



URINARY TRACT INFECTIONS DRIVEN BY MULTI-DRUG-RESISTANT
ESCHERICHIA COLI IN FELINES: A COMPREHENSIVE CLINICAL REVIEW OF
RESISTANCE MECHANISMS, PATHOGENESIS, AND FUTURE THERAPEUTICS

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ABSTRACT

Feline lower urinary tract disease (FLUTD) represents a highly complex and multifaceted clinical challenge in veterinary medicine, characterized by significant morbidity, economic burden on pet owners, and immense diagnostic complexity. Within the broad umbrella of FLUTD, bacterial urinary tract infections (UTIs) account for a substantial subset of cases, heavily disproportionate in older felines and those suffering from systemic comorbidities such as chronic kidney disease, diabetes mellitus, or hyperthyroidism. Decades of microbiological surveillance have unequivocally established *Escherichia coli* specifically the uropathogenic *E. coli* (UPEC) pathotype as the predominant etiological agent responsible for the vast majority of feline bacterial cystitis and pyelonephritis cases. Historically considered highly susceptible to empirical first-line antimicrobials, feline UPEC has recently undergone a dramatic evolutionary shift, mirroring the crisis seen in human medicine. The rapid and relentless emergence of multi-drug resistant (MDR) *E. coli*, particularly strains producing extended-spectrum beta-lactamases (ESBLs) such as the CTX-M family, alongside high-level fluoroquinolone resistance, severely complicates empirical therapy. This escalating resistance is driven by high-risk, intercontinental clonal lineages, most notably Sequence Type 131 (ST131), which has successfully breached the species barrier to transmit bidirectionally between humans and companion animals within shared households. This comprehensive, extended review critically synthesizes the contemporary literature surrounding the molecular pathogenesis of feline UPEC, providing an in-depth analysis of fimbria adhesion, intracellular bacterial community (IBC) formation, and host immune evasion. It extensively delineates the genetic and molecular mechanisms of antimicrobial resistance, focusing on plasmid-mediated horizontal gene transfer and chromosomal mutations in the quinolone resistance-determining regions. Furthermore, this review evaluates the current diagnostic paradigms established by the 2026 International Society for Companion Animal Infectious Diseases (ISCAID) guidelines, distinguishing subclinical bacteriuria from true infection. Finally, we explore the cutting edge of novel and alternative therapeutic interventions, including bacteriophage therapy and virulence inhibitors, establishing a vital framework for aggressive antimicrobial stewardship and the preservation of critical antimicrobial efficacy in companion animal medicine.

Keywords: Feline lower urinary tract disease; feline; pathogenesis; Multi-Drug Resistant; *Escherichia coli*; biosecurity; disease control

Introduction

Feline lower urinary tract disease (FLUTD) encapsulates a diverse spectrum of pathological conditions affecting the urinary bladder and urethra of the domestic cat. While sterile and idiopathic etiologies, such as feline idiopathic cystitis (FIC) and non-infectious urolithiasis, dominate the clinical landscape in younger, otherwise healthy felines, bacterial urinary tract infections (UTIs) emerge as a highly significant, persistent, and potentially life-threatening complication in specific patient demographics (Dorsch et al., 2015). According to highly comprehensive systematic reviews, including a pivotal 2024 meta-analysis encompassing thousands of veterinary clinical isolates across Europe and North America, the estimated prevalence of UTIs in cats presenting with signs of LUTD is approximately 18.6% (Hritcu et al., 2024). This figure, while lower than the corresponding prevalence in canines, escalates dramatically in geriatric feline populations or in those burdened with systemic immunosuppressive comorbidities.

Among the myriad of potential bacterial, fungal, and viral uropathogens, *Escherichia coli* consistently and overwhelmingly ranks as the undisputed primary etiological agent. Broad-scale epidemiological surveillance data derived from the extensive ComPath II and III European veterinary programs definitively reaffirm that *E. coli* accounts for 48.3% to 61.2% of all culture-positive feline UTIs, maintaining striking stability over recent decades (Moyaert et al., 2024). This absolute dominance is not merely circumstantial, nor is it simply a product of anatomical proximity to the gastrointestinal tract. Rather, it is the result of a highly evolved, multifaceted, and deeply entrenched parasitic relationship between the pathogen's sophisticated virulence machinery and the unique physiology of the feline uroepithelium. Spontaneously occurring bacterial UTIs in companion animals are predominantly driven by a highly specialized subset of extraintestinal pathogenic *E. coli* (ExPEC) strains, specifically designated as uropathogenic *E. coli* (UPEC).

