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The Neglected Role of *Klebsiella pneumoniae* in Food-Producing Animals and its Consequences for Food Safety and Public Health

Saddam[1]*, Muhsin Jamal[2], Ijaz Ahmad[3], Lalina Maroof[1], Sadeeq Ur Rahman[2], Muddasir Khan[4], M Jamil Khan[5], Khalid J Alzahrani[6], Ibrahim F Halawani[6], Khalaf F Alsharif[6].**

[1]*Department of Allied Health Sciences, City University of Health Science and Information Technology, Peshawar, Pakistan*

[2]*Department of Microbiology, Timergara Campus, Abdul Wali Khan University, Mardan, Pakistan*

[3]*Department of Health & Biological Sciences, Abasyn University Peshawar*

[4]*Centre of Biotechnology and Microbiology, University of Peshawar, Pakistan*

[5]*Department of Allied Health Sciences, Iqra National University Peshawar*

[6]*Department of Clinical Laboratories Sciences, College of Applied Medical Sciences, Taif University, P.O. Box 11099, Taif 21944, Saudi Arabia*

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***Corresponding author**: Sadeeq ur Rahman, Department of Microbiology, Timergara Campus, Abdul Wali Khan University, Mardan, Pakistan. Email: sadeeq@awkum.edu.pk

Abstract

Klebsiella pneumoniae is a versatile pathogen capable of causing infections in multiple species, yet its role in bovine mastitis remains underexplored. Although well known for hospital-acquired infections in humans, *K. pneumoniae* also affects dairy animals, leading to substantial economic losses. Its ability to acquire multiple antibiotic resistance (AR) determinants under selective pressure has contributed to the emergence of multidrug-resistant (MDR) and extensively drug-resistant (XDR) strains. Recent data indicate a rapid evolution and global dissemination of pathogenic Klebsiella lineages. In dairy herds, mastitis caused by Klebsiella spp. is increasingly difficult to manage due to the pathogen's virulence factors and efficient uptake of plasmid-mediated AR genes. Despite these concerns, the veterinary impact of *K. pneumoniae* has received limited attention. This review summarizes the epidemiology of *K. pneumoniae* in bovine mastitis and its AR acquisition mechanisms, emphasizing the urgent need for improved surveillance and targeted interventions to address this growing threat to animal and public health.

Keywords: *K. pneumoniae*; Mastitis; Pathogenicity; Virulence; Prevalence

Introduction

Klebsiella pneumoniae is an opportunistic pathogen responsible for a wide range of diseases in both animals and humans, including pneumonia, urinary tract infections, bacteremia, and liver abscesses [1,2]. Although extensive research has focused on *K. pneumoniae* infections in both clinical and hospital settings, the emergence of hyper virulent strains showing resistance to a broad range of antibiotics poses a serious challenge to health [3]. The development of antibiotic resistance (AR) is a multifaceted and complex phenomenon resulting from evolution in response to concomitant selective antibiotic pressure. This continuous use and frequent misuse of antibiotics over the last 80 years have resulted in the emergence of extremely drug-resistant (XDR) and multidrug-resistant (MDR) isolates of pathogenic bacteria. Recently, AR has become a world health problem and is particularly in underdeveloped countries, including Pakistan, where insufficient infection control measures further aggravate the issue. Recently, researchers reported a dramatic increase in AR among different bacterial species isolated in Pakistan [4-13]. However, despite these rising issues, epidemiological data regarding MDR *K. pneumoniae* in infecting animals is limited, as summarized in Table 1.

