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Klebsiella pneumoniae with its weaponry of virulence determinants - A comprehensive review

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Abstract:
Multidrug-resistance (MDR) and Hypervirulent *klebsiella pneumoniae (*hvKp*)* has created a global menace among clinicians as the former leads to therapeutic failure and the later invasive virulent variant causes community acquired infections. The pathogenic rivalry of *Klebsiella pneumoniae* is attributed to a diverse repertoire of virulence factors which facilitates adhesion, colonization, immune evasion, iron acquisition and biofilm formation. Understanding the pathogenic interplay and molecular basis of these virulence determinants is necessary for developing novel therapeutic interventions and for upgrading infection control measures. Hence, the significant virulence factors of *Klebsiella pneumoniae* and their role in bacterial pathogenicity. Thus, it sheds light on specific genes coding for virulence with a special mention about the prevalence of hypervirulent *K. pneumoniae* (hvKp) and its diagnostic determinants.

Keywords: Multidrug resistance (MDR), global menace, virulence determinants, therapeutic strategies, infection control, hypervirulent *K. pneumoniae (*hvKp*)*

Background:
Klebsiella pneumoniae, a gram-negative bacterium belonging to the family *Enterobacteriaceae*, is ubiquitously found in the environment and as a commensal colonizing skin, oropharynx and gastrointestinal tract of healthy humans and animals. It belongs to the normal gut microbiota but can gain colonization of other sites, such as the respiratory tract, urinary tract and bloodstream, causing pneumonia, UTI, bloodstream infection, *etc.* **[1]**. Though considered commensals, they tend to cause opportunistic infections in people with weakened immune systems/underlying medical conditions, resulting in fatal conditions **[2]**. It has become increasingly antibiotic-resistant, limiting treatment options. However, its emergence and spread are taking a toll on the healthy population in community settings, as the incidence of hypervirulent (hvKp) strains is being more commonly reported from Southeast Asian countries with severe outbreaks of invasive infections **[3]**. These clinical strains vary based on the presence or expression of the virulence factors in addition to their resistance profile**.** Therefore, it is of great clinical significance to describe the virulence determinants of such a pathogen, which has been declared a priority pathogen by the WHO **[4].** The pathogenicity of *K. pneumoniae* bacteria is associated with various types of virulence factors that enhance its invasion, colonization and enable it to evade host defense **[5]**. With the advancements in molecular testing and next generation sequencing, extensive knowledge on the virulence traits of *Klebsiella pneumoniae* have been well made by integrating genomic, transcriptomic and surrogate host models **[6]**. These advancements have shifted our understanding from observing basic traits to mapping the complex regulation, genetic plasticity and evolutionary convergence of highly invasive, multidrug-resistant strains **[7]**. Therefore, it is of interest to describe the virulence determinants with their pathogenic role in infectious diseases and their molecular analysis which further instigates our search for novel innovations in prevention, diagnosis and treatment against these virulent strains**.**

Virulence factors and their role in the pathogenesis of *K. pneumoniae*:
Numerous factors that contribute to the pathogenicity of *Klebsiella pneumoniae* are as follows: Adhesive fimbriae, capsule, lipopolysaccharide (LPS) associated with mucoviscosity, production of siderophore/iron carriers and Biofilm formation. The more recently identified virulence factors include porins, OMP's, mechanism of efflux pump, transport of iron and genes involved in allantoin metabolism **[8]**.

