

Bad Bug Book

Foodborne Pathogenic Microorganisms and Natural Toxins

Enterotoxigenic *Escherichia coli* (ETEC)

1. Organism

Enterotoxigenic *Escherichia coli* (ETEC) are highly motile, Gram-negative, rod-shaped bacteria. They are characterized by production of several virulence factors, including both heat-labile (LT) toxin and heat-stable (ST) toxins, as well as several colonization-factor antigens.

2. Disease

ETEC causes gastroenteritis in humans and is best known as the causative agent of travelers' diarrhea. It is also an important cause of diarrhea in infants, in less-developed countries.

- **Mortality:** The World Health Organization attributes 380,000 deaths (worldwide) to ETEC, mostly among children, each year.
- **Infective dose:** Volunteer feeding studies showed that a high dose, ranging from 10 million to 10 billion ETEC cells, may be needed to cause an infection in adults. Children may be affected by a smaller dose.
- **Onset:** Usually 26 hours after ingestion of contaminant, but can range from 8 to 44 hours.
- **Disease / complications:** Illness from ETEC is usually self-limiting, mild, and brief. However, some severe forms last longer and resemble cholera, with up to five or more daily passages of rice-water-like stools that result in severe dehydration. Antibiotic treatment usually is not required in ETEC infections, but seems to be effective in reducing the duration and severity of illness. In infants and elderly and debilitated patients, particularly, appropriate electrolyte replacement therapy may be necessary.
- **Symptoms:** Infection is characterized by sudden onset of diarrhea that is watery and without blood or mucus, rarely accompanied by high fever or vomiting. Other symptoms include abdominal cramps, low-grade fever, nausea, and malaise.

For Consumers: A Snapshot

This chapter is about the bacterium *E. coli*, but not the kind you've heard about in news reports about outbreaks. That kind of *E. coli* sometimes causes kidney failure and death, but the kind in this chapter, called ETEC for short, causes traveler's diarrhea. People in the U.S. usually don't get ETEC infections, unless they travel to areas of the world with poor sanitation. In most people, the illness goes away by itself, after a few days of bowel movements that look like rice-water, and cramps, perhaps a low fever, and nausea. But some people, especially infants and people with weak immune systems, can develop a severe illness more like cholera, which lasts up to 19 days. For these people, especially, getting treatment is very important. They can lose so much fluid from diarrhea that it upsets the chemical balance of their blood, which can lead to heartbeat disturbances and may even lead to death. In the U.S., this doesn't happen often, but in countries with poor sanitation, ETEC is a major cause of infant death. Contaminated water often is the source of ETEC, and that water or an infected food handler can contaminate food. Once it gets inside the bowels, the bacterium produces a toxic substance that causes the illness. You can help protect yourself from ETEC by drinking bottled water if you travel outside the U.S. and following basic [food-safety tips](#).

- **Duration of symptoms:** Most cases last a few days; however, severe forms can last up to 19 days.
- **Route of entry:** Oral.
- **Pathway:** After ingestion, ETEC colonizes the small intestine, where the toxins that induce fluid secretion are produced.

3. Frequency

ETEC outbreaks are infrequent in the United States, but infections are a more common occurrence among travelers to foreign countries. ETEC infections are endemic in many developing tropical countries and areas with poor hygiene standards. Infections are more prevalent in the warmer, wet months.

4. Sources

Most ETEC outbreaks are linked to consumption of contaminated food or water. ETEC is often found in feces of asymptomatic carriers, and humans appear to be the most likely source of ETEC. In 1975, a large outbreak affecting 2,000 people was traced to sewage-contaminated water at a national park. Contaminated well water in Japan and water supplies aboard cruise ships also have been implicated in ETEC outbreaks. Foodborne outbreaks of ETEC have occurred in restaurants and at various catered functions.

Examples of implicated foods include Brie cheese, curried turkey, [mayonnaise](#), crabmeat, deli food, and salads. In most of these cases, foods became contaminated with ETEC via infected food handlers or through the use of contaminated water during preparation. ETEC infection does not appear to be transmitted by person-to-person contact, but some hospital infections have occurred and probably were caused by unsanitary conditions.

5. Diagnosis

During the acute phase of infection, large numbers of ETEC cells are excreted in feces. Since generic *E. coli* are also abundantly present in the bowels, ETEC strains can be differentiated from other *E. coli* by *in vitro* immunochemical assays, tissue culture, or gene probes and PCR assays specific for LT and ST toxin genes. Commercial kits that use antibodies to detect these toxins are also available.

