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# KLEBSIELLA PNEUMONIAE: THE STEALTH PATHOGEN

**Arunima Ghosh[1], Kingshuk Mandal[2], Sanchaita Bera[3], Souparno Paria[5], Moulima Maity[4]***

[1]Laboratory Technician, [3]Laboratory Assistant, [2,4,5]Assistant Professor, [1,2,3]Department of Medical Laboratory Technology, Haldia Institute of Health Sciences; [4]Department of Nutrition, Haldia Institute of Health Sciences; [5]Haldia Institute of Pharmacy

Haldia, Purba Medinipur, 721657, West Bengal, India

***Corresponding Author: Moulima Maity**

Department of Nutrition, Haldia Institute of Health Sciences, Haldia, Purba Medinipur, 721657, West Bengal, India. **DOI:** https://doi.org/10.5281/zenodo.20963729

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## ABSTRACT

*Klebsiella pneumoniae* - gram-negative, non-motile bacteria which is fit in to the family of Enterobacteriaceae; found in the human digestive tract. This bacterium is significant adaptable pathogen and responsible for a wide range of infections such as pneumonia, UTI, septicaemia and wound infections, predominantly in immune-compromised persons and hospitalized patients. This organism is characterized by noticeable polysaccharide encoded capsule that develops virulence by protecting against phagocytosis and host immune responses. Right now, Klebsiella has gained attention due to the emergence of multidrug-resistant (MDR) and highly virulent strains, specifically producing broad-spectrum β-lactamases (ESBLs) and carbapenems. This resistance systems severely limit therapeutic options and main challenge to worldwide public health management. Transmission normally happens via unclean hands, medical instruments, and hospital circumstances. First diagnosis, suitable antimicrobial treatment, and exact adherence to hygiene performs are critical in managing infections caused by *Klebsiella*. Current research focuses on new therapy strategies, with different antibiotics, bacteriophage therapy procedure, and vaccines, to fight increasingly resistant pathogen.

**KEYWORDS:** *Klebsiella pneumoniae*, capsular polysaccharide, Biofilm formation, virulence-associated genes, gene cluster, glucocorticoid therapy.

## INTRODUCTION

An opportunistic pathogen that can cause a number of infections is *Klebsiella pneumoniae.*[1] Edwin klebs discovered *Klebsiella pneumoniae* in the airways of patients in 1875, but in 1882, Carl Friedlander, a German pathologist and microbiologist made a significant contribution to figure out the bacterial aetiology of pneumonia,[2] because of this *Klebsiella pneumoniae* was also known as Friedlander's disease for a while.[3] *Klebsiella pneumoniae* is a facultative anaerobe that is encapsulated and non- motile.[4] This bacterium belongs to Enterobacteriaceae family of bacteria.[5] Klebsiella species are widely found in nature, including soil, water and animals and can invade medical equipment and the healthcare environment.[6,7] In individuals with impaired immune systems or those who are regularly exposed to healthcare, *K. pneumoniae* has historically been known to cause pneumonia, UTIs, and bacteraemia.[1] In addition, *K. pneumoniae* has known to cause serious infections that are acquired in the community, including pyogenic liver abscess, community-acquired pneumonia, and meningitis infection.[1] It is responsible for roughly one-third of all Gram-negative infections.[8] In humans, Klebsiella frequently colonizes the digestive and nasal systems without producing any symptoms. However, when the host immunity is unable to stop the pathogen's growth, the colonization might become an infection. Patients with diabetes, those on glucocorticoid therapy and recipients of organ transplants are some examples of this.[9] This review will provide an overview of *Klebsiella pneumoniae* biology pertaining to its epidemiology, virulence factor, virulence-associated genes and control strategies.

