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Preface

The Antimicrobial Resistance Surveillance Program (ARSP), running on in its 38th year, is a routine antimicrobial resistance surveillance as per the World Health Organization Global Antimicrobial Resistance and Use Surveillance (WHO GLASS) framework. As a routine surveillance, ARSP tracks antimicrobial resistance on aerobic bacterial infections and collects data on culture and susceptibility tests performed as part of clinical management including corresponding demographic data. The surveillance has consistently provided data for action in the country's ongoing efforts to address AMR.



The 38th report of the Antimicrobial Resistance Surveillance Program (ARSP) contains the national estimates and trends of antimicrobial resistance rates of aerobic bacteria in the Philippines. For 2025, it is noted that antibiotic resistance in respiratory infections due to *S. pneumoniae* and *H. influenzae* remained low as in the past several years. Antibiotic resistance in infections due to *Salmonella* Typhi, *Shigella* sp and *V. cholerae* remained low as well. It is a welcome observation that no gonorrhea infections were noted to be resistant to ceftriaxone, cefixime nor azithromycin. Antibiotic resistance among infections due to gram-negative bacteria and the non-fermenters remained high with MDR and possible XDR rates noted to continuously increase over the past 5 years. ESBL positivity rates for *E. coli* and *K. pneumoniae* were 34% and 31%, respectively.

As has been the case for the past 2 years, the ARSP 2025 Annual Report includes genomic characterization of isolates of emerging resistance phenotypes. For 2025, a single isolate of phenotypically colistin resistant *E. coli* was noted not to carry the mobile genetic element *mcr* gene which suggest that resistance is most likely chromosomal and is not therefore expected to rapidly spread. On the other hand, a single isolate of *K. pneumoniae* was reported to carry *mcr*-1.1. Although detected sporadically, the presence of *mcr* genes among local isolates requires continued surveillance as this has potential for horizontal transmission across strains and bacterial species.

The ARSP 2025 annual report includes its first reports on novel betalactam-betalactamase inhibitors (BL/BLI) resistance data on Enterobacterales and the non-fermenters. Resistance among *E. coli* ranges from 28% to ceftazidime-avibactam to 60% for imipenem-relebactam which is driven by the New Delhi metallo-beta-lactamase (NDM) in almost all of the resistant isolates. Similarly, resistance to novel BL/BLIs among *K. pneumoniae* isolates were noted to range from 32.44% to ceftazidime-avibactam to 37.5% for imipenem-relebactam and is likewise driven by the NDM in almost all of the resistant isolates. Genomic analysis highlights epidemiological diversity of these isolates indicating that emerging BL/BLI resistance is not clonal and circulates within multiple distinct genetic backgrounds across the national network. The observation that most of these resistant infections were presumptive hospital acquired emphasizes the necessity for continued strengthening of infection prevention and control measures.

The surveillance report now likewise includes data on the genomic characterization of a limited number of select gram-negative and gram-positive bacteria of public health importance in the country. The granular data provided by whole genome sequencing provides information on the occurrence, distribution and population dynamics of bacterial pathogens in the country including resistance genes and transmissible elements. These genomic data provide critical evidence towards antimicrobial stewardship and infection and prevention measures. For example, it was noted that among the carbapenem resistant *E. coli* and *K. pneumoniae* isolates that were sequenced there was a predominance of NDM-type carbapenemases against which the new generation beta-lactam-beta-lactamase inhibitor combination antibiotics do not have activity. Given the evidence of the predominance of NDM-type carbapenemases conferring resistance to carbapenems and the novel BL/BLIs there is a perceived great value in testing for carbapenemase genes among gram negative clinical isolates to guide rational treatment options.

Finally, aside from SDG AMR targets monitoring on targets of the National Action Plan on AMR 2024-2028 is included in respective sections for target isolates in the ARSP 2025 Annual Report. Two years of NAP-AMR implementation shows that the country is on track in achieving the targets for four (4) isolates (*A. baumannii*, *S. aureus*, *N. gonorrhea*, and nontyphoidal *salmonella*) but not for *E. coli* and *K. pneumoniae*. A mid-year review on implementation as well as revisiting strategies may be warranted in order to ensure that the country achieves its targets in addressing AMR in the country.

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Chair, Committee on Antimicrobial Resistance Surveillance Program

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Executive Summary

Streptococcus pneumoniae

The resistance rate of *S. pneumoniae* infections to penicillin, based on the non-meningitis (NM) breakpoint, remains low at 1.55%. This indicates that penicillin continues to be an effective treatment option for non-meningitic infections. Resistance to ceftriaxone (NM) is low at 1.28%, ceftriaxone (M) at 1.60%, clindamycin at 9.77%, and levofloxacin at 1.03%. Compared to 2024, overall resistance rates for all *S. pneumoniae* infections increased in 2025, with the exceptions of ceftriaxone, co-trimoxazole.

Haemophilus influenzae

In 2025, *H. influenzae* had an ampicillin resistance rate of 10.72%, a decrease from 13.95% in 2024. The non-susceptibility rate of ceftriaxone remained at 0% and no resistance was detected for azithromycin or fluoroquinolones. Resistance rates for amoxicillin/clavulanic acid and ampicillin-sulbactam were 7.57% and 5.30%, respectively.

***Salmonella enterica* serovar Typhi**

Over the past decade, antimicrobial resistance rates for *Salmonella* Typhi isolates have remained consistently low. In 2025, ceftriaxone resistance was 3.33% (maintaining a baseline below 4% for the last 10 years), carbapenem resistance ranged from 0% to 4%, and no resistance was detected for ciprofloxacin. Despite these low baselines, co-trimoxazole and azithromycin both exhibited statistically significant changes in their resistance trends over time.

Non-typhoidal *Salmonella* species

In 2025, non-typhoidal *Salmonella* isolates continued to exhibit higher resistance rates than *Salmonella* Typhi for ampicillin (37.47%), ceftriaxone (17.92%), co-trimoxazole (11.01%), and azithromycin (6.45%). In contrast, ciprofloxacin resistance remained within its seven-year range of 9% to 12%, and was 9.12% for the year.

***Shigella* species**

Over the past decade, ampicillin and co-trimoxazole resistance remained elevated above 50%. *Shigella* isolates showed fluctuating trends over the past years especially for ceftriaxone. For 2025, ceftriaxone resistance is at 13.24% from 6.7% (2024) while ciprofloxacin resistance is 9.33% from 12.5% (2024). Meanwhile, azithromycin resistance remained below 10% in the past five years.

Vibrio cholerae

In 2025, *V. cholera* infections remained susceptible to azithromycin, tetracycline and chloramphenicol. Resistance to cotrimoxazole decreased at 4%.

Neisseria gonorrhoea

Over the past 10 years, resistance rates for ceftriaxone, cefixime, and azithromycin over the past decade; conversely, sustained resistance was observed for both tetracycline and ciprofloxacin.

Staphylococcus aureus

Over the past ten years, *Staphylococcus aureus* infections showed a statistically significant, steady decline in oxacillin resistance. During the same period, resistance rates for erythromycin and clindamycin remained stable at 13.21% and 11.30%, respectively, while linezolid resistance consistently remained below 2%. Regarding emerging resistance, suspected vancomycin resistance was recorded at 1% to 2% over the past five years; however, these cases remain unverified as they were not confirmed by the national reference laboratory. Additionally, two (2) infections demonstrating non-susceptibility to daptomycin, a last-resort antibiotic for *S. aureus*, were detected and confirmed this year. Sustainable Development Goal (SDG) indicator 3.d.2 tracks global antimicrobial resistance via the prevalence of methicillin-resistant *S. aureus* (MRSA) bloodstream infections (BSIs), a major driver of mortality, morbidity, and healthcare costs. The 2025 data demonstrate a downward trend in MRSA resistance among BSIs, falling from 45.2% in 2021 to 37.5% in 2025. Serving as an epidemiological proxy for MRSA BSIs, these ARSP metrics indicate measurable progress toward achieving the target.

***Enterococcus* species**

Resistance rates of *Enterococcus faecalis* infections to penicillin and ampicillin were recorded at 16.36% and 6.77%, respectively, while nitrofurantoin maintained a low resistance rate of below 5% over the past ten years. Notably, a case of vancomycin-resistant *E. faecalis* urinary tract infection was detected and confirmed; the isolate was susceptible to linezolid, nitrofurantoin, and peni-

cillin, but resistant to other relevant antibiotics. Additionally, high-level gentamicin resistance showed fluctuation, rising to 25.84% from the 17.2% reported in 2024. For *Enterococcus faecium* infections, a statistically significant decreasing trend in vancomycin resistance was observed over the past decade. Conversely, resistance to nitrofurantoin showed a statistically increasing trend during the same period. Meanwhile, resistance rates for both high-level gentamicin and high-level streptomycin fluctuated significantly over the ten-year timeframe. Meanwhile, linezolid resistance was at 0.96% from 1.1% the previous year, and the change was not statistically significant.

Escherichia coli

Escherichia coli isolates demonstrated stable, moderate-to-high resistance to fluoroquinolones and third-generation cephalosporins, with ciprofloxacin at 45.9%, levofloxacin at 42.6%, cefotaxime at 40.33%, and ceftriaxone at 38.99%. In contrast, resistance to carbapenems was relatively lower, with rates of 10.9% for imipenem, 10.2% for meropenem, and 9.9% for ertapenem. The yearly variations in resistance rates were statistically significant across all tested antibiotics over time. The ESBL rate for *E. coli* infections was at 33.68% and fluctuated over the past five years, with a notable upward trend observed from 2023 to 2025. Using these infections as a proxy for resistance to third-generation cephalosporins, this trend underscores the critical need to scale targeted stewardship interventions to align the Philippines with this SDG indicator. Meanwhile, colistin resistance remained below 2%, though observed fluctuations were based on limited sample sizes. For novel beta-lactam/beta-lactamase inhibitor combinations, relatively moderate resistance rates were detected, with ceftazidime-avibactam at 28.02% and imipenem-relebactam at 60%. A single isolate of phenotypically colistin resistant *E. coli* was noted not to carry the mobile genetic element *mcr* gene which suggest that resistance is most likely chromosomal and is not therefore expected to rapidly spread.

Klebsiella pneumoniae

Klebsiella pneumoniae isolates demonstrated persistently high resistance to cephalosporins, fluoroquinolones, co-trimoxazole, and ampicillin-sulbactam. Gradual upward trends were observed for both carbapenems (rising from 9%–12% in 2016 to 17%–18% in 2025) and piperacillin-tazobactam (increasing from 22.6% to 35.4%). The ESBL rate reached 31.2% in 2025, up marginally from 30.99% in 2024. While ESBL rates have consistently remained above 30% since 2022, multi-year analysis indicated that the overall changes across the ten-year period were not statistically significant. Meanwhile, colistin resistance remained low overall at 4% during the past three (3) years. Conversely, relatively high resistance rates were noted for novel beta-lactam/beta-lactamase inhibitor combinations, with ceftazidime-avibactam at 32.44% and imipenem-relebactam at 37.5%. Resistance to novel BL/BLIs is driven by the New Delhi metallo-beta-lactamase (NDM) in almost all of the resistant isolates.

Pseudomonas aeruginosa

In 2025, *Pseudomonas aeruginosa* isolates demonstrated significant decreases in resistance to ceftazidime (15.4% to 11.9%), fluoroquinolones (17.8% to 12.0%), and meropenem (17.3% to 12.7%). Piperacillin-tazobactam resistance exhibited a non-significant upward trend (18.8% to 19.7%). For novel beta-lactam/beta-lactamase inhibitors, phenotype resistance was recorded at 41.51% for imipenem-relebactam, 15.71% for ceftazidime-avibactam, and 6.86% for ceftolozane-tazobactam; however, these specific metrics require cautious interpretation due to limited dataset sample sizes.

Acinetobacter baumannii

In 2025, *Acinetobacter baumannii* maintained elevated resistance to most antimicrobials, including ceftazidime (45.2%), piperacillin-tazobactam (45.7%), meropenem (44.23%), and ampicillin-sulbactam (43.3%). Statistically significant decreases in resistance over time were observed for meropenem, imipenem, amikacin, gentamicin, co-trimoxazole, levofloxacin, and tobramycin. Conversely, colistin resistance increased significantly from 0.4% in 2024 to 3.5% in 2025. Temporal changes in resistance were non-significant for ampicillin-sulbactam, ceftazidime, and minocycline.

Reader's Guide

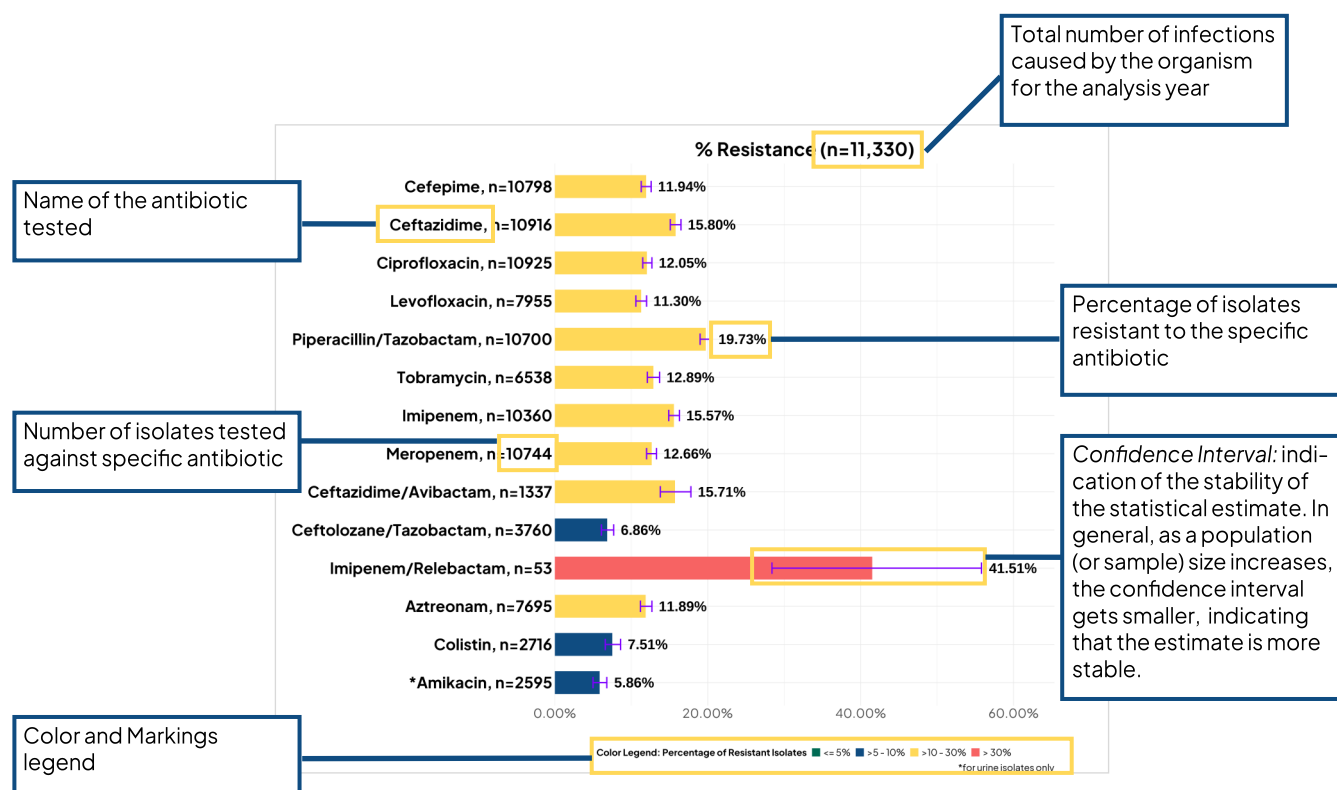


Figure 1. Components of the Percent Resistance Graph

Terminologies

Confidence Interval

Indication of the stability of the statistical estimate. In general, as a population (or sample) size increases, the confidence interval gets smaller, indicating that the estimate is more stable.

The purpose of a CI is to provide an estimate of how precise the observed %S (or %R) is when used to guide therapy and policy decisions. (CLSI M39-A4E). In the ARSP 2025 annual report, 95% confidence intervals are represented by purple lines overlapping the bars

Statistical significance

The difference between the resistance rate/s of infections to a particular antibiotic against 1) the same antibiotic from the previous year; 2) same antibiotic from a different subset; 3) the same antibiotic for a given duration (multi-year) were assessed statistically and represented by a p-value.

Introduction

Antimicrobial Resistance (AMR) is the process by which bacteria, viruses, fungi, and parasites evolve to resist the effects of medicines, making infections harder to treat and increasing the risk of severe illness and death^[1].

AMR is a serious public health threat because of its far reaching and serious implications in health care as well as economies. AMR hampers the control of infectious diseases because patients remain infectious for a longer time increasing the risk of spreading resistant microorganisms to others. AMR increases the cost of health care as more expensive therapies must be used when infections become resistant to first-line medicines. Infections due to resistant microorganisms increases economic burden to families and societies as it often results in longer duration of illness and treatment.

Antimicrobial medicines are the cornerstone of modern medicine. The emergence and spread of drug-resistant pathogens threatens our ability to treat common infections and to perform life-saving procedures including cancer chemotherapy and caesarean section, hip replacements, organ transplantation and other surgeries.

In addition, drug-resistant infections impact the health of animals and plants, reduce productivity in farms, and threaten food security.

The Philippine Committee on **Antimicrobial Resistance Surveillance Program (ARSP)** was created in 1988 through the Department of Health's Department Order 339-J. The program aims to provide critical inputs to the Department of Health's effort to promote rational drug use by determining the status and developing trends of antimicrobial resistance of selected bacteria to specific antimicrobials.

AMR surveillance remains an essential component in the control of AMR in the country. Surveillance data enable correct decisions to be made about treatment options and guide policy recommendations. The Philippine National Action Plan on Antimicrobial Resistance 2024-2028^[2] reiterates the importance of surveillance as it identifies the strengthening of surveillance and laboratory capacity as among its key strategies. Furthermore, these national data serve as a valuable input to the WHO GLASS report, contributing directly to the global AMR platform.

The culture and susceptibility reports submitted into the program were noted to have increased by 25.62% compared with the reports submitted in 2024. This difference must be kept in mind in the interpretation of the surveillance data in the past two years.

Consistent with the 2024 reporting manner, the 2025 ARSP annual report maintains a sample-based data approach covering all patients from whom clinical specimens were collected. By including laboratory-confirmed pathogens, negative cultures, and other microbial growth, this approach allows the calculation of infection frequencies. Consequently, instead of isolate resistance rates, the report presents pathogen resistance rates relative to the disease associated with the specimen type. The prevalence of specific infection types per surveillance site is also included to provide insight into pathogen frequency in each area.

However, inherent limitations persist, such as limited access to microbiological testing potentially excluding many individuals with infectious syndromes, which complicates obtaining a representative sample of the AMR-affected population, and the ongoing challenge of distinguishing between colonization, contamination, and true infection.

Surveillance, Testing Methods, Data Analysis, & Limitations

The Surveillance

The DOH-ARSP is a routine antimicrobial resistance surveillance on aerobic bacteria from clinical specimens.