In recent years, veterinary diagnostic laboratories and clinics worldwide have reported a profoundly worrying and accelerating surge in the isolation of multi-drug resistant (MDR) *E. coli* from feline urinary tracts. The uninhibited proliferation of these strains which are frequently resistant to critically important first-line veterinary agents such as amoxicillin-clavulanate, potentiated sulfonamides, and early-generation cephalosporins necessitates a much deeper, molecular-level understanding of the genetic drivers of resistance (Saputra et al., 2017). The veterinary profession is rapidly moving away from an era of reliable empirical success into a landscape fraught with therapeutic failures, chronic relapsing infections, and the daunting prospect of pan-drug resistant uropathogens. Furthermore, the intimate physical proximity between companion animals and their owners dictates that pets may serve as highly effective reservoirs

and amplifiers for MDR bacteria, transferring resistance plasmids and virulent clones to human counterparts. This reality firmly elevates feline MDR UTIs from an isolated veterinary issue to a paramount global public health concern falling directly under the 'One Health' dimension.

Feline Epidemiology and Predisposing Risk Factors

Unlike the canine urinary tract, where ascending bacterial infections are relatively commonplace across various life stages, the feline urinary tract particularly in young adult cats possesses highly effective innate defense mechanisms. Normal feline urine is typically highly concentrated (often exceeding a specific gravity of 1.035), highly acidic, and rich in urea and organic acids, creating an inherently hostile and bacteriostatic environment that vigorously suppresses bacterial colonization and multiplication. Consequently, the development of a bacterial UTI in a feline patient is rarely a primary event; it is almost invariably secondary to a breakdown in one or more of these robust local or systemic host defense mechanisms.

Demographics and Intrinsic Host Factors

Age is the single most powerful independent risk factor for the development of bacterial UTIs in cats. The incidence of culture-positive bacteriuria in cats younger than 10 years of age exhibiting signs of FLUTD (such as dysuria, pollakiuria, hematuria, or periuria) is remarkably low, frequently estimated at less than 2-5%. In stark contrast, cats over 10 years of age presenting with similar clinical signs demonstrate a UTI prevalence soaring to 40-50% (Dorsch et al., 2015). Female cats are generally considered at a slightly higher risk than intact or neutered males due to the comparatively shorter and wider anatomical structure of the female urethra, which more readily facilitates the physical ascent of fecal and perineal flora into the sterile bladder environment.

The Role of Comorbidities: Chronic Kidney Disease and Diabetes Mellitus

The dramatic age-related spike in UTI incidence is intrinsically linked to the parallel onset of geriatric diseases. Chronic kidney disease (CKD) is highly prevalent in older cats. As renal concentrating ability diminishes, the resulting isosthenuric or hyposthenuric urine (specific gravity < 1.025) loses its critical bacteriostatic properties, allowing opportunistic *E. coli* to thrive. Up to 30% of cats with stable, naturally occurring CKD are found to have subclinical bacteriuria or active UTIs upon screening. Similarly, Diabetes mellitus profoundly alters the urinary microenvironment. Glucosuria provides an abundant, easily accessible carbohydrate energy substrate that fuels rapid exponential bacterial replication. Furthermore, the systemic immunosuppression, altered neutrophil chemotaxis, and potential peripheral neuropathies (leading to incomplete bladder emptying) associated with diabetes synergistically compound the risk of infection. Hyperthyroidism, common in geriatric cats, also indirectly

predisposes them to UTIs through its secondary effects on renal hemodynamics and urine concentration.

Iatrogenic and Anatomic Interventions

Veterinary medical interventions often inadvertently breach host defenses. The placement of indwelling urinary catheters for the management of urethral obstruction (a common emergency in male cats) bypasses the natural flushing mechanism of the urethra, creating a direct physical conduit for nosocomial bacterial entry and providing an artificial surface highly conducive to bacterial biofilm formation. Surgical interventions, most notably perineal urethrostomy (PU), dramatically alter the lower urinary tract anatomy, widening the urethral orifice and removing the distal urethral high-pressure zone, significantly increasing the lifelong risk of ascending *E. coli* infections.

