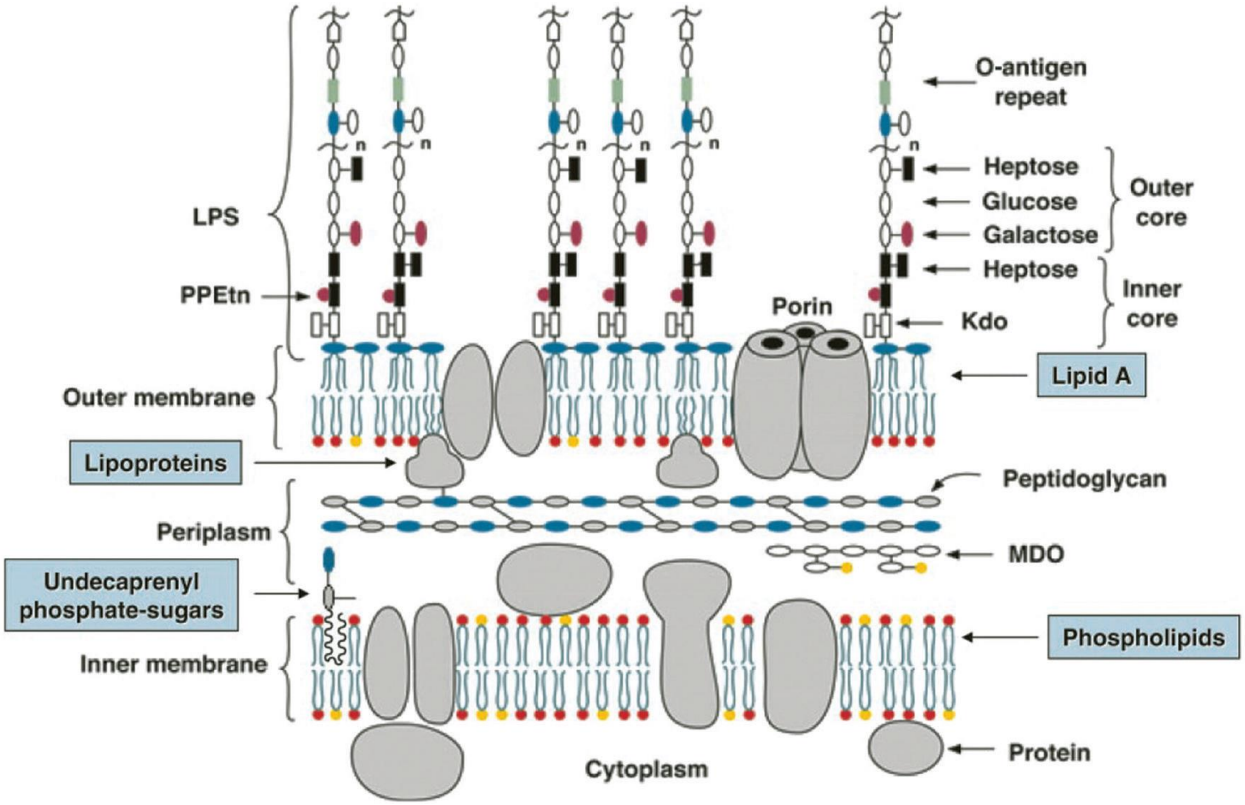
Enterobacteriace

Cellular Structure and Composition

Outer membrane: These organisms are gram-negative straight rods. The cell wall as other gram negative bacteria is consistent of inner cytoplasmic membrane, outer membrane (OM), and between them a thin peptidoglycan layer (Figure 1). The OM is an asymmetric bilayer with phospholipids on its inner surface, and lipid A (endotoxin), the hydrophobic anchor of lipopolysaccharide (LPS) on the outside. The outer most region of the LPS consists of polysaccharide called O antigen. The O antigen portion of the LPS molecule provides protection against phagocytosis.

Capsule: Enterobacteriaceae often expresses a capsule that consists of polysaccharide. Two types of capsular polysaccharides may be produced. The first type, known as M (mucous) antigen is produced by most strains. M antigen is thought to provide protection against desiccation. The second type, known as K antigen, K for *kapsel* (capsule in German), may provide antiphagocytic.

Pili: Many members of Enterobacteriaceae have fimbria (pili). Fimbriae are hair-like appendages that are arranged diffusely on the surface of the bacterial cells. Fimbriae are thinner and typically shorter and more numerous than flagella. The fimbriae bind to receptors on the surfaces of host cells, and different types of fimbriae vary in their binding specificities. A single bacterial isolate can express multiple fimbrial types. The term “pili” (plural) is used interchangeably with fimbriae.