Currently participating in the program are 28 surveillance sites, representing 17 out of the 18 regions of the country (Figure 2 and Table 1)

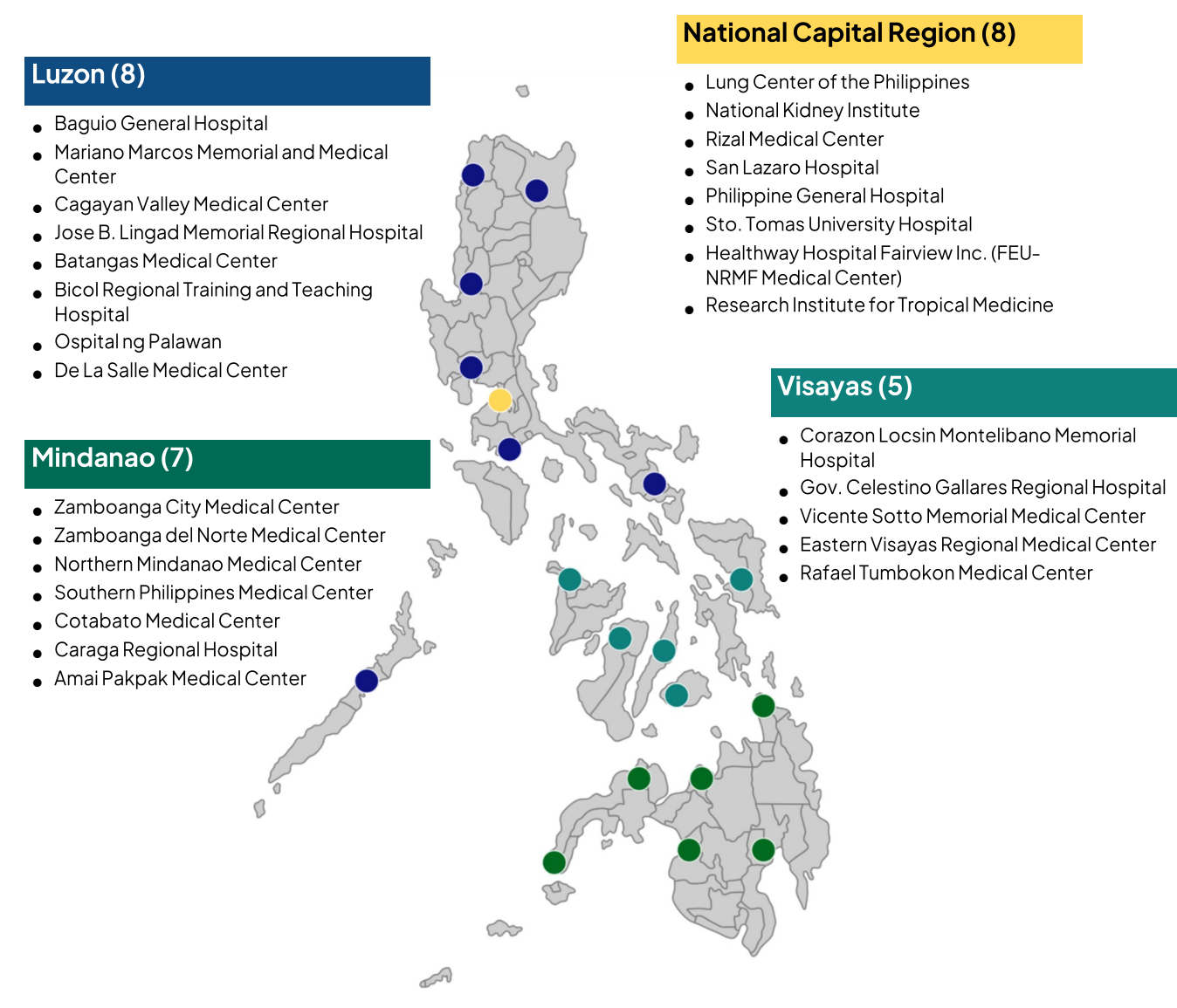


Figure 2. Regional representation in the ARSP 2025

Table 1. ARSP 2025 surveillance sites by region

Region	Surveillance Site
National Capital Region (NCR)	Lung Center of the Philippines National Kidney and Transplant Institute Rizal Medical Center San Lazaro Hospital Philippine General Hospital Research Institute for Tropical Medicine University of Santo Tomas Hospital Healthway Hospital Fairview Inc. (FEU-NRMF Medical Center)
Region 1—Ilocos Region	Mariano Marcos Memorial Hospital and Medical Center
Region 2—Cagayan Valley	Cagayan Valley Medical Center
Region 3—Central Luzon	Jose B. Lingad Memorial Regional Hospital
Region 4-A—CALABARZON	Batangas Medical Center De La Salle Medical Center
Region 4-B—MIMAROPA	Ospital ng Palawan
Region 5—Bicol Region	Bicol Regional Training and Teaching Hospital
Region 6—Western Visayas	Corazon Locsin Montelibano Memorial Regional Hospital Dr. Rafael S. Tumbokon Memorial Hospital
Region 7—Central Visayas	Celestino Gallares Memorial Hospital Vicente Sotto Memorial Medical Center
Region 8—Eastern Visayas	Eastern Visayas Regional Medical Center
Region 9—Zamboanga Peninsula	Zamboanga City Medical Center Zamboanga del Norte Medical Center
Region 10—Northern Mindanao	Northern Mindanao Medical Center
Region 11—Davao Region	Southern Philippines Medical Center
Region 12—SOCCSKSARGEN	Cotabato Regional Hospital and Medical Center
Region 13—CARAGA Region	Caraga Regional Hospital
Region 14 - CAR	Baguio General Hospital and Medical Center
Region 19 - BARMM	Amai Pakpak Medical Center

Legend: CALABARZON: Cavite, Laguna, Batangas, Rizal, Quezon; MIMAROPA: Mindoro, Marinduque, Romblon, Palawan; SOCCSKSARGEN: South Cotabato, Cotabato, Sultan Kudarat, Sarangani, General Santos City; Cordillera Administrative Region (CAR); Bangsamoro Autonomous Region in Muslim Mindanao (BARMM).

The surveillance collects data on culture and antimicrobial susceptibility and corresponding demographic data from its **28 surveillance sites**. Case finding is based on priority specimens sent routinely to surveillance sites laboratories for clinical purposes.

TESTING METHODS

All surveillance sites implement standard methods for culture and susceptibility testing based on the WHO Manual for the Laboratory Identification and Antimicrobial Susceptibility Testing of Bacterial Pathogens of

Public Health Importance in the Developing World [3] and the updated Clinical Laboratory Standards Institute (CLSI) references for antibiotic susceptibility testing (AST) and quality control [4].

Panel of antibiotics for testing is based on the latest CLSI recommendations. In the analysis of AST, an isolate is considered resistant to an antimicrobial agent when tested and interpreted as resistant (R) in accordance with the clinical breakpoint criteria based on the most recent Clinical Laboratory Standards Institute (CLSI) references for AST. The surveillance data are encoded using a database software called WHONET. WHONET is a Windows-based database software developed by the WHO Collaborating Centre for Surveillance of Antimicrobial Resistance based at the Brigham and Women's Hospital in Boston for the management and analysis of microbiology laboratory data with a special focus on the analysis of antimicrobial susceptibility test results.

Using a standard format, data are sent by the surveillance sites to the coordinating laboratory of the program – the Antimicrobial Resistance Surveillance Reference Laboratory (ARSRL) at the Research Institute for Tropical Medicine. Beginning January 2018, surveillance sites transmit data daily to the reference laboratory. The automated data transfer facilitates prompt identification of resistant isolates of public health importance as well as the identification of clustering of cases and potential outbreaks among surveillance sites. The ARSRL's Data Management Unit manages the cleaning, analysis, storage and security of the program's surveillance data.

Surveillance sites likewise send isolates with unusual AST patterns to ARSRL for phenotypic and genotypic confirmatory testing.

At the reference laboratory, all isolates with unusual susceptibility patterns received for confirmatory testing are re-identified using both automated (Vitek) and conventional methods. Both minimum inhibitor concentration (MIC) – via automated method (Vitek) and gradient E-test, and disk diffusion are employed in AST. As indicated, additional testing is done for specific antibiotics which are not included in the AST card in use in the reference laboratory and for susceptibility testing for specific bacteria such as *N. gonorrhoeae* which requires manual AST methods. Serotyping using whole genome sequencing (WGS) for *S. pneumoniae*, *H. influenzae*, *Salmonellae*, *Shigellae* and *Vibrio cholerae* was done for 2025.

Select isolates with resistance phenotype which have not been previously reported or have only been rarely reported to date underwent WGS at the ARSRL. The genomic characterization of these isolates is intended to determine their resistance mechanisms and assess their potential for dissemination, thereby guiding control and prevention interventions. Bacterial isolates were cultured overnight at 35°C in appropriate growth media. Genomic DNA was extracted using the NucleoMag® kit (Macherey-Nagel, Düren, Germany), following the manufacturer's instructions. DNA concentration was assessed using a Qubit fluorometer (Thermo Fisher Scientific, Waltham, MA, USA). Sequencing libraries were prepared using either the Illumina DNA Prep Kit (Illumina, San Diego, CA, USA) or the QIAseq FX DNA Library Kit (QIAGEN, Hilden, Germany). Whole genome sequencing was performed on the Illumina MiSeq platform, generating 2 × 150 base pair paired-end reads.

The program surveillance sites participate in an external quality assessment scheme (EQAS) conducted by the reference laboratory to ensure quality of laboratory results. Conditions permitting, periodic monitoring visits to surveillance sites were likewise done.

DATA ANALYSIS

Analysis is restricted to the first isolate received (per genus under surveillance) per patient in the calendar year. Data are expressed as a cumulative resistance percentage, i.e. the percentage of resistant isolates out of all isolates with AST information on that specific organism–antimicrobial agent combination. A 95% confidence interval is determined for the resistance percentage. Cumulative percentages of resistance are compared as proportions using either Chi square or Fischer's test, using a p value of <0.05 as statistically significant. Only species with testing data for 30 or more isolates are included in the analysis.

All analyses were performed using WHONET software (version 26.5.19). Antimicrobial susceptibility test results were interpreted according to Clinical and Laboratory Standards Institute (CLSI) 2025 breakpoints. Calculation of cumulative resistance percentages, 95% confidence intervals, and statistical comparisons were performed using WHONET's built-in analytic tools.

Bioinformatics Methods

Short-read WGS data were processed with Bactopia v4.0.0. Assemblies were produced by Shovill v1.1.0,

which wraps SPAdes v3.15.5 for de-novo assembly, and draft genomes were annotated with Prokka v1.14.6. Multi-locus sequence types were assigned using MLST v2.23.0 (PubMLST release 2025-02-25), and plasmid replicons were detected with PlasmidFinder v2.1.6. Species-specific modules comprised: SISTR v1.1.3 for *Salmonella enterica* serovar prediction; GPS pipeline v1.2.0 for *Streptococcus pneumoniae* serotyping; ECTyper v1.0.0 for *Escherichia coli* O:H serotyping; Kleborate v3.1.3 for *Klebsiella* virulence, resistance and capsule/outer-membrane-porin typing; Pasty v2.2.1 for *Pseudomonas aeruginosa* serotyping; Kaptive v3.1.0 for *Acinetobacter baumannii* capsule (K) and lipooligosaccharide outer-core (OCL) locus typing; and SCCmecFinder v1.2.0 together with spaTyper v0.2.1 for *Staphylococcus aureus* mec and spa typing. Core-genome single-nucleotide polymorphisms (SNPs) were called with Snippy v4.6.0, pairwise SNP distances were calculated with SNP-dists v0.8.2, and a maximum-likelihood phylogeny was inferred from the core-SNP alignment using IQ-TREE v2.4.0. Antimicrobial-resistance, virulence and stress genes were identified with AMRFinderPlus v4.2.7 (database 206-01-21.1). A complete list of software and versions are provided in the annex.

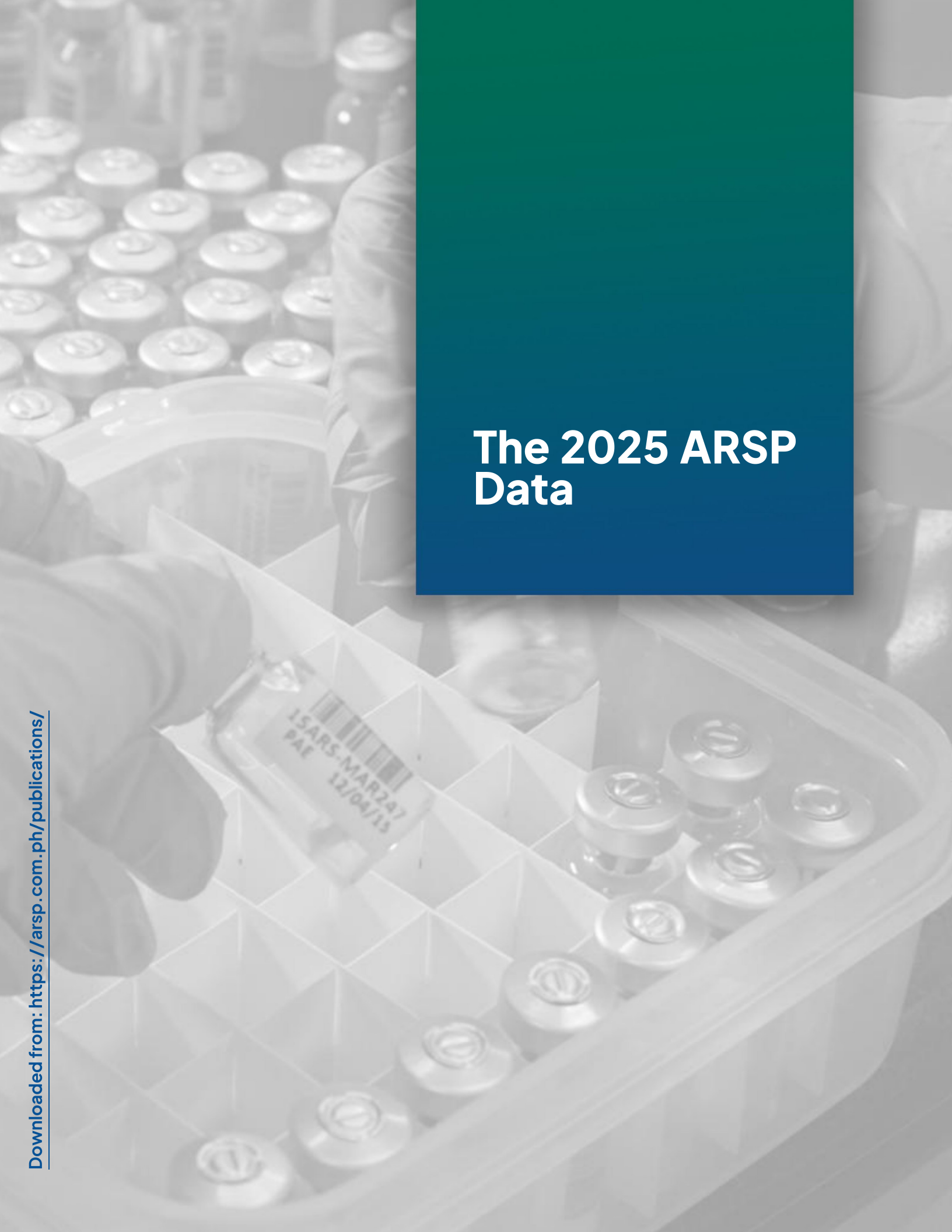
An annual report with a summary of the surveillance data focusing on aerobic bacterial pathogens of public health importance causing common infectious diseases with significant morbidity and mortality locally are disseminated to the program's stakeholders.

LIMITATIONS

Interpretation of data in this annual report should be undertaken with caution considering that there may be several factors that could influence and introduce bias to the data, resulting in over- or underestimation of resistance percentages. Potential sources of bias include population coverage, sampling, and laboratory capacity.

1. Most resistance data in the program come from regional hospitals serving nearby towns and cities, leaving local areas outside their coverage underrepresented.
2. Data for the National Capital Region came from 8 surveillance sites while data for other regions come from 1 or 2 surveillance sites.
3. Given that the program data are from routine clinical samples, differences in factors indicating need for microbiological cultures may introduce variations in the resistance data.
4. Performance of culture and susceptibility tests in the surveillance sites is dependent on the diagnostic habits of the clinicians as well as the financial capability of patients for such tests. Differential sampling can occur if cultures are typically only performed after empirical treatment shows no adequate therapeutic response. Predictably, this will lead to a serious overestimation of the percentage resistance by not including susceptible isolates in the denominator.
5. Lastly, the ability of the laboratory to identify the microorganism and its associated antimicrobial susceptibility pattern may differ.





The 2025 ARSP Data

The 2025 ARSP Data

The ARSP data corresponds to all surveillance data collected from the sites and submitted to the reference laboratory for the analysis year. Confirmatory testing for bacterial identification and antimicrobial susceptibility was performed on a subset of surveillance isolates to monitor organisms with emerging phenotypic resistance patterns, investigate outbreaks and for the conduct of structured genomic surveys.

In 2025, resistance data for 155,138 infections were reported and analyzed, reflecting an increase from the 123,949 cases reported in 2024. **Table 2** shows that 18 of 28 sites had an increase in data submission for 2025.

In 2025, all 28 ARSP sentinel sites representing 17 of 18 regions in the Philippines have data contribution. More than half (56.60%) of the reported infections were from Luzon, 23.83% from Mindanao and 19.57% from Visayas (**Figure 1**). Over half of the infections occurred in male patients (53.73%) and more than half (59.63%) belonged to the 20–64 age group. The most common infections were respiratory (52.16%), urinary (19.68%) and bloodstream (11.06%). **Table 3** shows the most common isolates per specimen type.

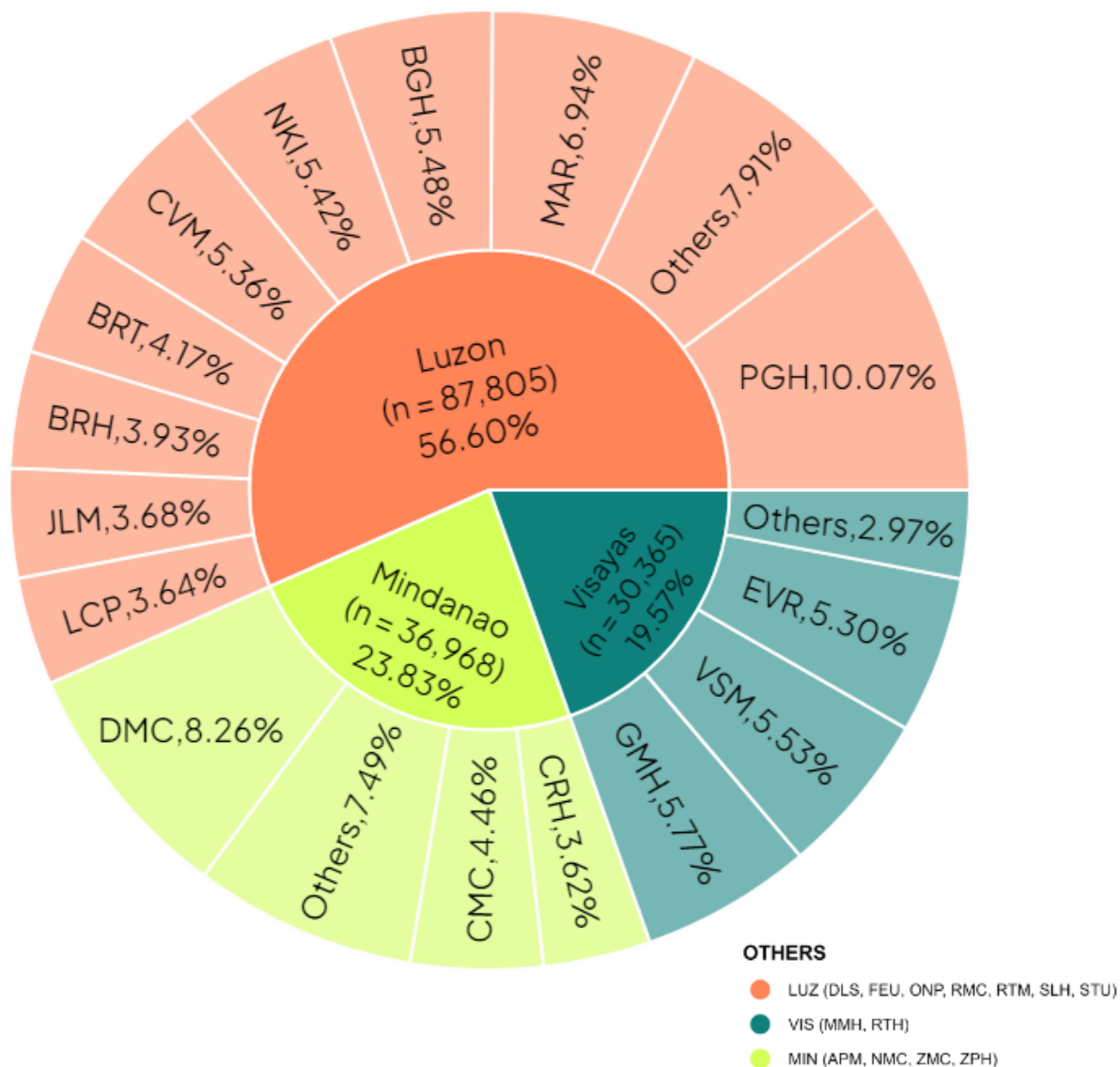


Figure 3. ARSP surveillance sites contribution , DOH-ARSP, 2025 (n=155, 138)

