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Escherichia coli, Pathogenicity of Escherichia coli,Virulence Factors of Escherichia coli, Secreted Virulence (Short Review)

Rana Talib Mohsen
University of Anbar, Al-Anbar, Iraq
rana2011@uoanbar.edu.iq

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Abstract

The Enterobacteriaceae family of facultative anaerobic, Gram-negative, glucose-fermenting bacteria that are catalase positive and oxidase negative is associated with almost all CUAI, whereas UTIs are particularly closely related. The bacterial species E. Coli is broad and varied. Some strains of Escherichia coli can cause lung infections, pneumonia, diarrhea, and urinary tract infections (UTIs). In natural settings, both biotic and abiotic variables can affect E. Coli's ability to survive and develop. The obtainability of nutrients and water, temperature, pH, and sunlight are examples of abiotic variables. E. Coli's capacity to obtain nutrients, contest with other microbes, and create biofilms outside is one example of a biologic challenge.

Keywords: Escherichia coli, Pathogenicity of Escherichia coli, Virulence Factors of Escherichia coli

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General Characteristics of *Escherichia coli*

Escherichia coli, the biological rock star, is the current term for the rapidly proliferating bacterium he discovered during this experiment, which he dubbed bacteria coli commune (1,2) E. Coli may grow rapidly and multiply in around 20 minutes under the right conditions. Numerous gene modification techniques that have created countless enzymes and other commercial products have employed The facultative anaerobic bacteria E. coli, which typically contains more than 106 cells per gram of feces (2,3). Throughout the staining procedure, E. Coli turns pink as a result of absorbing the color of the counterstain, safranin. Penicillin cannot damage E. coli because the outer layer of the cell wall serves as a barrier against certain antibiotics (4). The bacterial group E. Coli is diverse and wide-ranging. Some forms of E. coli can make you sick, but most are harmless. While certain strains of E. coli can cause respiratory diseases including pneumonia and UTIs, others can cause diarrhea. In a mutualistic relationship, harmless strains of bacteria that are a component of the gut's natural microbiota can support their hosts by preventing harmful bacteria from colonizing the intestine and creating vitamin K2, which aids in blood coagulation (5).

Pathogenicity of *Escherichia coli*

Escherichia coli is a human pathogen. Along with commensal strains, E. coli also harbors dangerous strains that cause about 2 million deaths and a variety of human diseases each year. E. coli has six well-studied intestinal pathotypes: Shigella strains are subtypes of Shiga-producing E. Coli, including those that are enterotoxigenic (ETEC), enteropathogenic (EPEC), enteroaggregative (EAEC), diffusely adherent (DAEC), enteroinvasive (EIEC), and enterotoxigenic (ETEC). These strains are categorized according to their mechanisms of pathogenicity and virulence. leading to diarrhea and other digestive issues. Enterohaemorrhagic E. coli (EHEC) is one kind of STEC that can cause serious enteric infections such hemolytic uraemic syndrome and hemorrhagic colitis, which can lead to sudden renal failure and often death (5,6,7). Pathogenic E. coli strains are concerned in a number of waterborne outbreaks, and STEC and EPEC have been well recognized as the source of waterborne epidemics worldwide (8,9). Numerous traits of the organism and host-related variables influence how UTIs develop. In other words, according to the genetic makeup of the strain, the virulence of the UPEC is influenced by the host's genetic

background as well as the functional and anatomical circumstances of the urinary tract (10). There are two ways that the microbes enter the urinary tract: the hematogenous route or the ascending route. The most popular path is the one that climbs. Here, the intestinal microbiota colonizes and moves up to the bladder and then the kidneys via the urethra. The haematogenous pathway of kidney infection is rare in healthy persons. Sometimes, in immunocompromised individuals with oral-sourced Candida fungemia or Staphylococcus aureus bacteremia, the renal parenchyma may be breached (1). Gram-negative UPEC is the most frequent causal agent of both complex and simple UTIs. Approximately 150 million persons globally have a UTI diagnosis each year (12). For instance, 90% of all simple UTIs, 65% of all severe UTIs, and up to 50% of all nosocomial UTIs are caused by community-acquired UTIs (13).

Virulence factors of *Escherichia coli*

In spite of strong host resistance, virulence factors allow the bacteria to infiltrate the host tissues and survive (14). the bacterial cell surface and those secreted and exported to the site of action are the two types of E. Coli virulence factors that have been identified as possibly important for colonization (15).

Surface virulence factors

elucidate how the variety of sticky organelles, such as type 1 fimbriae, P fimbriae, S fimbriae, and F1C fimbriae, that encourage bacterial adhesion to the host tissue is one of the surface virulence factors of E. coli, seen figure (1) (16).

1. Type 1 fimbriae

Bacteria's surface has filamentous projections called type 1 fimbriae, which are often protein-based. They can form biofilms and encourage targeted binding to receptors on the host's eukaryotic cells, including erythrocytes, urinary glycocalyx, and uroepithelial cells, on abiotic surfaces like plastic catheters. Type 1 fimbriae, the most prevalent adhesion factors in E. coli, are encoded by the fim gene cluster (17).

2. P Fimbriae

The first common components of UPEC are P fimbriae, which are encoded by pap genes and are important in the pathophysiology of ascending UTIs and pyelonephritis in humans (18). Adhesion is their responsibility. A particular galactose disaccharide, present on the

surface of uroepithelial cells in around 99 percent of the population, is bound by P fimbriae. The frequency of this host cell receptor's distribution contributes significantly to susceptibility and explains why some people get E. coli-caused UTIs repeatedly (19).