Molecular model of the inner and outer membranes of E. coli.

Escherichia

Escherichia is the type genus of the Enterobacteriaceae family. Many E. coli are commensals of the intestinal tract, especially the large intestine; however, many are opportunistic or primary pathogens too. Pathogenic E. coli are broadly divided into diarrhea genic and extraintestinal strains.

Cellular Structure and Composition

Escherichia are straight, cylindrical, gram-negative rods with rounded ends. The cell wall contains lipopolysaccharide (LPS), outer membrane proteins, lipoproteins and a thin peptidoglycan layer. Cells with a complete LPS layer will typically

express an O-antigen. Cells may express a capsule (K-antigen) and flagella (H-antigen).

Adhesins: fimbria (pili) Fimbrial adhesins found on *E. coli* that cause disease (colibacillosis) in animals include F4. F4 positive bacteria adhere to the full length of the small intestine, a feature that greatly increases the severity of disease.

Capsule is polysaccharide. The negative charge of the capsule helps protect the bacteria against phagocytosis, since the phagocytes also have a negative charge on their cell surfaces. The capsule is usually antigenic, and if so, listed as a K-antigen type. Some capsular antigen types (e.g., K87) make the bacteria with "serum resistance"; that is, it protects the outer cell membrane from the membrane attack complex of the complement. This property is especially important for survival of pathogenic strains causing extraintestinal infections. A thick O antigen layer may provide effects similar to a K-antigen capsule. These effects may include protection against phagocytosis and the membrane attack complex of the complement system. Thick O antigen layers that mediate these protective effects are some times referred to as an O antigen capsule.

Enterotoxins: Some pathogenic strains, especially those belonging to the diarrheagenic class produce one or more enterotoxins. These enterotoxins are protein exotoxins encoded by genes. *E. coli* produces two different enterotoxins: heat labile enterotoxin (LT) and heat-stable enterotoxin (ST). LT is labile when heated to 70 ◦C for 10 min, whereas ST is stable at 100 ◦C for 15min.

Serotyping

The O, H, and K antigens are used in serotyping a particular isolate. There are 174 O antigens, at least 80 K antigens, and 53 H antigens in the international serotyping scheme. For example, O8:K87:H19 describes an isolate with antigens

of the O antigen number 8, capsular antigen number 87, and flagellar antigen number 19.

Reservoir and Transmission

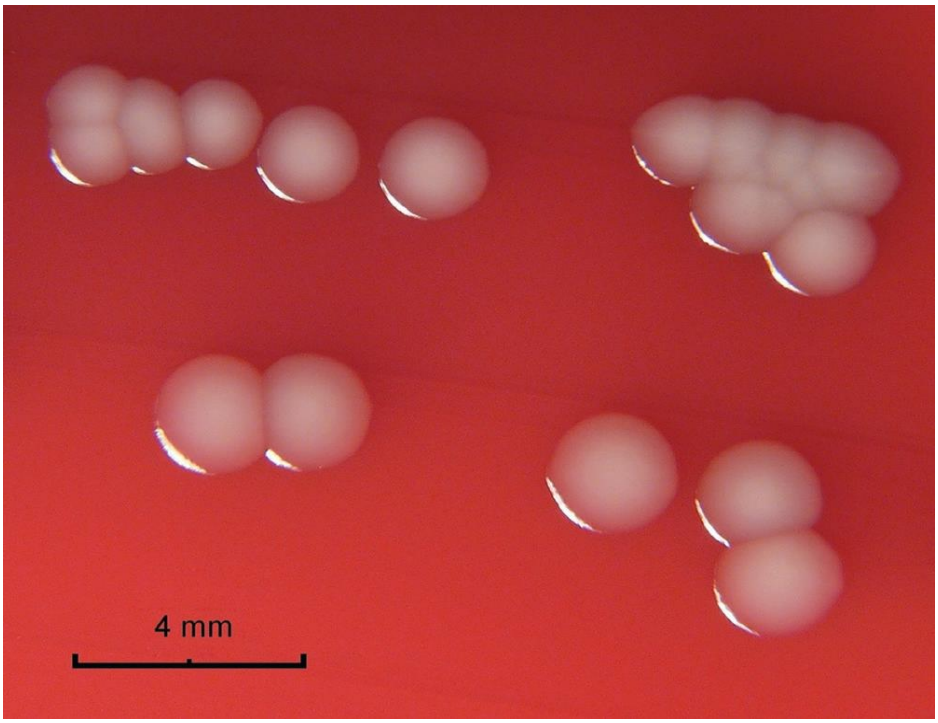
Strains of *E. coli* capable of producing disease reside in the larg gastrointestine and are abundant in environments inhabited by animals. Transmission is through the fecal-oral route. The larg intestine has been termed the “primary habitat” and the environment outside the animal, the “secondary habitat” of *E. coli*.

