWA PHL has been linked with the epidemiological data from the HPSC providing a composite picture of the epidemiology of VTEC in Ireland. As a result, data reported here is more comprehensive for both O157 and non-O157 infections.

Sixty-one VTEC cases were reported in 2004, a rate of 1.56 per 100000. When only VTEC O157 are considered, the rate was 1.33, considerably lower than was reported for Ireland for the last two years. This compares with provisional VTEC O157 incidence rates of 1.1/100,000 in Northern Ireland (CDSC NI personal communication),

4.1/100,000 in Scotland [3] and 1.33 in England and Wales (HPA Colindale, personal communication) in 2004.

Of particular interest are the nine (15%) VTEC infections reported in 2004 that were caused by non-O157 *E. coli* strains. All nine were reported in quarters 2 and 3, which is in keeping with the seasonal distribution noted both historically and this year for VTEC O157 in Ireland. Although the case numbers were small, non-O157 VTEC cases were reported from 6 of the 8 health board regions, indicating that they were widely distributed throughout the country. All non-O157 VTEC infections reported were in children less than 15 years or adults over 65 years. In fact, non-O157 serogroups comprised 30% of VTEC cases reported in children less than 5 years. In addition, 2 of the four VTEC-associated HUS cases reported in 2004 were caused by non-O157 VTEC. While these latter two observations presumably reflect a greater degree of screening for non-O157 VTEC in these higher risk groups, it is evident that non-O157 VTEC were an important cause of VTEC infections in Ireland in 2004. It is also notable that while in the past, VTEC O26 was the primary non-O157 VTEC reported in Ireland, the range of serogroups reported in 2004 was much greater.

A variety of sources and transmission routes have been demonstrated worldwide for VTEC, including food, water, environmental and direct animal contact as well as person-to-person transmission. In 2004 in Ireland, two outbreaks, one general and one family were linked epidemiologically and/or microbiologically with drinking water from private wells, demonstrating the potential of this type of water supply in the transmission of VTEC infection. A private well was also suspected as the route of transmission for a further family cluster. The general outbreak reported illustrates the danger that even a small private water supply can pose if it provides water to a large number of people in a short period of time, exposing them to infection if the water is contaminated.

No other sources or transmission routes were definitively identified for any of the other VTEC outbreaks reported in 2004 although person-to-person transmission and/or animal contact were suspected in a number of family/household clusters. In several case control studies internationally, contact with farm animals and farming environments has been shown to be a strong risk factor for VTEC infection among sporadic cases.[4] As a result of zoonotic disease outbreaks relating to farm animal contact in public settings in the US, the US National Association of State Public Health Veterinarians published a 'Compendium of Measures To Prevent Disease Associated with Animals in Public Settings, 2005' which outlines recommendations for public health officials, veterinarians, animal venue operators, animal exhibitors, visitors to animal venues and exhibits, and others concerned with disease-control and with minimizing risks associated with animals in public settings.[5]

The HPSC established a sub-committee to develop guidance for health professionals regarding human cases of VTEC infection. A report by this sub-committee is due to be published shortly and will provide guidance for clinicians, and laboratory, infection control and public health professionals, for the diagnosis, treatment and care of those

suffering from VTEC infection, and advice for the prevention of transmission of infection.

Acknowledgements
We wish to acknowledge the co-operation of microbiologists, medical scientists, SMOs, SPHMs, surveillance scientists, infection control nurses, PEHOs, and EHOs in providing the information on which this report is based.

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