## METHODOLOGY

We searched the databases PubMed and Google Scholar by using the following keywords in different combinations: "*Klebsiella pneumoniae*", "community-

acquired pneumonia", "Efflux pump", "capsular polysaccharide", "gene cluster", "Virulence", "colonization", "Serum resistance", "gram-negative bacteria", "Urinary tract infections", "mucoviscosity related genes", "control strategies", "glucocorticoid therapy" , "Biofilm formation", "virulence-associated genes", "diabetes", "immune response", "gastrointestinal tracts infections" and "Ayurveda" (last search conducted in MAY 2026). In our review paper, we focused our attention on *Klebsiella pneumoniae* – a gram-negative bacterium which creates various diseases, its characteristics, control strategies, immune response, virulence associated factors, epidemiology. Studies that were available in languages except English were excluded.

## VIRULENCE-ASSOCIATED FACTORS OF *Klebsiella pneumoniae*

*Klebsiella pneumoniae* has to astound the innate and humoral immunity[10] of host defence systems to move in and colonize inside the host. The virulence factors that *Klebsiella pneumoniae* uses to disseminate pathogenicity are listed below.

**Adhesion:** There are two types of fimbriae in *Klebsiella pneumoniae*: type1 and type 3. Urinary tract infections in *K. pneumoniae* are caused by the virulence protein type 1 fimbriae. Adhesion to mannose-containing sites on the host cell and in the host's extracellular matrix is mediated by type 1. The production of biofilms is significantly influenced by type 3 fimbriae.[11]

**Biofilm mediated colonization:** When pneumoniae are found in large biofilms, the entire mass of bacteria in the biofilm acts as a single entity due to their complex, unexplained signalling mechanism. They also have a comprehensive and virulent mechanism to infect the host and protect themselves from different antibacterial agents, which is a common occurrence in drug resistant conditions.

Biofilm deposition on medical equipment is a significant contributing factor to acquired bacterial resistance, which frequently results in nosocomial infections that are challenging to cure. *K. pneumoniae* medical isolates express type 3 fimbriae, thin, non-channelled belongs to chaperone- usher class of fimbriae.[12] By facilitating adherence to host tissues and abiotic surfaces, these fimbriae play a crucial role in virulence by encouraging the creation of biofilms. Type 3 fimbriae are therefore essential to the pathophysiology of infections linked to biofilms.

The type 3 fimbriae are encoded by the mrk gene cluster, which is made up of numerous essential genes that act synergistically to maintain their structural integrity and functionality. MrkA, MrkB, MrkC, MrkD and MrkF genes are associated with this cluster.[13] The mrkA gene codes for the major fimbrial subunit. The mrkB gene acts as a chaperone. The mrkC gene assist in the fimbrial assembly and export. The mrkD gene facilitates attachment to non-living surfaces and host tissues and mrkF gene contributes to stability and proper assembly.[13]

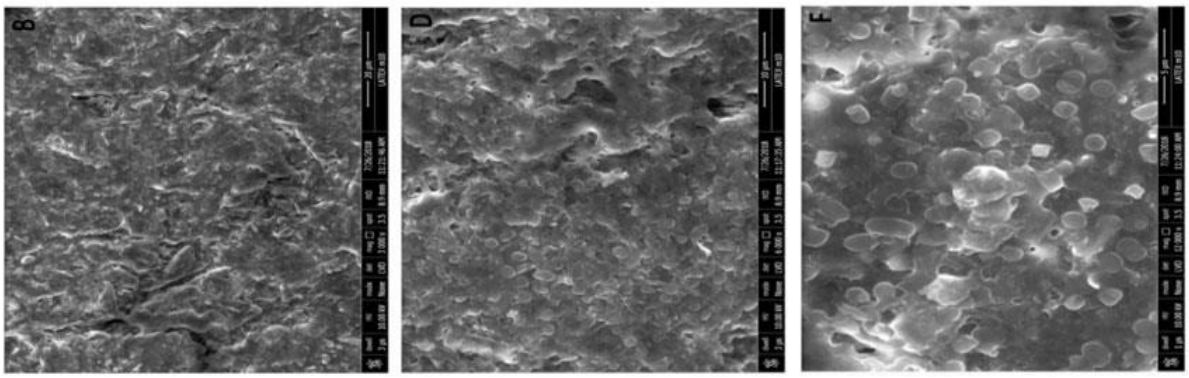

**Figure 1: SEM images of strong biofilm formed on the surface of silicon coated latex catheter.[14]**

**Efflux system:** The channels that the bacterial system created to get rid of antibiotics, dyes and detergents as well as aid in the development of bacterial resistance are called efflux pumps. Some *K. pneumoniae* strains have the AcrAB multidrug-resistant efflux mechanism, which is encoded by the acrRAB operon.[15]

