Shiga toxin-producing *Escherichia coli* (STEC) data: 2017
January 2019
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Contents

About Public Health England 2
Key points for 2017 4
Background 5
Methods 7
Microbiological case definitions 7
Epidemiological case definitions 8
Data 9
Cases of STEC in 2017 in England and Wales 9
Age, gender and seasonality of STEC O157 cases in England 10
Severity of illness 12
Transmission routes 12
Frequently reported subtypes of STEC O157 12
Non-O157 STEC 14
Outbreaks 15
Conclusions 16
PHE STEC publications in 2017 17
Acknowledgements 18
References 19
Key points for 2017

A total of 563 cases of STEC O157 were reported in England and Wales.

The incidence of STEC O157 in 2017 was at its lowest since universal screening of stools for STEC O157 began in 1996.

The lowest incidence of STEC O157 was in London (0.4 per 100,000 population) and the highest in Yorkshire & the Humber (1.34 per 100,000 population).

As per previous years, the highest incidence of infection was in children under 5 years of age.

A third of confirmed STEC O157 cases were hospitalised and 3% developed HUS.

In England, detection of non-O157 STEC increased in line with the growing number of NHS labs implementing GI diagnostics using PCR and 384 cases were reported in 2017.

The most commonly reported non-O157 STEC serogroup was O26 (13%).

Nine outbreaks of STEC involving 65 cases were investigated in 2017.
Background

Shiga toxin producing *Escherichia coli* (STEC), also known as Vero cytotoxin-producing *Escherichia coli* (VTEC), are bacteria that can cause gastroenteritis. Symptoms vary, from mild to bloody diarrhoea and, in severe cases, can cause haemolytic uremic syndrome (HUS), a serious and life threatening condition predominantly affecting the kidneys. A small proportion of patients, mainly children, develop HUS [1].

The main reservoir for STEC is cattle although it is also carried by other ruminants such as sheep, goats and deer. Transmission can occur through direct or indirect contact with animals or their environments, consumption of contaminated food or water, and person to person spread. STEC infections can present as sporadic cases or as outbreaks. Large national and multinational outbreaks have been associated with foodborne transmission [2-4].

The most common serogroup of STEC causing illness in England is O157. Other serogroups (termed non-O157) can also cause illness and have been implicated in outbreaks in England, and elsewhere.

Frontline laboratories in England use culture methods to detect STEC O157 by its inability to ferment sorbitol on selective media (MacConkey agar). However, non-O157 STEC ferment sorbitol and there is no culture method to differentiate non-O157 STEC from non-pathogenic *E.coli* in frontline laboratories. Therefore, detection of non-O157 STEC relies on Polymerase Chain Reaction (PCR).

The implementation and roll out of a GI PCR at frontline hospital laboratories began in December 2013, and by December 2017 around 10% of frontline laboratories were using it. As a consequence there has been a substantial increase in the detection of non-O157 cases in England. However, PCR is not used universally for detection of non-O157 STEC, and the true incidence remains unknown.

While non-O157 STEC can cause serious illness, variation exists among non-O157 STEC serogroups in their associations with severe disease, likely explained by differences in the virulence factors produced by different strains. STEC can produce 2 Shiga toxins (Stx), Stx1 (of which there are 4 subtypes 1a - 1d) and/or Stx2 (of which there are 7 subtypes stx2a – 2g). The presence of Stx2, specifically subtype stx2a, is more likely to cause HUS [1, 5]. The increasing numbers of non-O157 STEC has led to the need to prioritise the case load due to insufficient resources to follow up all cases. Risk assessment, based on clinical symptoms and risk group of the patient and potential pathogenicity of the strain of STEC infecting the patient, is challenging. In response, new guidelines on the public health management of O157 and non-O157 STEC cases were published by the STEC Guidelines Update Working Group in August

National enhanced surveillance of STEC in England and Wales has been undertaken since 2009. This report summarises the epidemiological data on confirmed cases of STEC O157 cases in England in 2017, and compares it to previous years. For the first time, data on cases of non-O157 STEC are also presented.
Methods

The National Enhanced Surveillance System for STEC infection in England (NESSS) began in January 2009 in order to supplement our understanding of the epidemiology of STEC infection. The system collects a standard dataset of clinical, epidemiological and microbiological data for all STEC cases, in order to improve outbreak recognition and facilitate public health investigations. The data is collected from enhanced surveillance questionnaires and reconciled with laboratory reports associated with cases.

