

Review About General Characteristics of *Escherichiae coli* Bacteria

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Abstract

Uropathogenic *Escherichia coli* (UPEC) identified as the primary causative agent. UPEC is responsible for the majority of both community-acquired and nosocomial UTIs, particularly in catheter-associated urinary tract infections (CAUTIs). The pathogenic success of UPEC is largely attributed to its diverse virulence factors, including adhesins, toxins, siderophores, and its ability to form biofilms. Among these virulence determinants, Type 1 fimbriae and P fimbriae play a critical role in bacterial adhesion to urinary tract epithelial cells and medical devices, facilitating colonization and persistence. Additionally, α -hemolysin contributes to host tissue damage and enhances bacterial survival by releasing essential nutrients such as iron. Iron acquisition systems, including siderophores like aerobactin, further support bacterial growth in the iron-limited environment of the urinary tract. Biofilm formation represents a key adaptive mechanism that enables UPEC to resist antimicrobial treatment and evade host immune responses. This complex, structured community enhances bacterial persistence and contributes to recurrent infections. Understanding the molecular and environmental factors that regulate UPEC virulence and biofilm formation is essential for developing effective therapeutic strategies and alternative interventions to combat antibiotic resistance.

1. Introduction

People of all ages, from birth to old age, can develop a urinary tract infection, which are one of the most common disorders encountered in medicine. Uropathogenic *Escherichia coli* (UPEC) are the major causative agent that could lead to either severe or mild urinary tract infections (UTI). In patients with catheter-associated urinary tract infections (CAUTI), it can be isolated from the urine of around thirty percent of the patients (Macleod and Stickler, 2007). Furthermore, it accounts for about eighty percent of the community-acquired UTIs and fifty percent of nosocomial UTIs.

According to Jacobsen *et al.* (2008), the opportunity for hospitalization, length of stay and antibiotic treatment duration are three times higher in catheterised residents than those not catheterised. In the treatment of community-acquired urinary tract infections, antibiotic resistance is a major health problem (Lartigue *et al.*, 2007). This implies that antibiotic resistance can have impact on severe as well as uncomplicated diseases. As a result of this, it is postulated that uropathogenic *Escherichia coli* isolates possess an array of available virulence factors correlating with the existence UTIs.

Ruiz *et al.* (2002) where siderophores, toxins, vesicles and fimbriae were considered as some of the parameters. Fimbria probably had a significant role in biofilm formation on the surface. Two major adhesins, Type 1 Fimbriae (Fim) and P Fimbriae (Pap - Pilus associated with pyelonephritis), are carried by most pathogenic *Escherichia coli*. These adhesins facilitate UPEC to adhere the urinary tract cells, catheters (Islam, 2008). The highly efficient adhesive organelles type 1 fimbriae are required for attachment to living and non-living surfaces, invasion and persistence in host cells as well as biofilm formation.

In the opinion of **Amalaradjou and Venkitanarayanan (2013)**, as well as **Reisner et al. (2014)** this is identified as an important virulence factor in catheter-induced urinary tract infections and also UTI. P fimbriae are the second-most common virulence factor found in uropathogenic *E. coli* and play a substantial role in the pathogenesis of ascending UTI and pyelonephritis among humans (**Bien et al., 2012**). According to **Snyder et al. (2005)**, UPEC first produces type 1 fimbriae, leading to increased adhesion in the bladder. Expressions of P fimbriae, which serve as adhesion receptors in the upper urinary tract, are next. The Antigen 43 (Ag43) protein, a member of the autotransporter family of surface-exposed proteins, is also has homology to fimbriae and mediates adherence of *E. coli* to epithelium of the urinary tract, and is urovirelence.

It also promotes the growth of the bacteria. According to **Aikman (2009)**, it also makes a major contribution to the aggregation of *E. coli* and the creation of biofilm on the urothelial cells of the host, which results in insufficient clearance, persistence, and increased bacterial resistance to the measures taken by the host to defend itself. It has been found that the development of haemolysin is often related with *E. coli* isolates, particularly those that cause urinary tract infections that are more severe (**Johnson, 1991**).

In addition to being associated with necrotoxicity and cytotoxicity, it liberates nutrients and other substances, notably iron, from lysed erythrocytes, which in turn enhances the proliferation of microorganisms using this method. The majority of bacterial species require iron as an important nutrient in order to perform their functions. Iron is essential for the metabolism of peroxide, Deoxyribonucleic acid (DNA) synthesis, electron transport, oxygen transport and storage, and other processes that *E. coli* must have in order to function properly. Due to the fact that the bacterium requires iron in order to proliferate, it has also been associated with the creation of biofilms for instance. In spite of this, **Johnson (1991)** found that the urinary system contains a minimal amount of this element. Because it is a hydroxamate siderophore, aerobactin is the most potent iron chelator in *E. coli* (**Lee et al., 2007**). Aerobactin is effective in chelating iron.

