Chapter 2
Outbreaks of Shiga Toxin-Related Poisoning

Abstract  Foodborne outbreaks of Shiga toxin-producing bacteria occur with disturbing regularity. The two most common Shiga toxin-producing bacteria are Shigella spp. and the Shiga toxin-producing Escherichia coli (STEC). Among foodborne pathogens, they result in a disproportionately large share of hospitalizations, serious sequelae, and deaths. In 1982 the first reported outbreak of STEC was caused by an E. coli O157:H7 serotype in undercooked hamburger, but by 2008 it was estimated that most foodborne STEC disease was caused by other serotypes. As the food service industry has adopted more stringent cooking practices and as diets have changed, the sources of outbreaks have shifted as well. The two largest outbreaks on record were caused by consumption of uncooked radish and fenugreek sprouts. There are at least 200 different known strains of STEC found in farm animals, where they can propagate “below the radar” because farm animals lack the receptors that would make them vulnerable to Shiga toxins. Future outbreaks are likely to involve other uncooked foods and different strains of STEC, and they may be triggered by the agricultural practices, food processing and transportation conditions, and ecological factors that bring them together.

Keywords  Shiga toxin poisoning • Quick serve restaurant • Effective dose • Pathogenic Escherichia coli • Shigellosis • Escherichia coli O157:H7 • Escherichia coli O104:H4 • Shigella spp. • Foodborne illness, Sakai, Japan • STEC outbreak, Germany

2.1  Human and Economic Impacts of STEC Outbreaks

The economic cost of Shiga toxin poisoning in humans is substantial. The situation in the United States can be used as an illustration of a worldwide problem. In 1996–1997, an estimated 110,000 people per year were infected with Stx-producing bacteria in the US (Mead et al. 1999). Of those, 2200 were hospitalized and 60 died. A subsequent analysis covering the years 2000–2008, estimates the number of STEC cases to be approximately 175,000 per year (Scallan et al. 2011). Between 2003 and
2012 in the US, outbreaks of disease caused by *E. coli* O157:H7 had routes of transmission that were foodborne (65%), person-to-person (10%), animal contact (10%), waterborne (4%) or undetermined (11%) (Heiman et al. 2015). These more recent estimates suggest that approximately 64% of STEC infections are caused by non-O157:H7 strains and that the majority (68–82%) of cases are foodborne (Scallan et al. 2011). The direct economic costs of Shiga toxin infections in the United States are estimated to be at least 400 million USD and possibly in excess of one billion USD per year (Frenzen et al. 2005; Hoffmann et al. 2015). The indirect costs are more difficult to estimate, but are substantial. These sums indicate that STEC infections represent a significant cost to the United States in terms of both misery and money.

STEC are a subset of pathogenic *Escherichia coli*, but they often possess virulence factors found in other pathogenic *E. coli* (Tozzoli and Scheutz 2014). Table 2.1 lists this diverse cast of characters and some of their most salient characteristics, including virulence factors such as intimin, fimbriae, heat-labile enterotoxin (LT), and heat-sta-