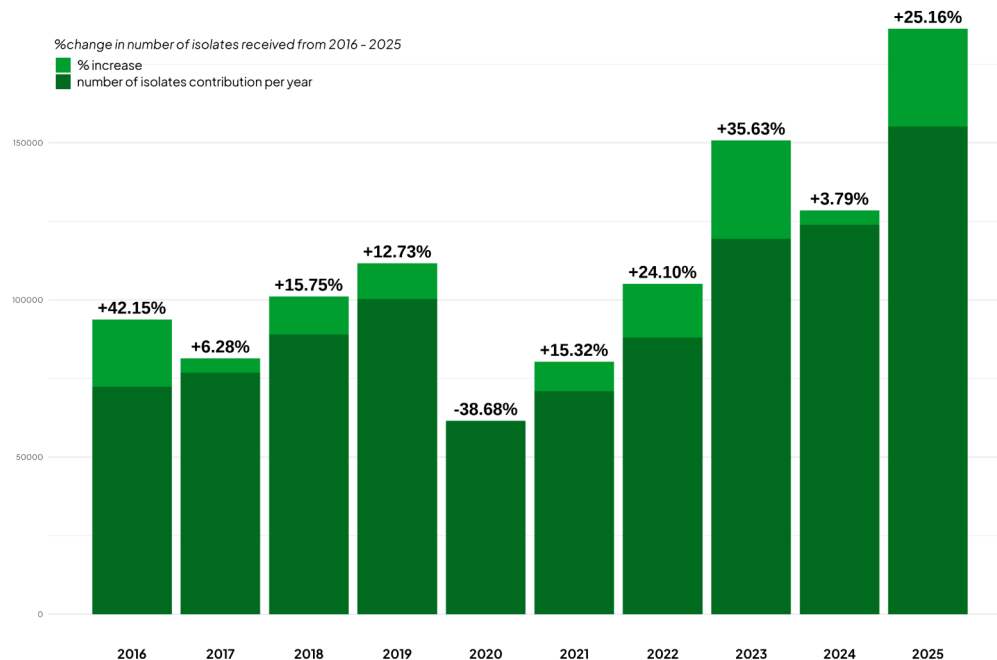


Figure 4. Number and percentage difference of infections received from 2016-2025

Table 2. Surveillance sites infection contribution, DOH-ARSP, 2016-2025

Surveillance Sites	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024	2025	Change %
APM											4416	-
BGH	3214	4628	4842	5775	5234	2968	3191	5459	8385	8796	8508	-3.27
BRH	1294	2075	2472	3133	3633	1569	1472	2570	3133	4924	6101	23.90
BRT	1251	1584	1640	1842	2521	1176	1903	3055	4131	5628	6466	14.89
CMC	1300	1599	1704	2642	3181	2076	2021	3257	4509	4526	6914	52.76
CRH	-	10	624	879	1205	1115	1314	2075	3885	4551	5617	23.42
CVM	1512	3473	4141	4276	5668	3782	2687	4207	6114	4961	8313	67.57
DLS											2662	-
DMC	5109	8058	8680	10762	12177	7412	8189	9135	11613	9767	12818	31.24
EVR	1514	1731	3303	3879	3874	4056	4584	5388	5793	6491	8230	26.79
FEU	712	810	1201	1173	548		322	413	785	745	980	31.54
GMH	1807	1669	3153	3258	3957	2624	2818	4254	6752	7106	8949	25.94
JLM	1266	2768	3261	3880	4824	3248	3753	5229	5890	5387	5715	6.09
LCP	2905	3115	1367	3098	4433	2713	3205		3999	3799	5653	48.80
MAR	1849	2759	3565	4293	4462	3581	3302	4492	6416	7718	10762	39.44
MMH	2940	2886	3133	3026	2539	1425	1140	1575	2062	1858	2124	14.32
NKI	1455	5894	627	2959	4358		5571	5911	6719	7488	8402	12.21
NMC	2237	3105	2245	2961	3409	3735	2780	4868	6893	6160	4292	-30.32
ONP	-	2	5	68	90	13	31	202	204	423	1158	173.76
PGH	11710	12860	14572	12808	13895	6818	9557	12343	15906	16304	15628	-4.15
RMC	1054	3252	3160	3241	2375	2027	1968	2298	2895	2545	3493	37.25
RTH	-	25	69	159	352	289	0	4	5	5	2486	49620
RTM	336	410	513	598	507	255	423	528	555	706	661	-6.37
SLH	824	1410	2460	2044	2371	1019	1157	1060	1352	1468	1595	8.65
STU	1923	2275	2088	2184	2722	1419	1250	854	1122	1684	1708	1.43
VSM	3834	4803	6838	8714	10286	6886	6971	7596	8390	8841	8576	-3.00
ZMC	841	1142	1222	1346	1644	1192	1341	1276	1913	2068	2550	23.31
ZPH	8	4	7	3	69	129	1	0	0	0	361	-
TOTAL	50,895	72,347	76,892	89,001	100,334	61,527	70,951	88,049	119,421	123,949	155,138	25.16

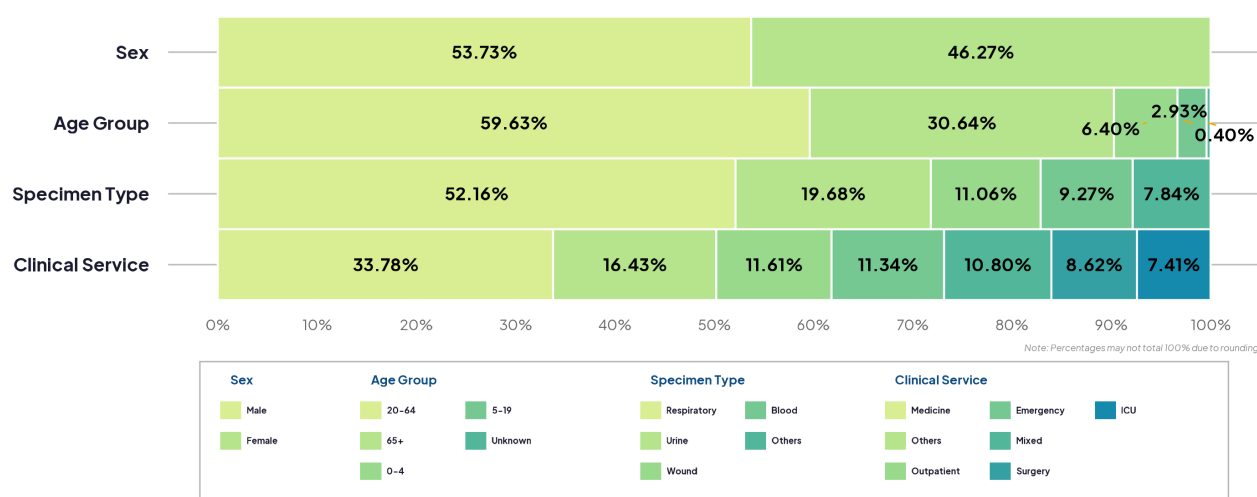
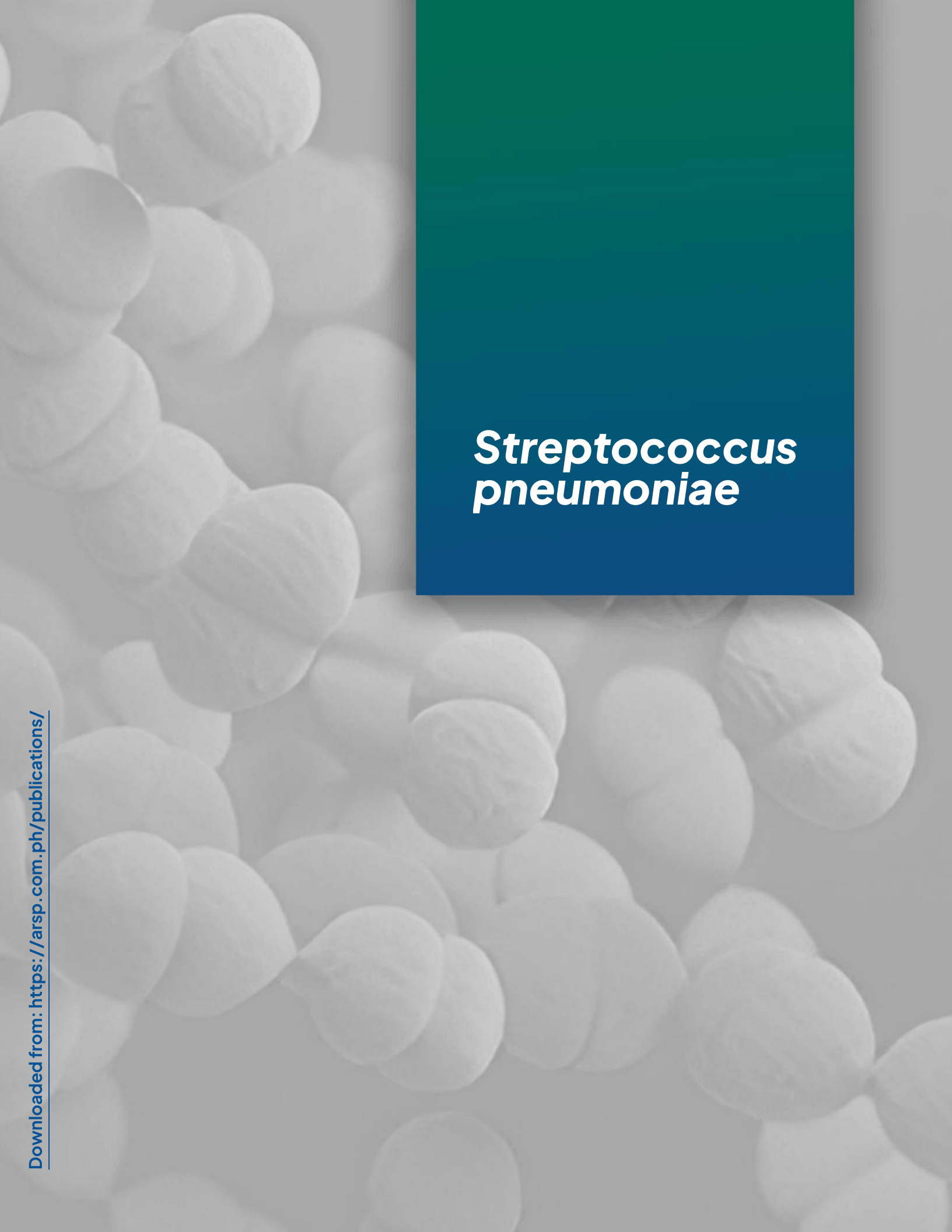


Figure 5. Patient demographics of 2025 analyzed and reported bacterial infections, DOH-ARSP (n=155,138)

Table 3. Most common pathogens among all infections analyzed and reported, DOH-ARSP, 2025 (n=155, 138)

	Rank	Organism
Bloodstream Infections	1	<i>Staphylococcus aureus</i> ss. <i>aureus</i>
	2	<i>Klebsiella pneumoniae</i> ss. <i>pneumoniae</i>
	3	<i>Escherichia coli</i>
Cerebrospinal Fluid Infections	1	<i>Acinetobacter baumannii</i>
	2	<i>Pseudomonas</i> sp.
	3	<i>Klebsiella pneumoniae</i> ss. <i>pneumoniae</i>
Respiratory Tract Infections	1	<i>Klebsiella pneumoniae</i> ss. <i>pneumoniae</i>
	2	<i>Pseudomonas aeruginosa</i>
	3	<i>Acinetobacter baumannii</i>
Diarrheal Disease	1	<i>Salmonella</i> sp.
	2	<i>Aeromonas</i> sp.
	3	<i>Vibrio Cholerae</i>
Urinary Tract Infection	1	<i>Escherichia coli</i>
	2	<i>Klebsiella pneumoniae</i> ss. <i>pneumoniae</i>
	3	<i>Enterococcus faecalis</i>
Skin and Soft Tissue Infection	1	<i>Klebsiella pneumoniae</i> ss. <i>pneumoniae</i>
	2	<i>Pseudomonas aeruginosa</i>
	3	<i>Enterobacter</i> sp



Streptococcus pneumoniae

Streptococcus pneumoniae

Streptococcus pneumoniae contributes significantly to global and local morbidity and mortality as a primary cause of pneumonia, meningitis, and sepsis. Treatment is increasingly challenged by the organism's growing resistance to commonly used antibiotics like penicillin and macrolides. The Antimicrobial Resistance Surveillance Program have documented evolving resistance patterns that highlight the need for continuous monitoring.

There were 439 *Streptococcus pneumoniae* infections reported for 2025. Surveillance sites within Luzon contributed most (65.60%) of the reported infections, 15.49% from Visayas and 18.91% from Mindanao (Figure 6).

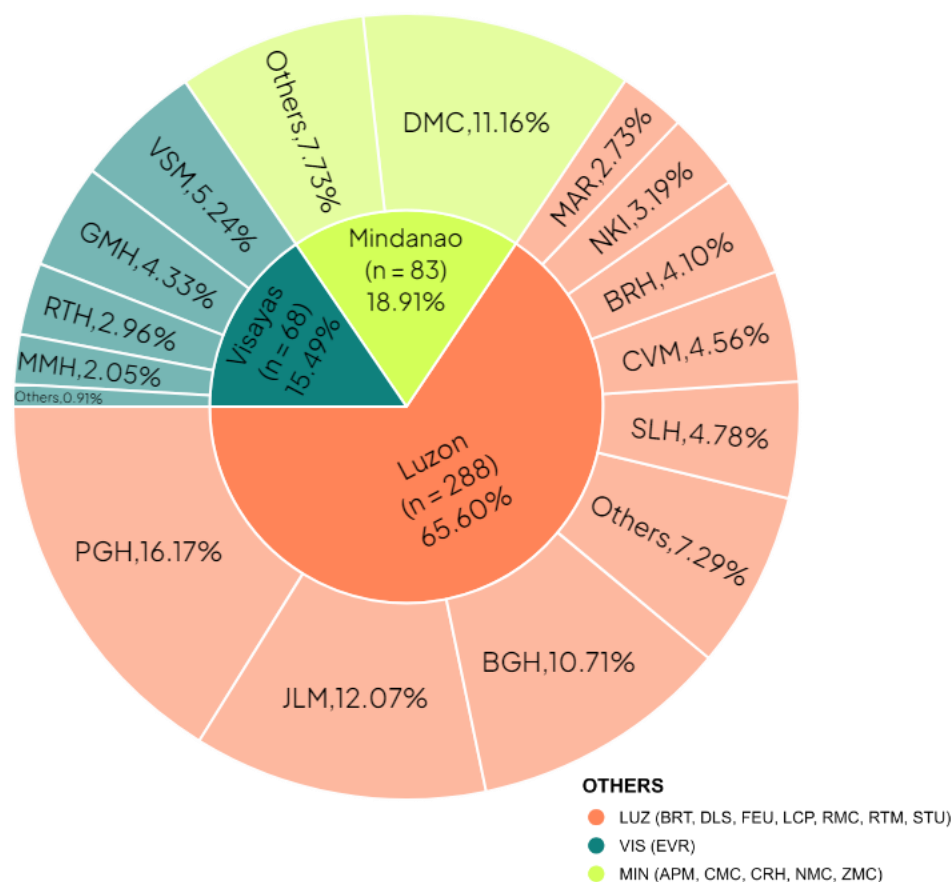


Figure 6. Distribution of *S. pneumoniae* infections, DOH-ARSP, 2025 (n=439)

Figure 7 illustrates that most (59.91%) of the infections were from male patients and most (55.13%) were from the 20-64 years old. Most common types of infections were respiratory (64.92%) and bloodstream (26.88%).

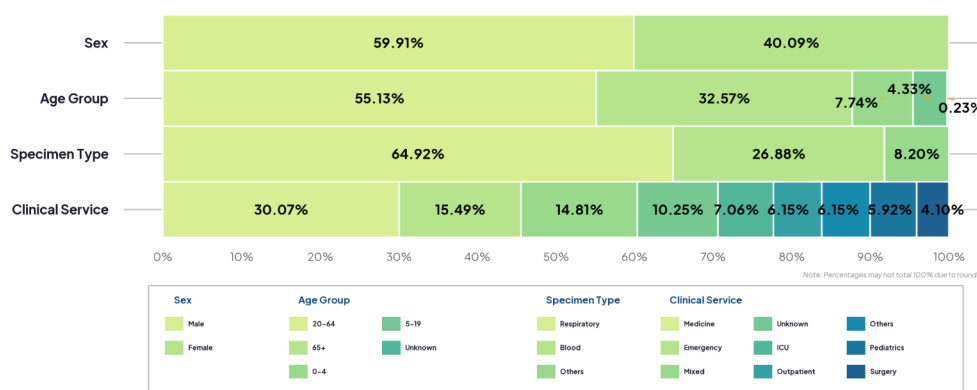


Figure 7. Patient characteristics of *S. pneumoniae* infections, DOH-ARSP, 2025 (n=439)

Figure 8 shows the resistance rates among all types of *S. pneumoniae* infections. Penicillin resistance is at 17.08% using meningitis breakpoint, 1.55% using non-meningitis breakpoint and 4.04% using oral breakpoint.

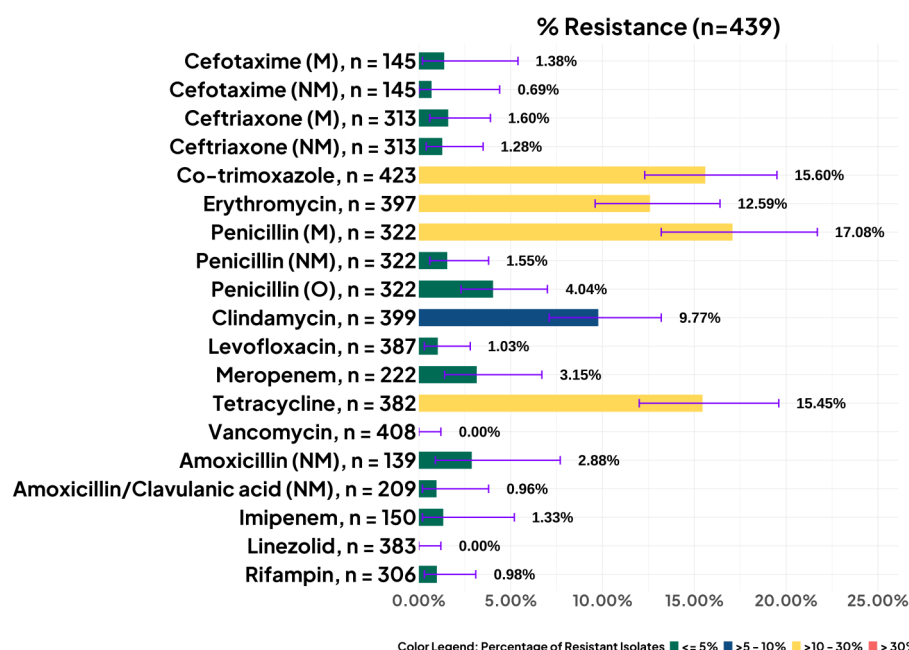


Figure 8. Proportion of all *S. pneumoniae* infections with resistance to tested antibiotics, DOH-ARSP, 2025 (n=439)

Multiple year analysis shows that the resistance rates over the past decade were statistically significant for all antibiotics tested.