Table 1: Global Prevalence of Resistant K. pneumoniae in Humans

Region	ESBL-Producing *K. pneumoniae* (%)	Carbapenem-Resistant *K. pneumoniae* (CRKP) (%)	Dominant Carbapenemase Genes	References
Global	20–50% (wide variation)	5–30% (high regional differences)	KPC, NDM, OXA-48, VIM	(Antimicrob Resist Infect Control 2021)
Africa	30–60% (high in North/West Africa)	10–50% (OXA-48 prevalent in North Africa)	OXA-48, NDM	(WHO AFRO 2021)
Asia	40–70% (India, China >60%)	15–60% (India >50% CRKP)	NDM, KPC, OXA-48	(Lancet Microbe, 2022)
Europe	10–40% (higher in South/East)	5–50% (Greece, Italy >25%)	KPC, OXA-48, VIM	(Euro Surveill. 2022)
North America	15–30% (US higher than Canada)	2–15% (US hotspots: NYC, LA)	KPC, NDM	(CDC NHSN 2023)
South America	30–60% (Brazil, Colombia high)	10–40% (KPC dominant)	KPC, NDM	(Antimicrob Resist Infect Control 2021)
Middle East	40–70% (Saudi Arabia, Lebanon)	20–60% (NDM/OXA-48 endemic)	NDM, OXA-48	(J Infect Public Health 2023)

Abbreviations: CRKP, carbapenem-resistant *K. pneumoniae*; ESBL, extended-spectrum beta-lactamase; US, United States; OXA-48, Oxacillinase-48; NYC, New York city; LA, Los Angeles; KPC, *K. pneumoniae* carbapenemase; NDM, New Delhi metallo-beta-lactamase; VIM, verona integron-encoded metallo-β-lactamase.

Table 2: Resistance Profiles of Veterinary K. pneumoniae Isolates

Host Species	Sample Type	Resistance Profile	Key Resistance Genes	Prevalence (%)	References
Dairy Cattle	Milk (mastitis)	ESBLs, fluoroquinolones, colistin	bla_{CTX-M-15}, bla_{SHV}, *mcr-1*	20–50% (ESBL)	(J Diary Sci 2024; Vet Microbial, 2023)
Beef Cattle	Feces, nasal swabs	MDR (3rd-gen cephalosporins, tetracyclines)	bla_{TEM}, tet(A), sul2	15–40% (MDR)	(Front Vet Sci 2023; Appl Environ Microbio1. 2024)
Poultry	Meat, cloacal swabs	ESBLs, carbapenems (rare)	bla_{CTX-M-1}, bla_{NDM-5}	30–60% (ESBL)	(EFSA J. 2024; Antimicrob Agents Chemother. 2023)
Swine	Feces, carcasses	Colistin, aminoglycosides	*Mcr-1*, *aac(6')-Ib-cr*, bla_{OXA-48}	10–30% (MCR)	(Emerg Infect Dis. 2023; J Glob Antimicrob Resist. 2024)
Companion Animals	Urine, wounds	MDR (carbapenems, fluoroquinolones)	bla_{KPC}, qnrS, bla_{VIM}	5–20% (CRKP)	(Vet Res. 2024; JAC Antimicrob Resist. 2023)

Abbreviations: ESBL, extended-spectrum beta-lactamases; MDR, multidrug-resistant; *bla_{CTX-M-1}, β-lactamase, cefotaximase-Munich group 1; bla_{NDM-5}, β-lactamase, New Delhi metallo-β-lactamase-5 variant; mcr-1, mobilized colistin resistance gene 1; aac(6')-Ib-cr, aminoglycoside acetyltransferase variant; bla_{OXA-48}, beta-lactamase oxacillinase-48 gene; bla_{KPC}; beta-lactamase, K. pneumoniae carbapenemase, qnrS, quinolone resistance protein S; bla_{VIM}, beta-lactamase, Verona integron-encoded metallo-beta-lactamase; MCR, mobilized colistin resistance; CRKP, carbapenem-resistant K. pneumoniae.*

Habitat and Transmission of Klebsiella pneumoniae

Klebsiella pneumoniae naturally inhabits the gastrointestinal and upper respiratory tracts of both humans and animals and is also commonly found in the environment [14]. Multiple transmission routes have been identified. In humans, person-to-person spread-particularly via the hands of healthcare workers-is a major source of transmission [15]. In animals, transmission occurs through fecal–oral routes, contaminated surfaces, direct contact, and farm equipment such as milking machines and drinking water systems [16].