<table>
<thead>
<tr>
<th><strong>E. coli</strong> group name</th>
<th>Acronym</th>
<th>Properties</th>
</tr>
</thead>
<tbody>
<tr>
<td>Attaching effacing</td>
<td>AEEC</td>
<td>Cause attaching and effacing lesions</td>
</tr>
<tr>
<td>Adherent-invasive</td>
<td>AIEC</td>
<td>Invade cells to cause their pathology</td>
</tr>
<tr>
<td>Avian pathogenic</td>
<td>APEC</td>
<td>Cause extraintestinal infections in birds</td>
</tr>
<tr>
<td>Diffusely adherent</td>
<td>DAEC</td>
<td>Cause diarrhea in children</td>
</tr>
<tr>
<td>Diarrheagenic</td>
<td>DEC</td>
<td>Cause the symptom of diarrhea, as part of pathology</td>
</tr>
<tr>
<td>Enteroaggregative</td>
<td>EAEC or EAggEC</td>
<td>Aggregate host cells, produce hemolysin, and heat-stable (ST) enterotoxin to cause pathology that leads to watery diarrhea without a fever</td>
</tr>
<tr>
<td>Enteroaggregative haemorrhagic</td>
<td>EAHEC</td>
<td>EAEC that also express Shiga toxin(s)</td>
</tr>
<tr>
<td>Enterohemorrhagic</td>
<td>EHEC</td>
<td>Express Shiga toxin(s) and encode a locus of enterocyte effacement (LEE)</td>
</tr>
<tr>
<td>Enteroinvasive</td>
<td>EIEC</td>
<td>Invade cells, causing diarrhea and high fever.</td>
</tr>
<tr>
<td>Enteropathogenic</td>
<td>EPEC</td>
<td>Attach to host cells via intimin (int); effacement of cells; may invade various tissues</td>
</tr>
<tr>
<td>Enterotoxigenic</td>
<td>ETEC</td>
<td>Noninvasive; attach to enterocytes; produce heat-labile (LT) and/or heat-stable (ST) enterotoxins; cause traveler’s diarrhea</td>
</tr>
<tr>
<td>Extraintestinal pathogenic</td>
<td>ExPEC</td>
<td>Leave the intestinal area to cause diseases such as sepsis and urinary tract infections</td>
</tr>
<tr>
<td>Intestinal pathogenic</td>
<td>IPEC</td>
<td>Cause pathology inside the intestine</td>
</tr>
<tr>
<td>Meningitis-associated</td>
<td>MNEC</td>
<td>ExPEC that cause meningitis and sepsis</td>
</tr>
<tr>
<td>Shiga toxin-producing</td>
<td>STEC/VTEC</td>
<td>Express Shiga toxin(s)</td>
</tr>
<tr>
<td>Uropathogenic</td>
<td>UPEC</td>
<td>ExPEC that cause uropathology</td>
</tr>
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ble toxin (ST). The most serious of these pathogens are the Shiga toxin-producing *E. coli* (STEC), also known as Verotoxin-producing *E. coli* or Verocytotoxin-producing *E. coli* (VTEC). Although STEC may have the same virulence factors found in other pathogens, they also produce Shiga toxin(s), their major virulence factor. Delivery of a single Shiga toxin molecule to the cytoplasm of a target cell is sufficient to kill it.

The symptoms of a STEC infection are highly varied, but most patients recover in 5–7 days without specialized treatment. Most suffer only moderate to severe stomach cramps with or without diarrhea. For example, a survey of patients infected with STEC (O157:H7 serotype) between 2003 and 2012 in the US revealed that 83% had symptoms that were not serious enough to require hospitalization (Heiman et al. 2015). The initial symptoms were moderate to severe abdominal discomfort with diarrhea. Throughout the disease course, there is usually no fever, but if it is present, it is not very high (<101 °F; <38 °C). The clinical manifestations of STEC infections can then progress from diarrhea to hemorrhagic colitis (HC, bloody diarrhea). Among children under the age of 10, approximately 15% of cases progress to the more serious hemolytic uremic syndrome (HUS) (Tarr et al. 2005). The overall mortality rate for infected patients is approximately 0.2%; for hospitalized patients, approximately 3%; and for patients with HUS, about 11%.

STEC infections are not the most common cause of foodborne illness, but they account for a disproportionately large share of the hospitalizations and deaths. For example, between 2009–2010 *Salmonella* accounted for 54% of reported US foodborne illnesses and 66% of the hospitalizations (Anonymous 2013). During the same time frame STEC accounted for less than 5% of reported cases, but 21% of hospitalizations (Anonymous 2013). Between 1998 and 2014 STEC accounted for 2.5% of total cases of foodborne illness, 13% of hospitalizations, and nearly 11% of the deaths associated with foodborne illness (FOODTool). The hospitalization rate for diseases associated with STEC infection is much higher than that seen for more common foodborne illnesses, such as those caused by *Salmonella* or *Campylobacter* (Mead et al. 1999).

STEC infections incur costs beyond those directly associated with treating the infected patients. The hamburger-associated STEC outbreak that occurred in the US in 1992 cost the QSR chain $160 million in lost sales and a 30% loss of its stock value (1992 USD) (McGrath 2009). In addition, there were costs related to litigation in excess of $40 million (1992 USD). A 2002 outbreak of STEC affecting 28 people led to the recall of 8.6 million kg of ground beef (Anonymous 2002). The estimated loss of bagged spinach sales as a result of a 2006 outbreak was in excess of $200 million (2006 USD) (Arnade et al. 2009). Even larger outbreaks of STEC have occurred in Japan and Germany (Frank et al. 2011; Michino et al. 1999). The sources of an outbreak can be limited to a single ingredient from a single farm, such as occurred in Japan, where one farm was responsible for the entire outbreak (Michino et al. 1999). A single supplier of contaminated fenugreek seeds was responsible for the outbreak in Germany (Buchholz et al. 2011). Since foods are often mixtures of ingredients, contamination from a single source can affect suppliers of the other ingredients, even if they were not contaminated. In this way the indirect economic effects of an outbreak can greatly exceed the direct costs associated with treating the afflicted patients.
2.2 Effectiveness of STEC in Causing Severe Disease Outbreaks

