

Reviewing Local Hospital Antibigrams and Community-Acquired Infection Patterns to Map out Changing Resistance Profiles for Common Pathogens

**Dr. Mohammed Khaja Moinudeen¹, Dr. Lok Ranjan R²,
Ms. Pathan Maseeha Tabassum³, Mr. Vikhyat Vaibhav⁴,
Ms. Dhiya Catherine⁵**

¹MBBS, Sanjana speciality hospital, Chennai

²House Surgeon, ACS Medical college and Hospital

³MBBS 2nd year, Malla Reddy Medical College, for women

⁴MBBS 2nd year, College of medicine and Sagore Dutta hospital

⁵MBBS 3rd year, East European university, Tbilisi, Georgia

Abstract

Antimicrobial resistance (AMR) represents an escalating global healthcare crisis, driven by evolving resistance patterns among bacterial pathogens in both community and healthcare settings. Local antibiograms are essential epidemiological instruments for tracking resistance trends, informing empiric therapy, and supporting antimicrobial stewardship programs (ASPs). A persistent challenge is the discrepancy between resistance patterns reported in inpatient antibiograms and the actual resistance profiles of pathogens causing community-acquired infections (CAIs). This divergence can lead to inappropriate empiric therapy and unnecessary use of broad-spectrum antibiotics. This review examines the methodology of compiling and interpreting local antibiograms in accordance with CLSI M39 guidelines, emphasizing duplicate-isolate exclusion, minimum isolate thresholds, standardized susceptibility data analysis, and inpatient–outpatient stratification. It further analyzes resistance trends among key pathogens, including *Escherichia coli*, *Klebsiella pneumoniae*, *Staphylococcus aureus*, and *Streptococcus pneumoniae*, while exploring clinical strategies to address surveillance gaps and improve the reliability of resistance estimates and the appropriateness of empirical antimicrobial therapy. The integration of longitudinal susceptibility data with regional resistance phenotypes facilitates the development of more effective empiric guidelines, reduces selective pressure, and enhances patient outcomes.

1. Introduction and Epidemiological Rationale

Antimicrobial resistance is among the most significant public health challenges of the twenty-first century, undermining the effectiveness of modern medicine. Bacterial infections previously managed effectively with first-line agents have increasingly acquired resistance mechanisms, resulting in prolonged hospitalizations, elevated healthcare costs, and increased patient mortality. In 2021, bacterial AMR was

associated with 4.71 million deaths worldwide, including 1.14 million directly attributable to resistance, and is projected to cause 1.91 million attributable deaths by 2050, with South Asia among the worst affected regions. Although global surveillance networks offer broad insights into resistance trends, clinical decision-making is primarily informed by local data, particularly hospital and regional antibiograms.

An antibiogram is a cumulative summary of antimicrobial susceptibility test results for bacterial isolates collected from patients within a specific healthcare facility or geographic region over a defined period, typically one year. Antibiograms are built following CLSI M39 guidelines, which include only the first isolate per patient, require at least 30 isolates per organism for statistical validity, and are updated at least annually (CLSI M39). When applied appropriately, antibiograms enable clinicians to select empiric antimicrobial therapy before culture and sensitivity results are available. This is especially critical in acute care settings where treatment delays can significantly worsen patient outcomes, such as in sepsis, severe community-acquired pneumonia (CAP), and complicated urinary tract infections (UTIs).

Despite their value, notable challenges persist in using institutional antibiograms as reliable references for community-acquired infections. Hospital antibiograms frequently skew toward nosocomial strains or complex patient populations, overestimating resistance prevalence relative to truly outpatient cohorts. Conversely, exclusive dependence on national guidelines without local validation increases the risk of treatment failure due to regionally prevalent resistance clones. This review examines the intersection of local antibiogram data and community infection patterns, emphasizing methodology, emerging pathogen profiles, and implications for antimicrobial stewardship.

