Shiga toxin-producing Escherichia coli (STEC) food-borne outbreaks

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Shiga toxin–producing *Escherichia coli* (STEC) food-borne outbreaks

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**ABSTRACT.** *Escherichia coli* (*E. coli*) are Gram negative, non-sporulating bacteria, which belong to the normal intestinal flora of humans and animals. Shiga toxin-producing *E. coli* (STEC) are a group of *E. coli* that is defined by the capacity to produce toxins called Shiga toxins (Stx). Following ingestion of STEC, the significant risk of two serious and potentially life-threatening complications of infection, hemorrhagic colitis and hemolytic uremic syndrome (HUS), makes STEC food poisoning a serious public health problem. Besides Stx, human pathogenic STEC harbor additional virulence factors that are important for their pathogenicity. Although human infection may also be acquired by direct transmission from person to person or by direct contact of humans with animal carriers, the majority of STEC infections are food-borne in origin. The gastrointestinal tract of healthy ruminants seems to be the foremost important reservoir for STEC and ingestion of undercooked beef one of the most likely routes of transmission to humans. Other important food sources include fecally contaminated vegetables and drinking water.

The serogroup classification of STEC is based on the somatic (O) and flagellar (H) antigens, and, to date, more than 200 STEC serogroups have been identified. Human STEC infections are, however, associated with a minor subset of O:H serotypes. Of these, the O157:H7 or the O157:H- serogroups (STEC O157) are the ones most frequently reported to be associated with food-borne outbreaks. However other non-O157 STEC serogroups such as *E. coli* O26, O103, O111, O121, O45 and O145 have caused several outbreaks in recent years.

Two outbreaks of gastroenteritis caused by *E. coli* O157:H7 were first reported in the US, following the consumption of undercooked hamburgers, in 1982. Since then several outbreaks were reported worldwide. A major *E. coli* O157:H7 outbreak occurred in Japan and contaminated radish sprouts was identified as the vehicle of infection. More than 6,000 school children were affected, 101 people were hospitalized with HUS and 12 deaths were recorded. The recent outbreak of STEC O104:H4 infection and HUS reported in Germany in the spring of 2011 was one of the largest outbreaks worldwide. As of 27 July, 3126 cases of STEC infections, 773 cases of HUS including 46 deaths linked to the outbreak in Germany and occurring in the European Union (EU) (including Norway). Outside the EU 8 cases of STEC and 5 cases of HUS, including 1 death have been reported in the USA, Canada and Switzerland, all with recent travel history to Germany.

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The present review on major STEC food-borne outbreaks recorded worldwide highlights the need for control measures in order to prevent or at least minimize the occurrence of similar events in the future.

**Keywords:** Escherichia coli, Shiga toxin-producing Escherichia coli (STEC), food-borne outbreaks

**INTRODUCTION**

Shiga toxin–producing Escherichia coli (STEC) are known as enterohemorrhagic E. coli (EHEC) and are pathogens of both animals and humans. They are a subset of the larger group of verocytotoxin-producing E. coli (VTEC) which may be categorized into specific groups (pathotypes). These categories include pathogenic E. coli (EAggEC), which are responsible for diarrhea; and non-pathogenic E. coli (EPEC), which are responsible for diarrhea in foodborne outbreaks. Other categories include enteroaggregative E. coli (EAEC), enteroadherent E. coli (EAdEC), diffuse-adhhering E. coli (DAEC), and enteropathogenic E. coli (EPEC), enteroinvasive E. coli (EIEC), and enteroaggregative E. coli (EAEC).

Some strains of STEC can be pathogenic only to animals, while others can cause disease in both animals and humans. In most cases, they are asymptomatic or produce mild symptoms, such as gastrointestinal distress. However, certain strains can cause severe illness, including hemolytic-uremic syndrome (HUS) and thrombotic-thrombocytopenic purpura (TTP). STEC strains that cause severe illness are associated with specific serotypes, such as O157, O26, O111, and O104. In recent years, STEC O104:H4 has been responsible for a large outbreak in Germany, affecting over 150,000 people and resulting in 49 deaths.

**Escherichia coli** is a normal inhabitant of the mammalian intestinal tract and can cause disease in humans and animals. The mechanisms of pathogenicity include the production of toxins and/or host cell attachment factors, and the invasion of colonic mucosal cells. Usually, a given infection involves more than one virulence factor. Based on virulence properties, some strains can be pathogenic to humans and animals since they carry or produce a plethora of virulence factors.

