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Antibiotic-resistant *Klebsiella pneumoniae*: phenotypic mechanisms and epidemiological patterns

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Abstract

Klebsiella pneumoniae has become a pathogen of significant clinical relevance due to the increasing prevalence of antimicrobial resistance and its remarkable ability to adapt to diverse environments. This Gram-negative bacillus is associated with both hospital-acquired and community-acquired infections and contributes to the emergence of multidrug-resistant strains. This study presents a literature review conducted through a search of the PubMed database, in which articles published during the last six years related to resistance mechanisms, virulence factors, and the epidemiology of K. pneumoniae were selected. Available evidence indicates a global increase in multidrug-resistant strains, particularly those producing β-lactamases and carbapenemases. In addition, virulence factors such as the capsule, lipopolysaccharide, fimbriae, siderophores, and biofilm formation contribute to bacterial colonization. These mechanisms limit available therapeutic options and highlight the importance of strengthening epidemiological surveillance and promoting the rational use of antibiotics.

Key words: *Klebsiella pneumoniae*, antimicrobial resistance, β-lactamases, carbapenemases, virulence factors, multidrug resistance.

Introduction

Weaknesses within the healthcare system have not been limited to the management of emergency situations; they have also influenced the progression of longstanding epidemiological challenges in the country. Among these is the increasing prevalence and complexity of bacterial infections, driven by reduced diagnostic capacity, hospital overcrowding, and the widespread, often indiscriminate use of antibiotics without clear therapeutic indication during the pandemic. These conditions have facilitated the emer-

gence and spread of antimicrobial-resistant pathogens, thereby intensifying both the clinical management burden and the challenges associated with controlling their transmission.

Within this context, certain bacterial species have gained particular relevance due to their remarkable capacity for adaptation and resistance, notably *Klebsiella pneumoniae*.

The genus Klebsiella is classified within the phylum Proteobacteria, class Gammaproteobacteria, and family Enterobacteriaceae. These organisms are Gram-negative, rod-shaped, non-motile, and oxidase-negative bacteria, distinguished by a polysaccharide capsule that surrounds the cell and protects against host defense mechanisms. The genus was named in honor of the German microbiologist Edwin Klebs (1-3). *Klebsiella* species exhibit a broad distribution across diverse environments. They can be found in water, soil, and vegetation, as well as in animals and humans, where they form part of the normal microbiota of the skin, respiratory mucosa, and gastrointestinal tract. This ecological versatility underlies their ability to adapt to multiple niches and, under certain conditions, behave as opportunistic pathogens capable of causing clinically significant infections.

This bacterial species has become one of the most significant agents in the emergence and dissemination of multidrug-resistant organisms, owing to its ability to rapidly adapt to continuous antibiotic exposure and its efficient spread within clinical settings. Consequently, it has emerged as a high-impact pathogen, associated with severe infections and nosocomial outbreaks (4, 5).

In this context, the present review article aims to analyze antibiotic resistance in *K. pneumoniae,* addressing the main phenotypic mechanisms that contribute to this phenomenon, as well as its global distribution and epidemiological patterns.

Methodology

A comprehensive review of the scientific literature on antibiotic-resistant *K. pneumoniae* was conducted in PubMed. The search strategy incorporated a range of terms related to the microorganism, resistance mechanisms, and its epidemiological behavior, with the aim of broadening and refining the retrieved results.

The selection was limited to articles published within the past six years to ensure the inclusion of recent and relevant scientific evidence. The retrieved studies were subsequently reviewed and critically analyzed, prioritizing those that address phenotypic resistance mechanisms, epidemiological distribution, and the key challenges in controlling antibiotic-resistant *K. pneumoniae*. The information obtained was then synthesized and systematically organized for analysis within this review article.

Results

Clinical Overview

The ability of *K. pneumoniae* to transition from a commensal organism to a pathogen is influenced by multiple factors that increase the risk of infection, including immunosuppression, prolonged hospital stays, prior antibiotic use, invasive medical procedures, and the presence of comorbidities such as diabetes, hepatobiliary diseases, neoplasms, and chronic pulmonary conditions. These factors favor its occurrence primarily in hospitalized patients and in vulnerable populations, including preterm neonates, older adults, and immunocompromised indi-

viduals (6, 7).

This microorganism frequently colonizes the oropharynx and gastrointestinal tract as part of the host's normal microbiota. In addition, it has been identified in a variety of environmental reservoirs, including soil, water, and inert surfaces such as medical equipment, reflecting its remarkable capacity for adaptation and persistence across diverse environments (8, 9).