6. Target Populations

Infants and travelers to underdeveloped countries are most at risk of ETEC infection. As with other infections, people with weak immune systems are more likely than others to suffer severe, even life-threatening cases.

7. Food Analysis

Presence of ETEC in foods can be determined by plating culture enrichment of food samples onto media that are selective and differential for *E. coli* and testing the isolates for presence of LT and ST toxins, using PCR or commercial kits that use antibodies specific to the toxins. Because of its high infectious dosage, ETEC analyses usually are not performed unless generic *E. coli* levels are very high.

8. Examples of Outbreaks

See the Centers for Disease Control and Prevention's [Morbidity and Mortality Weekly Reports](#).

9. Other Resources

- Loci index for [genome](#)
- GenBank [Taxonomy Database](#)

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Foodborne Pathogenic Microorganisms and Natural Toxins

Enteropathogenic *Escherichia coli* (EPEC)

1. Organism

EPEC are Gram-negative, rod-shaped bacteria. They are characterized by the presence of the locus for enterocyte effacement (LEE) pathogenicity island, which carries multiple virulence factors, including the *eae* gene that encodes for intimin and, together with the *tir* gene (intimin receptor), allows intimate adherence of EPEC to intestinal epithelial cells. In the 1940s and 1950s, EPEC was a frequent cause of infantile diarrhea in the United States. Currently, EPEC infections are less important in developed countries, but continue to be a common cause of diarrhea in developing countries, especially in children less than 2 years old.

2. Disease

The disease usually associated with EPEC is infantile diarrhea.

- **Mortality:** Mortality rates from 25% to 50% have been reported in the past. In developed countries, better treatment and medical facilities have greatly reduced mortality, but some deaths still occur.
- **Infective dose:** EPEC is highly infective in infants, and the dose is presumably very low. Adults, however, are not as susceptible. Volunteer feeding studies showed that 10 million to 10 billion cells are needed to cause diarrhea in adults, provided that gastric acid first has been neutralized by bicarbonate.
- **Onset:** Onset of diarrhea is often rapid, occurring as soon as 4 hours post ingestion of EPEC.

For Consumers: A Snapshot

Many *E. coli* are harmless, but some cause serious illness if they contaminate foods and are eaten. This book covers four kinds of *E. coli* that cause foodborne illness. The one in this chapter, called “EPEC” for short, isn’t the one that causes many of the outbreaks that make headlines in newspapers. (That kind, sometimes called “EHEC,” includes *E. coli* O157:H7, can cause problems like kidney failure, and is especially serious.) Although EPEC mostly affects countries with poor sanitation and has become less of a problem in countries like the U.S., it still can occur here. Over the decades, with advances in medicine and sanitation, the death rate from foodborne EPEC has dropped in developed countries. In a recent estimate of illness from contaminated food in the U.S., the Centers for Disease Control and Prevention listed no deaths from EPEC. Still, this illness should be taken seriously, as it occurs most often in children under age 2. The symptoms are often mild, but sometimes are severe, lasting for weeks or months. In the most severe cases, it can cause so much watery diarrhea that a child loses dangerous amounts of body fluid and minerals, and medical treatment is needed. Day-care centers and pediatric wards in countries with poor sanitation are often high-risk places for EPEC outbreaks. In those countries, bottle-fed babies seem to be at highest risk – but any food or fluid contaminated with feces (from someone infected who doesn’t wash his or her hands after a bowel movement, for example) can spread the illness. To help protect yourself and others, follow basic [food-safety tips](#). For tips about how to handle baby bottles safely, [see the chapter called “Cronobacter,”](#) and look for the box called “For Consumers: A Snapshot.”

- **Illness / complications:** The diarrhea can be mild; however, the infection sometimes can be severe. Fluid and electrolyte imbalance may need to be corrected, to prevent dehydration.
- **Symptoms:** Profuse, watery diarrhea; vomiting; and low-grade fever.
- **Duration:** Diarrhea occasionally is protracted, lasting from 21 to 120 days.
- **Route of entry:** Oral.
- **Pathway:** After ingestion, EPEC adheres to intestinal mucosa and causes extensive disarrangement of the digestive-absorptive enzyme system, resulting in nutrient malabsorption.

3. Frequency

Foodborne outbreaks of EPEC are sporadic. Incidence varies on a worldwide basis, but countries with poor sanitation practices have the most frequent outbreaks. Many of these occur in day-care centers and pediatric wards.