**KEYWORDS:** Escherichia coli, Shiga toxin-producing Escherichia coli (STEC), food-borne outbreaks

**INTRODUCTION**

Shiga toxin–producing Escherichia coli (STEC), sometimes referred to as verocytotoxin-producing E. coli (VTEC) have emerged as significant etiological agents of food-borne infectious disease in various countries. A total of 3,573 confirmed STEC infections in humans were reported in the European Union in 2009 (EFSA 2011). In the United States it is estimated that STEC O157:H7 causes 63,153 illnesses annually. Non-O157 STEC is responsible for 112,752 illnesses. Foodborne ETEC is responsible for 17,894. Finally diarrheagenic E. coli other than STEC and ETEC are responsible for 11,982 illnesses annually (Scallan et al. 2011 a,b)

E. coli is a normal inhabitant of the mammalian intestinal tract (Karmali et al. 2010). Many strains are harmless or even beneficial to the host; however, some strains can be pathogenic to humans and animals since they carry or produce a plethora of virulence factors (EFSA 2011). The mechanisms of pathogenicity include the production of toxins and/or host cell attachment factors, and the invasion of colonic mucosal cells. Usually, a given infection involves more than one virulence factor. Based on virulence properties, some strains can be pathogenic to humans and animals since they carry or produce a plethora of virulence factors.

**REFERENCES:**


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referred to as Shiga toxins (Stx) producing *E. coli* (STEC) especially those causing haemorrhagic illnesses acquired significance for the food industry in the last 30 years (EFSA 2011, Bell and Kyriakidis 2009, Meng et al. 2007, Fratamico and Smith 2006).

STEC classification is based on the somatic (O) and flagellar (H) antigens, and more than 600 STEC serotypes (including 160 O serogroups and 60 H serogroups) have been identified to date. In humans, over 130 STEC serotypes can cause severe inflammation of the large intestine accompanied by haemorrhages of the intestinal mucosa and severe diarrhoea (haemorrhagic colitis, HC), or haemytic uraeic syndrome (HUS), which can lead to renal failure and even death (Thorpe 2004).

The vast majority of STEC reported outbreaks worldwide have been attributed to *E. coli* serogroup O157 and particularly to the *E. coli* O157:H7 serotype. However, other non-O157 *E. coli* serogroups such as *E. coli* O26, O103, O111, O121, O45 and O145 have been implicated in several outbreaks (Karmali 2004). The aim of the present work was to review selected major reported food-borne STEC outbreaks worldwide.

**Characteristics of STEC**

*E. coli* is a Gram-negative, facultative anaerobic, non-sporulating, motile bacterium and a member of the Enterobacteriaceae family. Most *E. coli* strains can grow at a temperature range from 10 to 46°C. Although the minimum temperature for growth of *E. coli* O157 in milk has been determined to be 8°C, certain *E. coli* strains can grow in milk at temperatures as low as 6.5 °C (D’Aoust et al. 1988). Optimum growth occurs at aw 0.995 and the minimum aw value is 0.950. Salt (NaCl) at 8.5 % inhibits the growth of *E. coli* O157. The optimum pH for growth is 6.0 to 8.0. Reported range for *E. coli* is 4.4 to 9.0 (EFSA 2011, Bell and Kyriakidis 2009, Meng et al. 2007, Fratamico and Smith 2006).

STEC are not heat resistant microorganisms. For *E. coli* O157, a D57°C value of 5 min, and a D63°C value of 0.5 min have been reported in meat. Results reported by D’Aoust et al. (1988) confirm that the high-temperature, short time process (71.7°C, 15 s), which is commonly used to pasteurize milk, is sufficient to kill approximately 1×10⁶ *E. coli* O157:H7 ml⁻¹.

Most of STEC strains (including *E. coli* O157:H7) are unusually acid tolerant, being able to withstand exposure to extremely acidic conditions (pH 2.0), particularly when they are previously incubated at acidic pH environments (Brudzinski and Harrison 1998). For example O157:H7 survived for up to 2 months with only two decimal reductions in cell population during fermentation, drying and storage of fermented sausage; for 5-7 weeks in mayonnaise at 5°C and for 10-31 days in apple cider (Fratamico and Smith 2006). The acid resistance of most of STEC strains enables them to survive the acidic milieu of various foods as well as passage through the human stomach. Bacteria that are more resistant to the gastric environment have a greater opportunity to survive and therefore ultimately colonize the intestinal tract and cause illness.