The Arsenal of UPEC: Virulence Factors and Pathogenesis

The profound ability of UPEC to successfully colonize the highly dynamic and hostile environment of the feline urinary tract is entirely dependent on an expansive and tightly regulated arsenal of virulence factors. The pathogenesis of UPEC infection is a complex, multi-stage cascade involving adhesion, invasion, immune evasion, toxigenesis, and nutrient acquisition (Babacan & İzgür, 2020).

Adhesion Mechanics: Fimbrial and Afimbrial Structures

The critical first step in pathogenesis is the firm adherence of the bacterium to the transitional epithelial cells (umbrella cells) lining the bladder mucosa. This adhesion must be strong enough to withstand the immense mechanical shear forces generated by the periodic flushing of urine during micturition. UPEC achieves this primarily through the expression of hair-like proteinaceous appendages known as fimbriae or pili. Type 1 fimbriae are the most universally expressed and critically important adhesins in the lower urinary tract. At the distal tip of the Type 1 fimbria sits the FimH adhesin protein, which possesses a highly specific binding affinity for mannosylated uroplakin receptors (specifically uroplakin Ia and Ib) abundantly coating the surface of feline umbrella cells. P fimbriae, tipped with the PapG adhesin, bind to digalactoside receptors and are more closely associated with ascending infections and pyelonephritis, as these receptors are highly expressed in the renal pelvis and tubular epithelium.

Intracellular Bacterial Communities (IBCs) and Immune Evasion

iron away from host binding proteins and transporting it back into the bacterial cell via highly specific outer membrane receptors (e.g., IutA for aerobactin). The robust

The interaction between FimH and host uroplakins is not merely adhesive; it triggers a profound signaling cascade within the host cell. This interaction induces localized reorganization of the host actin cytoskeleton, prompting the umbrella cell to actively engulf the attached *E. coli*. By invading the host cell, UPEC achieves several critical survival objectives: it escapes the mechanical washout of urine, it hides from circulating host immune cells (such as polymorphonuclear neutrophils), and it physically shields itself from the action of many antimicrobial agents that possess poor intracellular penetration. Once inside the cytoplasm, UPEC rapidly replicates to form massive, biofilm-like aggregates known as Intracellular Bacterial Communities (IBCs). As the IBC matures, the bacteria detach, become highly motile, and flux out of the host cell often triggering host cell apoptosis and exfoliation to rapidly infect adjacent naïve epithelial cells. Furthermore, a subset of UPEC can invade deeper layers of the transitional epithelium, entering a dormant, metabolically inactive state known as Quiescent Intracellular Reservoirs (QIRs). These QIRs are entirely refractory to antibiotic therapy and can persist for months or years, ultimately serving as the hidden source for chronic, relapsing UTIs in felines long after the initial infection was seemingly cured.

Toxigenesis and Tissue Damage

To acquire vital nutrients, particularly iron, and to facilitate deeper tissue invasion, UPEC secretes a variety of potent exotoxins. Alpha-hemolysin (HlyA) is a pore-forming lipoprotein toxin that inserts itself into host cell membranes. At low concentrations, HlyA interferes with host cell signaling and induces apoptosis; at high concentrations, it causes outright lysis of erythrocytes, uroepithelial cells, and immune cells, releasing a massive influx of host nutrients into the surrounding environment. Cytotoxic Necrotizing Factor 1 (CNF1) is another critical toxin that constitutively activates host Rho GTPases. This manipulation results in profound cytoskeletal alterations, inhibiting host cell division, promoting cellular multinucleation, and significantly enhancing the intracellular invasion efficiency of UPEC, while simultaneously blunting the phagocytic capacity of responding neutrophils.

Siderophores

Iron is an absolute requirement for bacterial metabolism, yet the mammalian urinary tract is an extremely iron-poor environment, as host proteins like transferrin and lactoferrin tightly sequester the metal. To overcome this severe nutritional limitation, UPEC synthesizes and secretes specialized, high-affinity iron-chelating molecules known as siderophores. Aerobactin, enterobactin, and yersiniabactin are the primary siderophores utilized by feline UPEC. These molecules are capable of tearing ferric

expression of multiple redundant siderophore systems is a hallmark of highly virulent UPEC strains, enabling them to thrive in the face of intense host nutritional immunity.

