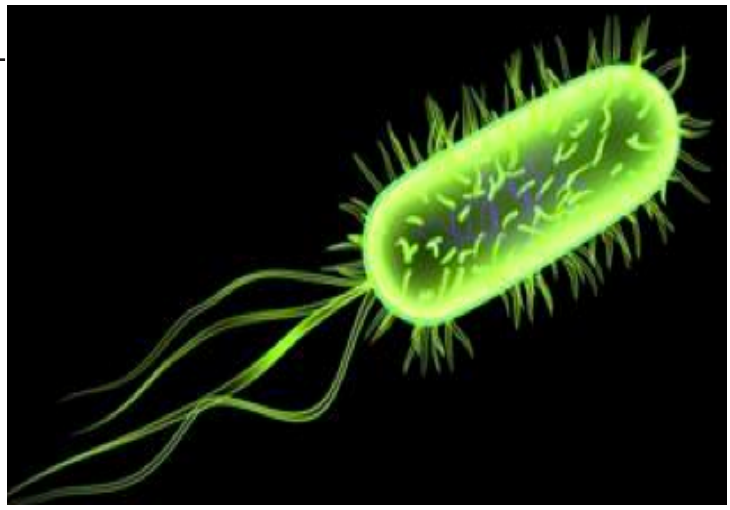
Escherichia coli

Characteristics, Diagnosis and Treatment

CONTENT



I. GENERAL INFORMATION

E. coli is a Gram-negative (bacteria which do not retain crystal violet dye), facultative anaerobic (that makes ATP by aerobic respiration if oxygen is present, but is capable of switching to fermentation or anaerobic respiration if oxygen is absent) and nonsporulating bacteria. Cells are typically rod-shaped, and are about 2.0 micrometers (μm) long and 0.25–1.0 μm in diameter, with a cell volume of 0.6–0.7 μm^3. E. coli uses mixed-acid fermentation in anaerobic conditions, producing lactate, succinate, ethanol, acetate, and carbon dioxide.

Since many pathways in mixed-acid fermentation produce hydrogen gas, these pathways require the levels of hydrogen to be low, as is the case when E. coli lives together with hydrogen-consuming organisms, such as methanogens or sulphate-reducing bacteria.

Optimal growth of E. coli occurs at 37 °C (98.6 °F), but some laboratory strains can multiply at temperatures of up to 49 °C. Growth can be driven by aerobic or anaerobic respiration, using a large variety of redox pairs, including the oxidation of pyruvic acid, formic acid, hydrogen, and amino acids, and the reduction of substrates such as oxygen, nitrate, fumarate, dimethyl sulfoxide, and trimethylamine N-oxide.