4. Sources

Source(s) and prevalence of EPEC are controversial, because foodborne outbreaks are sporadic. Foods implicated in past EPEC outbreaks have included raw beef and chicken, but any food exposed to fecal contamination is strongly suspect. In the mid 1990s, an EPEC outbreak in Minnesota was traced to a buffet, but no specific food item was identified. In 1995, two outbreaks in France affected 59 people and were traced to mayonnaise, lettuce, and pickles.

5. Diagnosis

Diagnosis consists of culturing for *E. coli* from stools of infected people and testing the isolates for the ability to cause attachment and effacing (A/E) lesions on tissue culture cells. PCR assays can also be used to test the isolates for LEE genes, but since Enterohemorrhagic *E. coli* (EHEC) also carry LEE, the isolates also have to be tested for Shiga toxins (Stx). EPEC are distinguished from EHEC by the presence of LEE and absence of Stx.

6. Target Populations

EPEC infections most often occur in infants, especially those who are being bottle fed. Poor-quality water used to rehydrate infant formulae in underdeveloped countries may be the source of EPEC in bottle-fed infants.

7. Food Analysis

Presence of EPEC in foods can be determined by plating culture enrichment of food samples onto media that are selective and differential for *E. coli* and testing the isolates for EPEC traits by tissue culture or PCR. Shiga toxins (Stx) assays are also essential to distinguish EHEC from EPEC. EPEC are characterized by the presence of LEE and absence of Stx.

8. Examples of Outbreaks

Check the CDC's [Morbidity and Mortality Weekly Reports](#) for articles about outbreaks.

9. Resources

- Loci index for [genome](#)
- GenBank [Taxonomy Database](#)

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Foodborne Pathogenic Microorganisms and Natural Toxins

Enterohemorrhagic *Escherichia coli* (EHEC)

1. Organism

Like generic *E. coli*, toxin-producing Shiga-toxigenic *Escherichia coli* (STEC) are Gram-negative, rod-shaped bacteria, but are characterized by the production of Shiga toxins (Stx). Depending on the reference cited, there are 200 to 400 STEC serotypes, many of which have not been implicated in human illness; however, a subset of STEC called enterohemorrhagic *Escherichia coli* (EHEC), the topic of this chapter, includes only those that cause serious illness. Serotype O157:H7 is the prototypic EHEC strain.

Although O157:H7 is currently the predominant strain and accounts for ~75% of the EHEC infections worldwide, other non-O157 EHEC serotypes are emerging as a cause of foodborne illnesses. In the United States a group often referred to as the “big 6” (O111, O26, O121, O103, O145, and O45) accounts for the majority of the non-O157:H7

serotypes isolated from clinical infections and, therefore, is currently a focus of concern. However, other EHEC serotypes, such as O113, O91, and others, also can cause severe illness. As a result, the non-O157 EHEC serotypes of public health concern can change quickly, depending on outbreak incidents, and can vary with countries and geographic regions.

A recent example is the large outbreak, in 2011, that was centered in Germany, but also affected various other countries in the European Union. The pathogen was identified as an *E. coli* strain of serotype O104:H4 that produced Shiga toxin and, therefore, was thought to be an EHEC. However, genetic analysis showed that this pathogen had 93% genetic homology with a strain of Enteroaggregative *E. coli* (EAEC), which is known for causing persistent diarrhea in underdeveloped countries, but has seldom been implicated in major foodborne incidents. Hence, the O104:H4 strain that caused the outbreak appears to be an EAEC strain that acquired the ability to produce Shiga toxin.

For Consumers: A Snapshot

Most *E. coli* bacteria are harmless, but some produce a toxin (Shiga toxin) that can cause serious illness, including bloody diarrhea, blood-clotting problems, kidney failure, and death. Not all of the Shiga-producing *E. coli* can cause these problems, but the subset called enterohemorrhagic *E. coli* (EHEC) can. You might have heard news reports about these EHEC bacteria, such as *E. coli* O157:H7, when they’ve caused outbreaks of foodborne illness. EHEC outbreaks have been traced to many kinds of foods; for example, ground meats, **unpasteurized** (“raw”) milk, unpasteurized fruit juice, lettuce, spinach, sprouts, and, more recently, commercially manufactured frozen cookie dough. Some people get the less serious form of the infection, which can range from no symptoms to diarrhea that starts out watery, then turns bloody. But the infection sometimes progresses into the life-threatening form of the illness that causes kidney failure and other problems, with children and people with weak immune systems being at especially high risk. Cooking ground beef well; washing raw fruits and vegetables under clean, running water; and **not** drinking **unpasteurized** (“raw”) milk or eating certain cheeses made from it are some of the things you can do to help [protect yourself](#).