Antibiotic Resistance and Mechanisms

The acquisition of antimicrobial resistance (AR) determinants by pathogenic *K. pneumoniae* leads to resistance against multiple antibiotics, resulting in prolonged hospitalization and increased risk of healthcare-associated infections [17]. Resistance mechanisms include enzymatic drug inactivation, reduced membrane permeability, and modification of antimicrobial target sites.

Clinical Importance in Humans and Animals

K. pneumoniae causes a range of infections in humans and various animal species. In bovines, it is particularly associated with pneumonia, mastitis, and skin infections [18–22]. Immunocompromised hosts-both human and animal-are at higher risk of severe infection. Despite its clinical significance,

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limited information is available regarding the impact of *K. pneumoniae* on cattle welfare, productivity, epidemiology, and resistance development in non-human isolates. *K. pneumoniae* also contributes to neonatal sepsis [23]. Environmental factors, including increased rainfall and damp bedding, are associated with a higher prevalence of Klebsiella mastitis [24]. The bacterium is frequently isolated from milking machines, wash water, and farm drinking water, highlighting its diverse reservoirs and transmission points [21, 25].

Klebsiella Mastitis: Epidemiology and Impact

Diagnosing the causative agent of bovine mastitis can be challenging. Literature consistently reports Klebsiella spp., Escherichia coli, followed by Enterobacter and Serratia species, as the most frequently isolated Gram-negative pathogens in bovine clinical mastitis [26,27]. The reported incidence ratio of Klebsiella to E. coli mastitis ranges from 1:10 to 1:1 [28, 29]. *K. pneumoniae* can be highly infectious. Entry typically occurs through the teat canal, allowing the bacteria to proliferate in the udder, causing inflammation and milk loss. Its survival within the mammary gland is promoted by availability of nutrients and its ability to evade host immune defenses. The risk of infection is highest during the dry period due to easier bacterial access to the teat canal, poor hygiene, and bacterial colonization of the teat skin. In the periparturient period, elevated cortisol levels may suppress immune responses, increasing susceptibility. Severe coliform mastitis caused by Klebsiella can compromise the blood–milk barrier, leading to bacteremia and septicemia [27].

Public Health Significance and Need for Further Research

Antimicrobial-resistant strains of *K. pneumoniae*, especially extended-spectrum β-lactamase (ESBL) and carbapenemase producers are identified by the World Health Organization as critical priority pathogens [30]. Losses caused by Klebsiella mastitis surpass those associated with E. coli mastitis in terms of milk production and animal survival [31]. Additionally, Klebsiella infections are often less responsive to treatment and tend to persist longer. Given these concerns, further research into virulence factors, AR mechanisms, plasmids, integrons, and gene cassettes involved in resistance transmission is urgently needed. This review aims to highlight the prevalence of *K. pneumoniae*-associated mastitis, the epidemiology of Klebsiella infection in dairy animals, and the genetic determinants contributing to antimicrobial resistance [32].

Disease Course and Pathogenesis

The pathogenesis of mastitis caused by *K. pneumoniae* and intra-mammary inflammation has not yet been adequately reported. The point source of infection includes bedding materials, manure, soil, and other organic cow-related environmental factors, such as sawdust and wood bedding materials. During the transition period of animals from nursing to weaning, the incidence of infection is significant. The teat canal serves as the entry point for bacteria. Once within the mammary gland, bacteria must utilize the available nutrients in the breast discharge while evading the host's defensive system. Coliform mastitis is frequently diagnosed based on clinical signs, coliform organism culture from milk, and a high somatic cell count [33]. However, in the acute instance, the milk sample may be negative because neutrophils have removed the organisms. At least four components capsule, lipopolysaccharide, fimbriae, and siderophore have been identified as crucial for virulence despite heterogeneity among the strains [34]. *K. pneumoniae* attaches to the host cell surface thanks to the cell wall receptor. Bacteria alter their surface so that polymorph nuclear leukocytes and macrophages cannot phagocytize them. This ability renders the host cell non-phagocytic and mediates its safe entry to the host cell [35]. Then, capsular polysaccharide helps in the second invasion of Klebsiella. In addition, the capsule also mediates its non-phagocytic entry to the host cell. Further, the capsule also acts as a barrier to protect *K. pneumoniae* from host phagocytic activity. Endotoxin production by *K. pneumoniae* also contributes to its pathogenicity. However, its production is not dependent on factors that determine other virulent factors of *K. pneumoniae*. All these factors contribute relatively to the virulence of *K. pneumoniae* in vivo, making it difficult to determine their individual contributions, as they all contribute to virulence [30,32].