Adhesive fimbriae:
K. pneumoniae is composed of two important types of fimbriae, type 1 encoded by fim AICDFGHK genes and type 3 by mrk ABCDF, which facilitates the adhesion of bacteria to the biotic (human host urothelium) and abiotic surfaces (medical devices, urinary catheter) and initiates colonization, biofilm formation and bacterial invasion **[9]** thereby eluding the host defense mechanism. Type 1 fimbriae, coded by fim H gene, expressed in 90% of *K. pneumoniae* are responsible for UTIs by invading the urothelium and subsequently form biofilm **[10]**. Type 3 fimbriae, containing the mrk gene, are expressed during biofilm formation on abiotic surfaces such as indwelling catheters and endotracheal tubes, facilitating Ventilator-Associated Pneumonia **[11]**. Hence, the expression of type 3 fimbriae is a prognostic feature of device-associated and healthcare-associated infections caused by *K. pneumoniae*. A non-fimbrial adhesin encoded by the Ycfm gene also enables adhesion to the host cells and form biofilm.

Capsular polysaccharide and hypermucoviscosity:
Capsule guards the bacteria from opsonophagocytosis and other host-immune responses. It is hydrophilic in nature, favorable for mucoid colonies on agar plates. Based on antigenic variations in the capsular polysaccharide, *K. pneumoniae* has been classified into 79 capsular (K) types with different serological profiles. But recent advancements with whole-genome sequencing have expanded its classification to more than 180 capsular locus (KL) types. Certain capsular serotypes, particularly K1, K2, K5, K20, K54 and K57, are strongly associated with hypervirulent strains

and severe invasive infections. The extensive diversity of capsule types contributes to immune evasion, epidemiological variation and challenges in vaccine development **[12]**. Mucoviscosity-associated gene A (MagA) is associated with capsular serotype K1 and capsule-associated gene A (K2A) is confined to serotype K2, which are frequently reported in liver abscess **[13]**. Regulator of mucoid phenotype A (rmpA) gene is a plasmid-mediated gene coding for synthesis of capsular polysaccharide **[14]** and has been notably reported in enhancing capsule production in high–mucus phenotype of hypervirulent *K. pneumoniae* (hvKP), making it the most important molecular markers of hypervirulence in *K. pneumoniae*. Previous reports indicated that hvKp strains are hyperviscous due to overproduction of capsules and mediate their role in UTI infections **[15]**. Carbapenem-resistant hypermucoviscous *K. pneumoniae* reports a high mortality rate among the developing countries, indicating that hypermucoviscosity is one of the most potent virulence factors of *Klebsiella* species **[16].** Based on the research studies, CPS has also been found as a promising vaccine candidate with its immunomodulatory characteristics and capsule-targeting antibodies are effective in killing *K. pneumoniae* **[17]**. Recent studies suggest that CPS-based conjugate and bi conjugate vaccine instigate robust antibody responses against both classical and hypervirulent *K. pneumoniae* strains. Moreover, capsule-specific monoclonal and polyclonal antibodies have shown effective binding to capsular antigens, enhancing complement activation, opsonophagocytosis and bacterial clearance. These findings support the development of capsule-targeted immunotherapeutic strategies and multivalent CPS-based vaccines as potential approaches to combat multidrug-resistant *K. pneumoniae* infections.

Lipopolysaccharide (LPS)

Lipopolysaccharide (LPS) is an important component of the outer membrane of *Klebsiella pneumoniae* and contributes significantly to bacterial virulence, immune evasion and survival within the host. It is made up of O antigen which is encoded by the wb gene cluster and core oligosaccharide and lipid A which are encoded by the waa and lpx gene clusters, respectively. The O antigen of LPS underpins the retention of the capsule and has been researched as an attractive vaccine candidate **[18]**. The biosynthesis and structural integrity of LPS are dependent on the uridine diphosphate galacturonate 4-epimerase (uge) gene, which encodes an enzyme involved in the formation of the LPS core oligosaccharide. The uge gene catalyzes the conversion of UDP-glucuronic acid to UDP-galacturonic acid, a crucial precursor required for proper LPS assembly. Studies have demonstrated that disruption of the uge gene results in defective LPS and capsule production, leading to marked attenuation of virulence. Experimental infection models have shown that uge-deficient mutants are unable to establish urinary tract infections and exhibit significantly reduced pathogenicity in pneumonia and septicemia models. Recent investigations have further revealed that expression of the uge gene is regulated by iron availability through the ferric uptake regulator (Fur), highlighting its importance in the adaptation and virulence of *K. pneumoniae*. Thus, the uge gene plays a critical role in LPS biosynthesis and contributes substantially to the organism's ability to cause urinary tract infections, pneumonia, bacteremia and sepsis **[19].** In fact, LPS in the major strains, with some modification in part, circumvents the host immune system and in other strains, their capsular K antigen camouflages LPS to avoid detection by toll-like receptor 4 (TLR4), thereby failing to activate complement and inhibiting the inflammatory response **[20]**. A recent study also proved that O1 antigen provides resistance to complement-mediated killing by polymerizing the C9 molecule in the MAC complex, facilitating their increased survival in serum **[21]**. Hence, LPS-based vaccines may act as a promising immunotherapeutic agent in limiting this superbug soon.