**Evasion of host immune response:** The coating of acidic sugar polymers known as the capsular polysaccharide, which surrounds *K. pneumoniae*, is crucial to its pathogenicity because it provides defence against compliment-mediated destruction and macrophage phagocytosis.[16,17] The strains of *Klebsiella pneumoniae* that manufacture capsules range in serotype from k1 to k78. K1 and K2 serotype-containing capsules are more pathogenic.[11]

**Siderophore-mediated iron acquisition:** Iron is essential for *Klebsiella pneumoniae* to remain viable throughout an infection. However, it is difficult to retrieve iron from the host. In the host iron is present as a protein, transferrin. To acquire iron, bacteria like *Klebsiella pneumoniae* require the synthesis of specialized substances called siderophores. *K.*

*pneumoniae* generates a variety of siderophores, such as aerobactin, salmochelin, enterobactin and yersiniabactin (Ybt). These siderophores facilitate iron uptake in *K. pneumoniae* from the host.[11]

**Table 1: Genes blameable for the Virulence factors of *K. pneumoniae.***

| Virulence factor | Virulence gene | Reference |
|---|---|---|
| Hypermucoviscous phenotype and mucoviscosity related genes | rmpA, rmpA2, allS, wabG | [18] |
| Biosynthesis of lipopolysaccharide | Uge, wcaG | [19] |
| Iron uptake and transport | iutA, icuA, iroN, iroB, ybtA, irp2, kfu, entB | [19,18] |
| Adhesion | Cf29a, fimA, fimB, fimC, fimD, fimE, fimF, fimG, fimH, fimI, fimK | [20,19,18] |
| Efflux pump (AcrAB) | acrA; acrB | [11] |
| Biofilm formation (Type 3 fimbriae) | mrkA, mrkB, mrkC, mrkD, mrkF, mrkH, mrkI, mrkJ | [11] |
| Serum resistance | glf; kfoC; wbbM; wbbN; wbbO; wzm; wzt; uge; wabG | [11] |

## EPIDEMIOLOGY

In nature, Klebsiella species are widely distributed and frequently found in soil, water and other surfaces.[21] *K. pneumoniae* frequently colonises human mucosal surfaces, such as the gut and upper respiratory tract, where colonisation rates differ greatly between people depending on their exposures and habitat.[7,21,22] According to research, the incidence of Klebsiella colonisation varies between 5 and 35% in western nations and between 18.8 and 87.7% in Asia.[23,24] Klebsiella carrier frequencies in non-hospital settings vary from 5 to 38% in faecal samples and from 1 to 6% in nasopharyngeal samples.[6,25] Colonisation rates in hospitalised individuals might reach 77% in the gastrointestinal tract and 19% in the nasopharynx.[6,26]

*K. pneumoniae* is found as a saprophyte in the digestive tract and nasopharynx of humans. Carrier rates vary significantly between studies. The nasopharynx has a detection rate of 1 to 6 %, whereas stool samples have a detection rate of 5to 38%.[27-30] Klebsiella sp. are rarely identified on human skin and are thought of as merely passing members of the flora[31] since gram-negative bacteria do not find favourable growth conditions there.[32]

In a hospital setting, where colonisation rates rise in direct proportion to length of stay, these carrier rates are significantly altered. There are higher incidences of Klebsiella carriage among hospital staff as well. [33-35] The gastrointestinal tracts of patients and the hands of hospital staff are the main sources of Klebsiella transmission in a hospital setting[36], aside from medical equipment (infected by improper sanitary practices) and blood products.[29,38,39]