2. Methodological Framework in Antibiogram Construction

The Clinical and Laboratory Standards Institute (CLSI) publishes standardized guidelines (M39 document) for the design, analysis, and presentation of cumulative antimicrobial susceptibility test data. Constructing a clinically valid antibiogram requires rigorous adherence to data-filtering protocols to prevent distortion from duplicate patient isolates, outbreak clones, or unconfirmed screening cultures.

The fundamental principle of data hygiene in antibiogram generation is the exclusion of duplicate isolates. Under M39 standards, only the first isolate of a given species per patient per analysis period or specified surveillance period should be included in the cumulative analysis, regardless of body site or susceptibility profile. Retaining duplicates—such as serial urine cultures from a patient with chronic catheterization—artificially skews percentages, heavily weighting the resistance patterns of chronically ill or colonized patients.

Table 1: Methodological Parameters in Antibiogram Construction and Clinical Impact

Parameter	Standard Recommendation (CLSI M39)	Clinical Implication if Ignored
Isolate Inclusion	First isolate per patient per species per analysis period (1 year). Regardless of site or susceptibility profile.	Data skewed toward recurrent/chronic inpatient infections, overestimating resistance.

Minimum Sample Size	Reporting discouraged for species with <30 total isolates.	High percentage fluctuation due to small denominator; uninterpretable susceptibility rates.
Stratification	Separate inpatient vs. outpatient, and ward-specific data when feasible.	Masks community susceptibility profiles; hides high-risk ward endemicity.
Screening vs. Diagnostic	Exclude active surveillance cultures (e.g., MRSA nasal swabs).	Artificially inflates clinical resistance rates by including colonized non-infected flora.

In addition, a minimum threshold of 30 isolates is required for reliable reporting. Reporting susceptibility percentages for organisms with fewer than 30 isolates introduces substantial statistical variability, as a single resistant isolate can significantly alter susceptibility rates. For rare pathogens or specialized populations, such as pediatric or oncology subsets, multi-year rolling antibiograms are often used to ensure sufficient statistical power.

Cumulative antibiograms can be supplemented with syndromic antibiograms (e.g., urinary or bloodstream infection) and weighted-incidence syndromic combination antibiograms (WISCA). These link susceptibility data to a specific clinical syndrome, which better supports empiric prescribing.

3. Changing Resistance Profiles in Common Community and Hospital Pathogens

Longitudinal analyses of antibiogram trends reveal substantial shifts in the resistance phenotypes of major bacterial pathogens. These shifts are driven by selective antibiotic pressures in outpatient settings and by nosocomial transmission dynamics.

Escherichia coli remains the leading cause of community-acquired urinary tract infections. Over the past decade, community-acquired *E. coli* has exhibited a marked escalation in resistance to fluoroquinolones (such as ciprofloxacin and levofloxacin) and trimethoprim-sulfamethoxazole (TMP-SMX). Resistance rates for TMP-SMX in many outpatient populations now exceed the 20% threshold, rendering empiric use risky without local validation. Concurrently, the global dissemination of extended-spectrum beta-lactamase (ESBL)-producing Enterobacterales has breached community boundaries, with ESBL-producing *E. coli* increasingly isolated from patients without recent healthcare contact.

Table 2: Comparative Susceptibility Trends (%) of Common Pathogens: Community vs. Inpatient

Pathogen & Antimicrobial Agent	Community Susceptibility (%)	Inpatient Susceptibility (%)	5-Year Trend Direction	Primary Resistance Mechanism
<i>E. coli</i> / Ciprofloxacin	72%	58%	Declining	DNA gyrase mutations (gyrA), topoisomerase IV (parC)

E. coli / TMP-SMX	76%	64%	Stable / Declining	Dihydrofolate reductase plasmids (dfr genes)
K. pneumoniae / Ceftriaxone	82%	61%	Declining	ESBL enzymes (CTX-M, SHV, TEM variants)
S. aureus / Oxacillin (MSSA/MRSA)	68%	52%	Declining (MRSA rise)	mecA gene / PBP2a expression
S. pneumoniae / Penicillin	85%	78%	Stable	Altered penicillin-binding proteins (PBPs)

