

Salmonellosis in Connecticut, 1998-2004

Salmonella species cause an estimated 1.4 million infections, 15,000 hospitalizations, and 400 deaths annually in the United States (1). In Connecticut, salmonellosis is health care provider and laboratory reportable.

The Foodborne Diseases Active Surveillance Network (FoodNet), part of the Centers for Disease Control and Prevention's (CDC) Emerging Infections Program, collects data from 10 CDC-funded U.S. sites on diseases caused by enteric pathogens transmitted commonly through food (2). In Connecticut, FoodNet is a collaborative effort between the Connecticut Department of Public Health (DPH) and the Department of Epidemiology and Public Health at Yale University School of Medicine.

Salmonella spp., FoodNet, 1998-2004

During 1998-2004, 3,214 cases of salmonellosis were reported to the DPH. Of these, five serotypes accounted for 60% of infections: *S. Typhimurium* 770 (24%), *S. Enteritidis* 707 (22%), *S. Newport* 231 (7%), *S. Heidelberg* 150 (5%), and *S. Thompson* 65 (2%). The annual rate ranged from 11.6-16.4 cases per 100,000 population.

Salmonellosis cases declined by 10% over the 7-year period (Figure 1). The decline varied by serotype (Figure 2).

The average annual incidence by age group showed the highest rates among infants < 1 year of age (Figure 3). Cases were more commonly reported during July – September (44%).

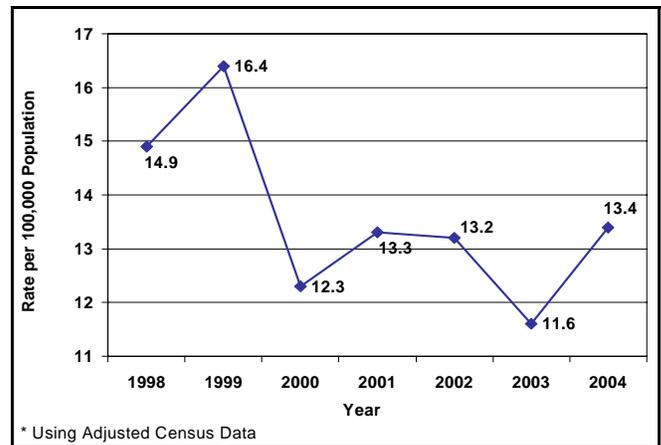
National Antimicrobial Resistance Monitoring System

The National Antimicrobial Resistance Monitoring System (NARMS) for Enteric Bacteria Laboratory at the CDC conducts antimicrobial susceptibility testing on a variety of foodborne pathogens received from state public health laboratories, including *Salmonella* spp. The DPH Laboratory routinely sends every 20th non-Typhi *Salmonella* and every *Salmonella typhi* isolate to NARMS.

In this issue...

Salmonellosis in Connecticut, 1998-2004	9
Shiga toxin-producing <i>Escherichia coli</i>	11

Figure 1: Salmonellosis Case Rate*, Connecticut, 1998-2004



Fluoroquinolones (e.g., ciprofloxacin) and third generation cephalosporins (e.g., ceftriaxone) are commonly used to treat severe infections caused by *Salmonella*. National data demonstrate slowly increasing antimicrobial resistance in non-typhoidal *Salmonella*. For ciprofloxacin,

Figure 2: Five Most Frequent *Salmonella* Serotypes Isolated by Year, Connecticut, 1998-2004