**STEC infection**

STEC infection is usually acquired by the ingestion of contaminated food or water, by person-to-person transmission or contact with carrier animals (Karmali 2004). The main reservoirs for STEC are cattle and other farm ruminants such as sheep and goats. Wild ruminants, such as deer may also carry the pathogen (Karmali 2010). Foods of animal origin, such as meat (especially ground beef) and unpasteurized milk, are probably the major vehicles of human STEC infection (Karmali 2010). Other types of foods, however, such as unpasteurized fruit juices or raw vegetables contaminated with STEC may also act as vehicles of transmission (EFSA 2011, Karmali et al. 2010). STEC can also be transmitted by direct contact with carrier animals. Person-to person transmission is not uncommon. Waterborne transmission is also becoming increasingly recognized. Outbreaks have been associated with sharing of paddling pools and other communal bathing waters (Coia 1998). Thus, although infection may initially be acquired from a contaminated foodstuff, subsequent outbreak spread can occur by various routes (secondary infections) especially during the late stages of an outbreak.

The infectious dose of *E. coli* O157:H7 is very low, 10 to 100 cells. In one outbreak, the contamination level of *E. coli* O157:H7 in uncooked hamburger meat was found to be less than 700 cells/patty (Griffin 1998). In another outbreak the infectious dose of O157:H7 was found to be less than 50 cells (Tilden et al. 1996), and for the EHEC O111, less than 1 cell/10 g of salami was sufficient to induce HC (Paton et al. 2010).
1996). Because the infectious dose is so low, the capacity of *E. coli* strains to survive gastric exposure directly impacts their ability to cause illness.

After ingestion, *E. coli* bacteria rapidly multiply in the large intestine and bind tightly to cells in the intestinal lining. Inflammation caused by the toxins is believed to be the cause of HIC, the first manifestation of *E. coli* infection, which is characterized by sudden onset of abdominal pain and severe cramps, followed by diarrhea within 24 hours (Fratamico and Smith 2006). HIC typically occurs within 2 to 5 days of ingestion of STEC and is often complicated by potentially fatal systemic sequelae such as neurological damage and HUS, which occur within 2–14 days after the onset of diarrhea (Nishikawa 2011). HUS is defined by the triad of acute renal failure, thrombocytopenia, and microangiopathic hemolytic anemia and occurs in about one-tenth to one-fourth of cases (Karmali 2004). STEC infections that progress to HUS tend to be symptomatic, and there is a bimodal distribution in susceptibility to HUS, with children and the elderly being at highest risk. STEC infections can also result in a variant form of HUS, sometimes referred to as thrombotic thrombocytopenic purpura (TTP). This “diarrhea-associated TTP” is more common in adults, and patients are more often febrile with marked neurological involvement (Paton and Paton 1998). Non-renal complications of STEC infections include ischemic colitis with colonic stenosis and, rarely, persistent pancreatitis with diabetes.

By definition, all STEC must produce Stx, but other virulence factors are also involved in pathogenesis and it is the possession of these that seems to determine the virulence of any given serotype. Such well-known factors include the ability to adhere to intestinal cells (*eaeA* gene), and the ability to produce haemolysin (*hlyA* gene) (EFSA 2011, Jaeger and Acheson 2000). The principal virulence factors associated with the severe sequelae of STEC infection are Stx. The Stx family consists of a number of structurally and functionally related protein toxins. The prototype of the family is elaborated by *Shigella dysenteriae* type 1 (Thorpe 2004). Stx are AB5 toxins consisting of an enzymatically active A subunit covalently associated with a pentameric B subunit that mediates binding to host cells. Briefly, Stx act as ribosome-inactivating toxins and inhibit synthesis of critical host proteins needed by the cell to survive and/or properly function (Thorpe 2004). The production of Stx in the gut lumen is strongly associated to HIC. A small portion of Stx traverses the epithelium and then translocates to the circulation, where it causes vascular damage in specific target tissues such as the brain and kidneys, resulting in systemic complications (Griffin 1998). Stx are classified into two subgroups, Stx1 and Stx2. A variant of Stx1, named Stx1e, and several variants of Stx2, named Stx2c, Stx2d, Stx2e, and Stx2f, which differ in their biological activities and association with disease, have been reported (Nishikawa 2011, Thorpe 2004).