Therefore, the haemolysin and heme transport systems may be able to utilise the plentiful heme supply of the host as a source of iron in a synergistic manner (**Torres et al., 2001**). Uropathogenic *E. coli* is capable of forming intracellular bacterial communities, also known as (IBCs), which are structures that resemble biofilms that are found within host cells and urinary catheters. In the case of human urinary tract infections, the formation of biofilm by uropathogenic *E. coli* is identified as a virulence factor. Microorganisms in the urinary tract have a greater capacity for pathogenicity and stay alive for longer as a result of this.

On the other hand, according to **Nair et al. (2013)**, there is no direct association between the development of biofilm and any specific virulence factor. *Escherichia coli* is characterised by its general characteristics. *Escherichia coli* were initially recognised as *Bacterium coli*; however, the nomenclature was later changed to *Escherichia coli* in order to honour the person who discovered it according to **Feng and Peter (2002)**. Gram-negative bacilli that are straight and cylindrical in shape and have rounded ends are known as *E. coli*. They range in diameter from (1.1 - 1.5) μm and in length from (2.0 - 6.0) μm (**Mohamed, 2022**). The organisms can be found singly or in pairs, they are mobile with flagella that are peritrichous, and they do not produce spores. Meet the requirements of the generic definition of the Enterobacteriaceae family. At 37 degrees Celsius, the DNA develops in a facultatively anaerobic environment and has a mol% G + C of 48.5-52.1. Both a respiratory and a fermentative metabolism are present in this organism, which is neutrophilic.

According to **Scheutz and Strockbine (2005)**, it is also possible for it to be chemoorganotrophic but anaerogenic. On the MacConkey, lactose fermented rapidly, leaving behind

a metallic green sheen on the EMB agar that was used for differential treatment. By observing the haemolysis of a urine isolate on blood agar, it is possible to determine without delay that the isolate is *E. coli* (Dash *et al.*, 2012). A positive methyl red test and a negative Voges-Proskauer reaction are characteristics of *Escherichia coli*, according to Abdallah *et al.*, (2016).

Additionally, *Escherichia coli* are oxidase negative, create indole, do not ferment citrate, and have a positive methyl red test. (Abbott *et al.*, 2003) They are capable of fermenting a wide variety of carbohydrates, which results in the production of both acid and gas. According to the Kauffman classification, these organisms have a wide range of serotypes, with roughly 700 different serotypes based on somatic lipopolysaccharide (O), capsular (K), and flagellar (H) antigens. The serotype O157:H7 is responsible for the development of haemolytic uremic syndrome. In the majority of cases, *E. coli* is discovered in the commensal normal gut microbiota. Particular strains, on the other hand, have acquired genes that make them susceptible to sickness. According to García-Aljaro *et al.*, (2019), it is also possible to detect it in earth and water, primarily as a result of contamination from faeces.

Variables that play a role in the occurrence of UPEC virulence It is absolutely necessary to have a comprehensive understanding of the properties that allow UPEC to successfully colonise its host in order to be able to properly combat this virus through the development of vaccines or alternative therapeutic options. There have been a great number of virulence factors discovered, in addition to adhesins, capsules, siderophores, and poisons (Johnson, 2017).

2. Type 1 Fimbriae (T1F)

Fimbriae-mediated adhesion is a necessary condition for the pathogenicity of *E. coli* in the urinary system. According to Johnson (1991), uropathogenic *E. coli* strains have the ability to express a wide variety of fimbrial adherence factors. These factors include P Fimbriae, S Fimbriae, Afa/Dr Fimbriae, and type 1 fimbriae. It is necessary for pathogenic microorganisms to attach themselves to particular host cells in order to initiate an inflammation. According to Proft and Baker (2009), the pathogen's tendency to gravitate towards tissues that have certain surface receptors is controlled by diverse adhesion molecules that are located on the surface of different bacterial cells. According to Lindhorst (2011), bacteria that live in a wide range of habitats make use of type 1 fimbriae as extremely efficient adhesion organelles that encompass both biotic and abiotic surfaces.