STEC is notifiable under the Public Health (Control of Diseases) Act 1984 and the Health Protection (Notification) Regulations 2010. In England, local diagnostic laboratories report presumptive cases of STEC to PHE Health Protection Teams (HPTs) and then refer samples to the Gastrointestinal Bacteria Reference Unit (GBRU) for confirmation and further testing. Each HPT arranges for the STEC Enhanced surveillance questionnaire (ESQ) to be completed for all cases to obtain a detailed history for the 7 days prior to onset of illness. The ESQ collects: demographic details; risk status; clinical conditions, exposures including travel, food and water consumption, environmental exposures and outbreak status. Completed questionnaires are submitted to the national Gastrointestinal Infections team at PHE to be included in NESSS.

Data included in this report were validated and extracted from NESSS and cases meeting the case definitions below were included in analyses. Laboratory data for cases in Wales were extracted and validated from the PHE Gastro Data Warehouse (GDW). Welsh data are included in Figure 1 and 2 as only laboratory data was available and are excluded from other sections of the report.

Data from the 2017 Office for National Statistics (ONS) mid-year population estimates were used to provide denominators for the calculation of incidence rates. All dates for the figures are based on the receipt date of a sample specimen at the GBRU.

Microbiological case definitions

Confirmed: Positive STEC culture confirmed by GBRU.

Probable: Suspect case with serum antibodies to lipopolysaccharide of *E. coli* O157 or other *STEC*, detected at the GBRU, as the only evidence of infection.
Epidemiological case definitions

Primary case: A symptomatic case with no history of close contact with a confirmed case in the 7 days prior to onset of illness.

Secondary case: Case with a date of onset more than 4 days after the primary case or where transmission is believed to be through exposure to a primary case.

Unsure: It is not possible to determine whether the case is primary or secondary with the information available. This may be because the patient was lost to follow-up, is asymptomatic or in an outbreak where it is not possible to identify the primary case(s).

Travel-acquired case: Case who has reported any travel outside of the UK in the 7 days prior to their date of onset of illness.

Asymptomatic case: A person from whom STEC was identified through contact screening procedures but who is asymptomatic.
Data

Cases of STEC in 2017 in England and Wales

In 2017, 948 confirmed cases of STEC were reported in England and Wales. These comprised 563 laboratory confirmed cases of STEC serogroup O157 (532 cases in England and 31 in Wales) and 385 cases with a serogroup other than O157 isolated (non-O157) (384 in England; 1 in Wales).

There were 20 probable cases with serological evidence of STEC infection, with antibodies detected to O157 lipopolysaccharides in 12 cases, and for non-O157 STEC lipopolysaccharides in 8 cases (one O111, three O26, four O103 cases).

The crude incidence rate of confirmed STEC O157 in England and Wales, was 0.96 per 100,000 cases (95% CI 0.88 – 1.04) - a continuation of the downward trend observed since 2015 (Figure 1). It is the lowest number of cases reported since 1996 when testing for STEC O157 on all faecal specimens from patients with suspected gastrointestinal infection in England began [6].

Figure 1. Incidence rate of Shiga toxin producing *Escherichia coli* (STEC) O157 cases by year, England and Wales, 2006 to 2017
Figure 2. Incidence of STEC O157 in England by region, 2017

The highest incidence of STEC O157 was in Yorkshire & Humber (1.34 per 100,000 cases per population, CI 1.05-1.68) and the North West (1.27 per 100,000 cases per population, CI 1.02-1.55) and the lowest was in London (0.4 per 100,000 cases per population, CI 0.3-0.58) (Figure 2). Data by local authority and PHE Centre level are available on the PHE fingertips website: fingertips.phe.org.uk/profile/health-protection

Age, gender and seasonality of STEC O157 cases in England

Of 532 confirmed STEC O157 cases in England, 279 (52%) were female. Children aged 1 to 4 years had the highest incidence of infection (3.6 per 100,000 population, CI 95% 2.98-4.45). Overall females had a higher incidence across all age groups, apart from 5 to 9 and 80 plus age groups (Figure 3).
As per previous years, STEC O157 infections displayed a distinct seasonality with the peak of infection in the summer months (Figure 4).

Figure 3. Age specific incidence rate of STEC O157 cases in England, 2017

Figure 4. Seasonal trend of laboratory confirmed cases of STEC O157 in England, 2015 to 2017
Severity of illness

Of 532 STEC O157 cases, 520 (98%) questionnaires were received. For cases where a questionnaire was completed, 508 (98%) cases reported symptoms; 93% (n=471) had reported diarrhoea, including 60% (n=304) with bloody diarrhoea. Other symptoms included abdominal pain (420, 83%), nausea (244, 48%), fever (190, 37%) and vomiting (187, 37%). Hospitalisation occurred in 35% of cases; duration of hospitalisation ranged from 1 to 21 days.

HUS occurred in 18 confirmed cases (3%). Most HUS cases (12, 67%) were under 5 years of age with a median age of 2 (range 1 to 67 years old). The incidence of HUS in children under 5 in 2017 was 0.31 per 100,000 cases per population (CI 0.17-0.54). There were 2 deaths due to STEC infection reported in 2017.