Siderophore production:

Siderophores are iron chelators of KP, through which they can steal iron from the host's iron chelating proteins, much needed for its survival, growth and replication. *K. pneumoniae* secretes siderophores, namely enterobactin (ent), salmochelin (iro), yersiniabactin (ybt) and aerobactin (iuc), which increase its growth efficiency, favoring it to overcome neutralization by the host. Enterobactin is the chief siderophore acquired, which gets inhibited by Lipocalin2, a host antimicrobial peptide produced by neutrophils during infection. Since, Yersiniabactin and salmochelin are lipocalin-2–resistant siderophores that facilitate iron acquisition during infection; their ability to evade host immunity enhances the survival of *K. pneumoniae* within host tissues, particularly the respiratory tract. Yersiniabactin-positive isolates are significantly more prevalent in respiratory infections than in blood, urine, or stool isolates, underscoring their role in pneumonia pathogenesis and tissue-specific virulence **[22]**. Salmochelin, encoded by the iroA gene cluster, is one of a hallmark siderophore of hypervirulent *K. pneumoniae* and is frequently detected in strains responsible for severe community-acquired infections such as pyogenic liver abscess, pneumonia, bacteremia and metastatic invasive disease **[23]**. Studies indicate that hvKp strains secrete large quantities of siderophores, compared with classical strains, specifically aerobactin, detected by iutA gene. Novel drugs based on iron acquisition systems and vaccines targeting siderophores, are expected to provide new treatment insights against hvKp phenotype **[24]**. The characteristic traits of virulence genes and their distribution were illustrated in detail in **Table 1**

Further determinants of pathogenic potential:

Besides these significant virulence determinants, outer membrane proteins, porins, efflux mechanism of antibiotic resistance and nitrogen utilization serve as additional pathogenic traits in the clinical events of *Klebsiella pneumoniae*. OMP of *Klebsiella* plays a key role in bacterial meningitis by facilitating blood-brain barrier penetration and influences pulmonary pathogenesis by inducing apoptosis and flourishes biofilm formation. Furthermore, OmpK35 and OmpK36 turn on hydrophilic molecules inside the bacterium. The presence of AcrAB-TolC multidrug efflux system exports antibiotics and

host-derived antimicrobial compounds, significantly contributing to both antimicrobial resistance and virulence. Colibactin, a toxic substance encoded by the pks gene cluster, reported with enhanced virulency, is associated with host DNA damage and has been detected as a significant marker in bloodstream infections (BSIs) **[25]**. A more recent study reported that *K. pneumoniae* meningitis remains associated with significant mortality and poor clinical outcomes, particularly when caused by carbapenem-resistant and hypervirulent strains. They highlighted the prevalence of hvKp producing kpc-2 gene causing mortality among the Asian population **[26].** The Klebsiella ferric iron uptake (*Kfu*) gene, a key gene involved in the acquisition of iron, is most often reported with hypervirulent strains, indicating its enhanced pathogenicity. The *Kfu* gene has also been linked to capsule development, hypermucoviscosity and purulent tissue infections. Besides its virulence trait, the *Kfu* protein also serves as a potential therapeutic target, as humans do not possess similar ABC transporters **[27]**. Research has also proved the presence of the *Kfu* gene influences the susceptibility of *K. pneumoniae* to certain antibiotics, such as CFDC, ceftiofur hydrochloride **[28].** The presence of allantoin metabolic genes, particularly the (allS) activator, enhances the bacteria to avail carbon and nitrogen from the external environment and is associated with virulence in *K. pneumoniae*, especially in the context of hvKp strains linked to liver abscesses.