Animal and human transmission of *K. pneumoniae*

K. pneumoniae is a zoonotic pathogen that causes septicemic, pneumonic, and urinary tract infections in hospitals. Pathogenic *K. pneumoniae* strains with a hypermucoviscosity (HMV) phenotype have recently been linked to multisystem abs cessation in both humans and nonhuman primates. Although *K. pneumoniae* is a well-known zoonotic agent, general information, including effective diagnostic tools and therapies for nonhuman primates, is lacking [31]. Similar molecular types and behaviors across *K. pneumoniae* strains from various sources, including humans and animals, suggest that these strains may be transmitted between humans and animals, as shown in the Figure 1. To prevent the spread of *K. pneumoniae* between humans and animals, cautious antibiotic administration is required in both human clinical treatment and animal production [33,35].

ESBL and Carbapenemase-producing *K. pneumoniae*

ESBL producers can efficiently hydrolyze third generation cephalosporins, making them difficult to treat and resulting in severe damage [36]. In 1983, isolates of multi-drug-resistant ESBL-producing *K. pneumoniae* encoding blaSHV and blaTEM were first described in Europe [3738]. A short while later, *K. pneumoniae* reported another plasmid-mediated ESBL variation blaTEM-3 [39]. This was followed by several reports describing MDR *K. pneumoniae* from different parts of the world, including the US and South America [40,41]. Since the emergence of ESBLs in *K. pneumoniae*, this pathogen has become the primary source of ESBL dissemination. The recent discovery of MDR pathogenic *K. pneumoniae* that encodes ESBL/AmpC in broiler chickens,

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soil, boots, slurry surfaces, and ambient air in the United States indicates its persistent and rapid spread [42]. In addition, genes that produce beta-lactamase enzymes that are partially inhibited by clavulanic acid and tazobactam were discovered [43]. Over the last three decades, ESBL-producing Klebsiella has become a global epidemic in many countries. In conclusion, a literature study reveals that ESBL-producing *K. pneumoniae* has been reported from most parts of the world, indicating its international presence and ability to disseminate worldwide [44].