HUS occurred in 4/20 (20%) probable cases. Eleven cases (58%) were female. Ages ranged from 2 to 76 years old with a median of 28.

Transmission routes

There were 453 primary cases, 20 secondary cases, and 12 asymptomatic contacts. For 35 cases epidemiological case definitions were not determined. Forty-one cases (8%) were travel acquired; the most frequently reported travel destinations included Spain, Turkey and Egypt.

Frequently reported subtypes of STEC O157

As with previous years, phage type (PT) 8 and 21/28 were the most commonly reported STEC O157 phage types (Figure 5) with a combined proportion of 48%. These were followed by PT54 (9%), PT32 (34, 6%) and PT34 (31, 6%). The peak in PT34 in 2016 was due to a large outbreak linked to mixed salad leaves where 165 cases were reported between 31 May and 29 July 2016[4]. The most common phage type among travel associated cases was PT 8 (20, 49%) followed by PT 34 (6, 15%).
Among isolates from STEC O157 cases in 2017, most (65%, 346/532) had Stx2 shiga-toxin only, 34% (n=182) had Stx1+2 and 1% (n=4) had stx1 only. Of those isolates with Stx2 or Stx1+2, 45% (n=210) had stx2a; the subtype more likely to cause HUS. All 532 isolates had the eae gene.
Non-O157 STEC

Historically, cases of non-O157 STEC have been under-ascertained, with 89 cases of STEC non-O157 cases reported between 2009 and 2013, prior to PCR being implemented.

Following the recent increase in frontline laboratories using PCR, there has been a significant increase in the detection of non-O157 cases in England and in 2017, 384 non-O157 STEC cases of 62 different serotypes were reported. One case was reported in Wales in 2017 (STEC O26).

The 5 most common non-O157 serogroups detected were O26 (48, 13%) followed by O91 (47, 12%), O146 (40, 10%), O103 (25, 7%) and O128ab (24, 6%) (Table 1) See link for full list of non-O157 STEC by Stx type reported in 2017. Comparisons with previous years are not valid due to the changes in detection methods.

In 2017, questionnaires were received for 69% (266) of non-O157 STEC cases. Of those symptomatic, 80% (n=213) reported diarrhoea, including bloody diarrhoea in 26% (n=69) cases. This was accompanied by abdominal pain (189, 71%), nausea (116, 44%), fever (76, 29%), and vomiting (72, 27%).

Fifty cases (19%) were hospitalised and 6 (2%) cases developed HUS. No deaths were reported amongst cases of non-O157 STEC.

Among all cases of non-O157 STEC in 2017, 23% (n=90) of strains possessed the Stx2 toxin either alone or in combination with Stx1 (112, 29%), (Full list of Non-O157 serogroups). This included 52 (14%) cases where strains possessed the stx2a subtype. A hundred and forty strains (36%) possessed the eae gene.

Table 1. Serotype, shiga toxin (stx) and shiga toxin 2 subtype profiles for the 10 most frequently reported strains amongst non-O157 cases, 2017

<table>
<thead>
<tr>
<th>Serogroup</th>
<th>stx1</th>
<th>stx2</th>
<th>stx1 + 2</th>
<th>Total</th>
<th>stx2a</th>
<th>stx2a%</th>
<th>HUS</th>
<th>HUS %</th>
</tr>
</thead>
<tbody>
<tr>
<td>O26</td>
<td>26</td>
<td>8</td>
<td>14</td>
<td>48</td>
<td>22</td>
<td>46</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td>O91</td>
<td>3</td>
<td>8</td>
<td>36</td>
<td>47</td>
<td>1</td>
<td>3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>O146</td>
<td>21</td>
<td>6</td>
<td>13</td>
<td>40</td>
<td>25</td>
<td>25</td>
<td>2*</td>
<td>8</td>
</tr>
<tr>
<td>O103</td>
<td>25</td>
<td>6</td>
<td>13</td>
<td>40</td>
<td>1</td>
<td>3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>O128ab</td>
<td>4</td>
<td>7</td>
<td>13</td>
<td>24</td>
<td>16</td>
<td>16</td>
<td></td>
<td></td>
</tr>
<tr>
<td>O117</td>
<td>16</td>
<td>6</td>
<td>13</td>
<td>33</td>
<td>2</td>
<td>13</td>
<td>1</td>
<td>7</td>
</tr>
<tr>
<td>Unidentifiable</td>
<td>9</td>
<td>5</td>
<td>1</td>
<td>15</td>
<td>2</td>
<td>13</td>
<td>1</td>
<td>7</td>
</tr>
<tr>
<td>O38</td>
<td>7</td>
<td>6</td>
<td>13</td>
<td>13</td>
<td>1</td>
<td>9</td>
<td>1</td>
<td>9</td>
</tr>
<tr>
<td>O111</td>
<td>10</td>
<td>1</td>
<td>11</td>
<td>11</td>
<td>1</td>
<td>9</td>
<td>1</td>
<td>9</td>
</tr>
<tr>
<td>O145</td>
<td>1</td>
<td>10</td>
<td>11</td>
<td>11</td>
<td>10</td>
<td>91</td>
<td>1</td>
<td>9</td>
</tr>
</tbody>
</table>