### STEC food-borne outbreaks

Food-borne outbreaks of STEC infection, some involving hundreds of cases, have been documented in various countries and in a variety of settings (Karmali 2010). *E. coli* Serogroup O157 and particularly serotype O157:H7 were identified as the causative agents in most of them. Outbreaks have been caused by both O157 and non-O157 STEC (Tables 1 and 2).

### STEC O157 food-borne outbreaks

*E. coli* O157:H7 was initially recognized as a food-borne pathogen in 1982, during outbreaks that occurred in the US following consumption of undercooked hamburger (Riley et al. 1983). Since then, many additional outbreaks of *E. coli* O157:H7 infections have been linked to the consumption of inadequately cooked hamburger meat. In the US, from 1982 to 2002, 350 outbreaks were reported, 183 (52%) were food-borne and the food vehicle for 41% of these was ground beef (Rangel et al. 2005). A state-wide *E. coli* O157:H7 outbreak in Washington State (US) from December 1992, through February 1993 was associated with the consumption of undercooked hamburger meat at a fast-food chain. In total, 501 cases were reported, including 151 hospitalizations (31%), 45 cases of HUS (9%), and three deaths (Bell et al. 1994). From December 1995 to March 1996, 28 children with HUS in Bavaria, Germany, were identified. This outbreak was caused by *E. coli* O157:H - and the consumption of two types of sausages, including one containing raw beef, was statistically related to illness (Ammon et al. 1999). In France, one outbreak of STEC O157 (69 cases, 17 HUS cases) occurred in 2005, linked to the consumption of contaminated ground beef (King et al. 2009). The same year, in the Netherlands steak
Table 1. Major recorded STEC 0157 foodborne outbreaks worldwide.

<table>
<thead>
<tr>
<th>Year</th>
<th>Country</th>
<th>STEC serogroup /serotype</th>
<th>Implicated food</th>
<th>No of people affected</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>1992-2002</td>
<td>USA</td>
<td>O157:H7</td>
<td>Various (41% ground beef)</td>
<td>183 foodborne outbreaks</td>
<td>Rangel et al. 2005</td>
</tr>
<tr>
<td>1992-1993</td>
<td>Washington State, USA</td>
<td>O157:H7</td>
<td>Undercooked hamburger meat</td>
<td>501 cases, 151 hospitalizations 45 HUS cases, 3 deaths</td>
<td>Bell et al. 1994</td>
</tr>
<tr>
<td>1992-2008</td>
<td>England-Wales</td>
<td>STEC 0157</td>
<td>Poultry meat (6.8%), bovine and Sheep meat (38.6%), salads, fruit</td>
<td>84 outbreaks; 1,168 cases (286 hospitalizations, 12 deaths)</td>
<td>Gorley et al. 2011</td>
</tr>
<tr>
<td>1994</td>
<td>Scotland</td>
<td>STEC 0157</td>
<td>Milk</td>
<td>69 cases, 1 death</td>
<td>Upton and Coia, 1994</td>
</tr>
<tr>
<td>1995</td>
<td>France</td>
<td>STEC 0157</td>
<td>Frozen beef burgers</td>
<td>69 cases, 17 HUS cases</td>
<td>King et al. 2009</td>
</tr>
<tr>
<td>1995</td>
<td>Netherlands</td>
<td>STEC 0157</td>
<td>Steak tartare (raw beef product)</td>
<td>21 confirmed, 11 probable cases</td>
<td>Doorduyn et al. 2006</td>
</tr>
<tr>
<td>1996</td>
<td>Japan</td>
<td>O157:H7</td>
<td>Radish sprouts</td>
<td>&gt; 6,000 (101 HUS cases, 11 deaths)</td>
<td>Michino et al. 1999</td>
</tr>
<tr>
<td>1996-2006</td>
<td>France</td>
<td>STEC 0157 (66.67%)</td>
<td>Bovine meat, raw milk cheeses</td>
<td>641</td>
<td>Espie et al. 2008</td>
</tr>
<tr>
<td>1999</td>
<td>Canada</td>
<td>O157:H7</td>
<td>Sausage</td>
<td>143</td>
<td>McDonald et al. 2004</td>
</tr>
<tr>
<td>1999-2008</td>
<td>Scotland</td>
<td>STEC 0157</td>
<td>Various types</td>
<td>2,298</td>
<td>Locking et al. 2011</td>
</tr>
<tr>
<td>2003</td>
<td>Slovakia</td>
<td>STEC 0157</td>
<td>Unpasteurised cow milk</td>
<td>9 (3 HUS cases)</td>
<td>Liptakova et al. 2004</td>
</tr>
<tr>
<td>2004-2007</td>
<td>New Mexico, USA</td>
<td>STEC 0157</td>
<td>No data</td>
<td>4</td>
<td>Lathrop et al. 2009</td>
</tr>
<tr>
<td>2005</td>
<td>Holland</td>
<td>STEC 0157</td>
<td>Steak tartare (raw bovine meat)</td>
<td>32</td>
<td>Doorduyn et al. 2006</td>
</tr>
<tr>
<td>2007</td>
<td>Scotland</td>
<td>STEC 0157</td>
<td>Bovine meat</td>
<td>10</td>
<td>McCartney et al. 2010</td>
</tr>
<tr>
<td>2007</td>
<td>Iceland</td>
<td>STEC 0157</td>
<td>Lettuce</td>
<td>9</td>
<td>Sigmundsdottir et al. 2007</td>
</tr>
<tr>
<td>2007</td>
<td>Netherlands</td>
<td>STEC 0157</td>
<td>Iceberg lettuce</td>
<td>35</td>
<td>Friesema et al. 2007</td>
</tr>
<tr>
<td>2008</td>
<td>Connecticut, USA</td>
<td>STEC 0157</td>
<td>Raw milk</td>
<td>14 cases (7 confirmed) (5 hospitalizations, 3 HUS cases)</td>
<td>Guh et al. 2010</td>
</tr>
<tr>
<td>2008</td>
<td>USA</td>
<td>STEC 0157</td>
<td>Bovine meat</td>
<td>99</td>
<td>Nowicki et al. 2010</td>
</tr>
<tr>
<td>2009</td>
<td>Germany</td>
<td>O157:H-</td>
<td>No data</td>
<td>8 (4 HUS cases, 1 death)</td>
<td>Nielsen et al. 2011</td>
</tr>
</tbody>
</table>