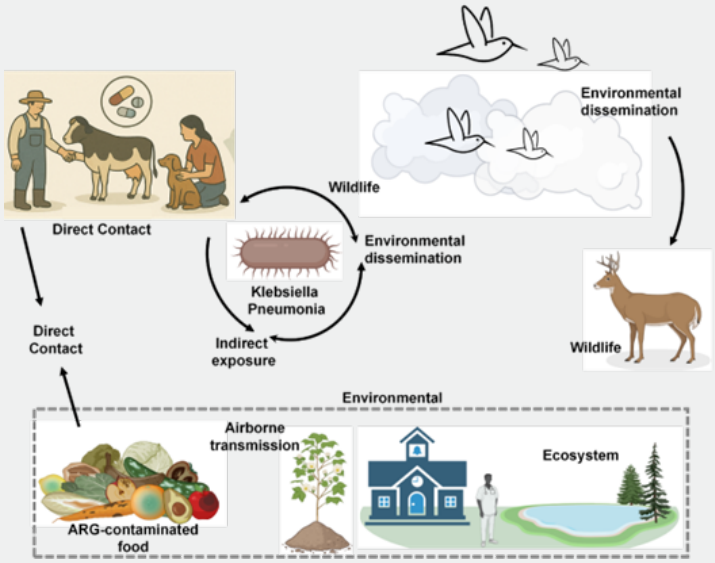

Figure 1: This figure delineates essential transmission pathways of K. pneumoniae, including direct contact (farm workers, human-pet interaction), indirect exposure (ARG-contaminated food, animal manure as fertilizer, and wastewater treatment plants), and environmental dissemination (migrating birds, wildlife, and airborne transmission), thereby elucidating the complex dynamics among animals, humans, and the environment. The use of antibiotics in humans, pets, and agricultural animals' results in the emergence of resistant bacteria, which contaminate unspoiled habitats or ascend the food chain, creating cyclic transmission loops. Healthcare systems, agricultural settings, and ecosystems with coexisting human, animal, and environmental interactions are examples of hotspots https://onlinelibrary.wiley.com/doi/full/10.1002/mlf2.12101.

The endemic increase in *K. pneumoniae*-producing ESBL enzymes, which exhibit reduced or no response to many essential and effective antibiotics, has led to a significant increase in the use of carbapenem drugs. The development of plasmid-mediated carbapenemases is a result of the widespread usage of carbapenem medicines. Carbapenem drugs are considered the last resort of antibacterials that can eradicate ESBL-producing pathogens. Imipenem was the first carbapenem to be used against Klebsiella infections in 1983, and the first carbapenem-resistant strain was reported only two years after its use [45,46]. A decade later, the extensive use of carbapenem drugs, *K. pneumoniae* carbapenemase (KPC), was described in the United State (US) [47]. Clinicians face a significant therapeutic challenge since KPC is well known for its resistance to all varieties of β-lactams and β-lactamase inhibitors [48].

Genetic Elements Responsible for Uptake and Dissemination of AR

Generally, Enterobacteriaceae, including *K. pneumoniae*, have adopted an array of mechanisms for the uptake and dissemination of drug resistance elements. This includes but is not limited to plasmids encoding drug resistance elements, uptake of gene cassettes, production of enzymes and other metabolites, and efflux of active drug molecules, etc. [17,49]. A successful bacterial strain should behave as an incredibly efficient vector for the spread of antibiotic resistance traits in the event of antibiotic resistance. This can be accomplished if the organism is capable of vertical AR element transmission and serves as a direct donor of horizontally mobile genetic elements, such as plasmids and transposons (Woodford et al., 2011). *K. pneumoniae* possesses a flexible genome that can mutate in response to stress. This mutation helps them to maintain their genome integrity and maintain their natural resistance. *K. pneumoniae* isolates have frequently been shown to contain transposons, plasmids, and integrons that carry genes for antibiotic resistance [50-52].

Epidemiology of *K. pneumoniae* causing mastitis in dairy animals

Clinical mastitis–causing pathogens vary geographically. Historically, Gram-positive organisms such as Staphylococcus aureus and Streptococcus agalactiae were the major agents in Europe and other regions; however, strict hygiene protocols have significantly reduced their prevalence. In contrast, systematic yearly epidemiological data on Klebsiella mastitis remain limited,

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with most available information coming from isolated studies worldwide [27, 53].

In the United States, approximately 40% of mastitis cases are attributed to Gram-negative bacteria [54], predominantly coliforms such as E. coli and *K. pneumoniae*. Klebsiella mastitis is more frequently observed in cattle kept indoors during winter, whereas its occurrence in pasture-grazed animals is uncommon [55]. A recent study from Japan reported the bla_{CTX-M-2} gene in most ESBL-producing *K. pneumoniae* isolated from mastitic milk, consistent with earlier findings of MDR Klebsiella in livestock, food sources, and the environment [56]. In several outbreaks, Klebsiella has shown remarkable pathogenicity. For example, in the US in 2006, the susceptibility of dairy herds to *K. pneumoniae* increased sharply, and 59% of 410 milk samples from cows with clinical mastitis yielded *K. pneumoniae* [57]. High prevalence has also been reported in Bangladesh, where Klebsiella species were detected in 62.5% of clinical mastitis cases [58]. In Southwest China, isolates exhibited high resistance to commonly used antibiotics, showing greater resistance patterns than isolates from Iran [59, 60]. In India, Klebsiella mastitis incidence has ranged from 20.16% to 24% [60, 61].