Avian-Pathogenic E. coli.

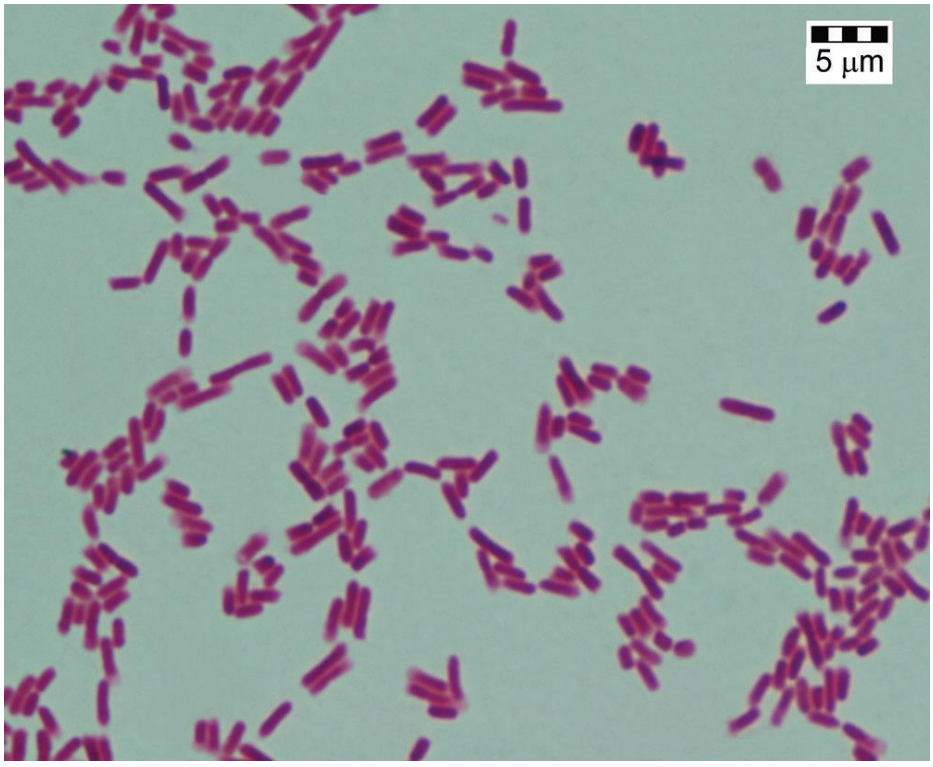
Avian-pathogenic E. Coli (APEC) cause colibacillosis of fowl, which is economically important to the poultry industry. APEC are invasive, extraintestinal strains of E. coli that are usually of certain serotypes, and bear several virulence genes. One common serotype is O1:K1:H7. The disease takes many forms in fowl, depending upon the age of the host and mode of infection. In the case of egg infections, the egg surface may be contaminated with potentially pathogenic strains at the time they are laid. The bacteria penetrate the shell and infect the yolk sac. If the bacteria grow, the embryo dies, usually late in incubation. Embryos that survive may die shortly after, with losses occurring as late as 3 weeks after hatching. A very important clinical manifestation in poultry is respiratory and septicemic disease. The course may be rapidly fatal or chronic, manifested by debilitation, diarrhea, and respiratory distress. Air sacculitis and pneumonia are common presentations. Other clinical syndromes caused by APEC include cellulitis, synovitis, pericarditis, salpingitis, and ophthalmitis.

Laboratory Diagnosis

Differentiation within enterbacteriace family is accomplished by some or all of the following: culture,biochemical tests, immunological tests (i.e., serotyping of O : K : H antigens and detection of virulence products), and PCR. In the case of intestinal samples, commensal strains of *E. coli* will be present and cannot be differentiated from pathogenic strains of *E. coli* simply by colony phenotype on such standard culture media as blood agar or MacConkey agar. In the case of some targeted serotypes (e.g., *E. coli* O157:H7), commercially available chromogenic media are available, and these media are designed to allow for presumptive differentiation of the pathogen from other flora on the plates. The microbiological diagnosis of extraintestinal disease is based upon the demonstration of *E. coli* in normally sterile sites or locations (joint, bone marrow, spleen, or blood). In fowl, the same sites are cultured, plus those grossly affected (lung, air sac). Dead in-shell embryos are cultured.



Colonies of E. coli growing on blood agar culture medium



E. coli gram-negative rods