Klebsiella pneumoniae is responsible for both healthcare-associated and community-acquired infections. In clinical settings, it is associated with pneumonia, bacteremia, urinary tract infections, endocarditis, central nervous system infections, and surgical site infections, predominantly affecting vulnerable patients.

Outside the hospital, it is linked to pneumonia and primary liver abscesses with the potential for systemic dissemination, including the so-called Friedländer's pneumonia, more commonly observed in middle-aged or older men with a history of alcohol use disorder.

Due to its clinical significance, rising antimicrobial resistance, and high mortality associated with its infections, the World Health Organization has classified *K. pneumoniae* as a critical priority pathogen (6, 8-10). This classification is supported by its role as one of the leading causes of Gram-negative bacillary bacteremia, second only to Escherichia coli (8).

K. pneumoniae bacteremia may present as a primary infection when the microorganism is detected in the bloodstream without an identifiable source; however, it more commonly occurs secondary to infections of the urinary, gastrointestinal, or respiratory tract, and is often associated with invasive procedures such as the use of intravenous or urinary catheters.

The severity of these infections lies in their high mortality, which can approach 50% even with appropriate antimicrobial therapy and may rise to nearly 100% in patients with a history of alcohol use disorder. In addition, they can lead to severe metastatic complications, including pyogenic brain abscesses, meningitis, and endophthalmitis, significantly worsening prognosis and complicating clinical management (2).

From a clinical and epidemiological perspective, *K. pneumoniae* can be divided into three main groups: classical strains cKp, commonly associated with opportunistic healthcare-associated infections; hypervirulent strains hvKp, related to community-acquired infections and capable of causing liver abscesses with systemic dissemination even in previously healthy individuals; and multidrug-resistant strains MDR, responsible for severe hospital-acquired infections with high rates of morbidity and mortality (11).

The latter represent a growing concern, as studies of genomic evolution have shown that MDR clones exhibit a greater tendency to acquire virulence genes, whereas hypervirulent strains display a lower likelihood of acquiring antimicrobial resistance determinants (11).

Epidemiology

The presence and colonization of *K. pneumoniae* are influenced by multiple factors operating both in community and healthcare settings, with their frequency varying across regions. These include soci-

odemographic characteristics such as age, sex, and hospitalization status, as well as environmental factors like sources of household water and contact with domestic or production animals. In addition, conditions such as malnutrition, underlying diseases, and inadequate practices in the use of detergents and antiseptics may favor its establishment.

At a global level, an increase in resistance to antibiotics classified within the AWaRe "Watch" group, particularly carbapenems and fluoroquinolones, has been observed among major Gram-negative pathogens, including *Acinetobacter spp., Escherichia coli, K. pneumoniae,* and *Salmonella spp.* (12). This trend represents a significant public health concern, as several of these microorganisms, especially carbapenem-resistant *K. pneumoniae* and *Acinetobacter spp.,* are associated with severe clinical outcomes and mortality rates that may exceed 30% (12).

In the hospital setting, *K. pneumoniae* accounts for a significant proportion of healthcare-associated infections, representing approximately 7–14% of pneumonia cases and 6–17% of nosocomial urinary tract infections, which may progress to complications such as pyelonephritis, recurrent infection, or permanent renal damage (2, 6).

Within this context, infections caused by carbapenem-resistant *K. pneumoniae* CRKP represent an increasing threat to public health due to their high lethality, estimated to range between 37% and 42% according to the meta-analysis by Lin and collaborators (13), with more than 90,000 cases reported annually in Europe and over 7,000 associated deaths; meanwhile, in resource-limited countries such as Malawi, up to 75% of *K. pneumoniae* bacteremia cases correspond to multidrug-resistant strains.

The same study indicates that the frequency of CRKP in hospital-acquired infections varies according to geographic region, the period analyzed, and the characteristics of healthcare institutions, with an increase observed in China from 3% in 2005 to 21% in 2017, while in Europe surveillance systems reported a rise from 6.2% in 2012 to 8.1% in 2015.

At a global level, the most pronounced increase in resistance among pathogens causing bloodstream infections has been observed in *K. pneumoniae* against imipenem, with an estimated annual growth of 15.3%. This trend has been documented across most WHO regions, with the highest increase in the African Region at 20.2%, whereas no overall increase was identified in the Region of the Americas (12). In contrast, resistance to cefotaxime in *K. pneumoniae* bacteremia did not show an increasing trend (12).