*E. coli* O157:H7 serotype is an extraordinarily effective organism for delivering Shiga toxins to the human intestine. The O157:H7 serotype has a number of mechanisms that make it very acid resistant, enabling it to pass through the human digestive tract unscathed (Miszczycha et al. 2014). It is so effective at surviving the stomach that the number of bacteria that need to be ingested to cause disease in humans is estimated to be less than 50 and perhaps as few as two STEC cells (Tilden et al. 1996), compared to 1000 colony-forming units (CFU) for ETEC and 1 million CFU for EIEC. Other Shiga toxin-producing bacteria associated with foodborne illness, such as *Shigella dysenteriae* type 1, are also able to cause human infection with inocula as low as 10–100 CFU (DuPont et al. 1989). These bacteria are an efficient means of delivering the toxin to the intestine, where it inflicts its damage.

Shiga toxin itself is not well acquitted to survive the journey through the stomach when orally delivered by STEC. Other toxins, such as botulinum toxins, have both structural elements and accessory molecules that allow them to survive journey through the human stomach and intestine to the bloodstream (Miyata et al. 2009). The intraperitoneal (IP) median lethal dose (LD$_{50}$) for type 2 Shiga toxin is 50 ng/kg or approximately 1 ng/mouse (Fuller et al. 2011; Tesh et al. 1993). However, when Stx1 or Stx2 are transmitted orally, the LD$_{50}$ for Stx2a is approximately 3 μg/mouse and greater than 150 μg/mouse for Stx1a (Russo et al. 2014). Thus, the LD$_{50}$ of a Shiga toxin is increased 1000-fold when delivered through the stomach. This is to be expected, since Shiga toxins are not stable at the pH of the stomach (Skinner et al. 2013). Further along the digestive tract, Stx has structural features that protect it from proteolytic digestive enzymes that are secreted into the intestine. Overall, these complex processes ensure that Shiga toxins are able to cause maximal damage to a human patient.

2.3 History of STEC Outbreaks and Their Continuing Evolution

The earliest reports of STEC disease were sporadic cases reported in the early 1980s (Anonymous 1982; Karmali et al. 1983). The first outbreak of STEC food poisoning was reported in the US in 1982 and was linked to a QSR chain in the states of Oregon and Michigan (Riley et al. 1983; Wells et al. 1983). In 1993, a more substantial and highly publicized outbreak occurred in another QSR chain in the state of Washington and clearly demonstrated the significant consequences of STEC (Bell et al. 1994). The cause of these outbreaks was eventually traced back to undercooked hamburger. The magnitude of the outbreak led to mandatory reporting of the O157:H7 serotype of *E. coli* in the United States (CSTE). A more recent outbreak,
in 2015, occurred in a fast casual restaurant chain (QSRs with more varied menu, where the meal is assembled in a separate location, before the customer relocates it to a table). The outbreak was caused by the O26 serogroup (Anonymous 2016), but the source of the contaminated food was not determined. This series of episodes made the general public aware that the once obscure STEC pathogens can be transmitted by a changing variety of foods.

Worldwide awareness of foodborne STEC-related infection paralleled the experience in the US. In the United Kingdom, the first reported outbreak of STEC (O157:H7 serotype) occurred in 1982 (Taylor et al. 1986), and the first foodborne outbreak occurred in 1985. Surprisingly, the associated food was potatoes, not beef (Morgan et al. 1988). In England and Wales, STEC outbreaks have remained relatively constant; those caused by infected meat and dairy declined, while those associated with children’s activities (nurseries, schools, petting farms) increased from 1983–2012 (Adams et al. 2016). The largest outbreak reported so far occurred in 1996, in Japan where more than 8300 school children were sickened by eating contaminated, uncooked white radish sprouts (Michino et al. 1999). In 2011, an outbreak of STEC sickened more than 3800 people in Germany (Frank et al. 2011). The source of the disease was traced back to fenugreek sprouts. STEC infection is now a major worldwide health concern (Adams et al. 2016; Caprioli et al. 2014; Heiman et al. 2015; Terajima et al. 2014), with sources of the outbreaks reflecting the distribution of STEC and changing national food preferences.