Currently, it is difficult to determine which serotypes of *E. coli* are EHEC and equally challenging to predict the emergence of strains that can acquire the genes for Shiga toxin production or other virulence factors and so cause human illness. EHEC are characterized by:

- production of Stx, including Stx1 and/or Stx2. Stx1 is nearly identical to the toxin produced by *Shigella dysenteriae* Type I. There are many subtypes of both toxins, and some subtypes of Stx2 appear to be implicated in human illness. Stx2 is most often associated with severe sequelae, such as hemolytic uremic syndrome (HUS), which is characterized by acute renal failure.
- presence of LEE (“locus for enterocyte effacement”; pathogenicity island that encodes for intimin, a protein that enables bacterial attachment to epithelial cells).

There are also several other putative virulence factors, including enterohemolysin, but the role of these factors in pathogenesis remains undetermined.

2. Disease

- **Mortality:** Patients whose illness progresses to HUS have a mortality rate of 3% to 5%.
- **Infective dose:** The infective dose of EHEC O157:H7 is estimated to be very low, in the range of 10 to 100 cells. The infective dose of other EHEC serotypes is suspected to be slightly higher.
- **Onset:** Symptoms usually begin 3 to 4 days after exposure, but the time may range from 1 to 9 days.
- **Disease / complications:** Infections from EHEC may range from asymptomatic-to-mild diarrhea to severe complications. The acute symptoms are called hemorrhagic colitis (HC), characterized by severe abdominal cramps and bloody diarrhea, which may progress to such life-threatening complications as HUS or thrombotic thrombocytopenia purpura (TTP) – conditions that are most often associated with O157:H7, but that also can occur with other EHEC serotypes. About 3% to 7% of HC cases progress to HUS or TTP.

Some evidence suggests that Stx2 and intimin are associated with progression to severe disease, such as HUS. Kidney cells have a high concentration of Stx receptors; hence, the kidney is a common site of damage. Some survivors may have permanent disabilities, such as renal insufficiency and neurological deficits.

Antibiotic therapy for EHEC infection has had mixed results and, in some instances, seems to increase the patient’s risk of HUS. One speculation is that antibiotics lyse EHEC cells, releasing more Stx into the host.

- **Symptoms:** Hemorrhagic colitis is characterized by severe cramping (abdominal pain), nausea or vomiting, and diarrhea that initially is watery, but becomes grossly bloody. In some cases, the diarrhea may be extreme, appearing to consist entirely of blood and occurring every 15 to 30 minutes. Fever typically is low-grade or absent.
- **Duration:** In uncomplicated cases, duration of symptoms is 2 to 9 days, with an average of 8 days.
- **Route of entry:** Oral (e.g., ingestion of contaminated food, water, or fecal particles).

- **Pathway:** After ingestion, EHEC attaches to intestinal epithelial cells via LEE-encoded factors and produces Stx that are internalized, activated, and can pass into the bloodstream to become systemic.

3. Frequency

There are about 63,000 cases of EHEC infections in the U.S. yearly, according to a [report](#) by the Centers for Disease Control and Prevention (CDC). Ground beef and beef products continue to be implicated in most infections; however, contaminated produce increasingly has been implicated as a vehicle. As for STEC non-O157, the CDC estimates that 112,752 cases, per year, are attributed to foodborne illness in the U.S.

EHEC O157:H7 was first identified in an outbreak, in 1982, in which hamburgers from a fast-food restaurant were the vehicle. In 1991, hamburgers from fast-food restaurants were implicated in another outbreak, which affected about 700 people in four states. In the mid 1990s, a large outbreak was traced to unpasteurized juices. The largest O157:H7 outbreak on record took place in Japan; radish sprouts were implicated and about 10,000 people were affected. Since then, O157:H7 has been implicated in numerous outbreaks that involved lettuce, salads, various types of sprouts, and, in 2006, bagged spinach. In 2009, an O157:H7 outbreak in the U.S. was traced to frozen, raw cookie dough.

About a dozen non-O157:H7 EHEC outbreaks have been recorded in the U.S., but incidences may be underestimated due to lack of routine testing and appropriate testing methods.