Similarly, resistance to imipenem in urinary tract infections caused by *K. pneumoniae* has shown a global annual increase of 12.9% across most regions, with the exception of the Americas, with the highest increase recorded in Europe at 29.4%, followed by the African Region at 20.2% (12). In addition, a 5.2% decrease in cefotaxime resistance was observed in the Eastern Mediterranean Region (12).

In Mexico, several studies have described the epidemiology of *K. pneumoniae* and its antimicrobial resistance patterns; however, the available information remains limited to establish a clear national epidemiological overview. This is largely because most reports are concentrated in specific hospitals

or regions and employ different sampling and characterization methodologies, making it difficult to compare results and identify consistent trends.

In particular, extended-spectrum beta-lactamase ESBL-producing strains have shown an increasing trend in the country. A multicenter study conducted between 2005 and 2012 across 14 hospitals in eight states reported that 27.5% of hospital isolates corresponded to these strains; subsequently, between 2014 and 2015, a prevalence of 6.4% was identified in urinary tract infections at the Centro Médico Nacional de Occidente, while a national report covering the period 2016–2017 documented frequencies of 21.9% in bacteremia and 8.5% in urinary tract infections (14).

More recently, during the period 2020–2021, an apparent reduction in the frequency of resistant strains was observed, followed by a notable increase between 2022 and 2023. This pattern may be associated with the impact of the COVID-19 pandemic, as the widespread use of antibiotics in infected patients, despite low rates of bacterial coinfection, likely contributed to the selection and dissemination of multidrug-resistant microorganisms, including ESBL-producing strains (15).

Antimicrobial Resistance Mechanisms

Over the past two decades, *K. pneumoniae* has increased its clinical relevance due to the sustained rise in antimicrobial resistance and the emergence of multidrug-resistant strains. As a result, it has become one of the most important Gram-negative bacilli in infections worldwide, particularly in hospital settings where selective pressure from antibiotic use favors the spread of resistant strains.

In Mexico, this situation has been reported by hospitals within the PUCRA network between 2017 and 2020, as shown in Figure 1. According to these data, blood cultures analyzed in 2020 revealed a concerning resistance pattern in *K. pneumoniae*, with 52% of the 755 recorded isolates classified as multidrug-resistant MDR, defined as resistance to at least one antibiotic in three or more therapeutic categories (16). These findings highlight the substantial burden of resistance associated with this pathogen in the national hospital setting.

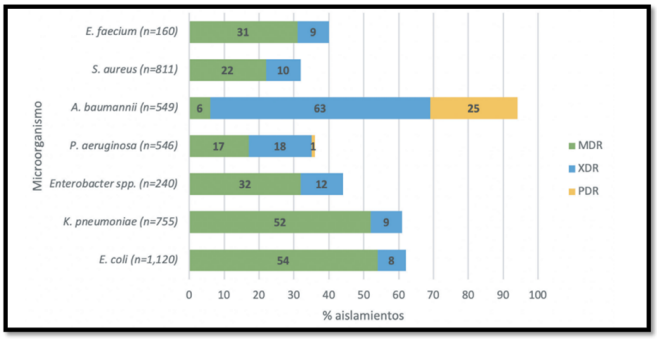

Figure 1. Percentage of MDR, XDR, and PDR isolates among ESKAPE group bacteria from blood cultures in 2020 (16).

Bacterial resistance can develop through various mechanisms that enable bacteria to evade the action of antibiotics. Among the most relevant are modifications of the cell surface, alterations in drug transport into or out of the cell, changes in antibiotic target sites, and drug inactivation processes (17).

Among these mechanisms, mutations in the lipopolysaccharide LPS of the outer membrane in Gram-negative bacteria are particularly notable, as they can alter cell permeability and hinder the entry of antibiotics. Similarly, the loss or modification of porins reduces the influx of these compounds into the bacterial cell, thereby decreasing their therapeutic effectiveness (17).

In addition, bacteria can acquire resistance plas-

mids, extrachromosomal DNA molecules that carry genes conferring different resistance mechanisms and can be transferred between bacteria, thereby facilitating the spread of resistance (17).

Among the mechanisms associated with these genes are enzymatic inactivation of antibiotics, mutations at target sites that reduce the drug's affinity for its cellular target, and efflux pump activity, which expels antibiotics from the cell and lowers their intracellular concentration (17).

