Enterohemorrhagic E. Coli (EHEC)

Enterohemorrhagic Escherichia coli (EHEC), commonly called E. coli O157:H7 is an important cause of foodbourne illness in the United States. In addition to bloody diarrhea, individuals can develop hemolytic uremic syndrome with anemia, thrombocytopenia, and acute kidney injury, especially in young children and elderly persons. This bacterial strain is most commonly transmitted via the fecal-oral route and typically associated with eating contaminated ground beef in undercooked hamburgers. It is a specific serotype of E. coli. The O refers to the cell wall antigen water, while H refers to the flagella antigen. This strain may produce Shiga-like toxin, which catalytically inactivates the 60S ribosomal subunit of eukaryotic cells. This blocks mRNA translation and causes cell death in the mucosal cells of the GI tract. The toxin also enhances cytokine release, which can cause hemolytic uremic syndrome (HUS). Strains of E. coli that express Shiga-like toxins gained this ability through lysogeny, meaning infection with a prophage that contained the coding for the toxin. E. coli O157:H7 can be differentiated from other E. coli strains because it is typically non-sorbitol fermenting, whereas the majority of other E. coli strains are sorbitol fermenters.In patients, antibiotics are not part of the treatment of patients with EHEC disease and may possibly increase the risk of subsequent HUS. As toxins may be released by dead and dying bacterial cells, the risk of endotoxin release could add to the patient's already potentially lethal burden.

PLAY PICMONIC

Characteristics

O157:H7
O157:H7
Enterohemorrhagic Escherichia coli (EHEC), commonly called E. coli O157:H7, is an important cause of foodborne illness in the United States. It is a specific serotype of E. coli. The “O” antigen refers to the cell wall lipopolysaccharide antigen, while the “H” antigen refers to the flagellar antigen. This strain produces Shiga-like toxin, which inactivates the 60S ribosomal subunit of eukaryotic cells, leading to inhibition of protein synthesis and cell death.

Non-Sorbitol Fermenting
Nun-Sorbet Ferns
E. coli O157:H7 can be differentiated from other E. coli strains because it is typically non–sorbitol fermenting, whereas the majority of other E. coli strains are sorbitol fermenters. This characteristic is diagnostic when cultured on sorbitol-MacConkey agar, where EHEC colonies appear colorless, unlike pink colonies of typical E. coli.

Hamburger Meat
Hamburger
This bacterial strain is most commonly transmitted via the fecal-oral route, typically associated with eating contaminated ground beef in undercooked hamburgers. Other sources include unpasteurized milk, raw vegetables, and contaminated water. The low infectious dose of EHEC makes person-to-person transmission possible.

Produces Shiga-like Toxin
She-jello in Mirror
EHEC produces two toxin variants, Stx1 and Stx2. Stx1 is identical to the Shigella dysenteriae Shiga toxin, while Stx2 differs slightly but has the same mechanism of action. The toxin catalytically inactivates the 60S ribosomal subunit by removing an adenine residue from rRNA, thereby blocking mRNA translation and causing cell death in the intestinal mucosa. It also stimulates cytokine release and induces endothelial injury, which can result in hemolytic uremic syndrome (HUS). Stx2 is more strongly associated with HUS.

Gram-negative
Graham-cracker Negative-devil
E. coli is a Gram-negative rod belonging to the Enterobacteriaceae family. It does not retain the crystal violet stain due to its thin peptidoglycan layer and outer lipopolysaccharide membrane, which contributes to its endotoxic effects by activating macrophages and cytokine release via TLR4 signaling.

Lysogeny
Lasso-jeans
EHEC strains that express Shiga-like toxins acquire this virulence trait through lysogenic conversion, which involves infection with a temperate bacteriophage (prophage) that carries the Shiga toxin gene (stx). The phage integrates into the bacterial genome, allowing toxin expression during bacterial replication.

Inactivates 60S Ribosome

60 S Ribosome Being Hammered

The Shiga-like toxin produced by EHEC irreversibly inactivates the 60S ribosomal subunit by cleaving a specific adenine residue from 28S rRNA. This halts protein synthesis and causes cellular apoptosis in the colonic epithelium. The resulting mucosal necrosis and inflammation contribute to bloody diarrhea, while vascular injury leads to HUS.

Disease

Bloody Diarrhea

Red Toilet

EHEC infections cause hemorrhagic colitis with bloody diarrhea but no fecal leukocytes, as the bacteria do not invade mucosal cells like Shigella. Instead, the Shiga-like toxin damages endothelial cells and the intestinal mucosa, producing bloody stools without significant fever.

Hemolytic Uremic Syndrome (HUS)

(HUS) Hemolysing U-rainbow Anemone

Hemolytic uremic syndrome is a potentially fatal triad of microangiopathic hemolytic anemia, thrombocytopenia, and acute kidney injury (AKI). It results from Shiga toxin–induced endothelial injury, which activates platelets and causes microthrombi in small vessels, particularly in the kidneys. HUS typically follows diarrheal illness caused by E. coli O157:H7 or Shigella dysenteriae.

Hemolytic Anemia

Hemolysing-RBCs from Anemone

Hemolytic anemia in HUS is microangiopathic, caused by fragmentation of red blood cells as they pass through narrowed, damaged microvasculature lined with platelet-fibrin thrombi. This leads to schistocytes on peripheral smear, jaundice, and elevated LDH. Patients may present with fatigue, pallor, and dark urine.

Acute Kidney Injury (AKI)

Kidney

Hemolytic uremic syndrome can cause acute kidney injury (AKI) due to endothelial injury and microthrombi formation within renal arterioles and glomeruli. The resulting ischemia and reduced filtration capacity lead to elevated BUN and creatinine, oliguria, and fluid overload.

Thrombocytopenia

Trombone-side-toe-peanut

Thrombocytopenia in HUS results from platelet consumption within widespread microthrombi. Although platelet count is reduced, coagulation studies (PT, aPTT) are typically normal, distinguishing HUS from disseminated intravascular coagulation (DIC). This contributes to petechiae, purpura, and mucosal bleeding.