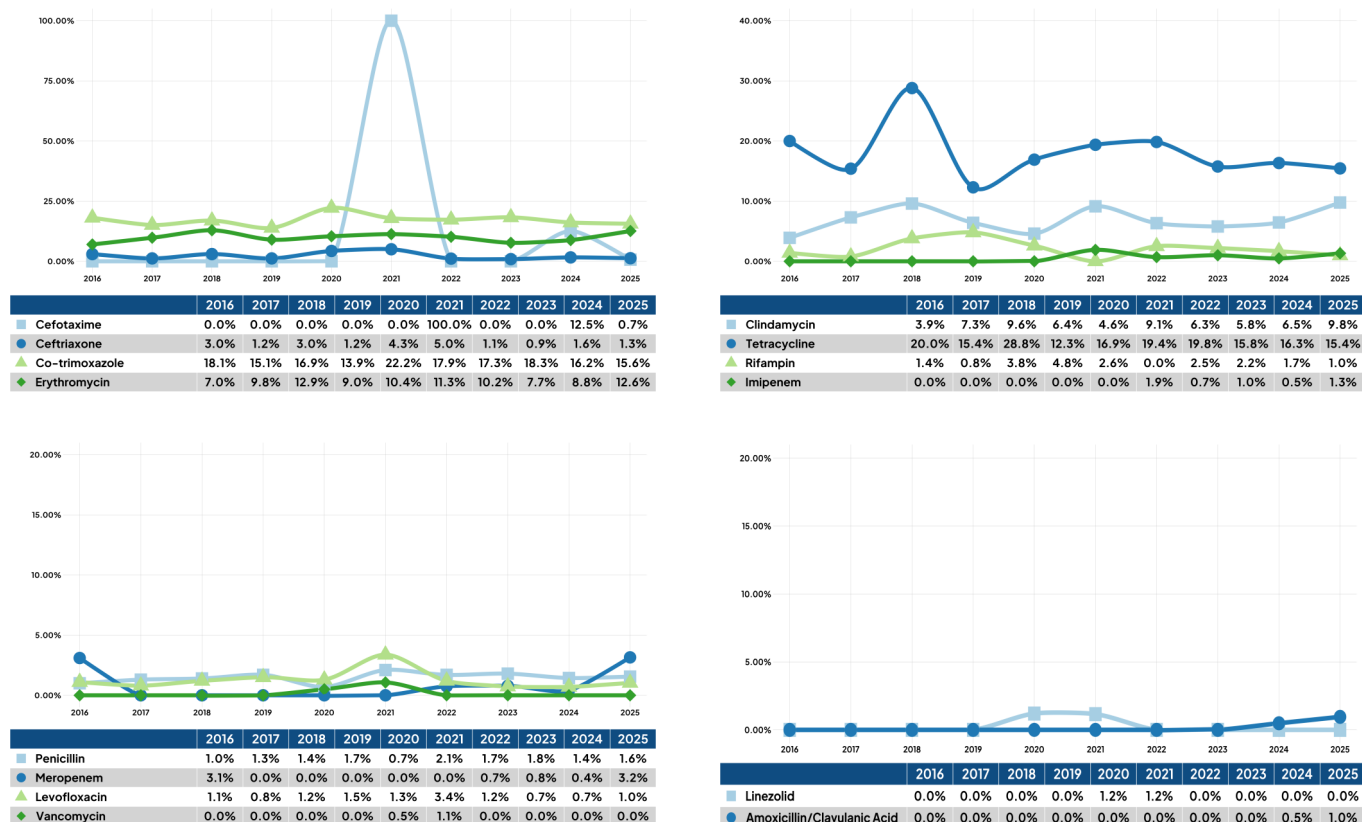


Figure 9. Yearly resistance rates for all *S. pneumoniae* infections, DOH-ARSP, 2025

In 2025, two rifampin-resistant *S. pneumoniae* infections were detected from respiratory specimens submitted for confirmation of identification (ID) and antimicrobial susceptibility testing (AST). Both cases occurred in elderly females from DMC and MAR. The isolates were observed to be resistant to co-trimoxazole but susceptible to amoxicillin-clavulanic acid, penicillin, clindamycin, ceftriaxone, levofloxacin, tetracycline, carbapenems, vancomycin.

Of the 70 reported cases of meropenem resistance, 12 isolates were submitted to the reference laboratory for ID and AST confirmation. Meropenem resistance was identified across respiratory (n=9), wound (n=2) and bloodstream (1) *S. pneumoniae* infections. The respiratory infections were mostly observed from adult patients (n=8) and a 1-month old patient, these two isolates were found to be susceptible to ceftriaxone, linezolid, levofloxacin, penicillin, rifampin, and vancomycin. The single invasive infection bloodstream infection occurred in an 80-year old male at BGH, this infection exhibited to have intermediate resistance to co-trimoxazole but susceptible to linezolid, levofloxacin, penicillin, tetracycline and erythromycin.

Figure 10 shows that penicillin resistance varies across surveillance sites. Elevated resistance rates were detected in surveillance sites in Visayas and Mindanao while relatively lower rates were observed in Luzon.

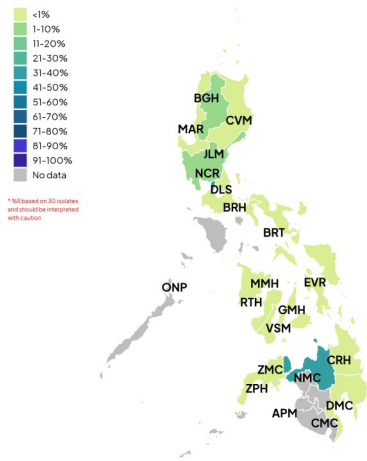


Figure 10. Resistance maps of *S. pneumoniae* infections for Penicillin (NM)



Lower Respiratory Tract Infections

Figure 11 shows resistance rates for lower respiratory tract infections. Complete susceptibility (0.00% resistance) was observed across multiple beta-lactams and other antibiotic classes. This includes cefotaxime and ceftriaxone (under both meningitis and non-meningitis criteria), amoxicillin/clavulanic acid, penicillin (non-meningitis), imipenem, vancomycin, and linezolid. Low resistance rates were observed for co-trimoxazole (16.88%), penicillin (M, 15.62%), tetracycline (12.00%), erythromycin (6.90%), and clindamycin (4.67%)

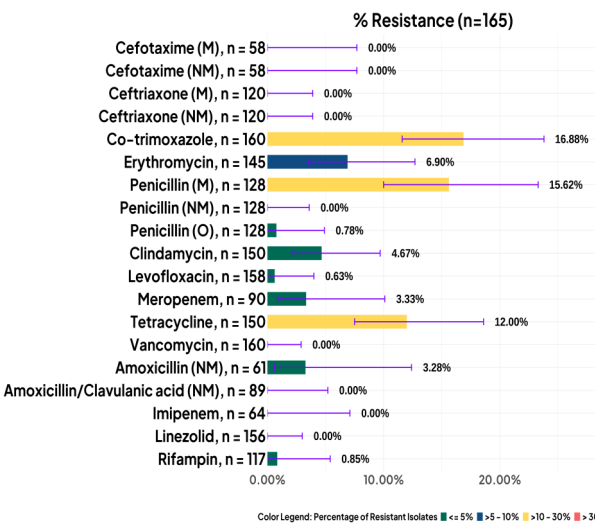
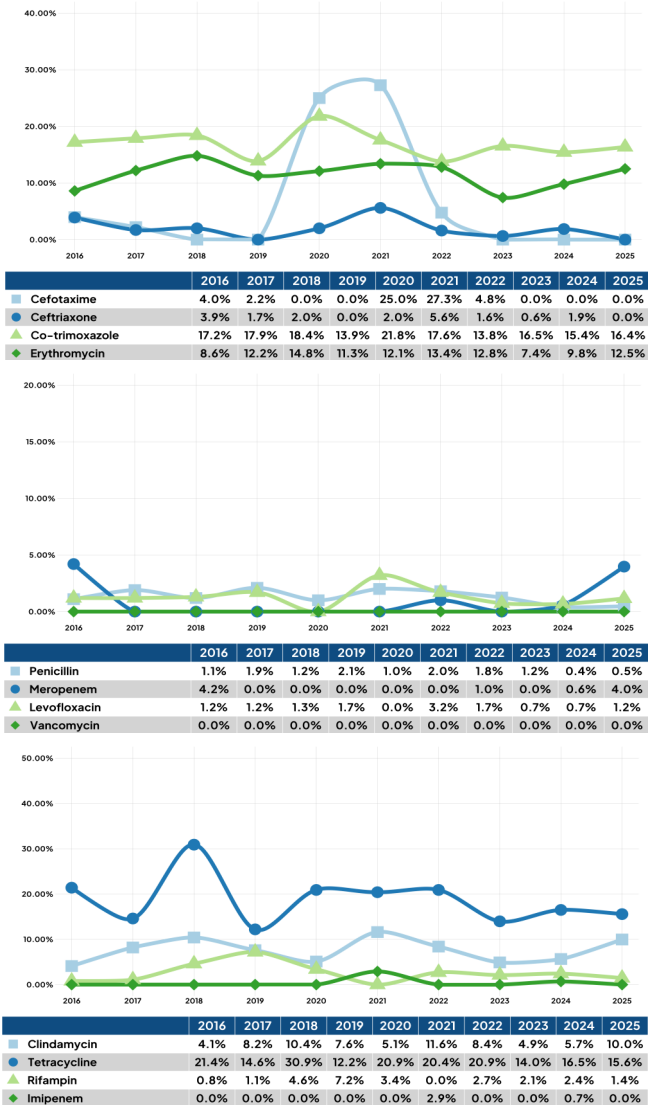


Figure 11. Proportion of *S. pneumoniae* lower respiratory tract infections with resistance to tested antibiotics, 2025 (n=165)

Multiple year analysis of *S. pneumoniae* respiratory infections shows that notable significant increases were observed for erythromycin (p=0.0008), clindamycin (p=0.0047), and penicillin (p=0.0000). No significant differences for co-trimoxazole, levofloxacin, ceftriaxone, meropenem, and tetracycline.



	2016	2017	2018	2019	2020	2021	2022	2023	2024	2025
Cefotaxime	4.0%	2.2%	0.0%	0.0%	25.0%	27.3%	4.8%	0.0%	0.0%	0.0%
Ceftriaxone	3.9%	1.7%	2.0%	0.0%	2.0%	5.6%	1.6%	0.6%	1.9%	0.0%
Co-trimoxazole	17.2%	17.9%	18.4%	13.9%	21.8%	17.6%	13.8%	16.5%	15.4%	16.4%
Erythromycin	8.6%	12.2%	14.8%	11.3%	12.1%	13.4%	12.8%	7.4%	9.8%	12.5%

	2016	2017	2018	2019	2020	2021	2022	2023	2024	2025
Penicillin	1.1%	1.9%	1.2%	2.1%	1.0%	2.0%	1.8%	1.2%	0.4%	0.5%
Meropenem	4.2%	0.0%	0.0%	0.0%	0.0%	0.0%	1.0%	0.0%	0.6%	4.0%
Levofloxacin	1.2%	1.2%	1.3%	1.7%	0.0%	3.2%	1.7%	0.7%	0.7%	1.2%
Vancomycin	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%

	2016	2017	2018	2019	2020	2021	2022	2023	2024	2025
Clindamycin	4.1%	8.2%	10.4%	7.6%	5.1%	11.6%	8.4%	4.9%	5.7%	10.0%
Tetracycline	21.4%	14.6%	30.9%	12.2%	20.9%	20.4%	20.9%	14.0%	16.5%	15.6%
Rifampin	0.8%	1.1%	4.6%	7.2%	3.4%	0.0%	2.7%	2.1%	2.4%	1.4%
Imipenem	0.0%	0.0%	0.0%	0.0%	0.0%	2.9%	0.0%	0.0%	0.7%	0.0%

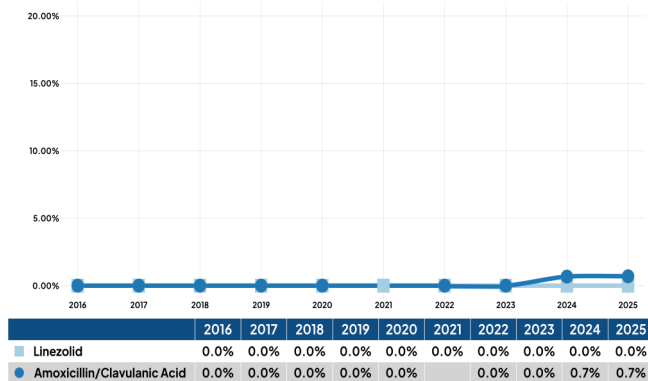


Figure 12. Yearly resistance rates of *S. pneumoniae* respiratory infections: DOH-ARSP, 2025



Invasive Infections

The resistance rates of invasive *S. pneumoniae* infections are shown in Figure 13. Penicillin (M) resistance was at (11.11%), co-trimoxazole resistance at 12.82% and tetracycline at 13.73%. Resistance to most antibiotics was lower for invasive *S. pneumoniae* infections compared to all *S. pneumoniae* infections except for ceftriaxone, penicillin (M), clindamycin, meropenem and tetracycline. No resistance was noted to vancomycin, linezolid and rifampin.

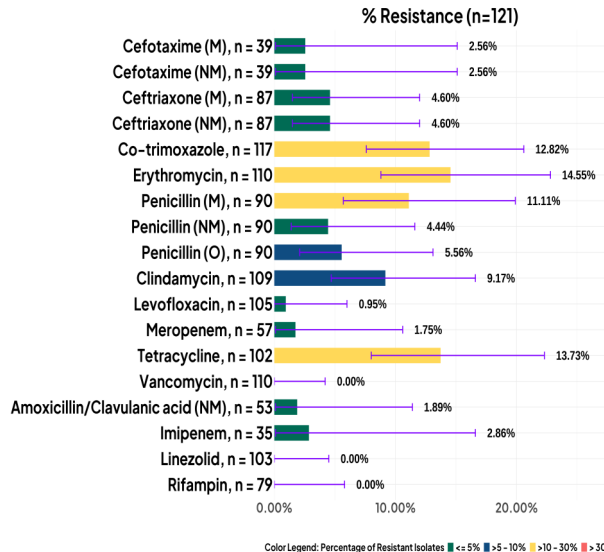


Figure 13. Proportion of invasive (bloodstream and CNS infections) *S. pneumoniae* infections with resistance to tested antibiotics, DOH-ARSP, 2025 (n=121)

Multi-year analysis shows that the resistance rates across all antibiotics over the past decade were statistically significant for invasive *S. pneumoniae* infections.

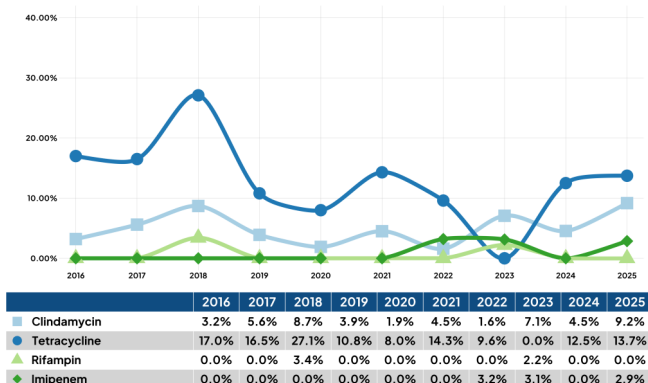
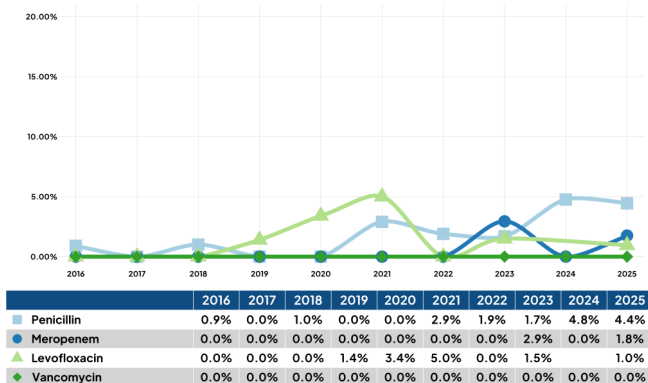
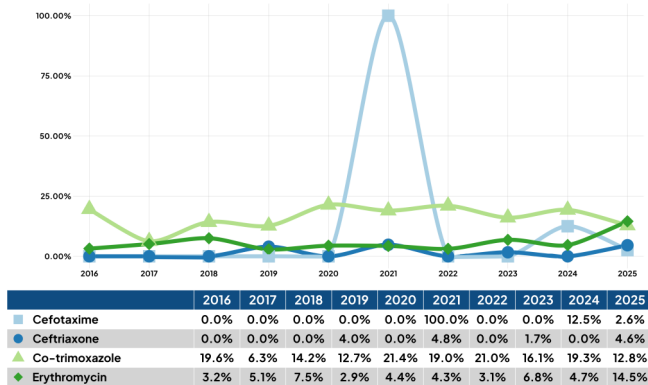


Figure 14. Yearly resistance rates of invasive *S. pneumoniae* infections, DOH-ARSP, 2025

Haemophilus influenzae

Haemophilus influenzae

Haemophilus influenzae is a Gram-negative coccobacillus that commonly causes respiratory tract infections such as otitis media, sinusitis, and pneumonia, as well as invasive diseases like meningitis and bacteremia. It remains an important pathogen despite the widespread use of Hib vaccination, particularly due to non-typeable strains that persist in both community and hospital settings. In the Philippines, surveillance data and regional studies indicate increasing antimicrobial resistance among *H. influenzae*, especially resistance to commonly used β -lactam antibiotics such as ampicillin. Ongoing monitoring of antimicrobial resistance trends is therefore essential to guide appropriate empirical therapy and support national antimicrobial stewardship efforts.

A total of **478** *H. influenzae* infections were reported for 2025. The highest contributors were BGH (25.94%), PGH (21.76%) and SLH (11.09%) (**Figure 15**). Based on island group distribution, Luzon was highest with 79.29% followed by Visayas at 10.46% and Mindanao at 10.25%.

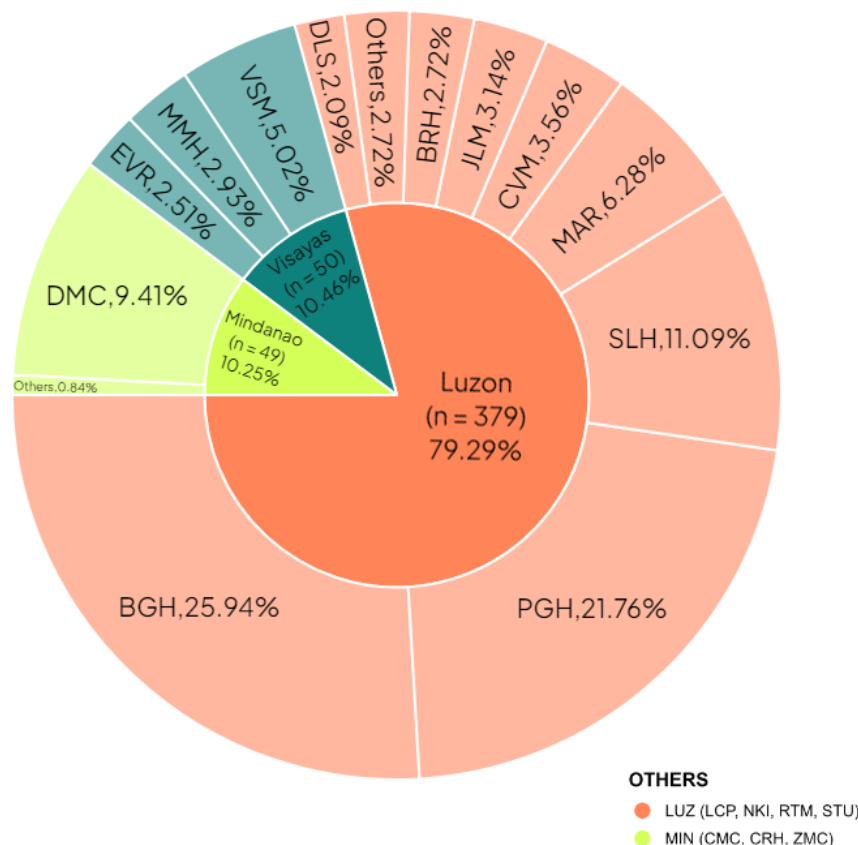


Figure 15. Distribution of *H. influenzae* infections, DOH-ARSP, 2025 (n=478)

More than half of the infections were among male patients at 58.79% and adult patients aged 20–64 years old at 53.56%. Respiratory infection remains the most common type of *H. influenzae* infections, accounting for 92.26%.