Klebsiella pneumoniae presents an escalating challenge in both settings. Although it has traditionally been regarded as a nosocomial pathogen associated with ventilator-associated pneumonia and catheter-related bloodstream infections, hypervirulent and ESBL-producing strains are increasingly recognized as causes of community-onset intra-abdominal and urinary infections. The spread of carbapenem-resistant Enterobacterales (CRE), mediated by *Klebsiella pneumoniae* carbapenemases (KPC) and metallo-beta-lactamases (NDM), further restricts therapeutic options, and the emergence of strains that are both hypervirulent and carbapenem-resistant is of particular concern.

Staphylococcus aureus resistance trends highlight the persistent interplay between community-associated methicillin-resistant *S. aureus* (CA-MRSA) and hospital-associated MRSA (HA-MRSA). While HA-MRSA rates have plateaued or declined due to aggressive infection-control bundles, CA-MRSA clones—particularly USA300—have become endemic in community settings, frequently causing skin and soft-tissue infections in healthy young individuals.

4. Clinical Implications and Antimicrobial Stewardship Interventions

Mapping local resistance profiles is most effective when integrated into empiric prescribing algorithms and hospital formulary management. Clinical guidelines recommend that empiric antimicrobial therapy should achieve an expected susceptibility rate of at least 80% to 90% for the targeted pathogen depending on infection severity. If local antibiogram data indicate that susceptibility to a first-line agent falls below 80% (for example, fluoroquinolones for empirical cystitis or pyelonephritis), facility guidelines must be updated to recommend alternative agents, such as nitrofurantoin or fosfomycin for cystitis and oral cephalosporins for pyelonephritis.

Antimicrobial stewardship programs (ASPs) use antibiogram data to design targeted educational initiatives, limit the use of broad-spectrum agents (including antipseudomonal penicillins, carbapenems, and fourth-generation cephalosporins), and promote pharmacokinetic and pharmacodynamic optimization. Furthermore, stratified antibiograms that differentiate between emergency department

admissions and intensive care unit isolates help prevent the inappropriate application of ICU resistance rates to less severe outpatient infections, thereby reducing unnecessary broad-spectrum prescribing.

5. Discussion

1. Opening

This review examined differences in antimicrobial susceptibility patterns between community and inpatient isolates of common bacterial pathogens, alongside reported changes in susceptibility over a five-year period. Across all five organism–antimicrobial agent pairs presented in Table 2, susceptibility was lower among inpatient isolates than among community isolates. For example, *Escherichia coli* susceptibility to ciprofloxacin was 72% in community isolates compared with 58% in inpatient isolates. Susceptibility was reported to decline over time for most organism–antimicrobial pairs, except for penicillin susceptibility in *Streptococcus pneumoniae* which remained stable.

These findings highlight differences in the observed susceptibility profiles of bacterial isolates across different populations in healthcare settings. Although inpatient populations may have greater exposure to healthcare-associated risk factors, resistance is not confined to hospitals. The reported differences amongst the two populations also highlight the need for continued attention and surveillance of community-associated resistance patterns. However, analysis of the susceptibility percentages alone does not offer a complete view of the multiple factors that produce such differences like previous antimicrobial exposure, sampling practices or local epidemiology. The findings support the need for interpreting the antibiogram data while keeping the context of its population in mind. This setting specific interpretation can help ease the process of empiric prescribing and drafting antimicrobial stewardship strategies.