Table 1. Primary Virulence Factors in Feline Uropathogenic Escherichia coli

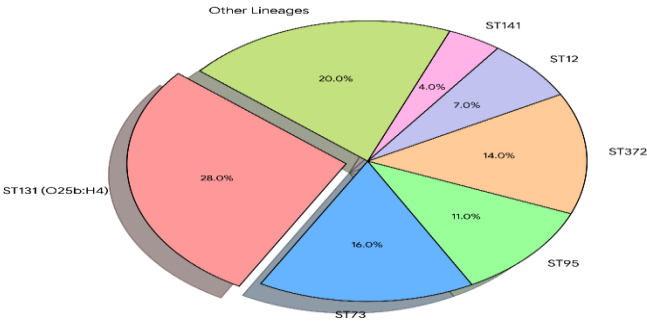
Virulence Factor Class	Specific Mechanism / Gene	Biological Function in Pathogenesis
Adhesins	Type 1 fimbriae (FimH)	Binds mannosylated uroplakins; prevents washout; triggers host cell invasion.
Adhesins	P fimbriae (PapG)	Binds digalactosides; facilitates ascent to kidneys (pyelonephritis).
Toxins	Alpha-hemolysin (hlyA)	Pore-forming toxin; lyses host cells to release nutrients (iron) and destroys immune cells.
Toxins	Cytotoxic Necrotizing Factor 1 (cnf1)	Modulates host cytoskeleton; promotes invasion and inhibits phagocytosis.
Iron Acquisition	Aerobactin (iucD / iutA)	High-affinity siderophore; scavenges iron from host transferrin in iron-poor urine.
Immune Evasion	Capsular polysaccharides (K antigens)	Provides a physical shield against complement-mediated lysis and opsonophagocytosis.

Genomic Epidemiology and the Zoonotic Threat: The ST131 Pandemic Clone

The global dissemination of multi-drug-resistant *E. coli* is not a random evolutionary event, but rather the result of the explosive international spread of highly successful, specific clonal lineages. When analyzing the phylogenetic

background of feline UPEC, the vast majority of isolates belong to the highly virulent phylogenetic group B2, and to a lesser extent, group D. Within these groups, advanced Multilocus Sequence Typing (MLST) has identified several dominant Sequence Types (STs) that heavily burden companion animals, including ST73, ST95, ST372, and ST12 (Bota et al., 2025).

Figure 2: Distribution of Major UPEC Sequence Types in Felines



However, the most formidable and clinically devastating lineage to emerge in recent decades is Sequence Type 131 (ST131). Originally identified as a major cause of severe, intractable extraintestinal infections in humans, the ST131 clone specifically the O25b:H4 serotype has now firmly established itself within the feline and canine populations worldwide. ST131 is uniquely dangerous because it represents a 'perfect storm' of microbiology: it combines an incredibly dense arsenal of virulence genes (facilitating aggressive tissue invasion and high survival rates) with high-level multidrug resistance. ST131 is intrinsically linked to the global spread of the blaCTX-M-15 extended-spectrum beta-lactamase gene, rendering it highly resistant to critical 3rd generation cephalosporins, whilst

concurrently carrying chromosomal mutations conferring profound resistance to fluoroquinolones.

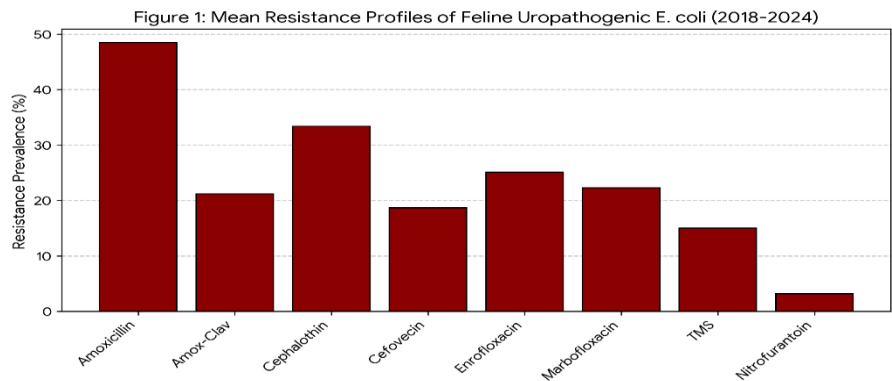
The zoonotic implications of ST131 in felines cannot be overstated. High-resolution whole-genome sequencing and core-genome MLST studies have definitively proven that companion animals and their human owners frequently share genetically indistinguishable ST131 strains. Research conducted by Platell et al. (2011) and Johnson et al. (2008) documented multiple instances of >94% pulsed-field gel electrophoresis (PFGE) profile similarity between human and pet ST131 isolates residing in the same household. This bidirectional transmission occurs through close physical contact, shared living environments, and the shedding of resistant bacteria in feline feces and urine,

creating an immense challenge for household infection control and public health surveillance.