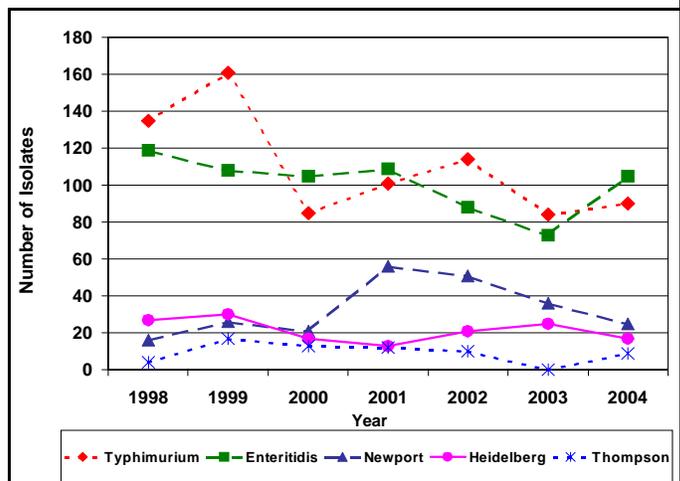
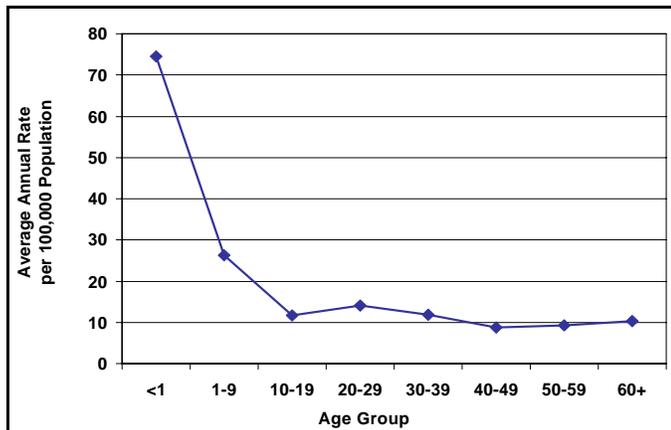


Figure 3: Average Annual Rate of Salmonellosis by Age Group, Connecticut, 1998-2004



decreased susceptibility increased from 0.4% in 1996 to 1% in 2002; resistance increased from 0% to 0.5%. For ceftriaxone, decreased susceptibility increased from 3% in 1996 to 4% in 2002; resistance increased from 0% to 0.2% (3).

Foodborne Outbreaks Caused by *Salmonella* spp, Connecticut, 1998 - 2004

Foodborne disease outbreaks are reportable to the DPH and the local health department. A foodborne disease outbreak is defined as “the occurrence of two or more cases of a similar illness resulting from the ingestion of a common food”.

During 1998-2004, the DPH Epidemiology and Emerging Infections Program identified 28 foodborne outbreaks due to *Salmonella* spp.. Of these, 10 (36%) involved residents of more than one state and were identified through molecular subtyping. Serotypes most commonly associated with outbreaks were *S. Enteritidis* (7), *S. Newport* (6), and *S. Typhimurium* (5).

The 28 outbreaks caused at least 213 illnesses, of which 192 were laboratory confirmed; 39 persons were hospitalized with no deaths reported. The median number of cases of illness identified per outbreak was 6 (range 3-23 persons).

Of the 28 outbreaks, 15 (54%) were linked to a specific food vehicle: produce items (6), cheese products (3), eggs (2), beef (2), a chicken product (1), and pork (1). The 6 produce items included tomatoes (3 outbreaks), mangoes (2 outbreaks), and cantaloupe.

Reported by: R. Marcus, MPH, S. Hurd, MPH, Yale University, Emerging Infections Program; Q. Phan, MPH, P. Mshar, MPH, Epidemiology and Emerging Infections Program, Connecticut Department of Public Health.

Editorial Note:

From 1998-2004, the rate of *Salmonella* infection showed a modest downward trend in Connecticut. Using 1996-1998 as a baseline compared to 2004, the incidence of salmonellosis declined 8% (95% CI = 1%-15%) in FoodNet sites (2) compared to 10% in Connecticut. Nationally, the largest and most significant decrease was also seen in *S. Typhimurium* (41% [CI=34%-48%]).

With the prevalence of antimicrobial resistant organisms on the rise, the risk for treatment failure in human infections is also increasing. Human health consequences of illness attributed to multidrug-resistant non-Typhi *Salmonella* will be examined in a multi-site study conducted by FoodNet later this year.

Connecticut is participating in PulseNet, a national network of public health laboratories that perform molecular subtyping of enteric pathogens using pulsed-field gel electrophoresis (PFGE). The network permits rapid electronic comparison of PFGE patterns. Closely related PFGE patterns suggest a common source. This capability has contributed significantly to detection and investigation of foodborne outbreaks in Connecticut and nationally.

Multiple case-control studies of *Salmonella* infection have identified eating meat, poultry, and eggs as risk factors for infection (4-5). The U.S. Department of Agriculture has implemented measures through its Hazard Analysis and Critical Control Points (HACCP) Program to reduce pathogens on these commodities. Recently, the Food and Drug Administration developed a plan to decrease illness associated with produce (6). Additional measures are needed across the farm-to-table continuum, including pathogen reduction during processing and education of consumers about risks and prevention measures. To reach the Healthy People 2010 Objective for *Salmonella* infection of 6.8 per 100,000 persons, additional efforts are needed (2).