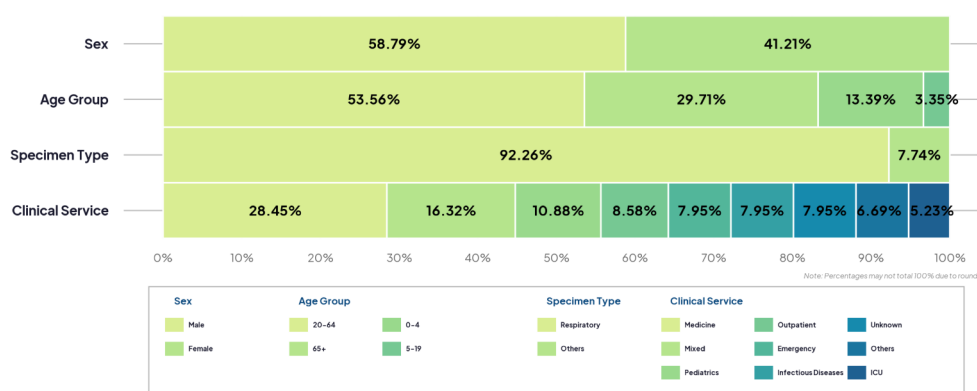


Figure 16. Patient characteristics of *H. influenzae* infections, DOH-ARSP, 2024 (n=478)

All types of Infections

Resistance to ampicillin, cefuroxime and ampicillin-sulbactam was at 10.72%, 9.63% and 5.30% respectively. Resistance to co-trimoxazole and tetracycline resistance was observed to be high across all infections.

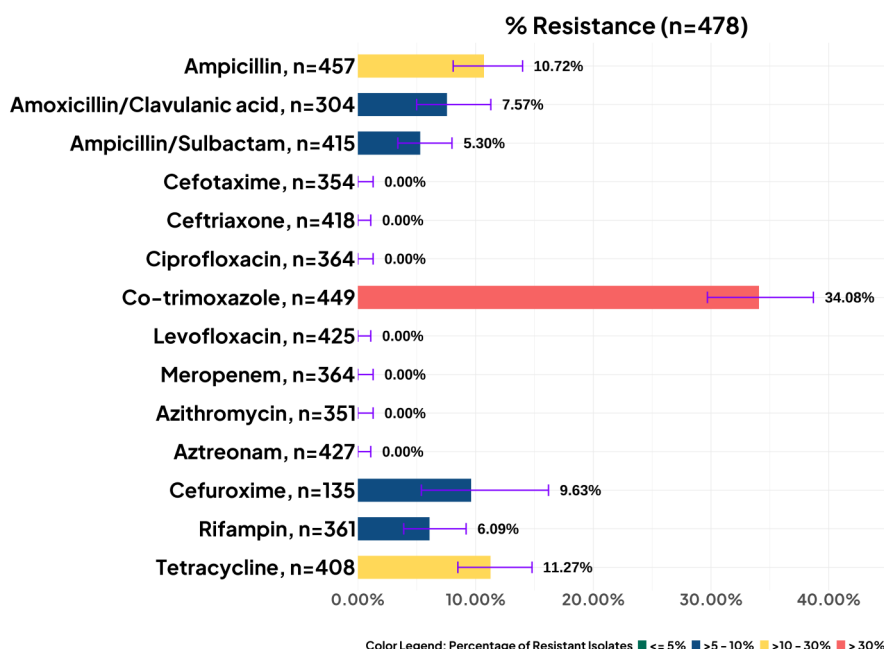


Figure 17. Proportion of all *H. influenzae* infections with resistance to tested antibiotics, DOH-ARSP, 2025 (n=478)

Figure 18 shows the trend in resistance rates of all *H. influenzae* infections over the last ten years. Multi-year analysis revealed that resistance rates were decreasing, except for amoxicillin-clavulanic acid, cefuroxime and rifampin; however, the changes in resistance rates for these three antibiotics were not statistically significant.

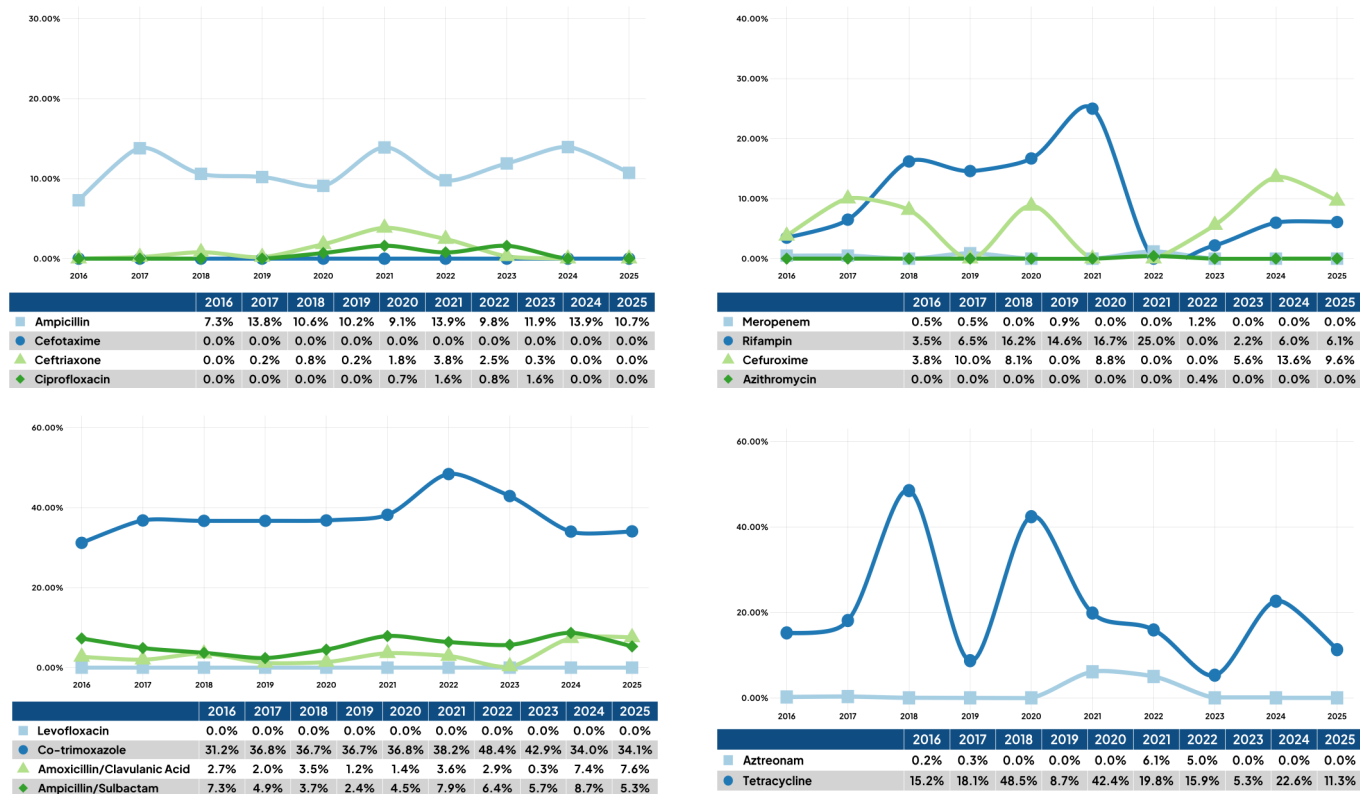


Figure 18. Yearly resistance rates of all *H. influenzae* infections, DOH-ARSP, 2025

Out of the 23 reported infections resistant to amoxicillin-clavulanic acid, eight (8) were sent to the laboratory for ID and AST confirmation. Most of these infections were seen among adult patients from 43-75 years old. These isolates showed variable susceptibility profiles for ampicillin and cefotaxime but were all susceptible to ceftazidime, ceftriaxone, cefotaxime, and fluoroquinolones. No notable susceptibility pattern among the detected amoxicillin-clavulanic resistant infections from 2023-2025.

A ceftazidime-resistant *H. influenzae* lower respiratory tract infection was identified in a 95-year old female from MAR. This isolate demonstrated non-susceptibility to ceftazidime and resistance to ampicillin, tetracycline and co-trimoxazole, but remained susceptible to amoxicillin-clavulanic acid, ceftriaxone, levofloxacin, and rifampin.

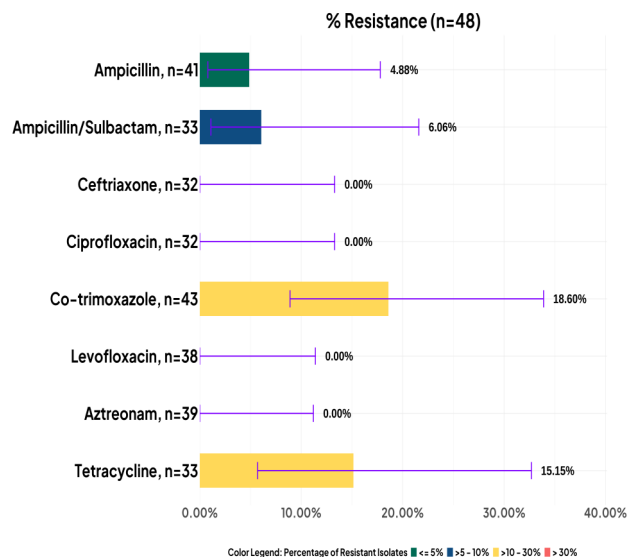


Figure 20. Proportion of invasive *H. influenzae* infections with resistance to tested antibiotics, DOH-ARSP, 2024-2025 (n=48)

Lower Respiratory Tract Infections

Figure 19 shows the resistance rates of lower respiratory infections caused by *H. influenzae*. Co-trimoxazole resistance was highest at 36.36% followed by tetracycline at 12.30% and ampicillin at 12.26%. Compared with the resistance rates of all infections, rates for lower respiratory infections were lower.

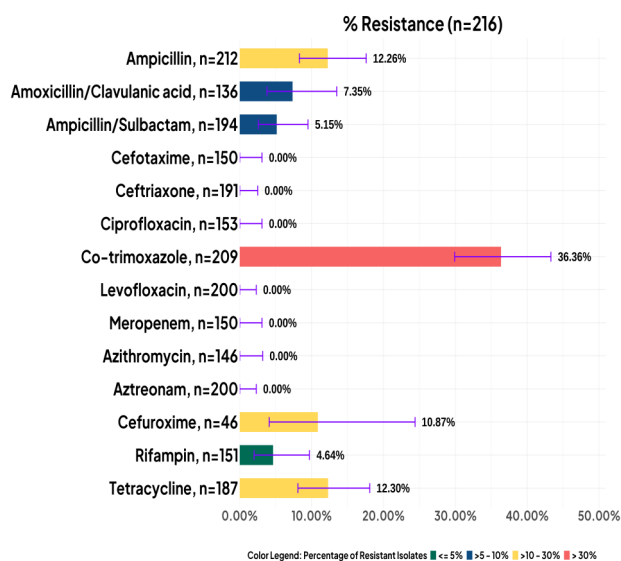


Figure 19. Proportion of *H. influenzae* lower respiratory infections with resistance to tested antibiotics, DOH-ARSP, 2025 (n=216)

Invasive Infections

The resistance rates of invasive *H. influenzae* infections (bloodstream and CNS infections) from 2024-2025 are shown in **Figure 20**. The data from the past three years were presented in order to obtain reasonable statistical estimates of resistance rates.

***Salmonella*
enterica
serovar Typhi**

Salmonella enterica serovar Typhi

Salmonella Typhi is a gram-negative, flagellated rod that is biochemically distinguished by its inability to ferment lactose and its minimal production of hydrogen sulfide. It initiates pathogenesis by invading the intestinal lining and surviving within host macrophages, leading to a systemic and potentially life-threatening infection. In the Philippines, the organism remains endemic due to persistent challenges in sanitation and clean water access, particularly in high-density urban areas. Vaccination and rigorous surveillance are now critical pillars for controlling its spread within the archipelago.

There were 63 *S. Typhi* infections detected in 2025. According to the island group distribution, 55.56% came from Mindanao, 41.27% from Luzon, and 3.17% from Visayas. The highest contributors were CMC (31.75%), and ZMC (9.52%).

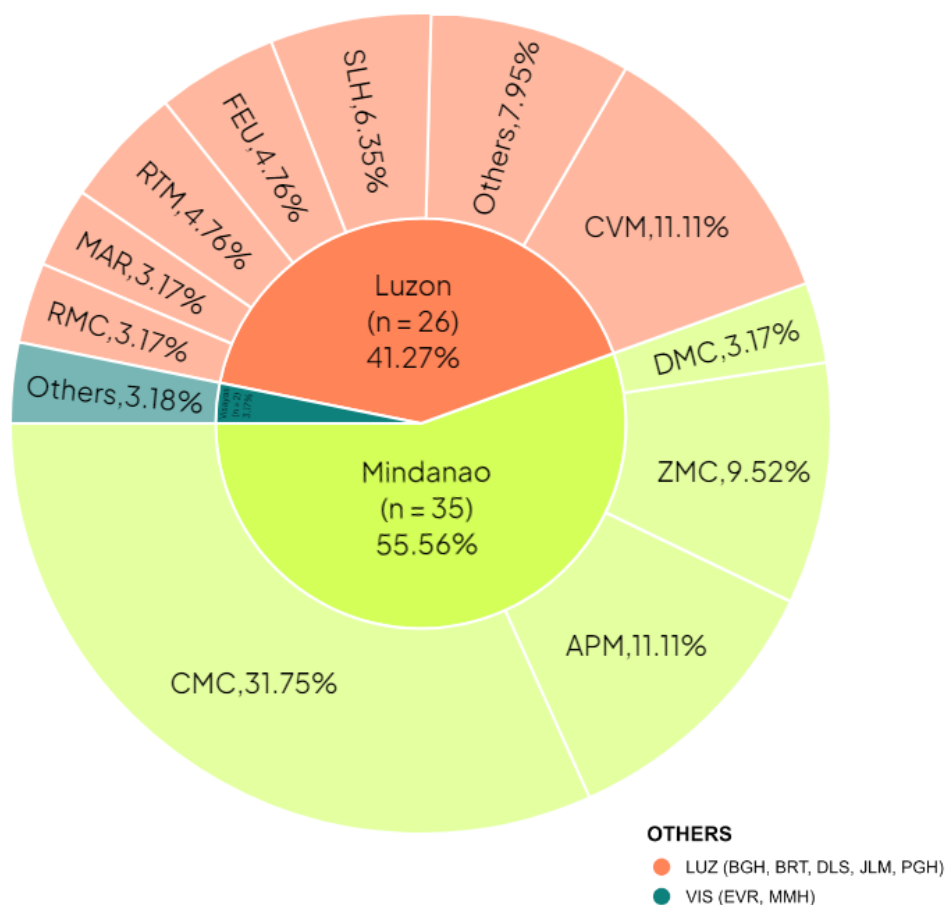


Figure 21. Distribution of *S. Typhi* infections, 2025 (n=63)

The majority (87.30%) of the infections were detected from blood specimens and occurred predominantly in male patients (57.14%).

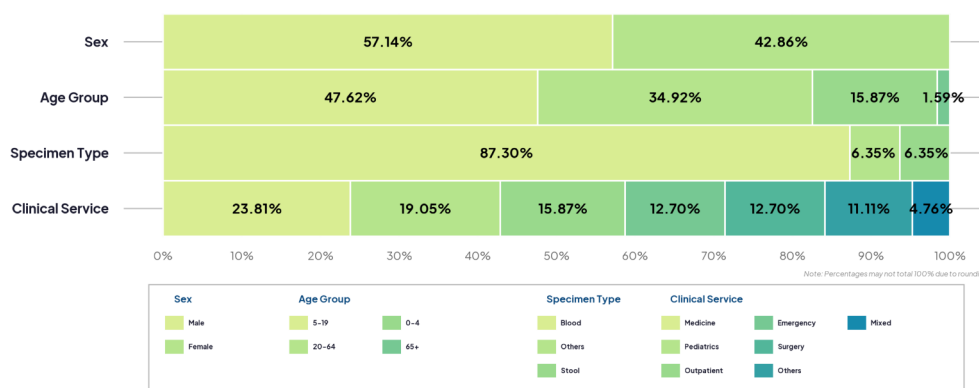


Figure 22. Patient characteristics in *S. Typhi* infections, DOH-ARSP, 2025 (n=63)

All types of Infections

Figure 23 displays the 2025 resistance rates for all *Salmonella enterica* serovar Typhi (S. Typhi) infections. Notably, resistance rates across all tested antibiotics including critical first-line and alternative therapeutic options remained consistently below 5%. Key watch-list agents like ciprofloxacin and azithromycin demonstrated complete susceptibility (0.00% resistance), while ceftriaxone resistance was maintained at a low baseline of 3.33% and carbapenems- imipenem (3.57%), ertapenem and meropenem (0.00%).

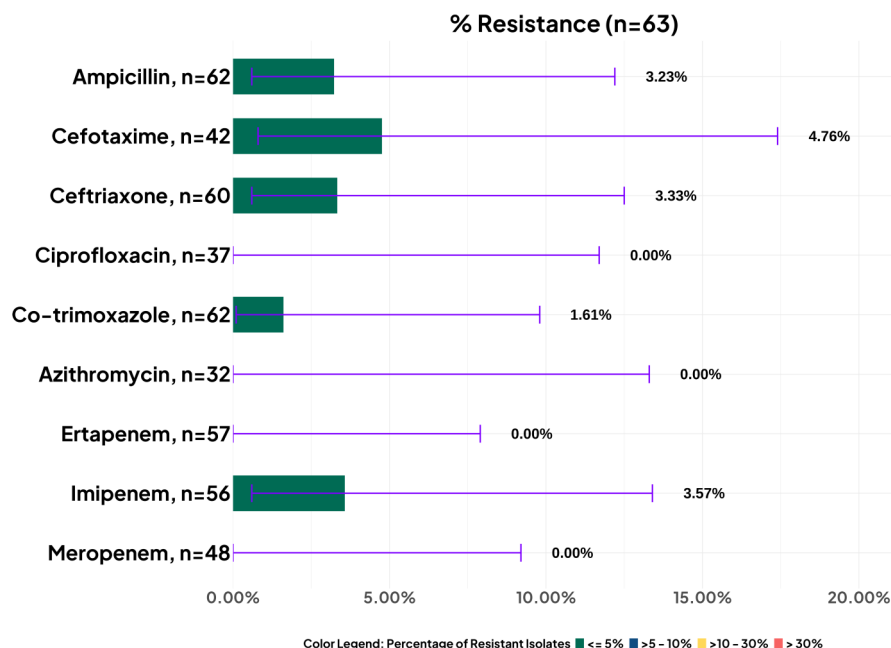


Figure 23. Proportion of all S. Typhi infections with resistance to tested antibiotics, DOH-ARSP 2025 (n=63)

Over the past decade, annual resistance rates for all *Salmonella enterica* serovar Typhi (S. Typhi) isolates to first-line and alternative therapeutic antibiotics have remained consistently low. Notably, the observed increase in resistance rates for chloramphenicol ($p=0.6840$) and decrease in azithromycin ($p=0.1000$) were both not statistically significant. No significant differences were observed for ciprofloxacin, co-trimoxazole, and ceftriaxone.