It is important to remember that several steps have been suggested to stop Klebsiella from spreading nosocomial. The spread of nosocomial Klebsiella infections can be stopped by strictly adhering to fundamental epidemiological guidelines for the treatment of urinary catheters, intravenous tracheostomies, and wounds; maintaining and caring for equipment; and practicing excellent hand washing.[35] Regulating the use of antibiotics in hospitals to avoid overuse and abuse is another way to control klebsiella infections. Additionally, in order to gather information for prevention and management of nosocomial klebsiella infection rates, nosocomial infection surveillance is essential.[6]

## CONTROL STRATEGIES

*K. pneumoniae* infections can be successfully prevented by locating and getting rid of their source. However, locating and eradicating the source of infection still presents significant obstacles. The majority of hospitals still primarily use specimen culture to check for K. pneumoniae.[6] According to certain reports, chromosomal genes including blaSHV, blaLEN, blaOKP and their side-chain genes (deor) can be identified using molecular techniques using multiplex polymerase chain reaction.[40]

To stop *k. pneumoniae* from spreading from the source, extensive screening, identification, education and multimodal intervention are crucial.[41] By promptly identifying those who are infected and taking basic precautions, such as wearing masks, gloves, exposure avoidance should be carried out. In both hospital and community settings, contact tracing should be used whenever feasible to reduce additional exposure to uninfected people. For medical professionals, hand hygiene education is equally essential.[42-44]

Strict criteria and principles should govern the administration of antibiotics, particularly for first empirical.[45,46] Antibiotics should be used rationally and consistently, which includes specific indications, an appropriate dosage, a long enough course of treatment, prudent antibiotic changes, and other interventions such surgical drainage and implant removal, if feasible.[47]

The polysaccharides that cover *Klebsiella pneumoniae*, such as the capsule and lipopolysaccharides, are perfect

candidate antigens that can be targeted by host immunity or vaccines.[6] There have been difficulties in the development of antibodies and vaccines against capsular polysaccharides as *K. pneumoniae* contains nine distinct LPS serotypes and a total of 77 distinct capsules.[48-50]

A healthy lifestyle, such as regular exercise, getting enough sleep, quitting smoking and eating a diet rich in fruits and vegetables can boost immunity.[51] Alternative methods should be investigated because of the high level of resistance to a variety of antibiotics. A novel treatment approach that can either enhance or replace conventional antibiotics is bacteriophage therapy.[52,53]

## ANTIBIOTIC THERAPY

When treating pneumonia cases, it is recommended to adhere to established antibiotic treatment guidelines because *K. pneumoniae* is uncommon in the general population. The antibiotics used should be customised to the particular sensitivities shown in the local area after a *K. pneumoniae* infection is suspected or confirmed. Currently, a 14- day course of either a respiratory quinolone or a third or fourth- generation cephalosporin as a monotherapy is advised for treating community – acquired *K. pneumoniae* infections. It is also possible to combine one of these regimens with an aminoglycoside. A course of aztreonam or a respiratory quinolone is the best option if the patient has a penicillin allergy.[54-56] Carbapenem therapy is advised after ESBL diagnosis because to its broad efficiency. Infectious disease specialists should be consulted for treating CRE (Carbapenem-resistant Enterobacteriaceae). Treatment options for CRE include dual therapy with carbapenem, tigecycline, fosfomycin, aminoglycosides and antibiotics from the polymyxin class. When two or more of these medications are used together in a treatment plan, death rates may be lower than when monotherapy is used alone.[57]

## CONCLUSION

In this article we may say that with developing antibiotic resistance *K. pneumoniae* is a foremost healthcare related pathogen. Rigorous lung, urinary and bloodstream infections are happened by it. *K. pneumoniae* is prevented by maintain the hygiene, use the antibiotic. Observation is the main key to control its pathogenic spread.

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