References:

1. Voetsch AC, Van Gilder TJ, Angulo FJ, Farley MM, Shallow S, Marcus R, et al. FoodNet estimate of the burden of illness caused by nontyphoidal *Salmonella* infections in the United States. *CID* 2004;38(Suppl 3): S127-34.

2. CDC. Preliminary FoodNet data on the incidence of infection with pathogens transmitted commonly through food-10 sites, United States, 2004. MMWR 2005;54:352-6.
3. CDC. National Antimicrobial Resistance Monitoring System for Enteric Bacteria (NARMS): 2002 Human Isolates Final Report. Atlanta, Georgia: U.S. Department of Health and Human Services, CDC, 2004.
4. Kimura AC, Reddy V, Marcus R, Cieslak PR, Mohle-Boetani JC, Kassenborg HD, et al. Chicken Consumption is a newly identified risk factor for sporadic *Salmonella enterica* serotype Enteritidis infections in the United States: a case-control study in FoodNet sites. CID 2004;38(Suppl 3):S244-52.
5. Hennessy TW, Cheng LH, Kassenborg H, Ahuja SD, Mohle-Boetani J, Marcus R, et al. Egg consumption is the principal risk factors for sporadic *Salmonella* serotype Heidelberg infections: a case-control study in FoodNet sites. CID 2004;38(Suppl 3):S237-43.
6. Food and Drug Administration. Produce safety from production to consumption: 2004 action plan to minimize foodborne illness associated with fresh produce consumption. Rockville, MD: US Department of Health and Human Services, Food and Drug Administration; 2004. Available at <http://www.cfsan.fda.gov/~dms/prodpla2.html>.

Shiga toxin-producing *Escherichia coli*

Shiga toxin-producing *Escherichia coli* (STEC) are emerging organisms capable of causing sporadic and epidemic disease. Manifestations of STEC infection can include asymptomatic infection, diarrhea, hemorrhagic colitis, and potentially life threatening hemolytic uremic syndrome (HUS). Production of Shiga toxins (Stx) is the defining characteristic of all STEC. *E. coli* O157:H7 is the most frequently identified STEC serotype in the United States, causing an estimated 60 deaths and 73,000 illnesses annually (1). In addition, over 100 different non-O157 STEC serotypes have been associated with human illness. The epidemiology of *E. coli* O157 has been well established while much less is known about non-O157 STEC.

Due to its inability to rapidly ferment sorbitol, *E. coli* O157 can be easily detected in stools if cultured using selective media, such as sorbitol-MacConkey agar (SMAC). However, routine laboratory testing for *E. coli* O157 does not detect non-O157 STEC. Since most non-O157 STEC serotypes ferment sorbitol, it is likely that they would remain undetected using routine culture methods. In contrast to culture, testing for the presence of Stx in stools using an EIA, non-culture based method detects both O157 and non-O157 STEC.

Methods for the rapid detection of Stx in stool samples have become commercially available and are increasingly used by clinical laboratories. In

2000, the Connecticut Department of Public Health (DPH) added Shiga toxin-related disease to the list of laboratory reportable findings. Clinical laboratories are also required to submit Stx-positive broths to the DPH Laboratory for confirmation and identification of the causative organism. Non-O157 STEC isolates are forwarded to the Centers for Disease Control and Prevention for confirmation and serotyping.

During 2000-2004, 340 STEC infections were reported to the DPH. Overall, 235 (69%) were O157, and 105 (31%) were non-O157 STEC; 169 (50%) of these infections were detected by Stx testing. Among the 169 infections, 65 (38%) were determined to be *E. coli* O157, and 104 (62%) were non-O157 STEC. A specific serotype was determined for 96 non-O157 STEC isolates. The four most common serogroups were O103 (24%), O111 (21%), O45 (17%), and O26 (15%).