Molecular Mechanisms of Antimicrobial Resistance

The clinical management of feline UTIs has been severely compromised by the rapid acquisition and horizontal

transfer of resistance genes. UPEC employs highly complex molecular strategies to neutralize almost every major class of antimicrobial agent utilized in veterinary practice (Poirel et al., 2018).



Beta-Lactam Resistance: ESBLs and AmpC Cephalosporinases

Resistance to beta-lactam antibiotics (penicillins, cephalosporins) is predominantly mediated by the bacterial production of beta-lactamase enzymes, which chemically hydrolyze the crucial four-membered beta-lactam ring, rendering the drug totally inactive. In feline UPEC, the most critical threat is the widespread emergence of Extended-Spectrum Beta-Lactamases (ESBLs). The CTX-M family (specifically CTX-M-15, CTX-M-1, and CTX-M-14) are now the most prevalent ESBLs worldwide (Zogg et al., 2018). These enzymes possess a massively expanded substrate profile, efficiently destroying penicillins, first- and second-generation cephalosporins, and critically, third-generation cephalosporins such as cefovecin (Convenia) and cefpodoxime, which are heavily marketed and utilized in feline medicine due to their convenient long-acting dosing regimens. Because ESBL genes are almost exclusively located on highly mobile conjugative plasmids, they rapidly disseminate horizontally between different E. coli strains and even across different bacterial species in the feline gut and urinary microbiome. In addition to ESBLs, the plasmid-mediated acquisition of AmpC beta-lactamases (such as CMY-2) confers robust resistance not only to cephalosporins but also to beta-lactam/beta-lactamase inhibitor combinations, such as amoxicillin-clavulanic acid

(Clavamox), stripping veterinarians of a primary empirical defence.

Fluoroquinolone Resistance: Chromosomal Mutations and PMQR

Fluoroquinolones (e.g., enrofloxacin, marbofloxacin, pradofloxacin) are critical, concentration-dependent bactericidal agents that act by directly inhibiting bacterial DNA gyrase and topoisomerase IV enzymes essential for DNA replication. High-level clinical resistance in feline UPEC is primarily driven by stepwise chromosomal point mutations occurring within highly conserved regions known as the Quinolone Resistance-Determining Regions (QRDRs) of the gyrA and parC genes. These specific mutations physically alter the target binding site, preventing the fluoroquinolone molecule from docking with the enzyme complex. Furthermore, the situation is exacerbated by the insidious spread of Plasmid-Mediated Quinolone Resistance (PMQR) determinants. Genes such as qnr produce small proteins that physically bind to and shield DNA gyrase from fluoroquinolones. While PMQR genes alone typically only confer low-level clinical resistance, they critically raise the Minimum Inhibitory Concentration (MIC) of the bacteria, allowing the strain to survive low-dose drug exposure and buying it the necessary time to rapidly acquire the high-level chromosomal QRDR mutations, inevitably leading to catastrophic treatment failure.

Table 2. Key Antimicrobial Resistance Mechanisms in Feline UPEC

Antimicrobial Class	Primary Molecular Mechanism	Key Genes / Mutations	Clinical Impact in Feline Medicine
Beta-lactams (Penicillins)	Enzymatic hydrolysis	blaTEM, blaSHV	High background resistance to ampicillin/amoxicillin.
3rd Gen Cephalosporins (Cefovecin)	Extended-Spectrum Beta-Lactamases (ESBL)	blaCTX-M-15, blaCTX-M-1	Total failure of long-acting injectable cefovecin therapies.

Amoxicillin-Clavulanate	AmpC Beta-lactamases	blaCMY-2	Neutralizes the protective effect of clavulanic acid.
Fluoroquinolones	Target site alteration & Shielding	gyrA, parC, qnr	Loss of critical 2nd-line bactericidal agents.
Trimethoprim-Sulfamethoxazole	Folate pathway bypass	sul1, sul2, dfrA	Limits options for highly penetrative oral therapy.