Tartare (a raw beef product) was revealed as the most likely cause of a STEC 0157 outbreak. A total of 21 laboratory-confirmed cases and another 11 probable cases were reported (Doorduyn et al., 2006). In 2007, an outbreak of *E. coli* O157 infection in Scotland, in which 10 people were affected, was linked to cooked meat from a supermarket delicatessen (McCartney et al. 2010). Two multi-state outbreaks of STEC O157 were recorded during 2008 in the US. A total of 99 persons (64 and 35 from the first and the second outbreak, respectively) had confirmed illness after consuming ground beef (Nowicki et al. 2010). In a study of food-borne outbreaks recorded in England and Wales from 1992-2008, 103 STEC outbreaks were reported, 1,168 people affected, with 286 hospitalizations and 12 deaths (Gormley et al. 2011). These outbreaks were associated with consumption of red meats (38.6%), milk and milk products (29.5%), salad, vegetables, fruit (6.8%) and poultry meat (6.8%).

Several outbreaks of STEC infections have been also attributed to the consumption of raw milk and associated dairy products, particularly raw-milk cheeses. Studies show that *E. coli* O157 strains can survive various stages during cheesemaking (Baylis 2009). A milk-borne STEC O157 outbreak (69 cases, 1 death) was recorded in Scotland during 1994 (Upton and Coia 1994). In 2003, an outbreak of STEC O157 infection occurred in Slovakia. Three children suffered from HUS, two children had bloody diarrhoea, and four adults were asymptomatic carriers. Unpasteurised cow’s milk was identified as the source of infection (Liptakova et al. 2004). Jensen et al. (2006) described the first outbreak of STEC (*E. coli* O157:H1-) with 25 patients in Denmark, after consumption of a particular
kind of organic milk from a small dairy. In 2008, 14
cases (seven confirmed) of STEC infection were iden­
tified after consuming raw milk purchased at a retail
market and a farm in Connecticut (US). Five patients
required hospitalization and three experienced 11US
(Guh et al. 2010). In a suspected milk-borne outbreak
of \textit{E. coli} 0157:H7-associated illness affecting kinder­
garten children, the organism was isolated from cattle
at a farm where raw milk was served (Borczyk et al.
1987). Other foods that have been incriminated in
STEC outbreaks include raw vegetables and salads, and
one of the major such outbreaks took place in Japan in
1996. More than 6,000 school children were affected
and radish sprouts contaminated with \textit{E. coli} 0157:H7
from a single farm were identified as the cause of infec­
tion. More than 101 people were hospitalized with HUS
and 12 deaths were recorded (Michino et al. 1999). In
2007, a STEC 0157 outbreak (33 patients) occurred
in the Netherlands. Pre-packaged, shredded iceberg
lettuce purchased from several supermarket chains was
reported as the possible source (Friesema et al. 2007).
Sigmundsdottir et al. (2007) reported that nine domesti­
cally acquired cases (seven hospitalizations) of STEC
0157 were diagnosed in Iceland. It is interesting that
the isolated strain, which was identified as the causa­
tive agent, was identical to the strain that caused the
outbreak in the Netherlands. Indeed, as verified either
by questionnaires or by supermarket purchase records,
five cases had consumed lettuce packaged and imported
from the Netherlands.