3. S Fimbriae and F1C Fimbriae

Both of these fimbriae types bind to kidney and lower urinary tract epithelial cells, which is how they are linked to UTIs (20). The E. coli strains that produce ascending UTIs are frequently linked to S fimbriae, which may aid in the spread of bacteria within host tissue (21).

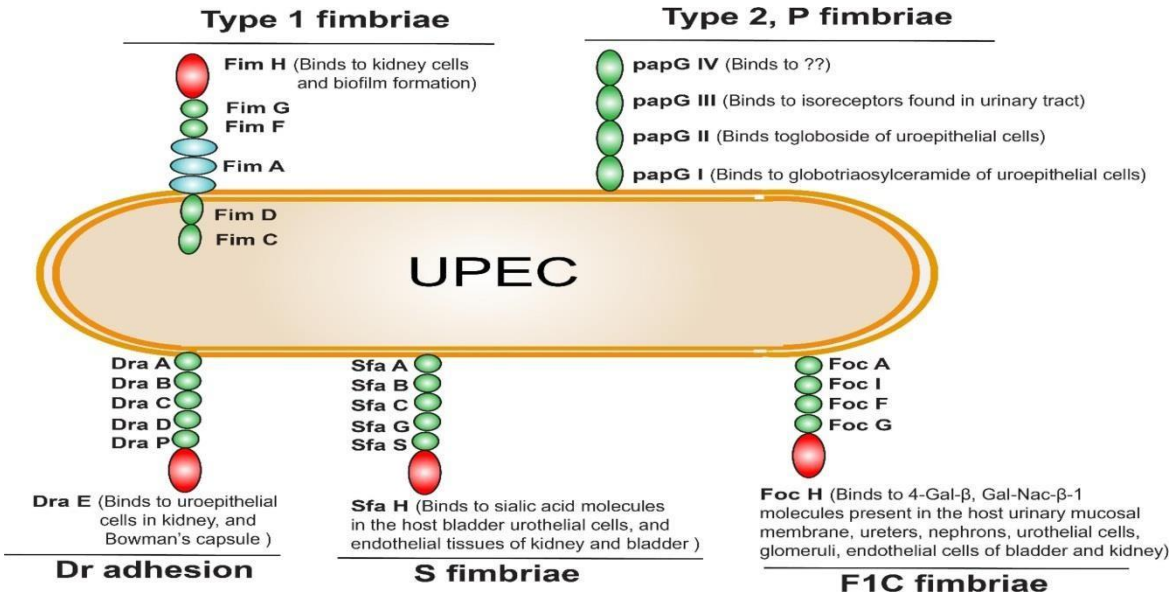


Figure (1): Fimbriae mediated adherence of Type 1 and Type 2, P fimbriae of uropathogenic *E. coli* (22).

4. Capsule

It is mostly covered and protected by a polysaccharide structure called a capsule. Complement and phagocytic engulfment are two host defensive mechanisms that the bacteria from the host immune system, the capsule, protects against (23). Capsular antigens (K antigen) are linked to upper urinary tract infections and aid in the pathophysiology of E. coli UTIs by encouraging bacterial populations in the host that resemble biofilms 24. By reducing complement and antibody binding to the bacterial surface, these capsules may increase bacterial pathogenicity (19).

5. Lipopolysaccharide

Gram-negative bacteria have lipopolysaccharide (LPS) in their outer membrane. The three covalently bound components of LPS are an interior disaccharide with several fatty acids called lipid A, which is what causes the toxicity of Gram-negative bacteria (endotoxin), an outer carbohydrate chain of 1–50 oligosaccharide units called the O antigen, and a core oligosaccharide (25).

6. Flagella

An organelle that is in charge of bacterial movement allows several harmful strains of Escherichia coli to interact with epithelial cells (26). Some studies suggest that E. Coli flagella may be crucial in allowing the bacteria to rise from the bladder and cause kidney infections in people. Seventy to ninety percent of all UTIs are caused by flagellated UPEC, and their pathogenesis involves bacterial interaction with the surface of the urinary tract's epithelial cells (27). Flagella have been proposed to increase bacterial pathogenicity by providing a selection advantage in the struggle for resources in the urine and promoting bacterial dispersal to the upper urinary tract (28).

Secreted Virulence Factors

1. Toxins

Numerous strains of E. coli release toxins that cause damage to the host's tissues or impair the host's immune system, therefore mediating infection (29). About 30–50% of UPEC isolates encode the pore-forming toxin α-hemolysin (HlyA), whose expression has been demonstrated to raise the clinical severity of UTIs (30). Since E. coli's alpha hemolysin also lyses lymphocytes and its beta hemolysin prevents phagocytosis, hemolysin's function is not restricted to red blood cells (19). Its significance as invasive strains may be indicated by the increased frequency of hemolysin-producing strains isolated from urine (30).

2. Protease enzyme

Proteases are thought to be significant virulence factors in infections that increase the invasiveness of organisms that harm host tissue and obstruct the host's defenses against microorganisms. The primary enzymes generated by microorganisms are proteases (31). The best microbial proteases for biotechnological processes are those with desired

properties (32). According to Iqbal et al. (2013), proteases are well-known virulence agents that help numerous infections evade the immune system and survive(33).

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