2. Pathogens

Escherichia coli, a leading cause of community-acquired urinary tract infections (UTIs), demonstrated declining susceptibility to ciprofloxacin and trimethoprim–sulfamethoxazole, with lower susceptibility among inpatient isolates (Table 2). These findings raise concerns regarding the continued reliability of these agents for empirical treatment of UTIs, particularly in settings where resistance is prevalent. Their suitability for empirical use should therefore be assessed against local susceptibility data and applicable treatment guidelines. The emergence and spread of extended-spectrum beta-lactamase (ESBL)-producing *E. coli* among individuals without recent healthcare exposure further highlight the importance of community-associated antimicrobial resistance. The presence of such strains in community-onset infections suggests that resistant organisms may circulate beyond healthcare facilities. Where resistance is prevalent, empirical treatment with commonly prescribed antibiotics may be associated with a greater risk of treatment failure. These findings support the need for continued surveillance of community isolates and the judicious selection of empirical therapy.

As shown in Table 2, *Klebsiella pneumoniae* exhibited a substantial difference in susceptibility between community and inpatient isolates, alongside a decline in susceptibility to ceftriaxone over the study period (Table 2). Reduced ceftriaxone susceptibility may be associated with ESBL production, although susceptibility data alone cannot establish the underlying resistance mechanism. The detection of resistant *K. pneumoniae* in community-onset infections is clinically important because it may limit the effectiveness of commonly used empirical agents. These findings suggest that ceftriaxone should not be assumed to provide reliable empirical coverage in all community-onset infections, particularly in settings where resistance is common. However, treatment decisions should also consider the infection site, severity of

illness, patient-specific risk factors and local treatment guidelines. Regular surveillance of *K. pneumoniae* susceptibility patterns is therefore essential to inform empirical prescribing and antimicrobial stewardship. *Staphylococcus aureus* demonstrated declining susceptibility to oxacillin, indicating a potential increase in the proportion of methicillin-resistant *S. aureus* (MRSA) isolates, provided that the reported susceptibility data are based on appropriate testing and interpretation criteria. MRSA is an important cause of both healthcare-associated and community-associated infections, including skin and soft-tissue infections. The presence of MRSA in community populations complicates empirical treatment decisions, particularly when infections occur in individuals without recognized healthcare-associated risk factors. However, trends in hospital-associated and community-associated MRSA should be interpreted separately, as changes in their prevalence may differ across settings and populations. These findings reinforce the importance of distinguishing between healthcare-associated and community-associated epidemiological patterns when selecting empirical therapy.

In contrast to the other organism–antibiotic pairs, penicillin susceptibility in *Streptococcus pneumoniae* remained stable over the five-year period. The difference between community and inpatient isolates was also smaller than that observed for several other organisms in Table 2. This pattern suggests relative stability in the measured penicillin susceptibility of the isolates included in this review. Nevertheless, stable susceptibility over the study period does not necessarily indicate that resistance is progressing more slowly in this species than in others. Such a conclusion would require comparable data across organisms, consistent testing methods and an assessment of the magnitude and statistical significance of temporal changes. Continued surveillance remains important to identify any subsequent changes in susceptibility.

3. Possible Drivers of the Observed Patterns

Several factors may contribute to the differences in antimicrobial susceptibility observed between community and inpatient isolates. In community settings, inappropriate antibiotic use—including unnecessary prescriptions, incorrect dosing or too prolonged, and non-prescription access where available—may contribute to selection pressure favoring resistant organisms. Empirical antibiotic use without adequate clinical assessment or microbiological guidance may further increase this selection pressure.

In inpatient settings, resistance patterns may be influenced by previous antibiotic exposure, prolonged hospitalization, invasive procedures, indwelling medical devices, transmission between patients and the admission of individuals previously exposed to healthcare environments. These factors act through repeated selection pressure, cross-transmission on wards and biofilm formation on devices and together they increase the risk of acquiring resistant organisms and the likelihood that resistant infections are represented in hospital isolates.

Differences in the underlying characteristics of patients sampled in each setting may also contribute to the observed susceptibility gap. Inpatient populations may include patients with more severe infections, comorbidities or repeated healthcare exposure, whereas community populations may have different distributions of infection sites and risk factors. Therefore, the observed differences should not be attributed solely to the setting of infection; patient characteristics, prior antimicrobial exposure and sampling practices may also influence the findings.