### Non-0157 STEC food-borne outbreaks

Non-0157 STEC serotypes are increasingly being
recognized as the causative agents in food-borne out­
breaks (Table 2). The most striking evidence is the out­
break of STEC O104:H4 infection and HUS reported
in Germany in the spring of 2011, which constituted
one of the largest outbreaks ever described worldwide.
As of 27 July, 3126 cases, including 17 deaths linked
to the outbreak in Germany and occurring in the EU
(including Norway). In addition, in the EU 773 cases of
11US (including 29 deaths) were linked to the German
outbreak. Outside the EU 8 cases of STEC and 5 cases
of HUS, including 1 death have been reported in the
USA, Canada and Switzerland, all with recent travel
history to Germany. The likely source of the outbreak
was contaminated raw sprouts from one farm in Ger­
many. An interesting characteristic in this outbreak is
that the incriminated \textit{E. coli} strain shows an unusual

<table>
<thead>
<tr>
<th>Year</th>
<th>Country</th>
<th>STEC serogroup /serotype</th>
<th>Implicated food</th>
<th>No of people affected</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>1983-2002</td>
<td>USA</td>
<td>non-0157 STEC</td>
<td>Various types</td>
<td>320 (22% O26:H11,16% O111:H8, 12% O103:H2/H11, 8% O121:H19/H7, 7% O45:H2, 5% O145)</td>
<td>Brooks et al. 2005</td>
</tr>
<tr>
<td>1986</td>
<td>Japan</td>
<td>O111:H-</td>
<td>No data</td>
<td>23 children affected, 1 HUS cases, 1 death</td>
<td>Tanaka et al. 1989</td>
</tr>
<tr>
<td>1992</td>
<td>Italy</td>
<td>O111</td>
<td>Unidentified</td>
<td>9 HUS cases; 1 child died</td>
<td>Caprioli et al. 1994</td>
</tr>
<tr>
<td>1999</td>
<td>USA</td>
<td>O111:H8</td>
<td>Lettuce</td>
<td>55 (2 HUS cases)</td>
<td>Brooks et al. 2004</td>
</tr>
<tr>
<td>2000</td>
<td>Germany</td>
<td>O26:H11</td>
<td>Bovine meat</td>
<td>11</td>
<td>Werber et al. 2002</td>
</tr>
<tr>
<td>2004-2007</td>
<td>New Mexico, USA</td>
<td>non-0157 STEC (64%)</td>
<td>No data</td>
<td>7 (18% O26, 13% O111, 33% O103, O121, O46, O177, O91)</td>
<td>Lathrop et al. 2009</td>
</tr>
<tr>
<td>2008</td>
<td>USA</td>
<td>O158</td>
<td>Raw cow milk</td>
<td>14 (3 HUS cases)</td>
<td>Guh et al. 2010</td>
</tr>
<tr>
<td>2011</td>
<td>Germany &amp; other countries</td>
<td>O104:H4</td>
<td>Raw vegetable sprouts</td>
<td>3.134 (778 HUS cases, 47 deaths)</td>
<td>EFSA 2011</td>
</tr>
</tbody>
</table>
combination of virulence factors of STEC and EAEC which has been reported sporadically in humans before. Sequence analysis and comparative genomics will be able to show if the German outbreak strain is an EAEC that acquired EHEC virulence determinants or if it is the other way around (EFSA 2011). According to the information reported to the European Center for Disease Prevention and Control (ECDC 2011), there were 10 reported cases of STEC O104 infection in the EU Member States and Norway between 2004 and 2010. Moreover, a case of HUS, which occurred in Italy in 2009 has now been associated with STEC O104, resulting in a total of 11 cases.