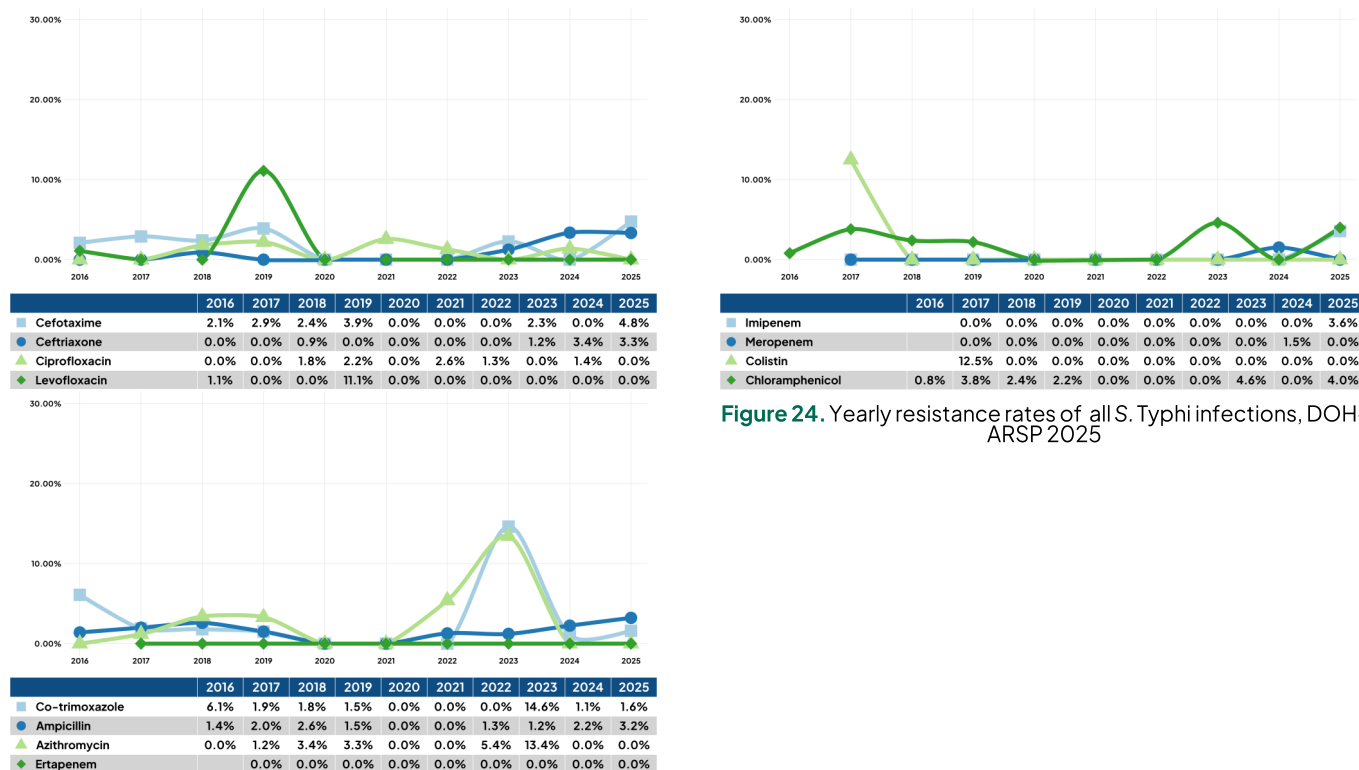


Figure 24. Yearly resistance rates of all S. Typhi infections, DOH-ARSP 2025



Bloodstream Infections

For *S. Typhi* bloodstream infections, antibiotic resistance rates were generally low, remaining below 5%, with several agents demonstrating 0% resistance. Resistance rates for cefotaxime, ceftriaxone, and ampicillin/co-trimoxazole were 2.78%, 1.89%, and 1.82%, respectively. Notably, the highest observed resistance was to imipenem at 4.08%.

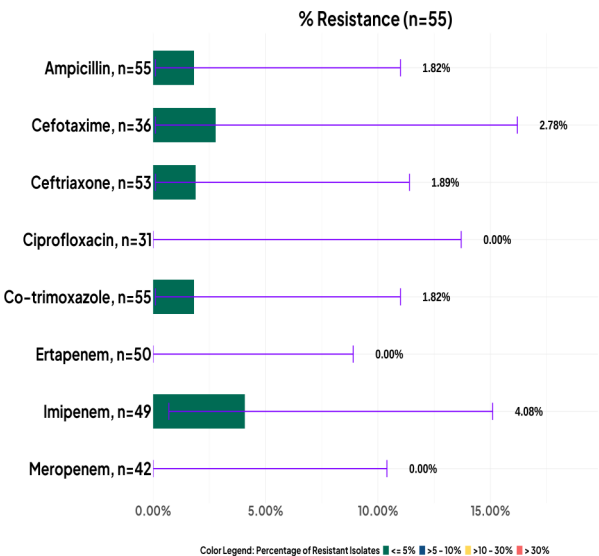


Figure 25. Proportion of *S. Typhi* bloodstream infections with resistance to tested antibiotics, DOH-ARSP, 2025 (n=55)



Diarrheal Disease

Figure 26 shows the resistance rates of *S. Typhi* diarrheal diseases. No resistance (0%) was observed to ciprofloxacin. Meanwhile, resistance to ceftriaxone was recorded at 5.71%, while ampicillin and co-trimoxazole resistance rates were 4.88% and 2.56%, respectively.

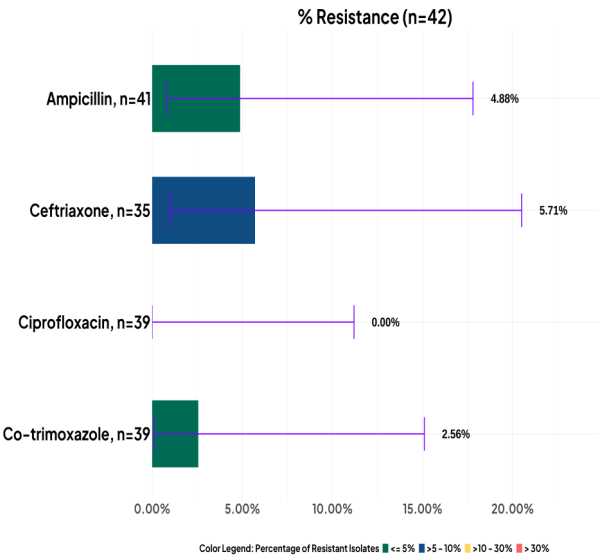
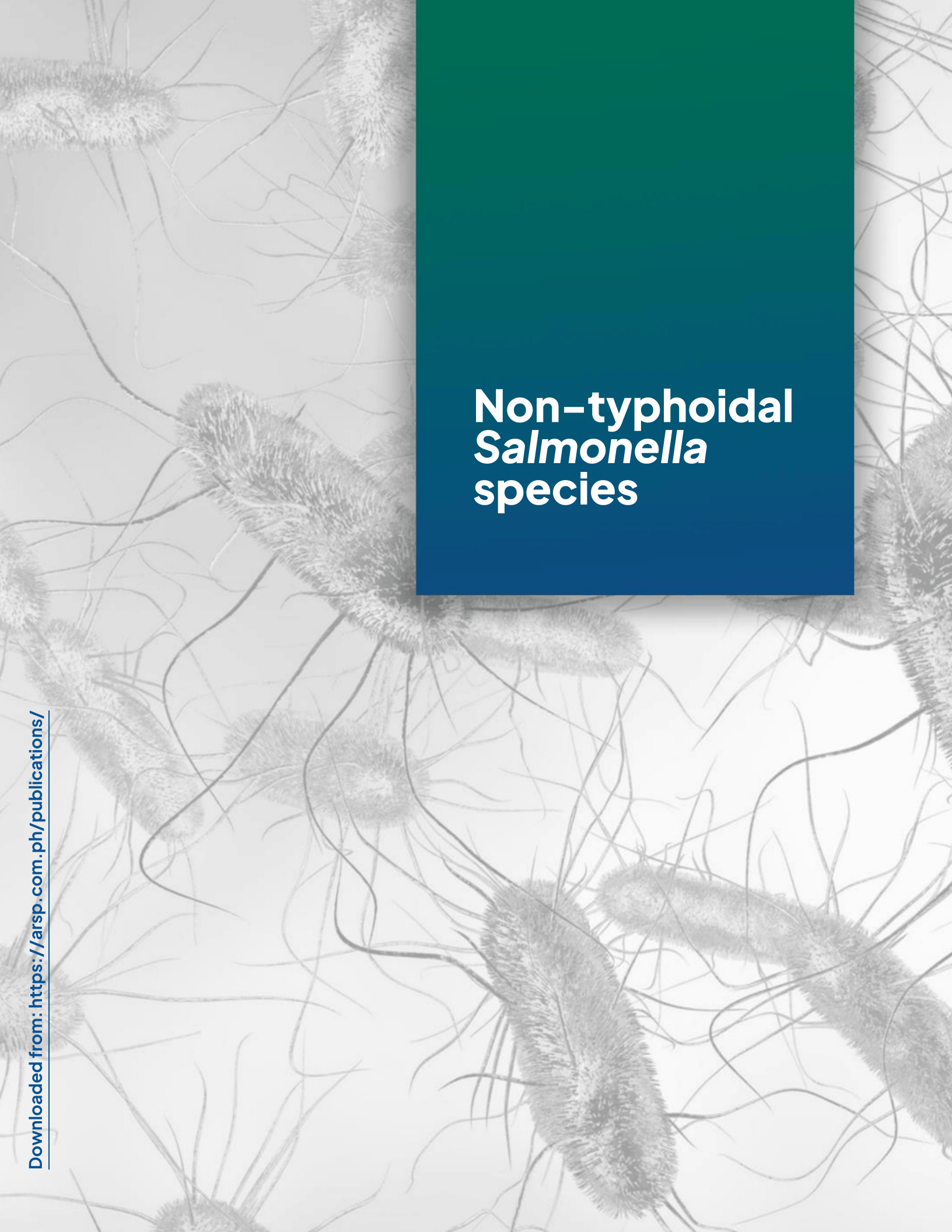


Figure 26. Proportion of *S. Typhi* diarrheal diseases with resistance to tested antibiotics from stool specimens, DOH-ARSP, 2017-2025 (n=42)



Non-typhoidal *Salmonella* species

Non-typhoidal *Salmonella* species

Non-typhoidal *Salmonella* (NTS) are gram-negative, motile bacilli that typically cause localized gastrointestinal inflammation rather than systemic fever. Their pathogenesis is driven by the invasion of intestinal epithelial cells, usually resulting in self-limiting diarrhea, though invasive cases are increasingly reported. The ARSP 2024 surveillance data showed that ceftriaxone resistance has surged to approximately 19.22%. These findings underscore a critical need for One Health interventions to curb the spread of multidrug-resistant NTS from the food chain to clinical environments.

A total of 452 non-typhoidal *Salmonella* (NTS) infections were reported for 2025. This was a 37.32% increase from the number of analyzed isolates in 2024. The two highest contributing sites were BRT (9.51%) and VSM (8.63%). Infections were mostly reported from Luzon (60.18%) followed by Mindanao (25.22%) and Visayas (14.60%).

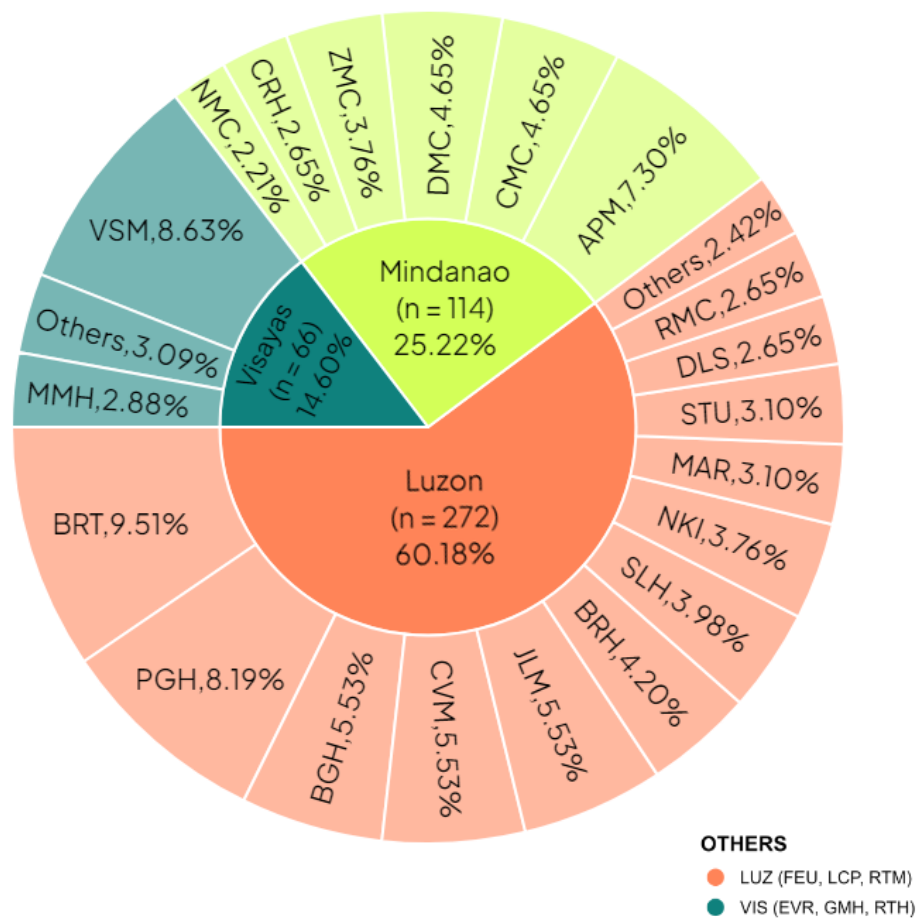


Figure 27. Distribution of NTS infections, DOH-ARSP, 2025 (n=452)

More than half (55.97%) of NTS infections occurred in male patients, and 50.00% were from the 20-64 age group. Many (45.35%) of the infections were reported bloodstream infections (Figure 28).

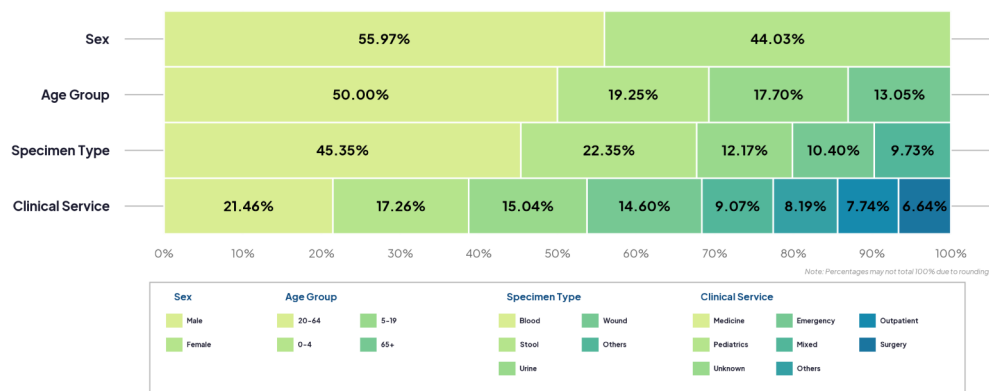


Figure 28. Patient characteristics in NTS infections, DOH-ARSP, 2025 (n=452)

All types of Infections

Figure 29 presents the 2025 resistance rates for all non-typhoidal *Salmonella* (NTS) infections. Compared to 2024, significant decreases in resistance were observed for both ciprofloxacin (9.12%, $p=0.000$) and ceftriaxone (17.92%, $p=0.0001$). Conversely, resistance rates for ampicillin, cefotaxime, and azithromycin were 37.47%, 20.22%, and 6.45%, respectively. Resistance to all tested carbapenems remained low, with rates below 2%.

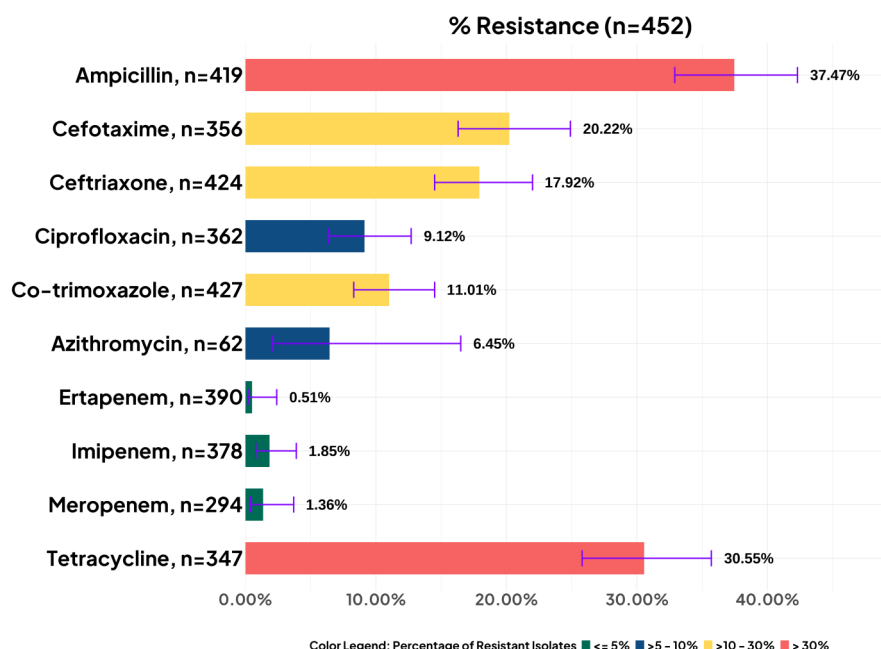


Figure 29. Proportion of all NTS infections with resistance to tested antibiotics, DOH-ARSP, 2025 (n=452)

Figure 30 shows the multi-year analysis of all NTS infections. Resistance rates to most infections decreased in 2025 however all changes were not statistically significant.