Among these mechanisms, the production of β-lactamases is one of the most important mechanisms of resistance to β-lactam antibiotics. These enzymes, primarily located in the periplasmic space, hydrolyze the β-lactam ring, preventing the antibiotic from binding to penicillin-binding proteins. The genes encoding these enzymes may be located on the bacterial chromosome or on mobile genetic elements such as integrons, transposons, and plasmids, which facilitate their transfer between bacteria (18).

Among the most relevant β-lactamases are extended-spectrum β-lactamases ESBL and AmpC enzymes, which represent key mechanisms of resistance to cephalosporins in Gram-negative bacteria, as they hydrolyze these antibiotics and reduce their therapeutic effectiveness.

Within this group, carbapenemases are particularly notable, as they are enzymes that can inactivate carbapenems. According to the Ambler classification, they are divided into several structural classes. Enterobacteriaceae producing these enzymes exhibit resistance to agents such as imipenem, meropenem, and ertapenem, which considerably limits therapeutic options (19, 20).

Class A carbapenemases, such as KPC, NmcA, IMI, SME, GES, and SFC, possess a serine residue at their active site, may be located on chromosomes or plasmids, and exhibit partial inhibition by clavulanic acid (19, 20).

Class B carbapenemases, or metallo-β-lactamases, which include NDM, IMP, VIM, GIM, and SPM-1, require a metal ion for their activity and are capable of hydrolyzing most β-lactams, with the exception of aztreonam. These enzymes can be inhibited by chelating agents such as EDTA, but not by clavulanic acid (19, 20).

Finally, class D carbapenemases, or oxacillinases, mainly from the OXA-48-like group, exhibit a lower capacity to hydrolyze carbapenems and cephalosporins and are not inhibited by clavulanic acid or EDTA, which complicates their detection and treatment (20).

Virulence Factors and Phenotypic Mechanisms

The pathogenicity of *K. pneumoniae* depends on a variety of virulence factors that enhance its ability to cause infection. Among the most important are capsular polysaccharides with K antigens, lipopolysaccharides with O antigens, adhesins, and siderophores such as aerobactin, enterobactin, salmochelin, and yersiniabactin, as well as toxins and the ability to form biofilms. Many of these determinants are encoded in the mosaic plasmid pLVPK (6, 7, 9, 21, 22).

Capsule

The capsule of *K. pneumoniae* is composed of polysaccharides and represents one of its main virulence factors, as it protects the bacterium against host defense mechanisms. The absence of this structure has been associated with less virulent

strains or those unable to cause infection, even when they exhibit multidrug-resistant profiles.

To date, 78–79 capsular types, known as K antigens, have been identified, differing in the composition of their repeating units (23-25).

Functionally, the capsule covers the bacterial surface, confers the characteristic mucoid phenotype, and contributes to evasion of processes such as phagocytosis, complement-mediated lysis, and antimicrobial peptide action. From an epidemiological perspective, serotypes K1 and K2 are more frequently associated with hypervirulent strains and account for approximately 70% of hvKp isolates (23, 24).

Lipopolysaccharide

Lipopolysaccharide LPS is an important factor in the pathogenicity of *K. pneumoniae*, particularly in hypervirulent strains, as it contributes to evasion of the host immune response and to the development of systemic infections (23, 26). This component of the outer membrane of Gram-negative bacteria is composed of lipid A, an oligosaccharide core, and the O-antigen chain, whose synthesis depends on the lpx, waa, and wb/rfb gene clusters (17, 27).

Functionally, LPS acts together with the capsule as a barrier that interferes with complement activity, reducing opsonization and phagocytosis, thereby promoting bacterial persistence in the bloodstream (23, 26).

From an epidemiological perspective, the O1 antigen has been associated with greater virulence and colonization capacity, whereas O2 is more frequently found in multidrug-resistant isolates, suggesting a relationship between certain serotypes and antimicrobial resistance (17, 28).

Fimbriae

Fimbriae are important adhesion structures in *K. pneumoniae*, as they facilitate the initial stages of host colonization. These proteinaceous appendages enable adherence to eukaryotic cells and abiotic surfaces, and also participate in processes such as intestinal persistence, interaction with macrophages, bacterial aggregation, and biofilm formation (25, 29).

Although approximately ten types of fimbriae have been identified in *K. pneumoniae*, the most studied and clinically relevant are types 1 and 3 (30).

Type 1 fimbriae are primarily associated with urinary tract infections, where their expression is elevated in bacteria present in the bladder and urine, while in the lung and gastrointestinal tract their expression is typically repressed (29, 31).