E. coli, *Klebsiella pneumoniae*, *Pseudomonas aeruginosa*, *Salmonella spp.*, *Serratia marcescens* and *Shigella flexneri* all belong to the Enterobacteriaceae family where they are primarily isolated from. According to Jones *et al.*, (1995), the synthesis of type 1 fimbriae is mediated by the chromosomally located *fim* gene cluster. This locus comprises nine genes, from (*fim A* to *fim I*), which include, (*fim* operon) and an invertible region; (*Fim H*) the adhesion subunit, and minor adapter subunits (*Fim F*) and (*Fim G*) are associated with the formation of a short linear tip fibrillum. The type 1 pilus rod the number of contacts between each (*Fim A*) subunit that belongs to a right-handed helical rod array representing each type 1 pilus, and the numbers of (*Fim A*) subunits per type 1 pilus are in an unsafe range generally due to its major component, (*Fim A*) subunits. (*Fim E*) and (*Fim B*) are independent transcription units that encode recombinases. In contrast, (*Fim C*) and (*Fim D*) are well described in pilus formation. The (*Fim I*) subunit role has not yet been specified, although it seems to be important for the synthesis of *E. coli* type 1 pili. According to Jonson *et al.* (2005) findings; type 1 pili are detected in over 90% of all uropathogenic *E. coli* identified.

2. α -Hemolysin (α -toxin or Hla)

A pore-forming exotoxin produced primarily by the bacterium *Staphylococcus aureus*, It functions as a key virulence factor by binding to the membranes of host cells such as red blood cells, epithelial

cells, and immune cells where it oligomerizes into a heptameric structure that forms transmembrane pores. These pores disrupt cellular ion balance, compromise membrane integrity, and ultimately lead to cell lysis or death. Also, plays a significant role in tissue damage, inflammation, and the pathogenesis of various infections, including skin infections, pneumonia, and sepsis (Tam *et al.*, 2019).

The α -Hemolysin virulence factor is a protein cytotoxin that is extracellular and cytolytic and secreted by many haemolytic *E. coli* strains. It is released by Gram-negative bacteria such as *Escherichia*, *Proteus*, *Morganella*, *Pasteurella*, *Actinobacillus* and *Bordetella* indicating its horizontal gene transfer acquisition (Burgos *et al.*, 2009). Named HlyA, also called α -Hemolysin, it is named due to its ability to lyse erythrocytes. This forms the basis of its best known assay with dramatic, evident zones of haemolysis around colonies on blood agar. The production of haemolysin is associated with human pathogenic genotypes in *E. coli*.

This is especially true for those genotypes that are accountable for the more severe clinical symptoms of urinary tract infections (Johnson 1991). It has been established that haemolysin is associated with necrotoxicity and cytotoxicity, both of which promote the proliferation of microorganisms by releasing nutrients and chemicals, including iron, from erythrocytes that have been lysed. As a consequence of this, the permeability of the erythrocyte membrane to Ca^{2+} , K^{+} , mannitol, and sucrose is increased. In addition to lysis of erythrocytes, haemolysin is extremely harmful to a wide range of host cells, which can lead to an increase in inflammation, damage to tissue, and a reduction in the host's ability to defend itself. It is possible that this harmful activity will make the renal impairment that is seen in pyelonephritis even worse. According to Coote (1996), α -hemolysin predominantly impacts the cells of the immune system of the host without causing any disruption to the cells themselves. However, it does drastically diminish the functionality of these cells.

3. Bacterial Biofilm

The biofilm of bacteria the term "**bacterial biofilm**" refers to a three-dimensional community of aggregated cells that are encased in an exopolysaccharide matrix that have been created by the bacteria themselves. The research conducted by Zalewska-Piateka *et al.*, (2013) demonstrates that this matrix has the ability to cling permanently to biotic or abiotic surfaces, as well as the air-liquid interface of liquid environments. When Antonie van Leeuwenhoek used a rudimentary microscope in 1674 to describe clumps of "animalcules" that were gathered from the surfaces of human teeth, she made the observation that biofilm production was taking place. The term "biofilm" gained widespread acceptance in the year 1984. The maturation of planktonic cells into a complex, multicellular structure is the process that results in the formation of biofilms, which can extend to depths of hundreds of microns. It is possible for biofilms to be formed as a consequence of the interaction between environmental stimuli and the genetic components of bacteria.

Environmental characteristics that have an effect on initial attachment include osmolarity, pH, nutritional circumstances, iron availability, oxygen tension, and temperature (Davey and O'Toole, 2000). Environmental parameters also include temperature. Research has established a connection between quorum sensing (QS) molecules and the regulation of biofilm growth. It is now widely acknowledged that quorum sensing is an essential component in the process of controlling the formation of biofilms. In the course of current molecular and genetic research, a great number of genes have been discovered, and the products of these genes are absolutely necessary for the composition of biofilms. According to Danese *et al.*, (2000), there is a correlation between the

adhesive requirements of the population and the differential gene expression that occurs between the two states of bacteria (either planktonic or sessile).