Table 1: Characteristic traits of virulence genes and their distribution

Virulence gene	Functions	Virulence Determination	Reference
Capsular genes			
RmpA (regulator of mucoid phenotype A)	Induces capsule production & hypermucoviscosity Inhibits the expression of the *mrkABCDF* cluster, controlling biofilm formation in hvkp strains Causes severe, invasive infections like liver abscesses, pneumonia and bacteremia in healthy individuals.	Key virulence factor of hvKp. Strains possessing both the hypervirulence features conferred by *rmpA* and antibiotic resistance have been recently reported.	**[29]**
Mag A	Confers a highly viscous,'' mucoid' phenotype specific to the K1 capsule serotype	Associated with the hypermucoviscous phenotype. Represented in strains causing liver abscesses.	**[30]**
wabG	Essential for the biosynthesis of core lipopolysaccharide (LPS) and the attachment of the cell-associated capsular polysaccharide	Target gene for the development of diagnostic assays	**[31]**
ycfM	Enhance capsule production with an antiphagocytic property. Facilitates urinary tract infections (UTIs) and biofilm formation.	Reported in almost all isolates exhibiting UTI	
Adhesin genes			
Fim H **Encodes FimH adhesin**	A key component of type 1 fimbriae, which mediates bacterial adhesion, colonization and pathogenesis, particularly in UTIs	*FimH* Strains with long-term residence within the urinary tract can be viewed as a therapeutic target to neutralize KP, causing UTIs	**[32]**
Mrk D **Encodes for Mrk D adhesin**	Enhances bacterial attachment to host cells and matrices, such as collagen and basement membrane in the kidney and lung. Enhancement of dense biofilms	Genomic screening of clinical *K. pneumoniae* genomes revealed that 98% of genomes carried mrkD.	**[33]**
MrkA **Encodes MrkA type 3 fimbrial protein**	Facilitates the growth of *K. pneumoniae* on abiotic surfaces. Enhances rapid Biofilm formation	Causes opportunistic urinary and respiratory infections Often reported in antibiotic-resistant strains.	
pgaC	Facilitates bacterial adhesion, intercellular aggregation and biofilm maturation Key marker in detecting biofilm-forming isolates	A rapid (RT-PCR) assay that uses *pgaC* to quickly identify biofilm-forming *K. pneumoniae* from clinical samples has been developed.	**[34]**
LuxR **LuxS**	Facilitates Biofilm formation, capsule synthesis and quorum-sensing mechanism Crucial for producing AI-2, a quorum-sensing molecule that regulates virulence, biofilm-related genes and the spread of antibiotic resistance plasmids	Studies suggest that mutations in *luxR* can affect biofilm formation in *K. pneumoniae*.	**[35]** **[36]**
wcaJ	Encodes an enzyme essential for synthesizing the bacterial capsule Plays a crucial role in virulence, biofilm formation and resistance to antibiotics	It acts as a negative regulator, where its absence indicates a high potential for biofilm-forming capacity in the strain.	**[37]**
Uge **Encodes uridine diphosphate galacturonate 4-epimerase**	Essential for virulence, LPS and capsule synthesis	The *uge* gene is present in all tested *Klebsiella pneumoniae* strains which speaks of its species specificity.	**[19]**
Siderophore genes			
ent B	Encodes for Enterobactin, iron-acquiring siderophore.	Observed with increased biofilm production, further pronouncing the infection at a higher rate. Key role in the virulence of hvKp & CRKP strains	**[38]**

