Chicago Department of Public Health

January, 2007

CDInfo

MONTHLY MORBIDITY REPORT

Communicable Disease Information

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CDInfo **is a surveillance newsletter intended to promote prevention of morbidity and mortality by providing useful data and practical recommendations for clinicians, laboratorians and infection control personnel who diagnose, treat and/or report infectious diseases in Chicago.**

Save the Date

The Chicago Department of Public Health

12th Annual Infection Control Conference

May 4, 2007

Holiday Inn City Center

300 E. Ohio

Shiga Toxin producing *Escherichia coli* – Local and National Surveillance

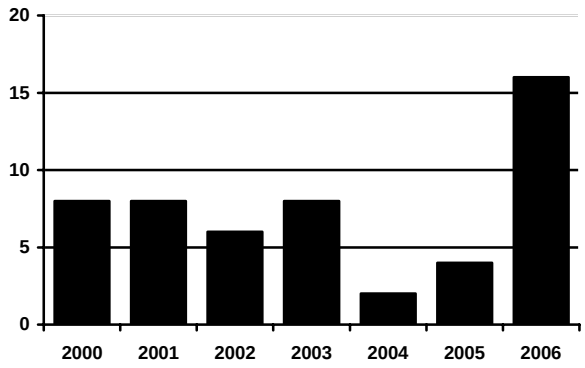
Background

Enterohemorrhagic *Escherichia coli* strains cause gastrointestinal disease of varying severity, including abdominal cramping, watery diarrhea, bloody diarrhea and hemorrhagic colitis. Fever is usually not present. Sequelae of infection may include hemolytic uremic syndrome (HUS), especially in children under 5 years and the elderly, thrombotic thrombocytopenic purpura (TTP) in adults (a disease spectrum often designated as TTP-HUS) and neurologic symptoms. HUS carries a 12% risk of end-stage renal disease or death, and 25% of survivors develop long term renal sequelae such as hypertension, proteinuria or renal insufficiency. The principal virulence factor of these organisms is a group of related cytotoxins called Shiga toxins (Stx), leading to the descriptive term "Shiga Toxin producing *E. coli*" (STEC). The STEC serotype most frequently associated with disease is O157:H7; however, other serotypes have been identified as the causes of sporadic disease and outbreaks. Non-O157 STEC cause disease of varying severity, including hemorrhagic colitis, HUS and death. The proportion of Stx-related disease attributed to non-O157 serotypes varies geographically, but the true incidence and burden of illness is not known.

The reservoir of STEC strains is the gastrointestinal tracts of young cattle and other herbivorous animals; however, these strains are stable in the environment and can proliferate in foods and beverages. The infectious dose is estimated at fewer than 100 organisms. The incubation period is usually 3 to 4 days but ranges from 1 to 8 days. Antimicrobial therapy is generally not recommended for the treatment of *E. coli* O157:H7 or STEC infections due to evidence that antibiotics may increase the risk of HUS.

E. coli O157:H7 was first recognized as a foodborne pathogen in 1982. Since then, *E. coli* O157:H7 has been responsible for at approximately 73,000 illnesses annually, with outbreaks linked to undercooked ground beef, other beef, unpasteurized milk or cider, sprouts, lettuce, salad, melons, grapes, coleslaw and contaminated water. Clusters of infection have also been linked to animal exposures, including farms and petting zoos. Person-to-person transmission can occur. In September – October, 2006, a large, multistate outbreak of *E. coli* O157:H7 infection attributed to fresh, bagged spinach led to the infection of at least 199 individuals from 26 states, including 31 cases (16%) of HUS and 3 deaths. More recently, an outbreak of *E. coli* O157:H7 has been linked to Taco Bell restaurants in the northeastern United States and has resulted in 71 reported illnesses from 5 states (as of December 14, 2006), including 8 cases (11%) of HUS. A second outbreak associated with Taco John's restaurants in Minnesota and Iowa has resulted in 65 cases (as of December 14, 2006). The sources of these outbreaks are still under investigation, but case control studies have suggested that shredded lettuce may be implicated in both outbreaks.

Reported *E. coli* O157:H7 Cases, Chicago, 2000-2006*



*2006 data is preliminary as of Dec. 27, 2006.