In contrast, type 3 fimbriae, present in more than 80% of strains, facilitate adhesion to extracellular matrix proteins and promote biofilm formation on abiotic surfaces, particularly on catheters and other medical devices (29-31).

Siderophores

Iron is an essential element for the metabolism of *K. pneumoniae*; however, in the human host its availability is limited due to nutritional immunity mechanisms, in which proteins such as transferrin, lactoferrin, ferritin, and siderocalin sequester it (26, 32, 33).

To overcome this limitation, *K. pneumoniae* produces siderophores, small molecules that capture iron in the extracellular environment and transport

it back into the bacterial cell through TonB-dependent systems, thereby ensuring its growth and persistence within the host (26, 33).

The most common siderophore is enterobactin, which is present in most Enterobacterales and is considered the primary iron acquisition system in *K. pneumoniae*. However, its function can be inhibited by lipocalin-2, which has driven the emergence of alternative siderophores that can evade this mechanism, such as salmochelin, yersiniabactin, and aerobactin (23, 33, 34).

Among these systems, aerobactin is considered the siderophore most strongly associated with the hypervirulent phenotype of *K. pneumoniae* (34, 35).

Biofilm

Biofilm formation is an important virulence factor in *K. pneumoniae*, frequently observed in clinical isolates and associated with infection persistence and increased antimicrobial resistance (17, 36-39).

These structures consist of organized bacterial communities embedded within an extracellular matrix composed primarily of polysaccharides and proteins. In *K. pneumoniae*, the polysaccharides include mannose, glucose, and their derivatives, while structures such as surface adhesins, fimbriae, and flagella contribute to adhesion and biofilm establishment (11, 37).

In clinical settings, biofilms commonly form on catheters and other medical devices, contributing to healthcare-associated infections, particularly in immunocompromised patients (11, 39).

In addition, the biofilm acts as a protective barrier against chemical agents, desiccation, and host immune mechanisms, including antibodies, antimicrobial peptides, complement, and phagocytic cells. Bacteria within biofilms can exhibit antimicrobial resistance levels 10 to 1000 times higher than planktonic cells, and their close proximity facilitates bacterial communication and the exchange of genetic material (37).

Genetic dissemination of resistance

According to Pu and collaborators (40), the emergence of *K. pneumoniae* strains that combine hypervirulence and carbapenem resistance can be explained by different mechanisms of horizontal plasmid transfer and genetic recombination between bacteria.

One of the most common scenarios occurs when hvKp and CRKP strains exchange genetic material, leading to two main outcomes. In some cases, hypervirulent strains acquire resistance plasmids, giving rise to carbapenem-resistant hypervirulent bacteria hv-CRKP. In other cases, resistant strains incorporate virulence plasmids, generating strains that are both resistant and hypervirulent CR-hvKP.

The transfer of these determinants may occur through individual plasmid transfer or through co-transfer, in which multiple plasmids are simultaneously mobilized between bacteria, as illustrated in Figure 2. Once inside the recipient cell, these genetic elements may be maintained independently or integrate with one another through genetic recombination, resulting in hybrid plasmids that carry both resistance and virulence genes in a single molecule.

Pu and collaborators (40) also describe that these recombination events can occur through homologous recombination, when there is sequence simi-

larity between DNA regions, or through site-specific recombination, which uses particular short sequences to integrate genetic fragments. Both mechanisms promote the generation of new genetic combinations that enhance bacterial adaptability.

In addition, the dissemination of these plasmids may involve not only *K. pneumoniae*, but also other Enterobacteriaceae such as *Escherichia coli*, thereby expanding the reservoir of resistance and virulence genes within the bacterial community.

Finally, Pu and collaborators (40) indicate in Figure 2 that certain plasmids, such as those of the IncN3 type, may participate in these transfer and recombination processes, facilitating the formation of hybrid plasmids. It has also been proposed that outer membrane vesicles may play a role in the mobilization of genetic material, further contributing to the spread of these determinants.