4. Sources

Raw or undercooked ground beef and beef products are the vehicles most often implicated in O157:H7 outbreaks. Earlier outbreaks also implicated consumption of raw milk. O157:H7 can develop acid tolerance, as evidenced by infections in which acid foods (<pH4.6) were implicated, such as yogurt, [mayonnaise](#), fermented sausages, cheeses, and unpasteurized fruit juices.

Various water sources, including potable, well, and recreational water, also have caused EHEC infections, as has contact with animals at farms or petting zoos.

Produce, including bagged lettuce, spinach, and alfalfa sprouts, increasingly is being implicated in O157:H7 infections.

Interestingly, infections in the U.S. by *non*-O157:H7 EHEC has been caused by many of these same vehicles, but, as of this writing, beef products have seldom been implicated.

Person-to-person transmission of infection is well documented.

Additional information is available from “*Escherichia coli* Serotype O157:H7: Novel Vehicles of Infection and Emergence of Phenotypic Variants,” by Dr. Peter Feng, FDA. [Emerging Infectious Diseases \(1995\) 1\(2\)](#)

5. Diagnosis

Unlike generic *E. coli*, EHEC O157:H7 do not ferment the sugar sorbitol, so an effective method is to plate patient's bloody diarrhea samples onto sorbitol MacConkey medium to screen for sorbitol non-fermenting isolates. These are then typed serologically using antibodies to the O157 and the H7 antigens. However, as other EHEC serotypes are increasingly causing illness, clinical samples are now simultaneously tested for the presence of Stx using commercially-available antibody kits. Any STEC strains found are then serotyped and identified. There are also many PCR assays specific for Stx genes that may be used for screening clinical samples.

6. Target Populations

All people are believed to be susceptible to hemorrhagic colitis, but young children and the elderly are more susceptible and at higher risk for the illness to progress to more severe complications. Others with weak immune systems also are at risk, such as people with some chronic diseases or AIDS, and people on immunosuppressive medications; for example, some drugs used for arthritis and cancer chemotherapy.

7. Food Analysis

Presence of EHEC O157:H7 in foods can be determined by plating culture enrichment of food samples onto selective and differential media. Unlike typical *E. coli*, O157:H7 do not ferment sorbitol and are negative with the MUG assay, so these tests are commonly used to distinguish O157:H7 strains from other *E. coli* prior to serological testing for the O157 and H7 antigens and also for the presence of Stx genes by PCR. Molecular assays also exist that can specifically detect O157:H7 strains using unique mutational markers.

Detection of non-O157:H7 EHEC, however, is more complex, due to the lack of unique traits. For non-O157 EHEC, food enrichment is first screened for Shiga toxin using an antibody assay or for Stx genes by PCR. Enrichment cultures that are positive for Stx are plated on agar media, and multiple isolates are then tested for Stx genes, in order to obtain a pure culture isolate. These putative STEC isolates are then retested for virulence genes and their serotype determined. This process is both time-consuming and labor-intensive and may require screening hundreds of isolates.

There are numerous commercially-available kits to test for Stx, O157, and a few other EHEC serotypes. However, there are several Stx subtypes and many EHEC serotypes, and not all of these can be detected by commercial test kits. The [Escherichia coli](#) link to the FDA Bacteriological Analytical Manual, Chapter 4, provides a description of methods to test for common *E. coli*. Methods for EHEC and O157:H7 are described in Chapter 4a.

8. Examples of Outbreaks

For more information about recent outbreaks see the Centers for Disease Control and Prevention (CDC) [Morbidity and Mortality Weekly Reports](#).

9. Other Resources

More information is available from the following sources.

- [USDA \(August 11 1998\)](#) – USDA Urges Consumers to Use Food Thermometer When Cooking Ground Beef Patties
- [CDC – General information about *Escherichia coli* O157:H7](#)
- [Produce Handling and Processing Practices](#), from *Emerging Infectious Diseases*, CDC
- [Risk assessment](#) of *E. coli* O157:H7 in ground beef, from the USDA Food Safety and Inspection Service

Bad Bug Book

Foodborne Pathogenic Microorganisms and Natural Toxins

Enteroinvasive *Escherichia coli* (EIEC)

1. Organism

EIEC is a Gram-negative, rod-shaped, enterotoxin-producing bacterium that closely resembles *Shigella*. Both are characterized by their ability to invade colonic epithelial cells. The genetic information required for the invasion phenotype is encoded within a 37 kilobase region on a virulence plasmid, which can vary in size from 180 kb in *S. sonnei* to 220 kb in *S. flexneri* and EIEC. There is a high degree of homology among these plasmids, and they are functionally interchangeable.