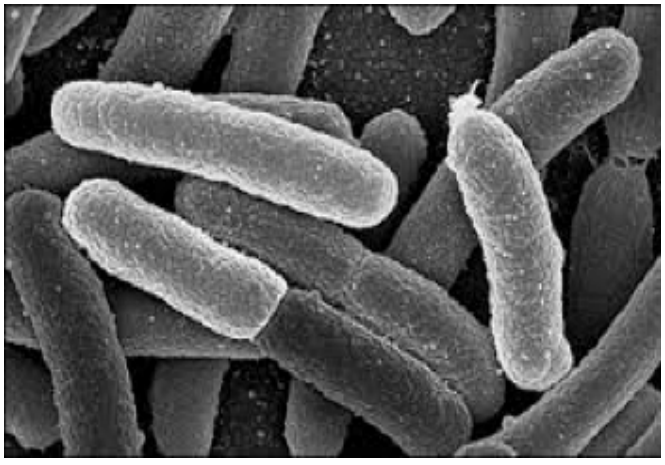


Figure 1. E.Coli cells under the view of microscope

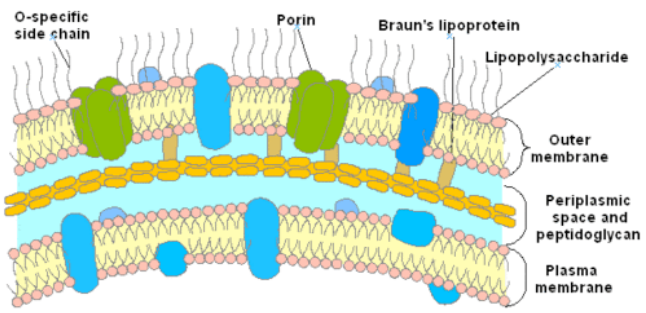


Figure 2. Gram-negative cell wall

II. STRAIN AND TYPE

E. coli is the type species of the genus (Escherichia). The first complete DNA sequence of an E. coli genome (laboratory strain K-12 derivative MG1655) was published in 1997. It was found to be a circular DNA molecule 4.6 million base pairs in length, containing 4288 annotated protein-coding genes (organized into 2584 operons), seven ribosomal RNA (rRNA) operons, anda 86 transfer RNA (tRNA) genes. Genes in E. coli are usually named by 4-letter acronyms that derive fom their function (when known). For instance, recA is named after its role in homologous recombination plus the letter A. Functionally related genes are named recB, recC, recD etc. The proteins are named by uppercase acronyms, e.g. RecA, RecB, etc. When the genome of E. coli was sequenced, all genes were numbered (more or less) in their order on the genome and abbreviated by b numbers, such as b2819 (=recD) etc.

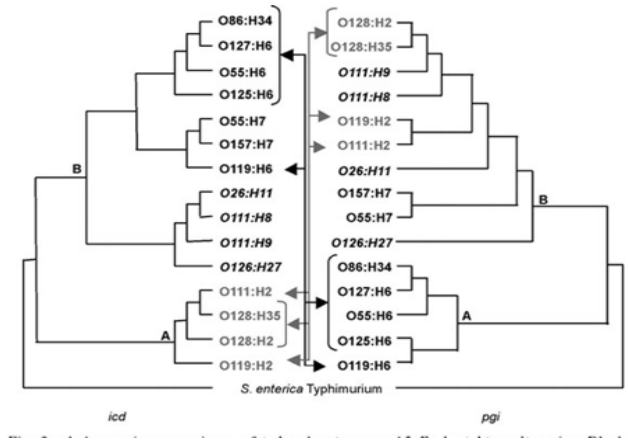


Figure 3. strain and type of E.Coli

The "b" names were created after Fred Blattner who led the genome sequence effort. Another numbering system was introduced with the sequence of another E. coli strain, W3110, which was sequenced in Japan and hence uses numbers starting by JW... (Japanese W3110), e.g. JW2787 (= recD). Hence, recD = b2819 = JW2787. Note, however, that most databases have their own numbering system, e.g. the EcoGene database uses EG10826 for recD. Finally, ECK numbers are specifically used for alleles in the MG1655 strain of E. coli K-12. Complete lists of genes and their synonyms can be obtained from databases such as EcoGene or Uniprot. Pathogenic E. coli strains are categorized into pathotypes. Six pathotypes are associated with diarrhea and collectively are referred to as diarrheagenic E. coli.

Six Strains of E.coli Considered Pathgenic:

- Shiga toxin-producing E. coli (STEC)—STEC may also be referred to as Verocytotoxin-producing E. coli (VTEC) or enterohemorrhagic E. coli (EHEC). This pathotype is the one most commonly heard about in the news in association with foodborne outbreaks.
- Enterotoxigenic E. coli (ETEC)
- Enteropathogenic E. coli (EPEC)
- Enteroaggregative E. coli (EAEC)
- Enteroinvasive E. coli (EIEC)
- Diffusely adherent E. coli (DAEC)

III. ROLE IN DISEASE

Most E. coli strains do not cause disease, but virulent strains can cause gastroenteritis, urinary tract infections, and neonatal meningitis. It can also be characterized by severe abdominal cramps, diarrhea that typically turns bloody within 24 hours, and sometimes fever. In rarer cases, virulent strains are also responsible for bowel necrosis (tissue death) and perforation without progressing to hemolytic-uremic syndrome, peritonitis, mastitis, septicemia, and Gram-negative pneumonia.

There is one strain, E.coli #0157:H7, that produces a toxin called the Shiga toxin (classified as a bioterrorist agent). This toxin causes premature destructiontion of