Clinical Management and 2026 ISCAID Guidelines

The shifting, highly volatile susceptibility landscape dictates that the clinical management of feline UTIs must be fundamentally re-evaluated. The era of indiscriminate empirical therapy has concluded. The International Society for Companion Animal Infectious Diseases (ISCAID) recently updated their comprehensive guidelines in 2026 to combat the AMR crisis directly (Weese et al., 2019; ISCAID, 2026 updates).

A cornerstone of the revised ISCAID framework is the absolute diagnostic differentiation between 'Subclinical Bacteriuria' and true 'Bacterial Cystitis'. Subclinical bacteriuria is defined by the presence of bacteria in the urine (often identified via culture) in the complete absence of lower urinary tract clinical signs. Current consensus strongly, unequivocally recommends against treating subclinical bacteriuria with antimicrobials in felines, even in cases of systemic disease like CKD or diabetes, unless there is compelling evidence of active tissue invasion (e.g., pyelonephritis). Treating asymptomatic bacterial carriage selectively eradicates susceptible commensals, creating a microbiological vacuum that is rapidly filled by highly resistant MDR strains, ultimately causing more harm than the initial colonization.

For true clinical infections, gold-standard diagnosis mandates aerobic urine culture and rigorous antimicrobial susceptibility testing (AST), optimally utilizing quantitative Minimum Inhibitory Concentration (MIC) data rather than simple disk diffusion, particularly for recurrent cases. Empirical therapy, if absolutely clinically necessitated while awaiting culture results, must prioritize narrow-spectrum agents (e.g., simple amoxicillin or trimethoprim-sulfonamides) over critically important human drugs (fluoroquinolones, 3rd generation cephalosporins). The guidelines also advocate for significantly shorter durations of therapy; uncomplicated bacterial cystitis often requires only 3 to 5 days of targeted antimicrobial therapy, drastically reducing the selective pressure for resistance, whereas deep tissue infections (pyelonephritis, prostatitis) necessitate prolonged, carefully monitored courses of 10-14 days with highly tissue-penetrant drugs.

Novel and Alternative Therapeutics

Given the terrifying trajectory of MDR UPEC, the veterinary pipeline must urgently diversify beyond traditional small-molecule antibiotics. Several promising non-antimicrobial therapeutics are currently under intense investigation. Bacteriophage therapy the targeted

utilization of lytic bacterial viruses to specifically infect, replicate within, and destroy *E. coli* is showing immense promise in compassionate-use veterinary trials. Because phages utilize entirely distinct killing mechanisms compared to antibiotics, they remain highly effective against even pan-resistant ESBL and ST131 strains. Furthermore, phages possess the unique ability to penetrate and degrade the extracellular polymeric matrix of bacterial biofilms and IBCs, feat traditional antibiotics struggle to achieve.

Additionally, anti-virulence strategies aim to disarm rather than kill the pathogen, theoretically applying far less selective evolutionary pressure for resistance. D-mannose is a simple sugar that acts as a highly effective competitive molecular decoy for the FimH adhesin of Type 1 fimbriae. By saturating the binding sites of the bacteria in the urinary lumen, D-mannose actively prevents UPEC from docking onto the uroepithelium, allowing the bacteria to be harmlessly flushed out during urination. Cranberry extracts, rich in proanthocyanidins (PACs), exert a similar anti-adhesive effect, specifically targeting the P fimbriae associated with ascending pyelonephritis. While current clinical data in felines remains mixed, these nutraceutical interventions offer highly attractive adjunctive therapies for the long-term management of chronic relapsing UTIs, potentially sparing felines from endless, highly toxic antibiotic cycles.

Conclusion

Urinary tract infections driven by multi-drug-resistant *Escherichia coli* represent a formidable, rapidly evolving, and profoundly complex threat in contemporary feline urology. The intricate intersection of highly adapted evolutionary virulence factors, capable of establishing impenetrable intracellular reservoirs, alongside highly mobile transmissible resistance plasmids, severely compromises traditional clinical management. The dissemination of pandemic lineages such as ST131 highlights an urgent One Health imperative, underscoring the reality that companion animals act as highly integrated reservoirs within the broader ecology of antimicrobial resistance. The veterinary profession must aggressively pivot toward rigorous, culture-driven diagnostic stewardship, strict adherence to updated ISCAID guidelines, and the intense pursuit of novel, non-antibiotic therapeutic modalities to preserve the efficacy of our diminishing antimicrobial arsenal and ensure the continued health of both feline patients and their human companions.

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