Escherichia coli O157:H7

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Escherichia coli is an important pathogen which can cause a variety of diseases including diarrhea, urinary tract infections, bacteremia, pneumonia, and meningitis. Most strains of *E. coli* are found surviving as avirulent commensals in the large bowel of mammals. However, specific virulence traits can be acquired from plasmids and other transferable elements which allow the organism to invade or attach to specific cellular types or to produce toxins. A loose correlation exists between strains of *E. coli* that cause specific disease and a serological classification scheme first described by Kauffman in 1944. This scheme is based on two highly variable cellular antigens, the somatic (O) antigen and the flagellar (H) antigen. Strains with a similar O antigen are of the same serogroup, while those strains with a similar O antigen and H antigen are of the same serotype. Additionally, strains can be characterized by the type of virulence genes they possess. Strains which possess the same virulence factors are said to be of the same virotype. Using serotyping and virotyping in conjunction, six different groups have evolved from those strains of *E. coli* that cause diarrhea. The first five groups include enterotoxigenic *E. coli* (ETEC), enteroaggregative *E. coli* (EAEC),enteroinvasive *E. coli* (EIEC), diffusely adherent *E. coli* (DAEC), and enteropathogenic *E. coli* (EPEC). These groups cause diarrheal disease by various combinations of virulence traits and mechanisms including the liberation of enterotoxins (ETEC), adherance to intestinal epithelial cells (EAEC, DAEC, and EPEC) and invasion of intestinal epithelial cells (EIEC). The sixth group, enterohemorrhagic *E. coli* or EHEC, is a more serious pathogen which not only causes bloody diarrhea but may also cause other serious diseases such as hemolytic uremic syndrome (HUS). EHEC was first recognized as an emerging pathogen in 1982 when an outbreak of diarrhea and HUS occured following ingestion of undercooked hamburgers from a fast-food restaurant. An epidemiological investigation found that the etiologic agent causing the disease was *E. coli* O157:H7, a serotype not previously recognized as a human pathogen. Since that time, large foodborne outbreaks caused by *E. coli* O157:H7 have occurred as well as sporadic cases of disease. The CDC has recently reported *E. coli* O157:H7 as the fourth most prevalent bacterial diarrheal pathogen behind *Campylobacter* sp., *Salmonella* sp., and *Shigella* sp.

EHEC are ingested from contaminated water or food and appear to colonize the lower intestine. A small infectious dose is required; studies have shown that as few as 200 bacteria are sufficient to cause disease. The major virulence factor of EHEC is a bacteriophage-encoded shiga -toxin. This toxin, in part, mediates diarrheal disease and presumably HC and HUS as well. There are two different types of immunologically distinct shiga-toxins produced in EHEC called Stx1 and Stx2. Stx1 is identical to the shigatoxin produced by *Shigella dysenteriae.*

Other virulence factors include an outer membrane protein called intimin, which is important in colonization of the intestine, and an enterohemolysin, encoded on a 60 Mda plasmid called pO157. Enterohemolysin may contribute to virulence by lysing red blood cells and therefore providing a source of iron to the bacterium.

Even though *E. coli* O157:H7 is the major serotype isolated from patients with HC and HUS, other serotypes have recently been isolated which express Stx1, Stx2, intimin and enterohemolysin. The most common non-O157:H7 serotypes isolated from humans with diarrheal disease are O26:H11, O103:H2, O111:NM, and O113:H21. Additionally, investigators have found Stx-producing *Citrobacter freundii* and *Enterobacter cloacae* from patients with HUS. Livestock animals, including cattle, sheep, pigs, chickens and goats, are the major reservoir of

Stx-producing *E. coli.* However, since the majority of these isolates are not of the O157:H7 serotype and do not contain other virulence factors such as *eae* or enterohemolysin, the virulence of these isolates is not known.

Treatment of patients with HUS is at best controversial. Some studies have suggested that EHEC infected patients who were treated with antibiotics had a greater chance of developing HUS than those patients who were not. Therefore, treatment is mostly supportive. However, screening for and detection of shiga-toxin producing *E. coli* is necessary for the detection of outbreaks so that appropriate infection control measures can be administered.

Detection of shiga-toxin producing *E. coli* is difficult in the clinical microbiology laboratory due to the large number of avirulent *E. coli* present in fecal specimens. However, since *E. coli* O157:H7 do not ferment sorbitol as strongly as other commensal *E. coli,* selective and differential media such as sorbitol-MacConkey agar (SMAC) or cefixime-tellurite-sorbitol-MacConkey agar (CT-SMAC) can be used to screen stool samples for this bacterium. Since other serotypes of EHEC ferment sorbitol strongly, they will not be detected using SMAC or CT-SMAC agars. To circumvent this problem, investigators have developed ELISA and PCR assays that will detect shiga toxin (both Stx1 and Stx2) from fecal samples. However, due to the expense of these assays, they are not widely used in clinical microbiology laboratories across the country. Therefore, the prevalence of non-*E. coli* O157:H7 EHEC is not known in most parts of the United States.

If your laboratory is not screening stool specimens for *E. coli* O157:H7, the NPHL suggests that you add SMAC or CT-SMAC to your routine stool culture procedure for a period of six months (preferably during the spring and summer). We do not suggest that you add a SMAC or CT -SMAC plate only to stools which are visibly bloody since blood in stools may not be visually evident. After the six month period, an assessment can be made as to whether continuing to screen for *E. coli* O157:H7 is appropriate for your laboratory. Our opinion is that even one case of diagnosed *E. coli* O157:H7 infection is worth the cost of the added SMAC or CT-SMAC agar plate.