The strains of STEC responsible for outbreaks have also changed over time. Domestic cattle alone are known to harbor more than 200 strains of STEC, and there are a large number of potential outbreak strains (Hussein and Bollinger 2005). It is therefore not surprising that while O157:H7 remains a serious threat, strains from other serotypes have emerged to rival that threat. During the survey period of 1996–1997, approximately 2/3 of STEC cases in the United States were caused by strains of the O157:H7 serotype (Mead et al. 1999). In contrast, a survey spanning the years from 2000–2008 estimated that the total number of STEC-caused disease cases increased, mainly due to non-O157:H7 serotypes (Scallan et al. 2011). By 2010, the actual incidence of non-O157:H7 serotype STEC infections equaled that of O157:H7 STEC infections in the US for the first time (Gould et al. 2013). Regulators have responded by classifying strains from six non-O157:H7 serotypes, O26, O45, O103, O111, O121, and O145 (the “Big Six”), along with O157:H7, as adulterants in ground beef that need to be screened for (Almanza 2012). However, the 2011 STEC outbreak that sickened more than 3800 people in Germany was caused by an O104:H4 serotype of STEC (Frank et al. 2011), a serovar not included among the Big Six. The German experience suggests that future outbreaks are likely to be caused by strains that are not on current regulatory lists.

Outbreaks of STEC in the US are recorded and available to researchers in a variety of databases and publications. The CDC produces the Morbidity and Mortality Weekly Report (MMWR), a weekly publication devoted to providing “timely, reliable, authoritative, accurate, objective, and useful public health information and recommendations” (MMWR). In addition to the information contained in the weekly report, the CDC maintains other web-based resources devoted to gathering
and disseminating information about disease outbreaks. These include the Foodborne Disease Outbreak Surveillance System (FDOSS) and the Waterborne Disease Outbreak Surveillance System (WBDOSS), now known as the Waterborne Disease & Outbreak Surveillance & Reporting (WDOSR). The Foodborne Outbreak Online Database (FOOD Tool) also provides an historical view of outbreaks (FOODTool). The National Outbreak Reporting System (NORS) allows health officials to report “waterborne and foodborne disease outbreaks and enteric disease outbreaks transmitted by contact with environmental sources, infected persons or animals, or unknown modes of transmission to CDC” (NORS). These websites provide useful, accurate information about outbreaks and archive their data for a number of years.

In addition to foodborne outbreaks, Shiga toxin-related disease caused by ingestion of STEC or *Shigella sonnei* has been transmitted by water (Auld et al. 2004; Bopp et al. 2003; Dev et al. 1991; Hrudey et al. 2003; Olsen et al. 2002; Swerdlow et al. 1992; Yarze and Chase 2000). In the US, most waterborne outbreaks are associated with recreational water use (Friedman et al. 1999; Keene et al. 1994; Paunio et al. 1999; Samadpour et al. 2002; Verma et al. 2007), but three large outbreaks occurred when Stx-producing bacteria (*S. sonnei* or *E. coli* O157:H7) contaminated drinking water in Florida, Missouri, and New York (Bopp et al. 2003; Swerdlow et al. 1992; Weissman et al. 1976). The source of waterborne STEC is often well water contaminated with fecal material. In 2000, nearly half the population of Walkerton, Ontario, Canada was sickened by drinking municipal water contaminated with STEC O157:H7 (Anonymous 2000; Hrudey et al. 2002). Subsequent investigation determined that water was contaminated with runoff from fields fertilized with STEC-containing bovine manure and then inadequately chlorinated before being supplied to consumers (Auld et al. 2004; Hrudey et al. 2003). The extent of waterborne outbreaks often dwarfs foodborne incidents because thousands of people may be exposed to a single water source. A survey of untreated water in Brazil found STEC, but not the O157:H7 strain (Lascowski et al. 2013). Again in the US, STEC is rarely a source of waterborne illness, but when it does occur, the health impact is disproportionately large compared to that seen with other microorganisms (Hynds et al. 2014).