2. Disease

The illness caused by EIEC is a mild form of bacillary dysentery, similar to that caused by *Shigella* spp.

- **Mortality:** A [recent estimate](#) of domestically acquired foodborne illness in the United States, by the Centers for Disease Control and Prevention (CDC), lists a death rate of zero for diarrheagenic *E. coli* other than Shiga-toxicogenic and enterotoxigenic *E. coli*.
- **Infective dose:** The infective dose of EIEC is thought to be in the range of 200 to 5,000 cells, somewhat higher than that of *Shigella*. The difference in the dose may depend on which virulence plasmid these pathogens harbor.
- **Onset:** The symptoms usually occur within 12 to 72 hrs after ingestion of contaminated food.
- **Illness / complications:** The illness generally is self-limiting, with no known complications.
- **Symptoms:** Mild dysentery; abdominal cramps, diarrhea, vomiting, fever, chills, and generalized malaise. Stools often contain blood and mucus.

For Consumers: A Snapshot

Many *E. coli* are harmless, but some can cause serious illness from contaminated food. We cover four kinds of *E. coli* that cause foodborne illness in this book. The one described in this chapter, called “EIEC” for short, isn’t the one that causes many of the outbreaks that make headlines in newspapers. (That kind, sometimes called “EHEC,” includes *E. coli* O157:H7, can cause problems like kidney failure, and is especially serious.) The disease caused by EIEC usually begins as watery diarrhea, then progresses to mild dysentery – diarrhea that often contains blood and mucus. Other symptoms may include cramps, vomiting, fever, chills, and a general sense of not feeling well. In people who are otherwise healthy, the illness usually goes away by itself, without medical treatment. In a recent estimate of cases of illness from contaminated food eaten in the U.S., the Centers for Disease Control and Prevention listed zero deaths from EIEC. But if a case turns severe, a health professional should be consulted, so that fluid and important minerals lost due to diarrhea can be replaced, if need be. It’s also important to know that EIEC can be passed not only by foods, but also by other people – for example, if infected people don’t wash their hands well after having a bowel movement, then objects or other people they touch may become infected. To help protect yourself and others, follow [good handwashing practices](#) and other basic [food-safety tips](#).

- **Duration:** Usually resolves in 5 to 7 days.
- **Route of entry:** Oral. Person-to-person transmission can also occur.
- **Pathway:** The pathogenesis of EIEC is similar to that of *Shigella* species. The process begins with cellular invasion via endocytic vacuoles. Once internalized, the vacuoles are lysed, the bacteria multiply intracellularly, and spread laterally to other cells. EIEC also produce an enterotoxin, which may be involved in causing the watery diarrhea that precedes the dysentery symptoms associated with EIEC.

3. Frequency

EIEC outbreaks are not frequent in the U.S. However, it may be misidentified or confused with shigellosis; therefore, its actual prevalence may be underestimated.

4. Sources

No specific foods are frequently associated with EIEC infections. Infected humans are the only known reservoirs of EIEC; hence, any food contaminated with human feces from an ill individual, either directly or via contaminated water, can be infectious. Imported Camembert cheese was implicated in an epidemic of gastroenteritis caused by EIEC that affected 226 people in 96 outbreaks, in 1971. A tofu product contaminated with EIEC affected 670 people in Japan, in 1988. In 1994, a restaurant-associated EIEC outbreak in Texas, which affected 370 people, was traced to contaminated guacamole.

5. Diagnosis

Diagnosis consists of culturing for *E. coli* from stools of infected individuals and testing the isolates for invasiveness using tissue cultures or animal models. EIEC isolates may also be identified using PCR assays to test for the presence of *inv* genes. These assays, however, will detect both EIEC and *Shigella* spp., so additional assays are needed for differentiation.

6. Target Populations

All populations are susceptible to EIEC infections.

7. Food Analysis

Presence of EIEC in foods can be determined by plating culture enrichment of food samples onto media that are selective and differential for *E. coli* and testing the isolates for the presence of *inv* genes. EIEC in foods can also be detected using *inv* gene-specific PCR assays, testing either directly or on food-sample enrichments.

8. Examples of Outbreaks

See Frequency section, above, and CDC's [Morbidity and Mortality Weekly Reports](#).

9. Resources

- Loci index for [genome](#)
- GenBank [Taxonomy Database](#)