Ybt-irp2	Codes for yersiniabactin, a siderophore for iron acquisition, immune evasion and enhanced colonization	Reported on extraintestinal infections	**[38]**
kfu	Involved in iron acquisition	Commonly associated with hypervirulent strains and additionally linked with capsule formation.	
iucABCD ***iutA***	Codes for siderophore aerobactin facilitate iron uptake mainly in hypervirulent strains	Aerobactin has been closely related to the invasive infection caused by hvKp strains	**[39]**
***pks* gene**	Encodes colibactin, which induces DNA damage and enhances virulence	Associated with hypervirulent strains, increased patient mortality, with meningitis	**[40]**
***allS* gene**	Encodes a protein that helps the bacteria utilize allantoin for carbon and nitrogen	Reported alongside with other virulence genes among hvkp strains.	
Identification genes			
Khe	Chromosomal hemolysin gene	Highly conserved among *K. pneumoniae* isolates, making it an excellent marker for identifying the bacterium	**[41]**
rpo B	Highly conserved housekeeping gene	Used for molecular identification and phylogenetic analysis	**[42]**

Biofilm production:

K. pneumoniae is a significant nosocomial pathogen with the potential to form biofilms on medical devices such as catheters, endotracheal tubes and artificial implants. Biofilms offer protection against host immune responses and antibiotic penetration, thereby contributing to drug resistance. The factors that contribute to biofilm formation in *K. pneumoniae* include the polysaccharide capsule, type 1 and type 3 fimbriae, quorum sensing and extracellular polymeric substance. *K. pneumoniae* biofilm formation is associated with a series of genes, including allantoin (allS), capsular polysaccharide (CPS) (treC, cpsD, wzc, wabG, wcaG, rmpA/A2, wzyk2 and magA), aerobactin (iutA), polysaccharides and adhesins (pgaA, pgaB, pgaC and bcsA), type 1 (fimA and fimH) and type 3 fimbriae (mrkD and mrkA), quorum sensing (QS) (luxS) and colonic acid. Type 3 fimbriae are composed of mrk A & mrk D; the former protein subunit instigates biofilm formation, and the latter induces adhesion, facilitating binding to PE and PVC surfaces. In a study evaluating the biofilm-producing *K. pneumoniae* isolates on medical devices, Devanga Ragupathi *et al.* specified that mrk (Type 3 fimbriae) and pgaC (polysaccharide adhesion) are the candidate genes associated with biofilm formation in *K. pneumoniae*, enhancing strong biofilm formation, promoting surface adhesion and also signified that 90% of strong biofilm-forming *K. pneumoniae* were resistant to carbapenem, significantly higher than the weak and negative biofilm-forming *K. pneumoniae* **[43]**. Ochońska *et al.* showed that *K. pneumoniae* formed differential biofilms on the surface of polyethylene (PE) and polyvinyl chloride (PVC) TTs in vitro using SEM analysis **[44]**. Devanga Ragupathi *et al.* also standardized a real-time (RT)-PCR assay for rapid detection of biofilm-forming *K. pneumoniae* from clinical samples in less than eight hours by taking mrk & pgaC as chosen targets in the detection of *K. pneumoniae* biofilms from clinical samples where whereas the wca J gene appears to act as a negative regulator, hence its absence indicates the high potential of the biofilm-forming capacity of the strain **[45]**. Thus, *K. pneumoniae* has been shown to form robust biofilms on abiotic surfaces such as invasive medical devices like catheters, ventilator tubing, as well as on host tissues like the respiratory, urinary and gastrointestinal tract mucosa, proceeding to nosocomial infections. However, these biofilm-associated Klebsiella pneumoniae infections are often chronic, recurrent and difficult to treat because of their increased resistance to antibiotics. Recent reports indicating a significant association between extensive drug resistance and biofilm formation in Klebsiella pneumoniae is quite worrying **[46].** Hence, recent strategies are aimed at disrupting the biofilm with the use of antimicrobial peptides, enzymes targeting biofilm matrix components, quorum-sensing inhibitors and modification of existing materials **[47]**. The strong virulence potential and biofilm-forming capacity of these isolates underscore a significant public health threat, particularly in vulnerable populations.