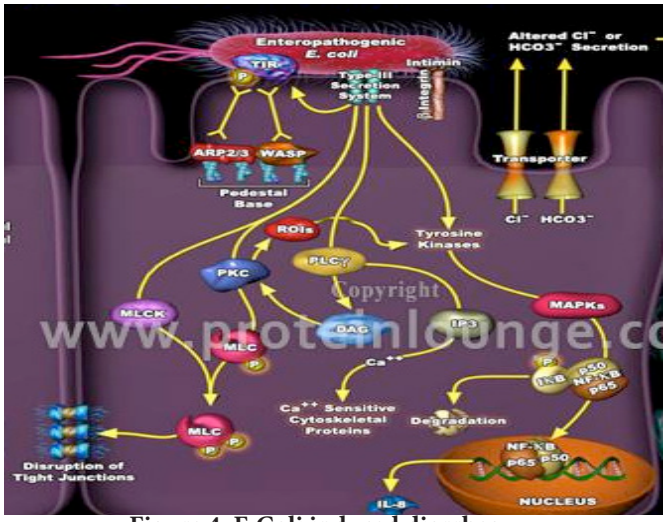


Figure 4. E.Coli induced diarrhea

the red blood cells which then clog the body's filtering system, the kidneys, causing **hemolytic-uremic syndrome (HUS)**. This in turn causes strokes due to small clots of blood which lodge in capillaries in the brain. This causes the body parts controlled by this region of the brain not to work properly. In addition, this strain causes the buildup of fluid (since the kidneys do not work) leading to edema around the lungs and legs and arms. This increase in fluid buildup especially around the lungs impedes the functioning of the heart, causing an increase in blood pressure.

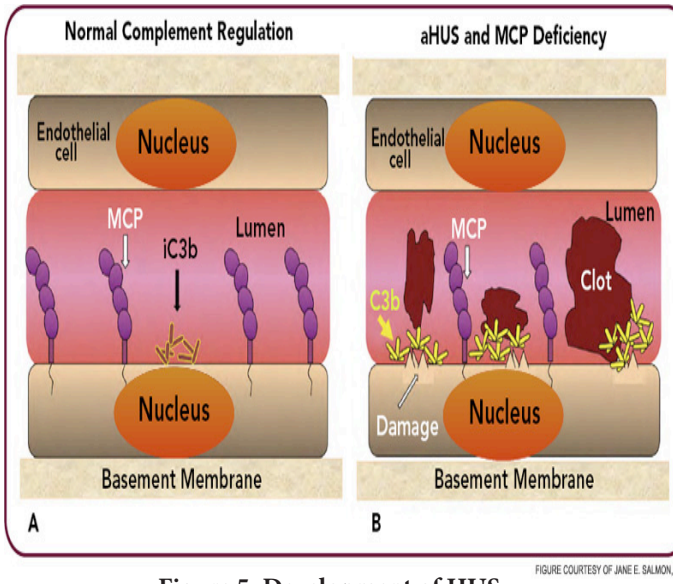


Figure 5. Development of HUS

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IV. DIAGNOSIS

Because other disease-causing bacteria (for example, Shigella and Salmonella) can give patients similar initial symptoms, a definite diagnosis is based on culture of E. coli 0157:H7 from the patient's sample of stool on special culturing plates that then are tested with antiserum (antibodies) that react only with E. coli O157H7. Not all clinics or hospitals have the diagnostic antiserum, so the testing may take a few days.