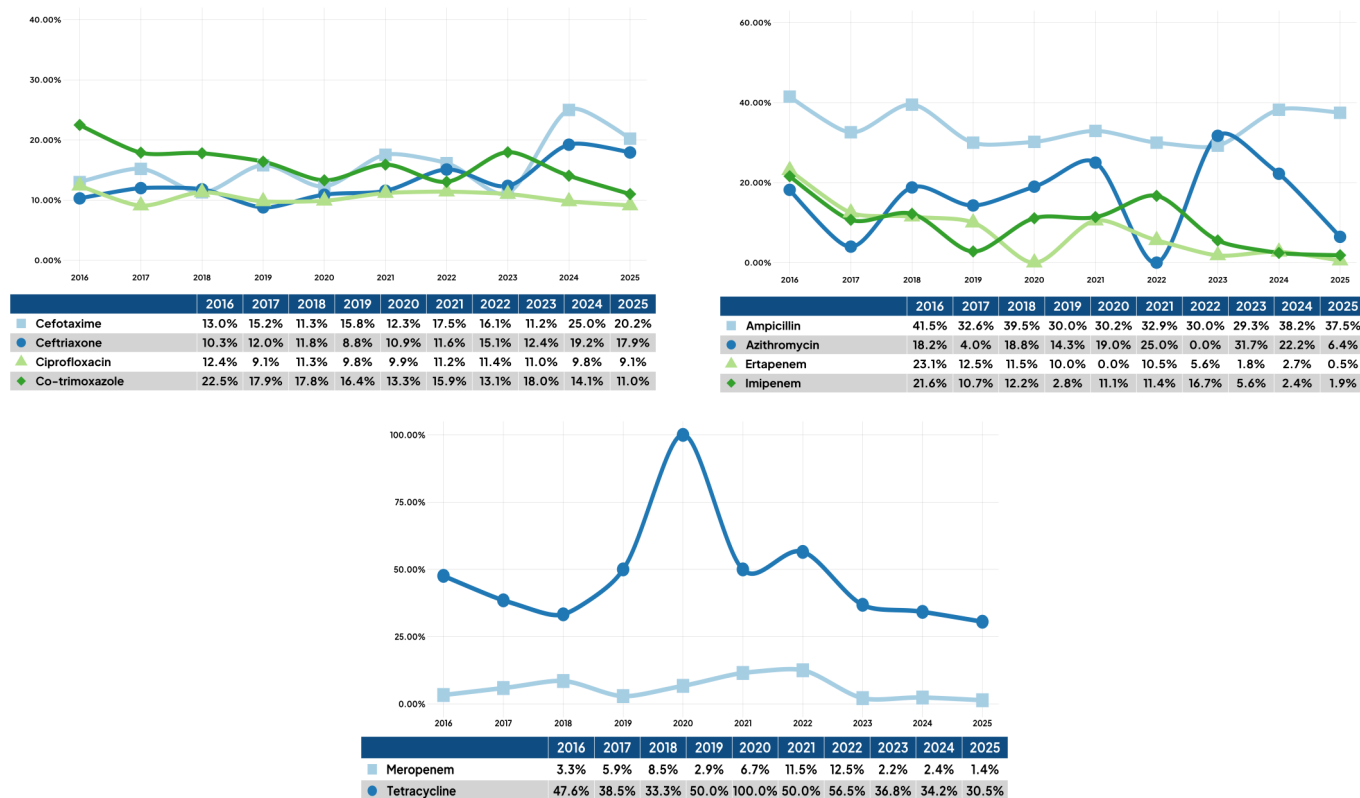
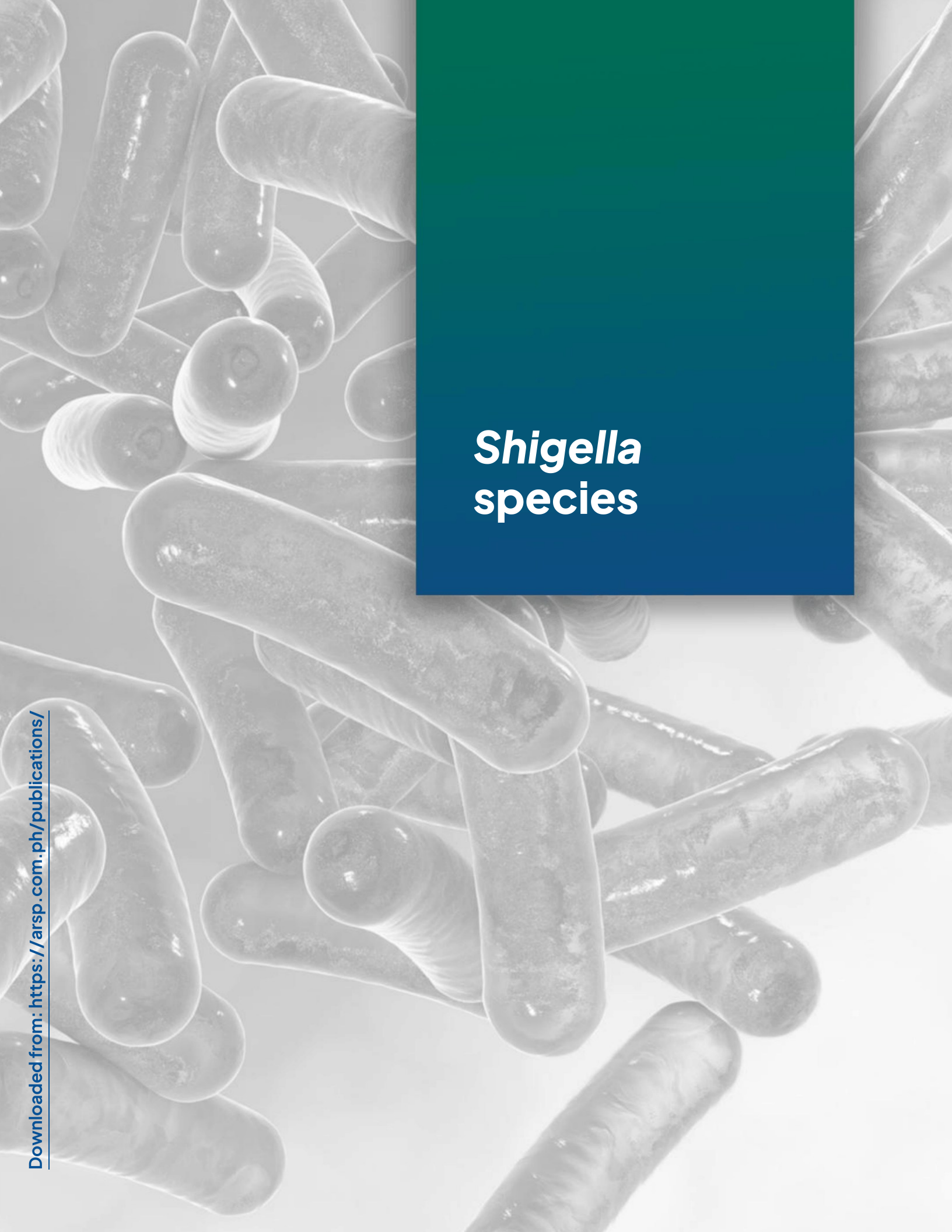


Figure 30. Yearly resistance rates of all NTS infections, DOH-ARSP 2025

A grayscale electron micrograph showing numerous Shigella bacteria. The bacteria are rod-shaped, with some appearing as single cells and others in pairs or small groups. They have a distinct, slightly textured surface and rounded ends. The background is a light, grainy gray.

***Shigella* species**

Shigella species

Shigella species present a major public health challenge due to a low infectious dose that drives rapid food, water, and person-to-person transmission. In the Philippines, shigellosis remains a critical cause of pediatric morbidity, disproportionately affecting communities with limited water, sanitation, and hygiene (WASH) infrastructure. National surveillance data indicates near-universal resistance to older therapies like ampicillin, alongside rising resistance to ciprofloxacin. This evolving resistance profile limits oral treatment alternatives, leaving ceftriaxone and azithromycin as the primary treatment options for severe clinical cases. Sustained surveillance and targeted interventions are therefore essential to monitor these patterns and preserve remaining treatment options.

There were **46** *Shigella* infections reported in 2025. Most infections were from VSM (34.78%). APM (10.87%) and CMC at 8.70%.

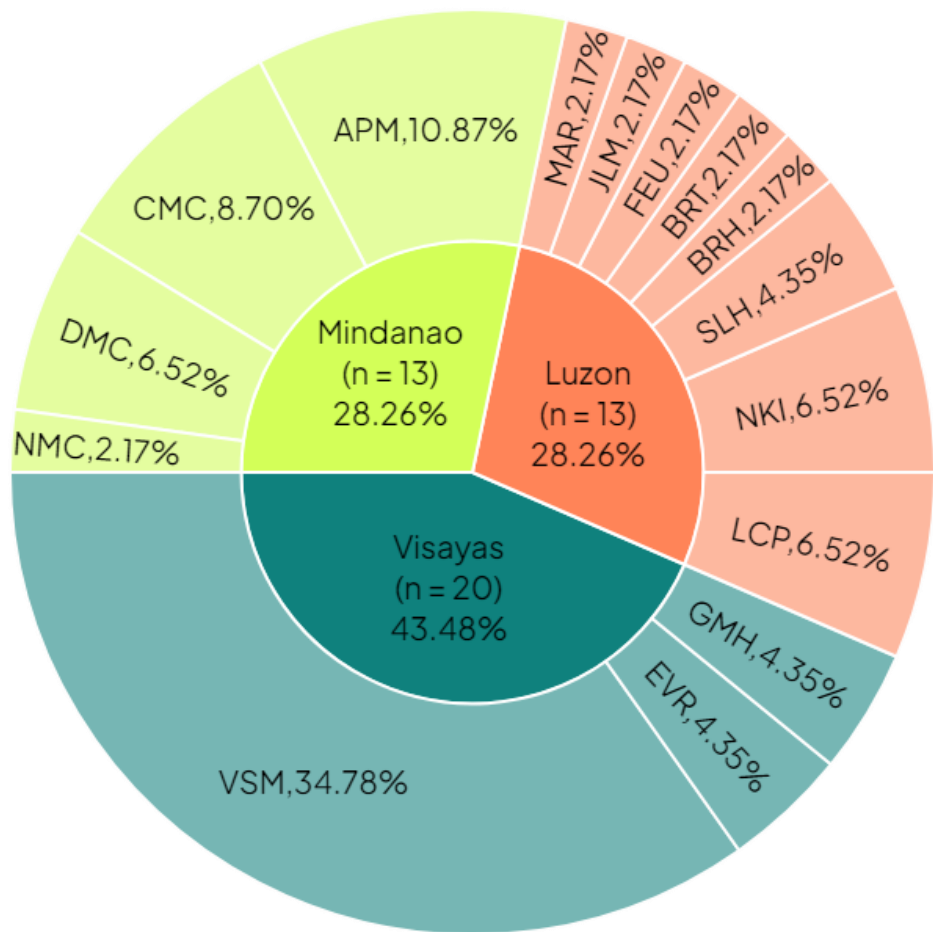


Figure 33. Distribution of *Shigella* infections, DOH-ARSP, 2025 (n=46)

More than half (52.17%) of the infections occurred among female patients and many are within the 20–64 (36.96%) and 0–4 (36.96%) age groups.

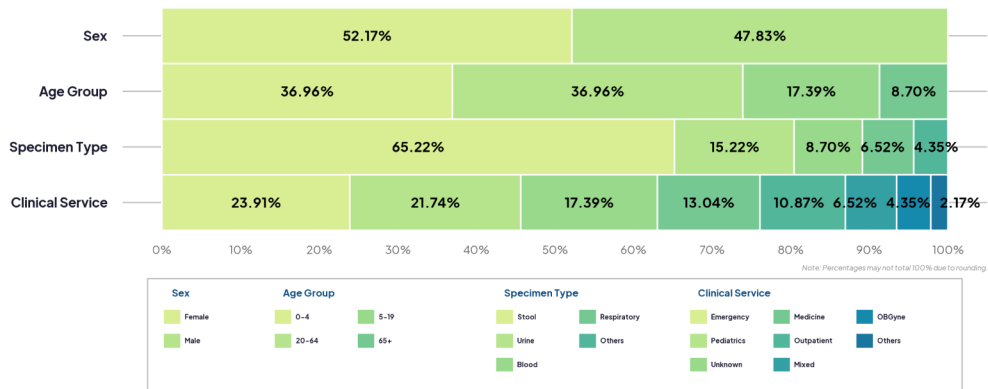


Figure 34. Patient characteristics in *Shigella* infections, DOH-ARSP, 2025 (n=46)

All types of Infections

Figure 35 shows the resistance rates of all *Shigella* infections. Resistance to ceftriaxone was at 13.24 %, and to ciprofloxacin at 9.33%. A comparison of the cumulative rates obtained from 2008 to 2024 (Figure 36) shows the changes in resistance rates of *Shigella* sp. Over the 10-year period, changes in the resistance rates for these infections were observed to be significant for ampicillin ($p = 0.0036$) and ceftriaxone ($p=0.0336$).

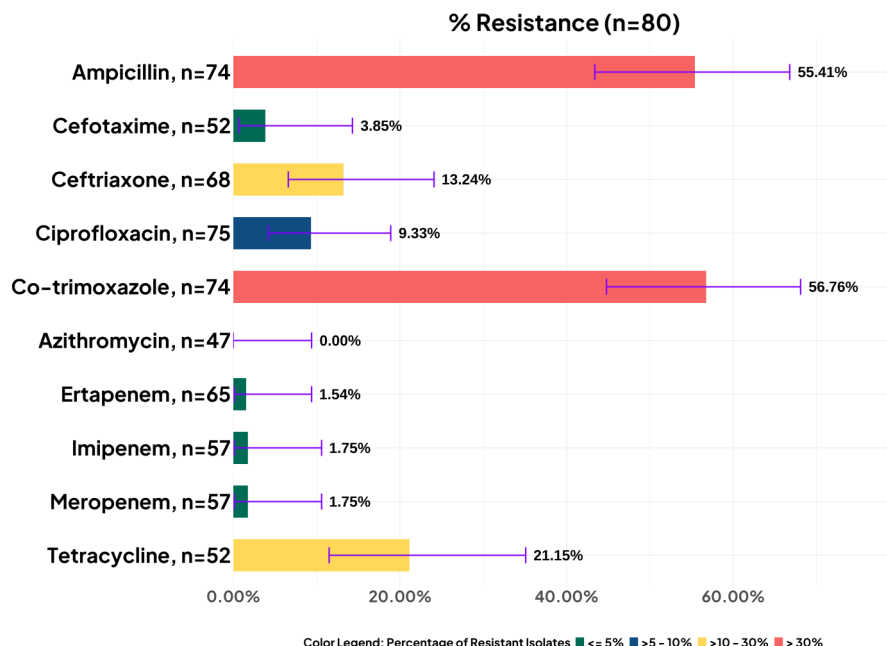


Figure 35. Proportion of all *Shigella* infections with resistance to tested antibiotics, DOH-ARSP, 2025 (n=46)

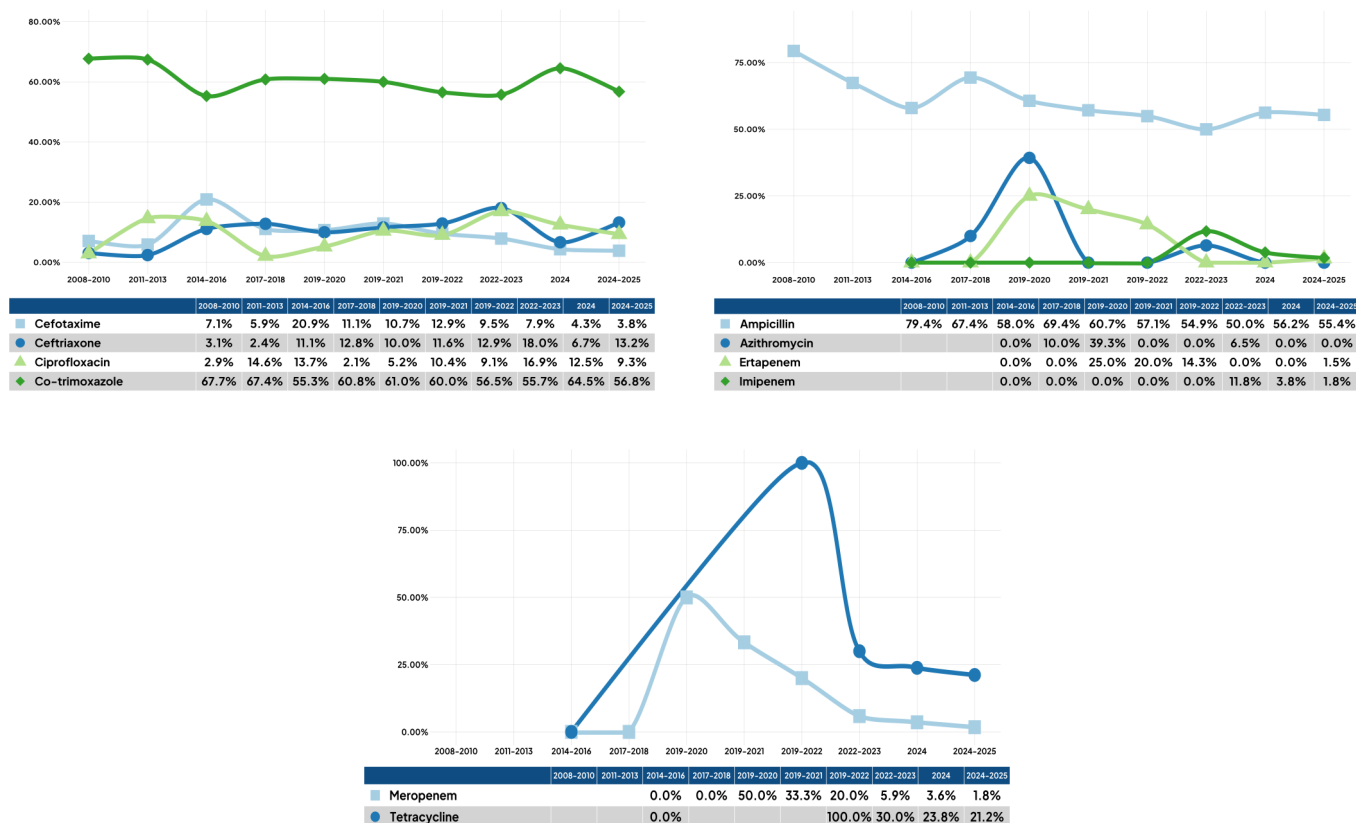


Figure 36. Yearly resistance rates of all *Shigella* infections, DOH-ARSP, 2025



Diarrheal Disease

Figure 37 shows the resistance rates for *Shigella* diarrheal diseases in 2025. High resistance rates of 60% were observed for both co-trimoxazole and ampicillin, however, these changes in resistance rates were not statistically significant. Conversely, resistance rates for ciprofloxacin, ceftriaxone, and azithromycin were all below 5%.

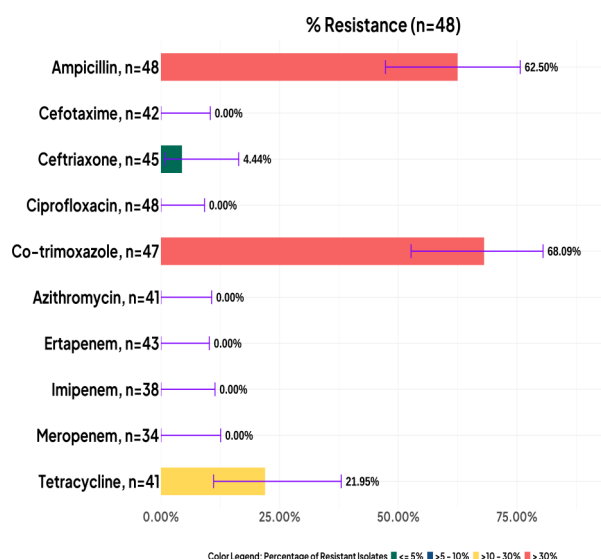
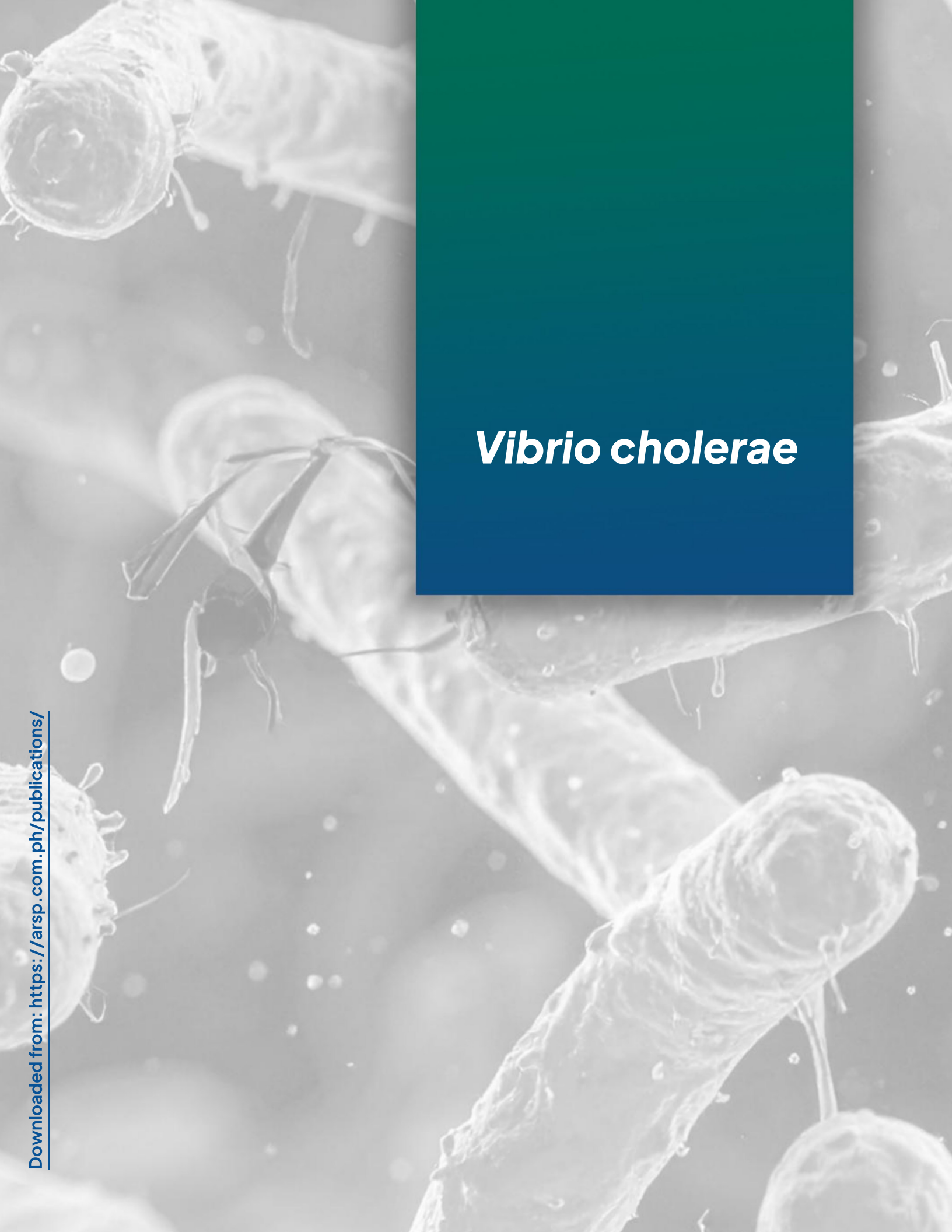


Figure 37. Proportion of *Shigella* diarrheal infections resistant to tested antibiotics, DOH-ARSP, 2025 (n=30)

Table 7. *Shigella* serotypes results, DOH-ARSP, 2025

Prediction	Number	%
<i>Shigella flexneri</i> serotype 1c (7a)	8	47.1
<i>Shigella sonnei</i> form II	5	29.4
<i>Shigella flexneri</i> serotype 1a	2	11.8
<i>Shigella flexneri</i> serotype 2b	2	11.8

A scanning electron micrograph (SEM) of Vibrio cholerae bacteria. The image shows several elongated, comma-shaped bacteria with prominent flagella at one end. The bacteria are set against a dark, textured background. A semi-transparent blue rectangular box is overlaid on the right side of the image, containing the text 'Vibrio cholerae' in white.