*Both O103 strains that cause HUS had the stx1a toxin.
Outbreaks

Nine STEC outbreaks affecting 65 people were investigated in 2017. Eight outbreaks, comprising 53 confirmed cases, were caused by STEC serogroup O157, and thus 10% of confirmed STEC O157 cases were attributed to outbreaks (Table 2). Most outbreaks were small in size (median 5 cases, range 3-17).

Despite epidemiological investigations, it was not possible to determine the vehicle and/or source of infection for 3 outbreaks, including the largest STEC O157 outbreak with 17 cases, and the outbreak of STEC O55:H7 Stx2. There were 11 HUS cases and 1 death associated with outbreaks in 2017.

Table 2. STEC O157 and non-O157 outbreaks in England, 2017

<table>
<thead>
<tr>
<th>Agent</th>
<th>Total number affected</th>
<th>Total laboratory confirmed</th>
<th>Hospitalised</th>
<th>Source</th>
<th>Region</th>
</tr>
</thead>
<tbody>
<tr>
<td>STEC O157 PT 21/28 Stx2</td>
<td>7</td>
<td>6</td>
<td>3</td>
<td>Raw drinking milk</td>
<td>South East</td>
</tr>
<tr>
<td>STEC O157 PT1 Stx2</td>
<td>6</td>
<td>6</td>
<td>0</td>
<td>Unknown</td>
<td>National</td>
</tr>
<tr>
<td>STEC O157 PT2 Stx2</td>
<td>11</td>
<td>11</td>
<td>8</td>
<td>Burgers</td>
<td>National</td>
</tr>
<tr>
<td>STEC O157 PT54 Stx2</td>
<td>5</td>
<td>4</td>
<td>0</td>
<td>Nursery</td>
<td>North West</td>
</tr>
<tr>
<td>STEC O157 PT21/28 Stx2</td>
<td>4</td>
<td>4</td>
<td>3</td>
<td>Raw pet food</td>
<td>National</td>
</tr>
<tr>
<td>STEC O157 RNDC Stx2</td>
<td>17</td>
<td>17</td>
<td>0</td>
<td>Unknown</td>
<td>National</td>
</tr>
<tr>
<td>STEC O55 Stx2</td>
<td>5</td>
<td>2</td>
<td>3</td>
<td>Unknown</td>
<td>South East</td>
</tr>
<tr>
<td>STEC O157 PT14</td>
<td>7</td>
<td>2</td>
<td>0</td>
<td>Private water supply</td>
<td>Wales</td>
</tr>
</tbody>
</table>
Conclusions

The number of STEC cases in England continued to decline in 2017 and reached the lowest figure since 1996 when screening for STEC O157 of all diarrhoeal faecal specimens submitted for testing began in England. The reason for this decline is unclear, although phage typing indicates a decrease in numbers of the 2 most frequently detected types (PT8 and PT 21/28) and suggest that a change in the predominate strains circulating in England to ones which cause less severe disease may be occurring.

The detection of non-O157 infections continued to rise in 2017 and reflects the more widespread use of PCR in frontline laboratories to detect a broader range of serogroups. Currently due to the limited availability of enhanced surveillance questionnaires it is difficult to know the true public health impact of non-O157 STEC.

While non-O157 STEC serogroups can cause serious illness and have been implicated in outbreaks, some types can cause more severe disease than others. Specific Stx subtypes such as stx2a and the presence of initimin (eae gene), along with host factors (such as age) affect the potential for the STEC to cause serious illness and complications. This can be challenging for follow up and the control of cases of infection. In response to managing these cases, the STEC Guidelines Update Working Group published new guidelines on the public health management of O157 and non-O157 cases in August 2018.

Further investigations are required to examine the public health significance and severity of disease associated with non-O157 infections.
Shiga toxin-producing Escherichia coli (STEC) data 2006 to 2017

PHE STEC publications in 2017


Rose TC, Adams NL, Barr B, Hawker J, O'Brien SJ, Violato M, Whitehead M, Taylor-Robinson DC. Socioeconomic status is associated with symptom severity and sickness absence in people with infectious intestinal disease in the UK. BMC Infect Dis. 2017 Jun 23;17(1)


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References