

E. coli O157:H7

E. coli O157:H7 is a Class B Disease and must be reported to the state within one business day.

Epidemiology

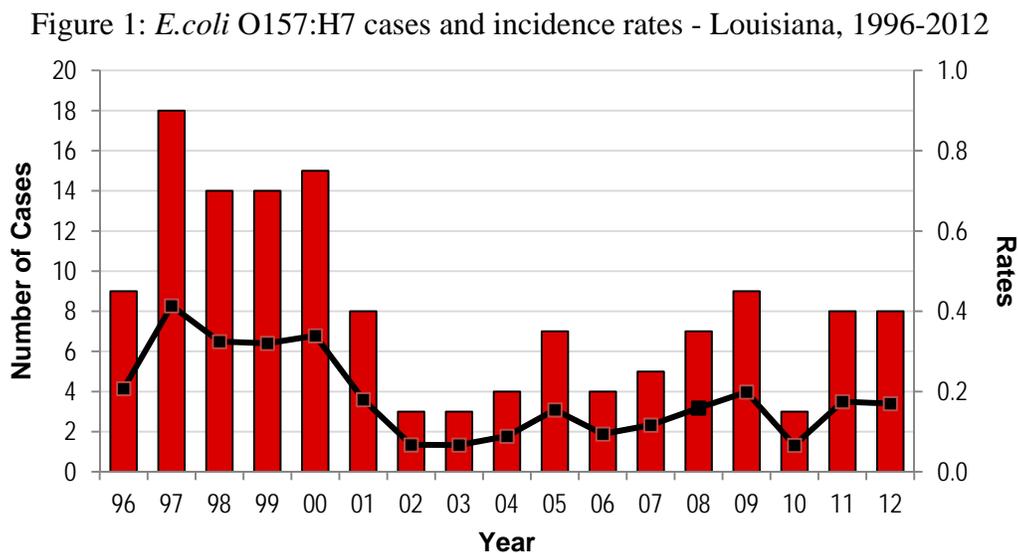
Enterohemorrhagic *Escherichia coli* (EHEC) or *E. coli* O57:H7 are bacteria that produce toxins (shiga-toxins) that cause illness. *E. coli* O157:H7 has a bovine reservoir and can be transmitted by undercooked ground beef and unpasteurized milk. These bacteria can also be spread from person-to-person by fecal-oral transmission, with person-to-person transmission most commonly being seen in families, child care centers and custodial institutions. Outbreaks from contaminated food and water have also occurred.

E. coli O157:H7 exists, at least intermittently, in both dairy and beef herds in the majority of cattle farms across the United States. Typically, O157 is detectable in the feces of fewer than 5% of cattle in the U.S. at any point in time. Despite this low detection rate, 60% of retail ground beef contains Shiga-Toxin producing *E. coli* (STEC). This is due in part to the way hamburger meat is produced. Hamburger meat is processed in bulk from a large number of animals. Up to a hundred cows may contribute to a single pack of hamburgers.

Symptoms of *E. coli* infection include diarrhea that ranges from mild and non-bloody to stools that are virtually all blood but contain no fecal leukocytes. Fever is not usually present.

Incidence

E. coli O157:H7 became reportable in Louisiana in 1996. The number of cases ranged from eight to almost 20 per year in the late nineties then decreased in recent years to less than ten (Figure 1).



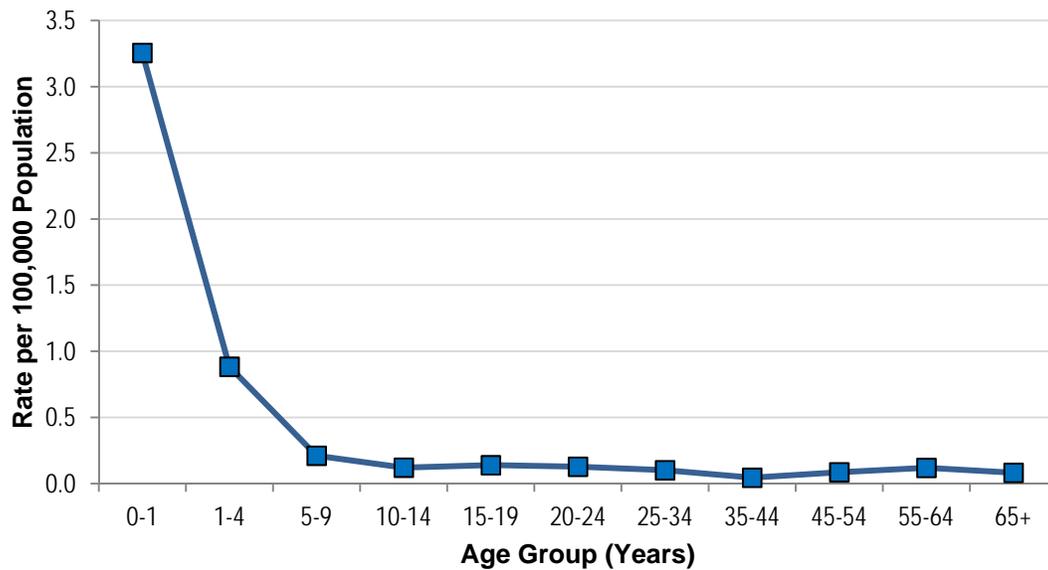
The most recent data (2010) from FoodNet* states shows a national incidence rate of STEC (O157) infections to be 0.9 per 100,000 population. The incidence rate of *E. coli* O157:H7 infections for Louisiana in 2010 was 0.07 per 100,000 population.