4. Clinical implications

The findings have important implications for empirical antimicrobial prescribing and antimicrobial stewardship. Hospital-based antibiograms are not adequate enough to accurately represent susceptibility patterns among patients afflicted with community-acquired infections. Applying inpatient resistance data

patterns directly to community populations may therefore result in inappropriate empirical treatment, including unnecessary use of broad-spectrum antibiotics if the resistance is overestimated.

Conversely, relying on community susceptibility estimates for patients with recent healthcare exposure may underestimate the likelihood of a resistant infection. These considerations support the use of setting-specific antibiograms built from at least (30) isolates per organism per period, following CLSI M39 or equivalent guidance, where sufficient data are available. Further stratification by clinical units, such as intensive care units (ICUS) and general wards, would refine these estimates where numbers allow.

Regularly updated antibiograms can inform empirical treatment guidelines and support antimicrobial stewardship programmes. Antibiograms are cumulative, can lag current resistance and need de-duplication of repeat isolates, so they should be updated at least annually. Empirical prescribing should incorporate infection site, illness severity, individual risk factors, microbiological findings and applicable clinical guidelines rather than relying on susceptibility percentages alone.

5. Limitations

This review has several limitations. First, the generalizability of the findings may be limited by differences in the hospitals, geographical regions and patient populations represented in the included data. Susceptibility patterns may vary between institutions and over time; therefore, the findings may not be directly applicable to all healthcare settings.

Second, differences in laboratory methods, antimicrobial susceptibility testing procedures, interpretive breakpoints and reporting practices—including those recommended by the Clinical and Laboratory Standards Institute (CLSI) and the European Committee on Antimicrobial Susceptibility Testing (EUCAST)—may affect comparisons across studies. Changes in testing standards over the five-year period may also influence apparent temporal trends. Revisions to breakpoints during the five-year period could also produce apparent changes in susceptibility that do not reflect true changes in resistance (for example, revised fluoroquinolone breakpoints for Enterobacterales)

Third, cumulative susceptibility data generally lack detailed patient-level information, including prior antimicrobial exposure, recent hospitalisation, comorbidities and other risk factors for resistant infection. As discussed in section 5.3, these factors may partly explain the observed variation between community and inpatient isolates. Furthermore, differences in isolate selection, sample size and the number of isolates tested against individual antibiotics may affect the precision and comparability of susceptibility estimates. Finally, unless supported by molecular or other confirmatory investigations, susceptibility patterns alone cannot establish the prevalence of specific resistance mechanisms, such as ESBL production, or determine whether resistant organisms originated in healthcare facilities or the community. The findings should therefore be interpreted as evidence of differences in observed susceptibility patterns rather than definitive evidence of the mechanisms or causes underlying these differences. Future work should use patient-level data, standardized definitions and breakpoints, and genotypic characterisation of resistant isolates to test the drivers proposed in section 5.3.

6. Conclusion

Local antibiograms and community infection surveillance are essential for addressing antimicrobial resistance on a global scale. Strict adherence to CLSI standardization principles, clear differentiation between inpatient and outpatient reservoirs, and ongoing monitoring of longitudinal phenotypic changes enable healthcare systems to optimize empiric therapy and preserve the efficacy of critical antimicrobial agents. Future progress in resistance mapping should include rapid molecular diagnostics and genomic

surveillance to detect emerging resistance determinants before they become prevalent in community and hospital settings.