Epidemiological Status in Pakistan

Mastitis is considered the most economically important disease of cattle in Pakistan, with incidence rates varying from 15% to 80% [62–64]. Despite this, country-wide data on pathogen-specific prevalence remain fragmented. A recent 2018–2019 study reported 20% mastitis in cattle, with 8% attributed to *K. pneumoniae* [65]. In buffaloes from Sindh, *K. pneumoniae* accounted for 11% of mastitis cases [66]. In goats from Kohat (Khyber Pakhtunkhwa), prevalence was 3.6% for Klebsiella and 11.5% for E. coli.

A four-year investigation in Peshawar (2010–2013) involving 2,791 milk samples showed 81% positivity for mastitis, with 6% caused by Klebsiella [67]. However, recent unpublished data generated from over 10,000 milk samples across 11 districts of Khyber Pakhtunkhwa suggest that the true incidence of Klebsiella mastitis may be significantly higher than previously reported. Limited surveillance, high diagnostic costs, and lack of technical capacity may have contributed to underreporting. Continuous monitoring and identification of pathogens are critical for mastitis control. Evidence indicates a shift in mastitis-causing agents, highlighting the need for surveillance programs to prevent the spread of contagious Klebsiella infections in dairy herds.

One Health Surveillance and Cross-Species Transmission Risks

A One Health approach emphasizes coordinated monitoring across human, animal, and environmental sectors. Integrated surveillance programs can track antimicrobial resistance (AMR) trends in animal reservoirs and evaluate their potential transmission risks to humans. Comprehensive strain characterization is needed to understand the diversity and epidemiological links between *K. pneumoniae* in livestock and human populations [68].

Clinical Impact and Economic Burden of K. pneumoniae Mastitis

K. pneumoniae mastitis poses serious clinical and economic challenges in dairy production. Clinically, affected animals develop severe udder inflammation, reduced milk yield, fever, and abnormal milk containing clots and flakes. The condition can lead to death or necessitate culling. Compared with E. coli mastitis, *K. pneumoniae* infections are generally more severe and associated with higher culling and mortality rates [69]. Economically, the estimated total failure cost of bovine mastitis is approximately $147 per cow per year, with milk production losses and culling accounting for 11–18% of the gross margin per cow. Damage to mammary tissue accounts for 70% of total losses. Treatment is complicated by poor antibiotic response, and costs are often driven by milk discard due to antibiotic residues.

Alternative therapies, including bacteriophage-based approaches, are being explored to reduce inflammation and bacterial load [1]. Vaccines have shown variable success, and further research is needed to develop effective immunization strategies against *K. pneumoniae* mastitis [70].

Future Direction and Research Gap

AMR studies should concentrate on examining the molecular mechanisms of AMR and developing novel therapeutic strategies [71]. Vaccine development should explore potential candidates to prevent *K. pneumoniae* infections. Extensive genomic studies are required to understand strain diversity and transmission dynamics [72]. Although *K. pneumoniae* is known to cause mastitis, significant gaps remain in understanding, including unclear AMR mechanisms, limited livestock surveillance, and virulent factors associated with *K. pneumoniae* strains. In the future, researchers should concentrate on One Health surveillance and genomic analyses to understand strain diversity, resistance, and host interactions.

Conclusion

ESBL-producing *K. pneumoniae* poses a major threat to dairy herds due to its severe disease impact, treatment difficulty, and ability to spread antimicrobial resistance. Preventive measures-especially during wet and winter seasons-should focus on strict hygiene, proper milking equipment sanitation, prudent antibiotic use, and improved bedding and moisture control. At a larger scale, a coordinated One Health approach with continuous surveillance and molecular monitoring is essential to detect resistant strains early and prevent their spread across animals, humans, and the environment.

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