* **FoodNet** is a collaborative project of the CDC, ten EIP sites, the U.S. Department of Agriculture (USDA), and the **Food and Drug Administration (FDA)**.

Age Group Distribution

The age group distribution shows a very high rate among infants and young children (Figure 2). There is no difference in incidence rates between males and females.

Figure 2: *E. coli* O157:H7 average annual incidence rates by age - Louisiana, 1996-2012



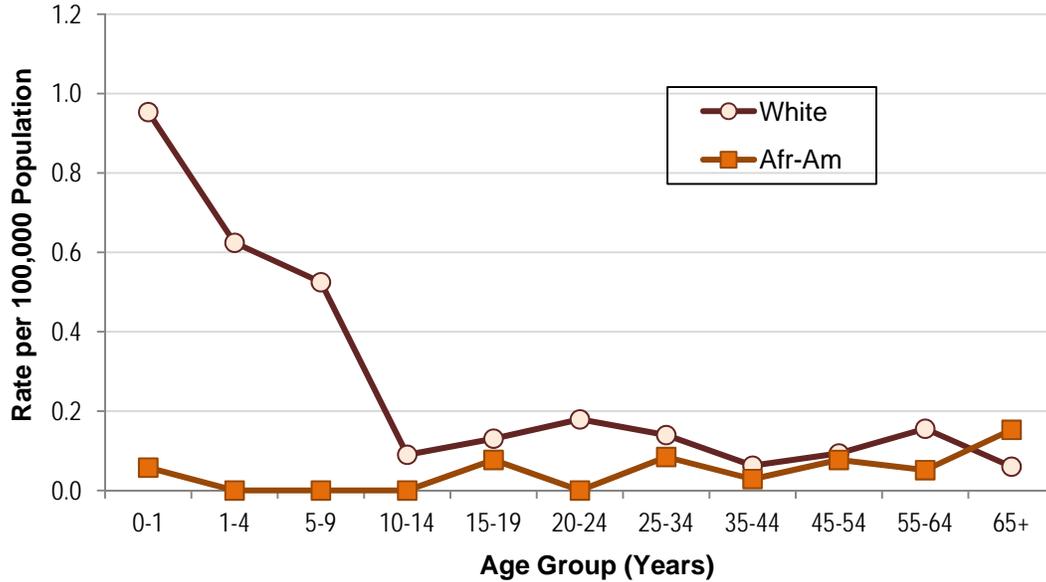
The highest rates are observed among infants who are not exposed to undercooked meat. These cases result from fecal-oral cross contamination when infants are fed. Detection is higher among infants than among older children and adults because infants with diarrhea are more likely to be brought to medical care and have stool cultured.

Those 65 years old and older, while not defined as a high risk group for *E. coli* infections, are at a higher risk for complications as a result of an *E. coli* infection.

Race Distribution

The race distribution shows a large discrepancy by race, with White infants and children having higher rates compared to African-Americans. These rates of *E. coli* O157:H7 are more reflective of diagnosis of diarrhea and access to medical care rather than true incidence (Figure 3).

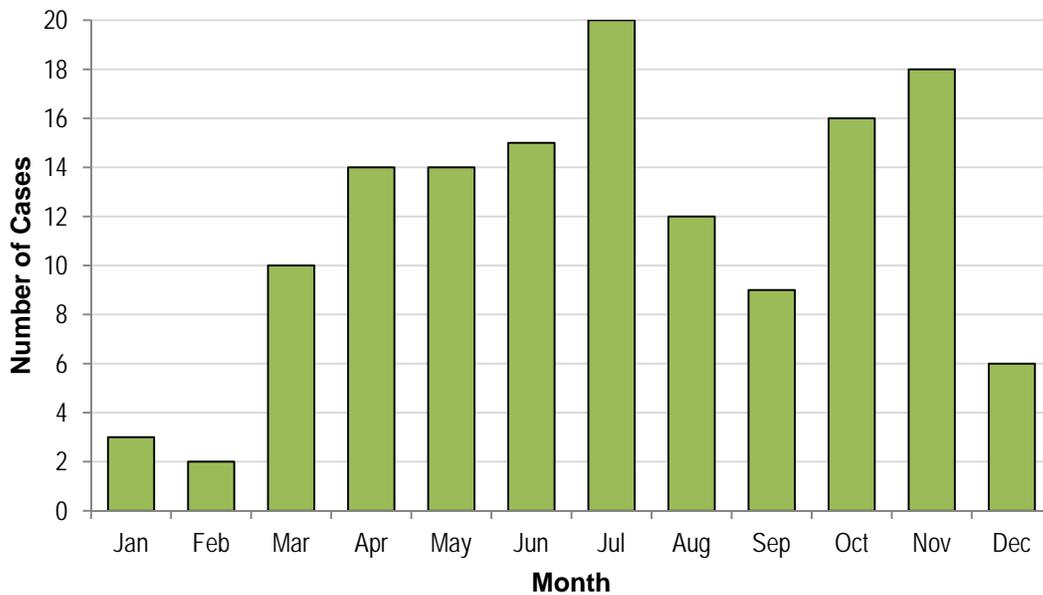
Figure 3: *E. coli* O157:H7 average incidence rates by race and age - Louisiana, 1996-2012