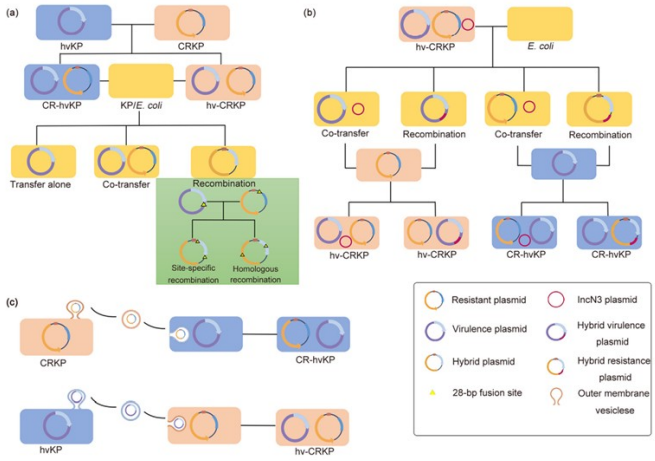

Figure 2. Convergence of virulence and resistance plasmids in K. pneumoniae (40).

Discussion

The evidence analyzed in this review confirms that *K. pneumoniae* has become one of the most relevant pathogens in the context of antimicrobial resistance worldwide, not only due to its high adaptive capacity, but also because of the convergence of resistance and virulence mechanisms that enhance its clinical impact. The limited availability of new antimicrobial agents effective against infections caused by carbapenem-resistant strains has led to the renewed use of polymyxins, such as colistin and polymyxin B, as last-resort therapeutic options (41).

Furthermore, the global increase in strains exhibiting widespread resistance to β-lactam antibiotics, including carbapenems, highlights the remarkable ability of this microorganism to adapt to environmental selective pressure. In particular, the indiscriminate use of these drugs has favored the selection of bacteria carrying plasmid-mediated carbapenemases that can inactivate even last-line treatments.

In addition, *K. pneumoniae's* ability to acquire and disseminate resistance genes through horizontal gene transfer is a key factor in the rapid spread of this phenomenon. The presence of plasmids facilitates not only the accumulation of multiple resistance determinants within a single strain, but also their transfer across different bacterial species, thereby expanding the reservoir of resistance in both hospital and community settings.

The problem is further exacerbated by the emergence of hypervirulent *K. pneumoniae* strains that, in addition to their high pathogenic potential, have begun to acquire antimicrobial resistance determinants, giving rise to variants that combine hypervirulence and multidrug resistance. These strains employ a wide range of virulence factors, including the capsule, siderophores, lipopolysaccharide, fimbriae, outer membrane proteins, and the type VI secretion system, which enhance their survival, invasion, and dissemination, further complicating

clinical management and limiting available therapeutic options (42).

This situation becomes particularly critical in low- and middle-income countries, where infections caused by *K. pneumoniae* are increasingly difficult to treat due to the rapid acquisition of extended-spectrum β-lactamases, posing a significant threat to public health. In this context, the recent emergence and dissemination of carbapenem-resistant strains have heightened global concern, to the extent that the World Health Organization has classified carbapenem-resistant Enterobacteriaceae as critical-priority pathogens for the development of new antimicrobials (43).

In this context, addressing resistant *K. pneumoniae* cannot be limited solely to the development of new antimicrobials; it requires a comprehensive approach that includes epidemiological surveillance strategies, infection control measures, and the optimization of antibiotic use. Furthermore, it is essential to strengthen research to understand the interaction between resistance and virulence, as this convergence is one of the main current challenges in the management of bacterial infections.

Conclusions

K. pneumoniae has emerged as a pathogen of major clinical importance due to its remarkable ability to adapt to and persist in diverse environments, as well as to acquire antimicrobial resistance mechanisms. The presence of multiple virulence factors, including the capsule, lipopolysaccharide, fimbriae, siderophores, and the ability to form biofilms, significantly contributes to its capacity to colonize, evade the host immune response, and cause infections that are difficult to manage. This is further compounded by the wide range of resistance mechanisms, particularly the production of β-lactamases and carbapenemases, which substantially limit available therapeutic options. Horizontal plasmid transfer and genetic recombination processes also promote the emergence of strains that combine hypervirulence and multidrug resistance, further complicating the control of these infections.

Strengthening epidemiological surveillance, promoting the rational use of antibiotics, and implementing appropriate infection control measures are key elements to reduce the dissemination of resistant *K. pneumoniae* and mitigate its impact on public health.

Ethics approval

The project on which this article is based was submitted to the Bioethics Committee of the Biomedical Research Center of the Universidad Autónoma de Coahuila as part of the development of the lead author's graduate thesis.

Informed consent

Not applicable

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Conflict of interests

The authors declare that there are no financial, personal, or professional conflicts of interest that could have influenced the preparation, interpretation, or presentation of the results in this manuscript. They further affirm that the content of this article was developed independently and without the influence of external entities.

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