The two largest foodborne STEC disease outbreaks occurred in Japan and Germany and were caused by consumption of contaminated fresh produce. In the US, the first outbreak of STEC transmitted by the consumption of fresh produce occurred in 1991, when apple cider was contaminated with *E. coli* O157:H7 (Besser et al. 1993). A foodborne outbreak occurred in 1996 via processed, but unpasteurized apple juice contaminated with *E. coli* O157:H7 (Cody et al. 1999), and fruit juices have remained a periodic source of outbreaks (Vojdani et al. 2008). Even the flour in cookie dough has been implicated in an STEC outbreak (Neil et al. 2012). The first reported produce-related outbreak of STEC-related disease caused by the O157:H7 serotype (1995) was traced back to contaminated lettuce (Ackers et al. 1998). Other outbreaks caused by this infamous STEC serotype (some discussed above) have been attributed to contaminated freshly bagged spinach and other fresh leafy produce in the US (Anonymous 2006; Grant et al. 2008; US-FDA 2006), the Netherlands, Iceland (Ackers et al. 1998; Friesema et al. 2008; Hilborn et al. 1999)
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and Sweden (Soderstrom et al. 2008). The potential hazards of consuming contaminated raw sprouts is a worldwide problem (Taormina et al. 1999). In the US, STEC outbreaks were associated with contaminated alfalfa sprouts from several producers in California (1996–1998) and more recently (2003) in Colorado and Minnesota (Breuer et al. 2001; Ferguson et al. 2005; Mohle-Boetani et al. 2001). Fresh produce has been a source of a number of STEC outbreaks in Canada (Bolduc et al. 2004; Kozak et al. 2013).

In the United States, contaminated fresh produce has become an increasing source of foodborne outbreaks (Fig. 2.1) (Sivapalasingam et al. 2004). Perhaps not a coincidence, this trend comes at a time when the consumer demand for fresh fruits and vegetables has increased by 25% over the years 1961–2000 (Pollack 2001). In contrast, between 1971 and 2000 there was a 20% per capita reduction in the demand for beef in the United States (Haley 2001). This shift in consumer preferences mirrors a change in the properties of the foodborne STEC contaminants, particularly related to their ability to attach to surfaces. The pathogenicity markers for the O157:H7 strains are related to the attachment and effacement mechanism used by the O157:H7 serovar to bind to intestinal cells. These markers are genes that were absent in the

Fig. 2.1 Sources of foodborne STEC outbreaks. Types of food associated with outbreaks are shown as percentages of the total number of STEC-related illnesses reported in 1998–2000 (illnesses, 4111; outbreaks, 97), 2005–2007 (illnesses, 1647; outbreaks, 83), and 2012–2014 (illnesses, 1225; outbreaks, 86). Source: Foodborne Outbreak Online Database (FOODTool)
STECC outbreak serotype in Germany in 2011 (Bielaszewska et al. 2011). Instead, that serovar had genes that encoded factors enabling it to aggregate. These trends underscore the evolutionary potential of STEC, capable of finding a niche in edible plants as well as in its more familiar habitat, the mammalian colon. In summary, uncooked fruits and vegetables can efficiently transmit STEC to consumers.

As mentioned above, the 2006 outbreak in the United States caused by the O157:H7 serotype was associated with the consumption of bagged spinach (Anonymous 2006). The strain of O157:H7 associated with the outbreak was also isolated from animals on a nearby ranch and from feral swine (Jay et al. 2007). This represented the first time the O157:H7 strain had been isolated from a feral swine in the United States. A detailed examination of the watershed revealed that *E. coli* O157:H7 could have been present in some fields at various times during the 19-month study (Cooley et al. 2007). This suggests that the outbreak may have resulted from contamination by run-off surface water from a nearby dairy farm or contamination by feral swine. Although the specific source of the outbreak was never pinpointed, these results emphasize that produce is grown in an environment that is dynamic and difficult to control.

### 2.4 Less Common Sources of STEC Outbreaks

Person-to-person contact is a significant factor in STEC outbreaks because such a low number of STEC cells can cause an infection. Between 2008 and 2009 there were a number of STEC outbreaks transmitted person-to-person (Wikswo and Hall 2012). Most of these cases involved facilities serving populations that are most susceptible to Stx: the very young, in nursery or daycare centers, and the elderly, in senior and geriatric care facilities. Documented person-to-person transmission of STEC remains relatively rare outside highly vulnerable populations.