Local Surveillance – Summer, 2006

On July 3, 2006, the Chicago Department of Public Health (CDPH) Communicable Disease Program received notification of two cases of *E. coli* O157:H7 from the microbiology laboratory of a Chicago hospital. The

two isolates were indistinguishable by pulsed field gel electrophoresis (PFGE). Hypothesis-generating interviews of the two case-patients revealed that the only exposure in common that could have accounted for the illnesses was that both had consumed foods from the same restaurant in the week prior to onset. A sanitary inspection of the facility conducted by the CDPH Food Protection Division found no critical violations. None of the workers reported recent history of gastrointestinal illness, and none had *E. coli* O157:H7 isolated from stool specimens. A health alert was sent to Chicago hospitals requesting heightened case surveillance for gastrointestinal illness compatible with *E. coli* O157:H7 infection or HUS. No additional cases associated with this restaurant were identified.

A review of *E. coli* O157:H7 in Chicago for the past seven years revealed an increase in the number of cases reported to CDPH in 2006 (see Figure). Further surveillance is needed to determine whether this increase represents a true increase in disease, or heightened awareness of the organism by clinicians leading to increased diagnostic testing and/or reporting of illness.

Issues related to laboratory surveillance for E. coli *O157:H7 and other STEC*

Review of case reports and discussions with local hospital laboratories revealed substantial variation in the ways that different hospitals test for these organisms. The classic method for identifying *E. coli* O157:H7 is to culture the specimen on a sorbitol-containing medium, such as sorbitol MacConkey agar (SMAC). Unlike most enteric organisms, *E. coli* O157:H7 is unable to metabolize sorbitol after overnight incubation, resulting in white or colorless colonies (as opposed to pink colonies for sorbitol fermenting organisms). This method is extremely sensitive for detecting *E. coli* O157:H7; however, no selective medium exists for the isolation of non-O157 STEC. Furthermore, although latex agglutination tests that rapidly identify *E. coli* O157:H7 are available commercially, there are no equivalent tests for non-O157 serotypes. Consequently, few clinical laboratories test stool specimens for these organisms.

In the past few years, alternate methodologies for detecting STEC have been developed, including enzyme immunoassays (EIA) and PCR that detect Stx or the genes that produce Stx directly. These techniques have the advantages of being quick and capable of identifying the presence of non-O157 STEC, but lack the specificity of culture and do not allow subsequent comparison of organisms by molecular techniques such as PFGE.

The Centers for Disease Control and Prevention recommend that laboratories use a combination of bacterial culture on sorbitol-containing medium and EIA or PCR for Stx to identify *E. coli* O157:H7 as well as non-O157 STEC in stool specimens. In addition to the public health implications of being able to rapidly identify outbreaks of foodborne disease, there are clinical implications to establishing a diagnosis and instituting appropriate therapy and supportive care that may result in improved patient outcome

As part of an initiative to improve our interpretation of foodborne illness surveillance data, the CDPH Communicable Disease Program is conducting a survey of hospital laboratories to create a clearer picture of *E. coli* O157:H7 and STEC diagnostic procedures in Chicago hospitals. We will subsequently survey commercial referral laboratories that receive specimens from Chicago hospitals. The results will be shared in aggregate form with participating hospitals and laboratories.

Reporting cases to CDPH

Cases of *E. coli* O157 or other STEC infections should be reported to the Communicable Disease Program within 24 hours. Report cases by calling Julio Fernandez (312-746-5925) or Loretta Miller (312-746-5377), or by faxing reports to 312-746-6388 or 312-743-1059. All specimens positive for *E. coli* O157:H7 or Stx should be sent to the State Laboratory for further characterization.

Additional Information

- Centers for Disease Control and Prevention. Importance of culture confirmation of Shiga toxin-producing *Escherichia coli* infection as illustrated by outbreaks of gastroenteritis – New York and North Carolina, 2005. Morb Mortal Wkly Rep 2006;55:1042-5.
- Johnson KE, Thorpe CM, Sears CL. The emerging clinical importance of non-O157 Shiga toxin-producing *Escherichia coli.* Clin Infect Dis 2006; 43:1587-95*.*
- Rangel JM, Sparling PH, Crowe C, et al. Epidemiology of *Escherichia coli* O157:H7 outbreaks, United States, 1982-2003. Emerg Infect Dis 2005;11:603-9.
- Tarr PI, Gordon CA, Chandler WL. Shiga toxin producing *Escherichia coli* and haemolytic uraemic syndrome, Lancet 2005; 365:1073-86.