An epidemiologic study was conducted during February 1, 2000-January 31, 2003 to determine the spectrum of clinical illness and risk factors associated with STEC infections. Overall, 164 O157 and 54 non-O157 STEC infections were identified. Compared to patients with O157 infection, patients with non-O157 were less likely to have bloody diarrhea (54% vs 88%, $p < 0.001$), be hospitalized (12% vs 49%, $p < 0.001$), and develop HUS (0% vs 10%, $p = 0.006$). Non-O157 STEC patients were more likely to be 20-39 years of age (26% vs 9%, $p < 0.001$).

Reported by: Q. Phan, MPH, Epidemiology and Emerging Infections Program, Connecticut Department of Public Health; S. Hurd, MPH, R. Marcus, MPH, Yale University, Emerging Infections Program; C. Welles, R. Howard, MS, State Public Health Laboratory, DPH.

Editorial

Shiga toxin is a family of toxins produced by a number of organisms, including Shiga toxin-producing *Escherichia coli* and *Shigella dysenteriae* type I. Since 2000, only one (<1%) isolate of *S. dysenteriae* was recovered from Stx-positive broths submitted to the DPH Laboratory. Thus, while the presence of Stx in stool may indicate infection with STEC or *Shigella*, a report of shiga toxin-related disease is more likely to mean infection caused by STEC.

Few studies have characterized the prevalence and epidemiology of non-O157 STEC infection.

In This Issue...

Salmonellosis CT, 1998-2004; Shiga toxin-producing *E. coli*

The use of culture-based methods to test for *E. coli* O157 does not detect non-O157 STEC. In the U.S., non-O157 STEC has been associated with HUS and at least three outbreaks: O104:H21 in 1994 in Montana, O111:H8 in 1999 in Texas, and O121 in 1999 in Connecticut (2-4).

In Connecticut, at least seven clinical laboratories have adopted Stx testing since 1999. Data from surveillance and epidemiologic study demonstrate that non-O157 STEC may be more prevalent than O157. Overall, non-O157 patients appear to have less severe complications, such as hospitalization and HUS, than O157 patients.

The use of Stx assays has several advantages over SMAC. First, Stx testing allows for more rapid diagnosis and thus prevents further exploratory, invasive procedures and unnecessary empirical treatment with antibiotics. Second, Stx testing enables the detection of non-O157 STEC. Third, evaluation studies have demonstrated that Stx assays are more sensitive and equally specific for detecting O157 compared to SMAC (5,6). For these reasons, clinical laboratories currently only culturing for O157 should consider using Stx

testing either solely or as an adjunct to culturing. Clinicians evaluating patients with diarrhea should consider infection with non-O157 STEC.

References

1. Mead PS, Slutsker L, Dietz V, et al. Food-related illness and death in the United States. *Emerg Infect Dis* 1999;5:607-25.
2. CDC. Outbreak of acute gastroenteritis attributable to *Escherichia coli* serotype O104:H21-Helena, Montana, 1994. *MMWR* 1995;44:501-503.
3. Brooks JT, Bergmire-Sweat D, Kennedy M, et al. Outbreak of Shiga toxin-producing *Escherichia coli* O111:H8 infections among attendees of a high school cheerleading camp. *Clin Infect Dis* 2004;38(2):190-8.
4. McCarthy TA, Barrett NL, Hadler JL, et al. Hemolytic Uremic Syndrome and *Escherichia coli* O121 at a Lake in Connecticut, 1999. *Pediatrics* 2001;108(4):E59.
5. Kehl KS, Havens P, Behnke CE, et al. Evaluation of the Premier EHEC assay for detection of Shiga toxin-producing *Escherichia coli*. *J Clin Microbiol* 1997;35:2051-4.
6. Gavin PJ, Peterson LR, Pasquariello AC, et al. Evaluation of performance and potential clinical impact of ProSpectT Shiga toxin *Escherichia coli* microplate assay for detection of Shiga Toxin-Producing *E. coli* in stool samples. *J Clin Microbiol* 2004;42:1652-6.

M. Jodi Rell, Governor
J. Robert Galvin, MD, MPH Commissioner of Health

James L. Hadler, MD, MPH
State Epidemiologist
Division of Infectious Diseases

HIV/AIDS Surveillance (860) 509-7900
Epidemiology (860) 509-7994
Immunizations (860) 509-7929
Pulmonary Diseases (860) 509-7722
Sexually Transmitted Diseases (STD) (860) 509-7920

Connecticut Epidemiologist

Editor: Matthew L. Cartter, MD, MPH

Assistant Editor & Producer:
Starr-Hope Ertel