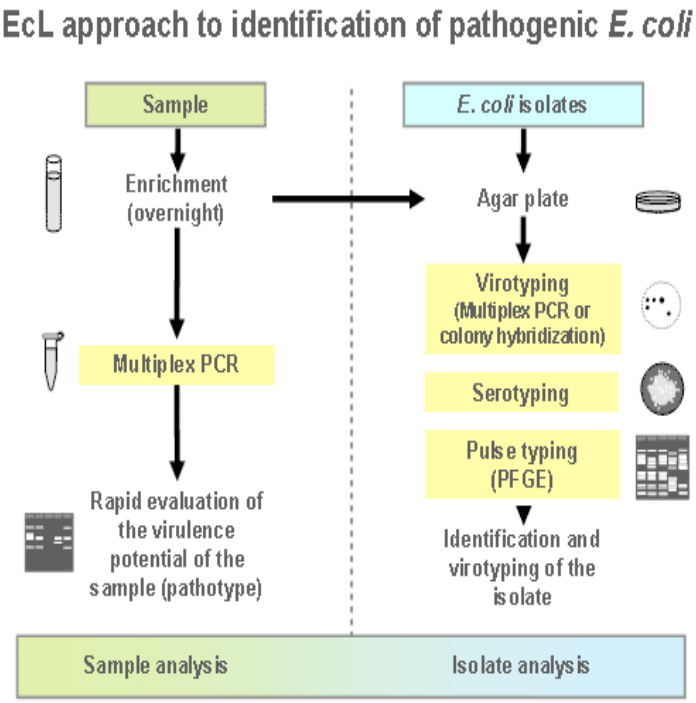

Figure 6. Diagnosis procedure of E.Coli

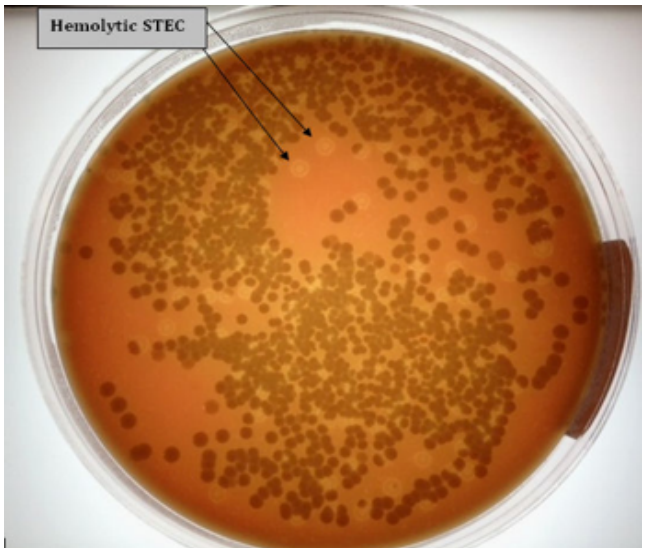

Figure 7. Use of antibodies to identify E.Coli

Because of the high frequency of outbreaks of E. coli 0157:H7, the CDC in 2009 recommended that all patients being screened for community-acquired diarrheal infections have their stool samples analyzed with antisera for Shiga toxins, the toxins that are produced by E. coli 0157:H7 and a few other bacteria (for example, E. coli 0104:H4), in addition to having cultures of their stool. This approach may result in faster diagnosis of E. coli 0157:H7 infections.

V. TREATMENT

Patients, especially healthy adults, often require no treatment for E. coli O157:H7 since many infections are self–limited. Moreover, for the acute diarrheal illness, antibiotics have not proven useful. In fact, some studies have shown that antibiotics may increase the chances of developing HUS (up to 17-fold). This effect is thought to occur because the antibiotic damages the bacteria, causing them to release even more toxin. Most investigators suggest antibiotic use only if a patient is septic, that is, there is evidence that the bacterium has spread to parts of the body other than the intestine. In addition, use of atropine and diphenoxylate (Lomotil), drugs that are commonly used to control diarrhea, also may increase symptoms and trigger complications.

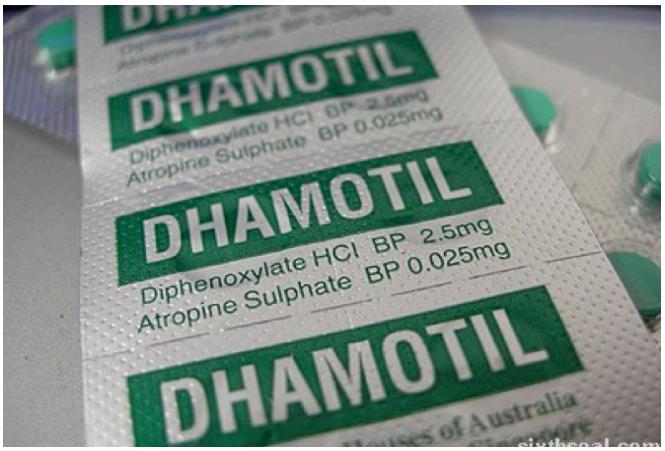

Figure 8. Diphenoxylate for E.Coli

When necessary, treatment includes the replacement of fluids and electrolytes to treat or prevent dehydration. Infection with E. coli 0157:H7 should be treated by a physician especially in children and the elderly. HUS and TTP require complex supportive care (for example, plasma exchange) in the hospital. Patients with kidney failure may need dialysis.

Treatments for Shiga toxin–associated hemolytic-uremia syndrome (Stx-HUS) have not proven of value. Instead, comprehensive supportive therapy is still the mainstay during the acute phase.

There is no clear consensus on the use of antibiotics. The evidence avoidance of antibiotics unless patient is septic. An in-vitro study demonstrated that although growth-inhibitory levels of antibiotics suppressed Stx production, subinhibitory levels of certain antibiotics. The FDA has approved the use of eculizumab for the treatmant of atypical hemolytic uremic syndrome. This monoclonal antibody inhibits complement-mediated thrombotic microangiopathy.

Other treatments during the acute phase of the disease, including plasma therapy and use of intravenously infused immunoglobulin (IgG), fibrinolytic agents, antiplatelet agents, corticosteroids, and antioxidants have proved ineffective in controlled clinical trials. Renal transplantation is safe and effective for children who progress to end-stage renal disease (ESRD). The recurrence rate in patients who undergo renal transplantation for HUS is 0-10%.