Werber et al. (2002) recorded a STEC O26:H111 outbreak (11 cases) in Germany in 2000 and a certain type of beef ("seemerrolle") was revealed as the probable source of infection. In France, in 2005, one outbreak of STEC O26 and O80 involving 16 HUS cases was linked to the consumption of unpasteurized cows’ cheese. Also, in France, evidence of STEC involvement in 590 (66%) of 900 HUS cases examined from 1996 to 2006 has been provided; thirty-five (6%) patients had been infected with STEC O26, 15 (2.5%) with O103, 11 (2%) with O145, 4 (0.7%) with O91, 4 (0.7%) with O111, and 4 (0.7%) with O55 (Espie et al. 2008). The most frequent STEC serogroups implicated in STEC food-borne outbreaks and HUS cases in Europe are shown in Table 3 (EFSA 2009).

During 2004-2007, in New Mexico (US) 71 of 111 cases (64%) of sporadic STEC infections were caused by non-O157 STEC with O26 (18%) and O111 (13%) being the most commonly identified serogroups. Serogroups O103, O121, O46, O177 and O91 were responsible for 33% of all STEC infections (Lathrop et al. 2009).

According to Brooks et al. (2005) STEC O111 is the second most common bacterial cause of HUS in the US, after STEC O157:H7. It was identified as the etiological agent in three of seven reported outbreaks of non-O157 STEC serotypes. Eleven (52%) of the 21 non-O157 STEC isolates associated with HUS were STEC O111. An outbreak of acute enteritis associated with STEC O111:H1- occurred in 1986 in Japan, with 23 children being affected, one developing HUS and dying (Tanaka et al. 1989). In 1992, nine children were hospitalized with HUS and one child died in Italy. The source of the outbreak was not identified, but STEC O111:NM was the most likely cause (Cappioli et al. 1994). In 1995, during an outbreak of HUS (21 children) in South Australia, STEC O111:H2 was identified as the principal cause and a locally produced semidry fermented sausage (mettwurst) as the incriminated vehicle (Paton et al. 1996). In 1999, a STEC O111:H18 outbreak at a youth camp was associated with consumption of lettuce. Fifty-five persons became ill and two women developed HUS (Brooks et al. 2004). In 2007, a mixed-serotype outbreak involving STEC O145:H28 and O26:H111 occurred in Belgium, after consumption of contaminated ice cream. Five girls developed HUS, and seven patients with bloody diarrhea were identified (Buvens et al. 2011). In 2006, a small but severe STEC O103:H25 outbreak, involving 17 persons (10 developing HUS, 1 child died), was reported in Norway, after the consumption of a traditional Norwegian sausage (morropoelse) made from sheep meat (Schimmer et al. 2008).

This literature review on STEC food-borne outbreaks highlights the need for measures in order to prevent similar events from happening in the future. The main focus in mitigating risks of food contamination and human infection from STEC should be on the prevention of contamination at all production stages reducing the likelihood of STEC to enter the food chain. The application of Good Agricultural Practices, Good Manufacturing Practices, Good Hygiene Practices and of the HACCP system in line with codes available from international organizations is recommended.

Table 3. The most frequent STEC serogroups implicated in STEC food-borne outbreaks and HUS cases in Europe.

<table>
<thead>
<tr>
<th>STEC</th>
<th>HUS</th>
</tr>
</thead>
<tbody>
<tr>
<td>No.*</td>
<td>%</td>
</tr>
<tr>
<td>O157</td>
<td>7,227</td>
</tr>
<tr>
<td>O26</td>
<td>732</td>
</tr>
<tr>
<td>O103</td>
<td>603</td>
</tr>
<tr>
<td>O91</td>
<td>425</td>
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<tr>
<td>O145</td>
<td>312</td>
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<td>O111</td>
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<td>O146</td>
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<td>O128</td>
<td>93</td>
</tr>
<tr>
<td>O55</td>
<td>74</td>
</tr>
<tr>
<td>Other</td>
<td>1,199</td>
</tr>
<tr>
<td>Total</td>
<td>10,998</td>
</tr>
</tbody>
</table>

(*Source: EFSA, 2009)
REFERENCES