Seasonal Distribution

Typically the seasonal distribution of *E. coli* infections shows a higher number of cases during the summer months. Data from 1996 to 2011 shows an increase in cases from April to November in Louisiana (Figure 4).

Figure 4: *E. coli* O157:H7 total cases by month - Louisiana, 1996-2012



Geographical Distribution

The geographic distribution of *E. coli* O157:H7 cases is a reflection of reporting patterns from medical providers (Table 1).

Table 1: Incidence rate of *E. coli* O157:H7 infections per 100,000 population by parish Louisiana, 1996-2012

Region	Parish	Inc. Rate 1996-2012	Region	Parish	Inc. Rate 1996-2011
1	Orleans	0.10	6	Rapides	0.63
	Jefferson	0.18		Avoyelles	0.42
	Plaquemines	0.24		Vernon	0.11
	St. Bernard	0.36		Grant	0
2	E. Baton Rouge	0.13		Winn	0.34
	W. Baton Rouge	0.55		La Salle	0
	E. Feliciana	0.28		Catahoula	0
	W. Feliciana	0		Concordia	0
	Ascension	0.21	7	Caddo	0.09
Iberville	0	De Soto		0	
Pointe Coupee	0	Sabine		0.24	
3	Lafourche	0.15		Bossier	0.06
	Terrebonne	0.05		Webster	0
	St. Mary	0.11		Claiborne	0
	St. John	0		Bienville	0
	St. Charles	0.63		Red River	0
	St. James	0.59		Natchitoches	0.15
	Assumption	0.76	8	Ouachita	0.43
4	Lafayette	0.10		Union	0.52
	St. Martin	0	Lincoln	0	
	Iberia	0	Jackson	0.37	
	Acadia	0	Morehouse	0.19	
	Vermillion	0.11	Caldwell	0	
	Evangeline	0	Richland	1.12	
	St. Landry	0.14	W. Carroll	0	
	5	Calcasieu	0.25		E. Carroll
Cameron		0		Madison	0
Beauregard		0		Franklin	0
Jefferson Davis		0		Tensas	0
Allen		0	9	St. Tammany	0.46
		Tangipahoa		0.22	
		Washington		0.41	
		St. Helena		0	
		Livingston		0.17	

Other *E. coli*

Most strains of *E. coli* are normal, harmless inhabitants of the intestinal tract.

There are a few Enterohemorrhagic *E. coli* (EHEC) strains beyond O157:H7, e.g., *E. coli* O26:H11. All of these strains produce cytotoxins resembling those found in *Shigella dysenteriae*, type 1. These toxins are referred to as shigalike toxins or verotoxins.

Enteroinvasive *E. coli* (EIEC) strains include these specific serotypes of *E. coli*: O28, O112, O115, O124, O136, O143, O144, O147, O152, O164 and O167. The EIEC strains resemble *Shigella* biochemically, and can invade intestinal epithelial cells.

Enteropathogenic *E. coli* (EPEC) strains traditionally have been defined as members of specific *E. coli* serotypes that have been epidemiologically incriminated as causes of infantile diarrhea. They include the following somatic serogroups: O44, O55, O86, O111, O114, O119, O125, O126, O127, O128, O142 and O158.

More recently, EPEC has been defined according to specific virulence properties. EPEC strains adhere to intestinal mucosa and produce a characteristic lesion in the gastrointestinal tract, termed an attaching and effacing lesion. EPEC do not produce enterotoxins and are not invasive.

Enterotoxigenic *E. coli* (ETEC) strains colonize the small intestine without invading it and produce either, or both, heat-labile and/or heat-stable enterotoxins. Examples of these strains include O6:H16 and O8:H9.

There is no tracking system for EPEC and ETEC; tracking of these strains is done only in research projects.

In 2001, national surveillance began for shiga-toxin producing *E. coli* under the name of EHEC. The case definition changed from EHEC to STEC (shiga-toxin producing *E. coli*) in 2006 and serotype specific reporting was implemented. From 2004 to 2011, the majority of the STEC cases in Louisiana were identified as shiga-toxin producing *E. coli*, non-O157:H7 (36.1%). The remaining cases were *E. coli* O157:H7 (33.6%), and STEC, not grouped (30.3%) (Figure 5).

Figure 5: Reported STEC cases by serotype - Louisiana, 2004-2012