*E. coli* O157:H7 has a high prevalence among domestic cattle and other domestic animals and is found in the feces, hides and hair of infected animals (Persad and LeJeune 2014). Consequently, gardening and visits to farms or petting zoos are also documented sources of STEC infection. The first case of STEC transmitted by animal contact was reported in 1992 (Renwick et al. 1993). STEC can be transmitted by touching the hair or hide of an infected animal (Elder et al. 2000). Two such outbreaks occurred in 2001, when children visiting farms in Pennsylvania and Washington state were infected with the O157:H7 serotype (Anonymous 2001). STEC outbreaks have frequently been associated with children and petting zoos in the US (Anonymous 2009; Goode et al. 2009; Heuvelink et al. 2002; Stirling et al. 2008). An outbreak in England involved children who came in contact with livestock during a “Lambing Live” event (Rowell et al. 2016), and petting zoos and farm tours remain a source of STEC infections in the UK (Stirling et al. 2008). Other outbreaks of *E. coli* O157:H7 infection resulted from farm visits by children, including one associated with a dairy farm in Japan (Anonymous 2001; Kassenborg et al. 2004; Muto et al. 2008).
The following discussion covers miscellaneous, but documented transmission of STEC-caused disease. The first case of STEC-caused disease being transmitted to a laboratory worker as an occupational hazard occurred in 2002. A garden fertilized with manure was another source of outbreak (Cieslak et al. 1993). Perhaps more startling was an outbreak of STEC O157:H7 that involved airborne dispersion and/or contaminated building surfaces at a County Fair, a traditional event in thousands of US counties in summertime (Varma et al. 2003). Studies have documented that many farm workers and their families have circulating antibodies to Shiga toxins and to the O and H antigens of *E. coli*, and some had apparently experienced infection with *E. coli* O157:H7, without clinical symptoms (Wilson et al. 1996).

Even though many STEC serotypes have been isolated from domestic cattle, not all of those isolated from humans are found in domestic cattle. A microbiological survey of the STEC present in domestic cattle revealed that approximately 261 STEC serotypes were present (Hussein and Bollinger 2005). By comparison, the number of different STEC serotypes found in human patients exceeds 400 (Tozzoli and Scheutz 2014). Domestic cattle are thought to be the major source of STEC outbreak serotypes (Karmali et al. 2010), but the STEC serotype (O104:H4) responsible for the outbreak in Germany has never been reported to be found in domestic cattle (Paddock et al. 2013; Shridhar et al. 2016). Even when researchers searched for it in domestic cattle in Germany after the 2011 outbreak, they were again unable to isolate the serotype (Wieler et al. 2011). It is not clear from which animal, if any, the O104:04 STEC serotype originated, but it does not appear to have been domestic cattle. Hence, identifying sources of origin for STEC, other than domestic cattle, is a pressing challenge.

In view of the low number of STEC cells necessary for infection, the source of an STEC outbreak can be quite prosaic, such as brief environmental exposure. Sources of future STEC outbreaks will undoubtedly include the infamous O157:H7 strain along with previously unrecognized strains. The ease of mobility and variability of the Shiga toxin-producing phages in domestic cattle will undoubtedly be the cause of many future outbreaks, but we can expect some surprises, too. It had been assumed that the attaching and effacing genes associated O157:H7 and other serotypes were required for an STEC serotype to cause serious disease. However, the 2011 outbreak in Germany was caused by an O104:H4 strain that does not have attaching and effacing genes (*vide infra*). Instead, the serotype possesses the enteroaggregative genes which are also known in other serotypes, but had never been associated with a large outbreak before (Bielaszewska et al. 2011). This experience suggests that future outbreaks may be caused by other serotypes that lack the genes that enable them to efficiently attach to intestinal cells. Since Shiga toxins do not cause disease in domestic cattle, Shiga toxin-producing phages can infect new *E. coli* serotypes and undergo recombination or modification by mobile genetic elements (discussed in Chapter 3), all without affecting bovine health. These facts suggest that domestic cattle will remain a reservoir for a constantly increasing number of STEC serotypes, whether they are passed to humans via consumption of plant- or animal-derived foods.
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