The formation of biofilm is a process that begins with five distinct stages. Planktonic cells are initially moved from the bulk liquid to the surface through the use of physical forces, chemotaxis, or bacterial appendages such as flagella. This allows for a connection that can be reversed during the process. The reversible adhesion of bacteria is affected by a wide variety of characteristics, such as the availability of nutrients, the qualities of the surface, the orientation of the bacteria, the concentration of iron, the temperature, and the oxygen levels. Type 1 pili, curli fibres and antigen 43 of *E. coli* that mediates firm adherence are among the contributing factors to promoting mediatory bacterial contacts (Cegelski *et al.*, 2009).

Polysaccharide synthesis is the mechanism leading to the production of the outer matrix. This matrix not only confers structural integrity, but also enables surface colonisation as well as adhesion and co-aggregation within the host. Among the *E. coli* matrix constituents are cellulose, polyglucosamine and colonic acid. The matrix may also include calcium ions, nucleic acids, proteins, surfactants, glycolipids and membrane vesicles. According to Soto, (2014), slime, as the matrix that describes this matrix comes to be referred to be responsible for more than 90% of the biomass.

Biofilm development the establishment of a spatial structure with 3D macrocolony formation is due to bacteria interactions, which produce an environment that varies significantly from that of planktonic cells (Beloin *et al.*, 2008). Bacteria don't actually grow biofilms, per se, they form over time when bacteria touch each other. In line with Zalewska-Piateka *et al.* (2013), biofilm microcolonies are distributed to various locations through aquatic conduits in which nutrients are transported and wastes expunged.

So in other words, for cells to get biofilm in new environs, they'd have to detach from the biofilm and then revert back to planktonic. The detachment of bacteria is believed to be caused by both active and passive means, such as enzymatic destruction, the action of environmental factors (shear forces and erosion etc.). The phenomenology is believed to involve both flavors of the mechanism. Soto, (2014) states that biofilm detachment is an essential step in the transmission of many bacterial taxa from the environment to humans, host-to-host, and within individuals (i.e., body sites), thus enabling infection establishment. Nearly of 99 percent of all bacteria on Earth can be found in biofilms ranging from recently established to more mature structures. The reprogramming of the expression of genes occurs in every phase under the influence of environmental signals. This is a requirement for each stage. This programme of cellular reprogramming influences the expression of genes in biofilms, which also impacts the expression of surface molecules, nutrient usage as well as virulence factors. According to Kostakioti *et al.*, (2013), it allows bacteria to acquire characteristics that facilitate survival in an environment that is hard to colonize, and therefore hard for the organism to survive on.

4. Conclusion:

Uropathogenic *Escherichia coli* remain the leading cause of urinary tract infections due to its remarkable ability to adapt, colonize, and persist within the urinary tract. This capability is mediated by a wide range of virulence factors, particularly adhesins such as Type 1 fimbriae, toxins like α -hemolysin, and efficient iron acquisition systems. The formation of biofilms significantly enhances bacterial survival by providing protection against antibiotics and host immune defenses, thereby contributing to chronic and recurrent infections. The increasing prevalence of antibiotic resistance among UPEC strains further complicates treatment and highlights the urgent need for alternative therapeutic approaches. Overall, the interaction between bacterial virulence factors and host

environmental conditions plays a crucial role in the pathogenesis of UTIs. A deeper understanding of these mechanisms is essential for improving prevention, diagnosis, and treatment strategies.

5. Recommendations

- **Development of Alternative Therapies:** There is an urgent need to explore non-antibiotic treatment strategies, such as anti-adhesion therapies, bacteriophage therapy, and plant-derived antimicrobial compounds, to combat antibiotic-resistant UPEC strains.
- **Targeting Virulence Factors:** Future research should focus on inhibiting key virulence factors such as fimbriae, hemolysin, and siderophore systems, which are essential for bacterial colonization and survival.
- **Biofilm Control Strategies:** Novel approaches should be developed to prevent or disrupt biofilm formation, especially on urinary catheters, including surface modifications and anti-biofilm agents.
- **Improved Infection Control in Healthcare Settings:** Strict catheter management protocols and hygiene practices should be implemented to reduce the incidence of catheter-associated urinary tract infections (CAUTIs).
- **Surveillance of Antibiotic Resistance:** Continuous monitoring of antimicrobial resistance patterns is necessary to guide appropriate antibiotic therapy and reduce treatment failure.
- **Further Molecular Studies:** Advanced molecular and genomic studies are recommended to better understand UPEC pathogenesis and identify new therapeutic targets.

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