Hypervirulent klebsiella pneumoniae:

Community-acquired infections among healthy populations have become a great cause of concern with the emergence of hypervirulent strains. A well-observed characteristic feature of hvKp is its hyper-mucoviscous phenotype, determined by the development of viscous strings exceeding 5 mm, also denoted as the string test. The combined presence of biomarkers like peg-344, rmpA/rmpA2 and iucA as genomic signatures along with string test as phenotypic confirmation is widely regarded as a hallmark identification signal of hypervirulent *K. pneumoniae* **[48, 49].** Some "high risk" types of *K. pneumoniae*, such as novel hvKp STs—particularly ST23, ST65 and ST86—are extensively drug resistant with enhanced virulence **[50]**. In these strains, many elements are carried on a large virulent plasmid (such as ST147 with blaNDM-1), which usually encodes iron vector genes (such as aerobactin, salmochelin and enterochelin), heavy metal resistance genes (encoding resistance for tellurite, silver, copper and lead) and capsule up-regulation genes, rmpA and rmpA2. Hence, the emergence of dual threat as a virulent and multidrug-resistant pathogen is quite worrying. A novel sequence type, ST7947, of carbapenem-resistant hvKP was recently identified in an Indian hospital **[51]**. A recent study from China substantiated that 22.8% of invasive *K. pneumoniae* infections were caused by multidrug-resistant hvKp (MDR-hvKP), with the ST11 clone being the most common associated with carbapenem resistance **[52]**. In Chile, researchers isolated an ST23 hvKP strain carrying KPC-2 and VIM-1 carbapenemases on a conjugative plasmid, demonstrating how virulence and resistance can combine. The European Centre for Disease Prevention and Control (ECDC) reports a rise in hvKp ST23. Large-scale plasmid analysis shows simultaneous global clusters of hvKp with carbapenem resistance, highlighting a worrying trend **[53]**. The global rise of

MDR-hvKp poses a dual threat of hypervirulence and antimicrobial resistance, emphasizing the urgency of robust epidemiological surveillance and genomic monitoring. In early 2024, The World Health Organization (WHO) reported the global emergence of hvKp ST23 strains harboring carbapenemase genes across six countries, including India, emphasizing the need for enhanced laboratory diagnostic capacities, molecular testing and surveillance to monitor and mitigate the spread of these high-risk pathogens. This review provides sustained knowledge for understanding the virulence genetics of *K. pneumoniae*, which helps in mapping their frequency and detection rate among the clinical strains. Yet another previous review by Riwu *et al.* (2022) also highlighted the significance of these classical virulence determinants in environmental settings, by documenting its role as a foodborne pathogen **[54]**. Altogether, these findings provided a framework of virulence-associated genes which can act as valuable markers for genomic surveillance. Targeted molecular monitoring of these genes may improve the early detection of high-risk clones and enhance infection control strategies in hospital settings where virulence and antimicrobial resistance coexist.

Conclusion:

The virulence determinants are much needed for unraveling the molecular basis of pathogenicity and to readily identify the high risk hypervirulent lineages. In addition, these virulence factors act as potential targets for novel treatment modalities like development of vaccines, monoclonal antibodies, antivirulence agents, Bacteriophage therapy etc. Futuristic research on virulence determinants will remain as a promising adjunct to traditional therapy in curbing this global burden.

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