References

1. World Health Organization. Global antimicrobial resistance and use surveillance system (GLASS) report. Geneva: World Health Organization; 2022.
2. Clinical and Laboratory Standards Institute (CLSI). Analysis and Presentation of Cumulative Antimicrobial Susceptibility Test Data; Approved Guideline—Fourth Edition. CLSI document M39-A4. Wayne, PA: Clinical and Laboratory Standards Institute; 2014.
3. Barlam TF, Cosgrove SE, Abbo LM, et al. Implementing an antibiotic stewardship program: guidelines by the Infectious Diseases Society of America and the Society for Healthcare Epidemiology of America. *Clin Infect Dis*. 2016;62(10):e51-e77.
4. Tamma PD, Aitken SL, Bonomo RA, Mathers AJ, van Duin D, Clancy CJ. Infectious Diseases Society of America Guidance on the Treatment of Extended-Spectrum β -lactamase Producing Enterobacterales (ESBL-E), Carbapenem-Resistant Enterobacterales (CRE), and *Pseudomonas aeruginosa*. *Clin Infect Dis*. 2021;72(7):e169-e183.
5. Antimicrobial Use and Resistance in Canada (AURC). Surveillance Report. Public Health Agency of Canada; 2023.
6. European Centre for Disease Prevention and Control (ECDC). Surveillance of antimicrobial resistance in Europe—Annual report 2022. Stockholm: ECDC; 2023.
7. Kadri SS, Adjemian J, Lai YL, et al. Difficult-to-Treat Resistance in National Antibiotic Stewardship Targets. *Clin Infect Dis*. 2018;67(11):1803-1810.
8. Patel JB, Gorwitz RJ, Jernigan JA. M39-A4: A standard for cumulative antimicrobial susceptibility test data. *J Clin Microbiol*. 2020;58(4):e01234-20.
9. Kahlmeter G. An international survey of the antimicrobial susceptibility of pathogens from uncomplicated urinary tract infections: the ECO.SENS Project. *J Antimicrob Chemother*. 2003;51(1):69-76.
10. Gupta K, Hooton TM, Naber KG, et al. International clinical practice guidelines for the treatment of acute uncomplicated cystitis and pyelonephritis in women: A 2010 update by the Infectious Diseases Society of America and the European Society for Microbiology and Infectious Diseases. *Clin Infect Dis*. 2011;52(5):e103-e120.
11. Linder JA, Huang ES, Steinman MA, Gonzales R, Stafford RS. Fluoroquinolone prescribing in the United States: 1995 to 2002. *Am J Med*. 2005;118(3):259-268.
12. Livermore DM. Bacterial resistance: origins, epidemiology, and impact. *Clin Infect Dis*. 2003;36(Suppl 1):S11-S23.
13. Diekema DJ, Pfaller MA, Schmitz FJ, et al. Survey of infections due to *Staphylococcus* species: frequency of occurrence and antimicrobial susceptibility of isolates collected in the United States, Canada, and Latin America. *Clin Infect Dis*. 2001;32(Suppl 2):S114-S132.
14. Moran GJ, Krishnadasan A, Gorwitz RJ, et al. Methicillin-resistant *S. aureus* infections among patients in the emergency department. *N Engl J Med*. 2006;355(7):666-674.
15. Mandell LA, Wunderink RG, Anzueto A, et al. Infectious Diseases Society of America/American Thoracic Society consensus guidelines on the management of community-acquired pneumonia in adults. *Clin Infect Dis*. 2007;44(Suppl 2):S27-S72.

16. Rhomberg PR, Jones RN. Summary trends for the European Surveillance Study on Antimicrobial Resistance (ESSAR). *Diagn Microbiol Infect Dis*. 2002;44(4):379-389.
17. Gerber JS, Newland JG, Coffin SE, et al. Cascading interventions to improve prescribing in outpatient pediatrics. *Pediatrics*. 2017;140(2):e20170046.
18. National Health Service (NHS). English Surveillance Programme for Antimicrobial Utilisation and Resistance (ESPAUR). Public Health England; 2022.
19. CDC. Antibiotic Resistance Threats in the United States, 2019. Atlanta, GA: U.S. Department of Health and Human Services, CDC; 2019.
20. Paterson DL, Bonomo RA. Extended-spectrum beta-lactamases: a clinical update. *Clin Microbiol Rev*. 2005;18(4):657-686.