Vibrio cholerae

Vibrio cholerae

Vibrio cholerae is a gram-negative, curved rod distinguished by its positive oxidase reaction and growth in alkaline conditions. Its pathogenesis centers on the cholera toxin, which triggers rapid, massive fluid loss and classic "rice-water" stools. In the Philippines, the O1 El Tor biotype persists as the primary cause of waterborne outbreaks in areas with poor sanitation. The 2024 ARSP data showed that tetracycline resistance remained below 7% over the past ten years. Resistance for azithromycin and chloramphenicol was less than 2% and co-trimoxazole showed a statistically significant increase in resistance rates.

There were reported 49 *Vibrio cholerae* infections for 2025 which mainly come from VSM in Visayas (57.14%), followed by Mindanao (40.82%) and Luzon (2.04%) (Figure 38).

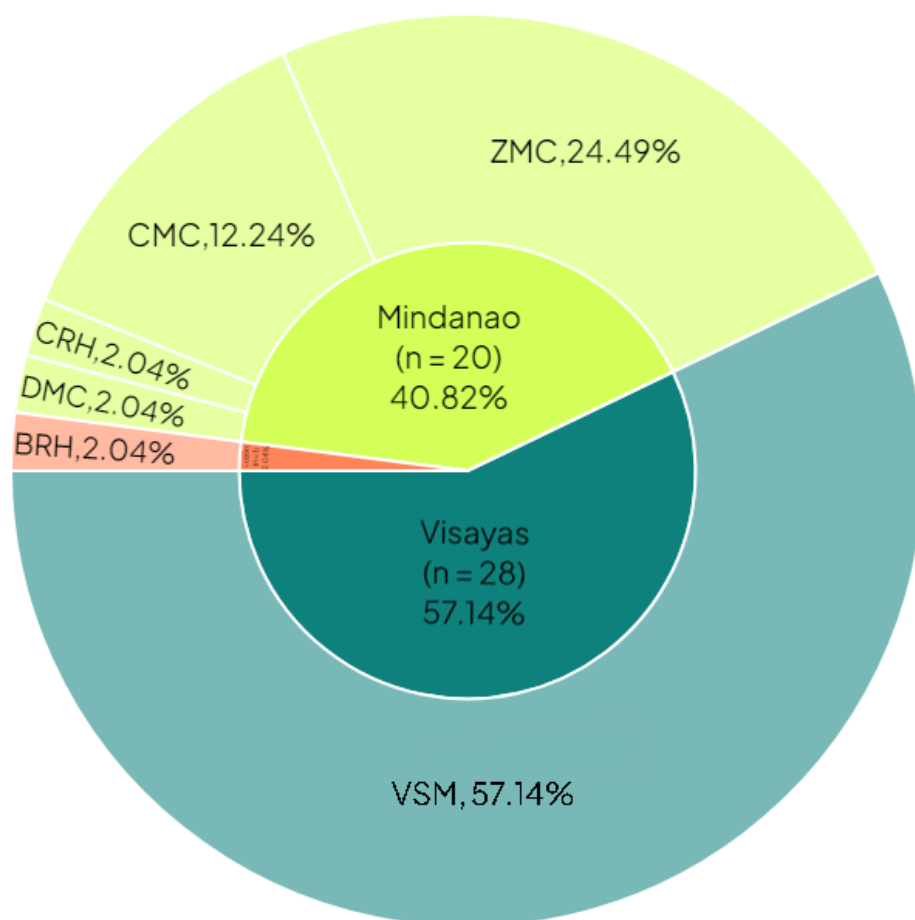


Figure 38. Distribution of *V. cholerae* infections, DOH-ARSP, 2025 (n=49)

Figure 39 shows that more than half (67.35%) of the *V. cholerae* infections were from male patients and were mostly seen among 5-19 (36.73%) and 0-4 (30.61%) age groups. Most (91.84%) of the isolates were detected from stool specimens.

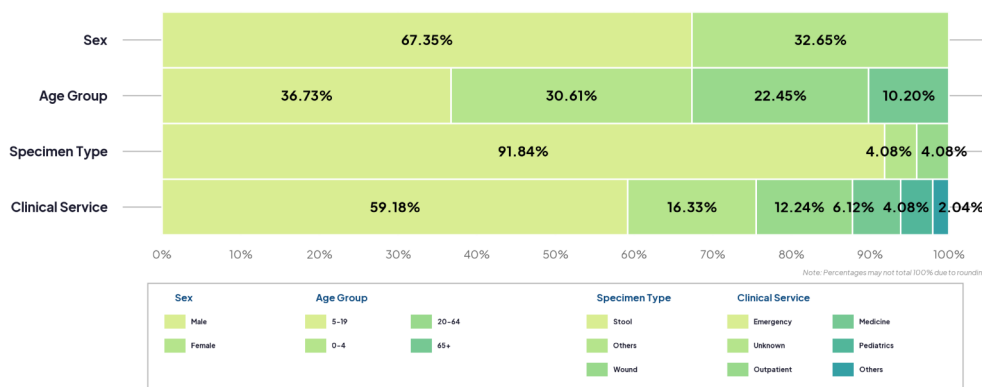


Figure 39. Patient characteristics in *V. cholerae* infections, DOH-ARSP, 2025 (n=49)

All types of Infections

In 2025, all *V. cholerae* infections were susceptible to chloramphenicol and azithromycin. A notable decline in resistance was observed for ampicillin, dropping from 28.4% during the 2023–2024 period to 10.20% in 2025. Similarly, co-trimoxazole resistance decreased to 4.08% from the 6.8% reported the previous year.

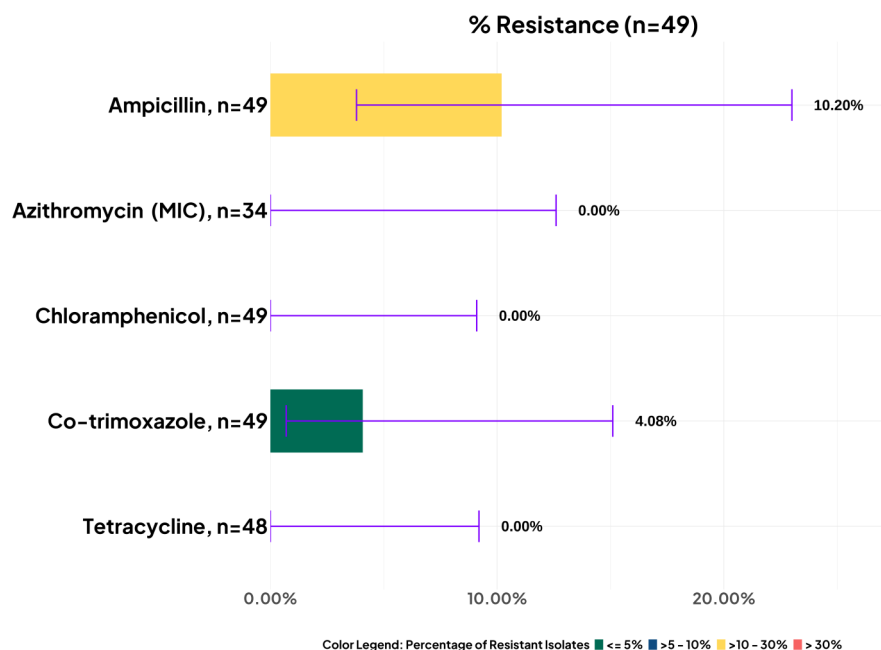


Figure 40. Proportion of all *V. cholerae* infections resistant to tested antibiotics, DOH-ARSP, 2025 (n=49)

Figure 41 shows the yearly resistance rates of all *V. cholerae* infections. The changes in resistance rate for co-trimoxazole was observed to be significant.

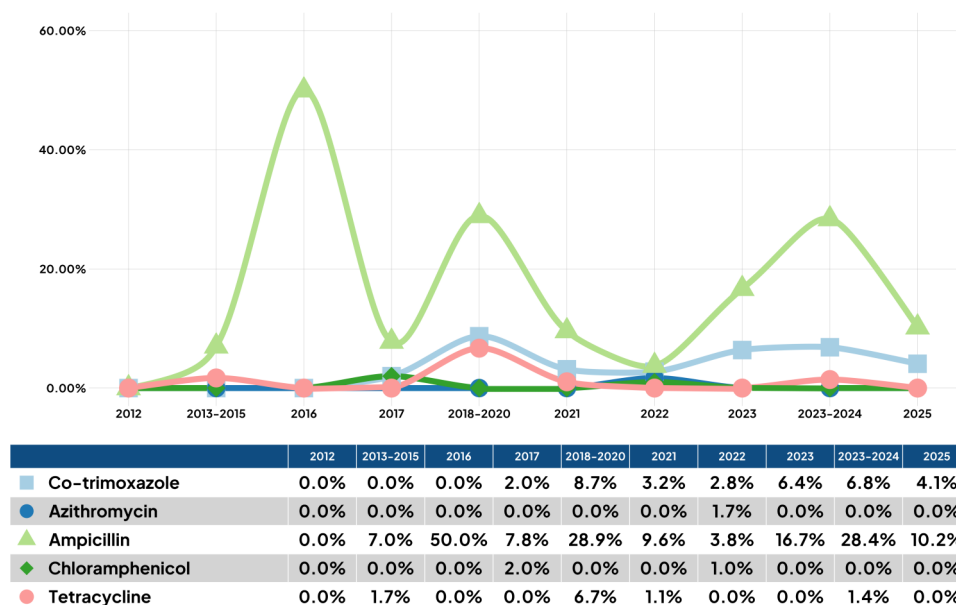
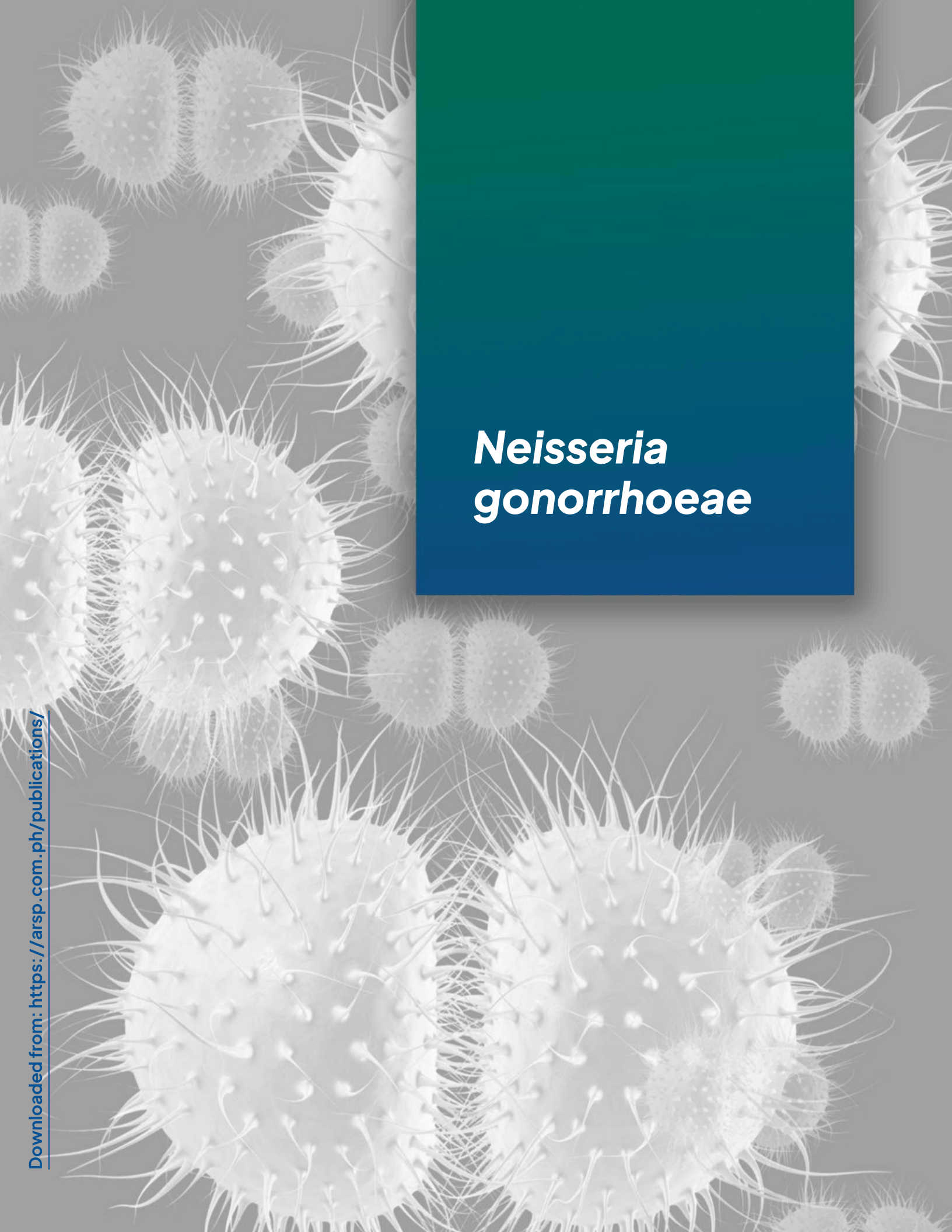


Figure 41. Yearly resistance rates of all *V. cholerae* infections, DOH-ARSP, 2025

The background of the slide is a grayscale electron micrograph of Neisseria gonorrhoeae. It shows numerous spherical bacteria, some appearing in pairs (diplococci) and others in small groups. Each bacterium is covered in fine, hair-like pili that radiate from its surface. The bacteria are set against a dark, textured background.

Neisseria gonorrhoeae

Neisseria gonorrhoeae

Neisseria gonorrhoeae remains a significant sexually transmitted infection challenge in the Philippines, with the epidemiological burden concentrated among the population aged 20–64. Regional data from the Enhanced Gonococcal Antimicrobial Surveillance Programme (EGASP) indicates declining ceftriaxone susceptibility across Southeast Asia; however, local surveillance has not yet detected resistance to ceftriaxone or azithromycin. In contrast, the therapeutic utility of older antimicrobial classes is severely limited, with 2025 surveillance data showing resistance rates of 81.56% for ciprofloxacin and 76.06% for tetracycline. These high baseline resistance rates underscore the importance of maintaining current frontline empirical treatment guidelines. Sustained national surveillance and structured infection control strategies are therefore necessary to monitor susceptibility shifts and mitigate the risk of emerging resistance.

A total of **164** *N. gonorrhoeae* infections were reported for 2025. This is a 12.33 % increase from 146 infections reported in 2024. The highest contributors are CVM (23.17%), DMC (15.85 %) and MAR (12.80%) (**Figure 42**). Based on island group distribution, the highest was Luzon with 56.71 % followed by Visayas at 25.61%, and Mindanao at 17.68%.

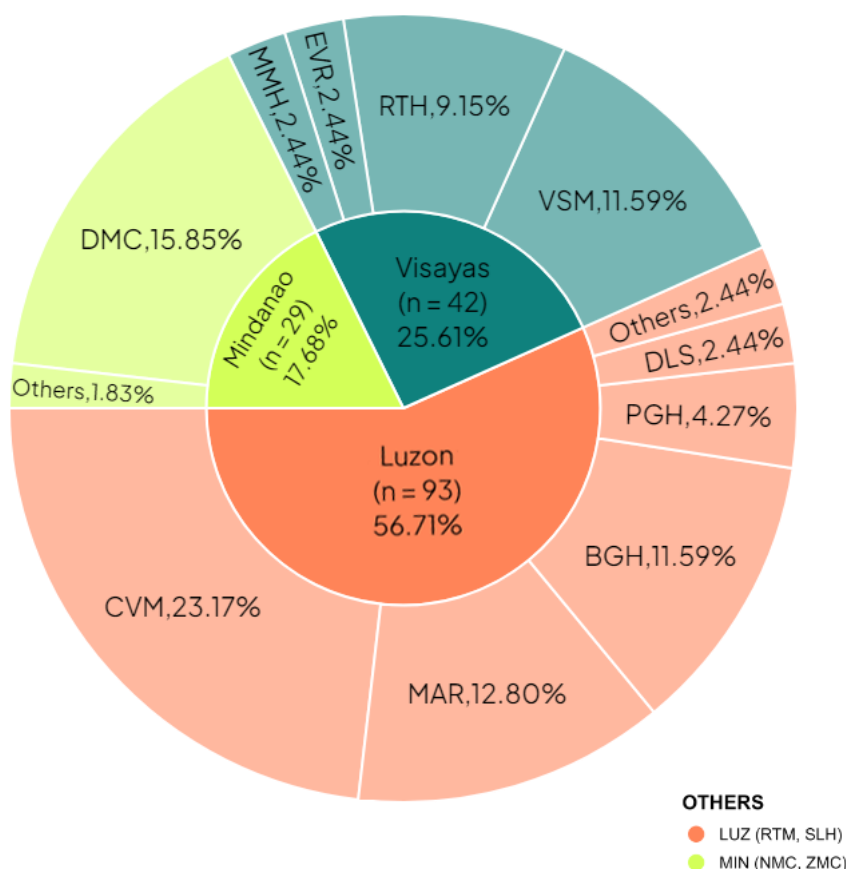


Figure 42. Distribution of *N. gonorrhoeae* infections, DOH-ARSP, 2025 (n=164)

The majority of infections reported were observed in male patients (91.46%), with the 20–64 age group comprising 89.02%. Genital specimens remain to be the most common source of *N. gonorrhoeae* isolates at 78.66%.

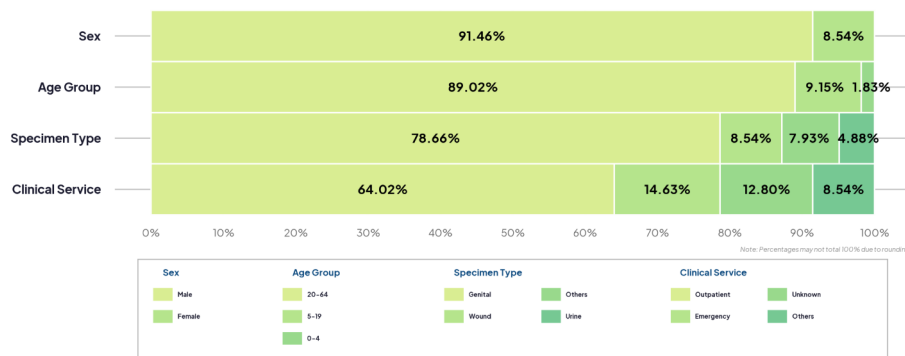
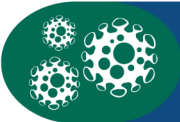


Figure 43. Patient characteristics in *N. gonorrhoeae* infections, DOH-ARSP, 2025 (n=164)



All types of Infections

The figure presents antimicrobial resistance rates for *N. gonorrhoeae*. While high resistance rates were observed for ciprofloxacin (81.56%) and tetracycline (76.06%), resistance to ceftriaxone, cefixime, and azithromycin remained at 0%, indicating complete susceptibility to these first-line treatment options.

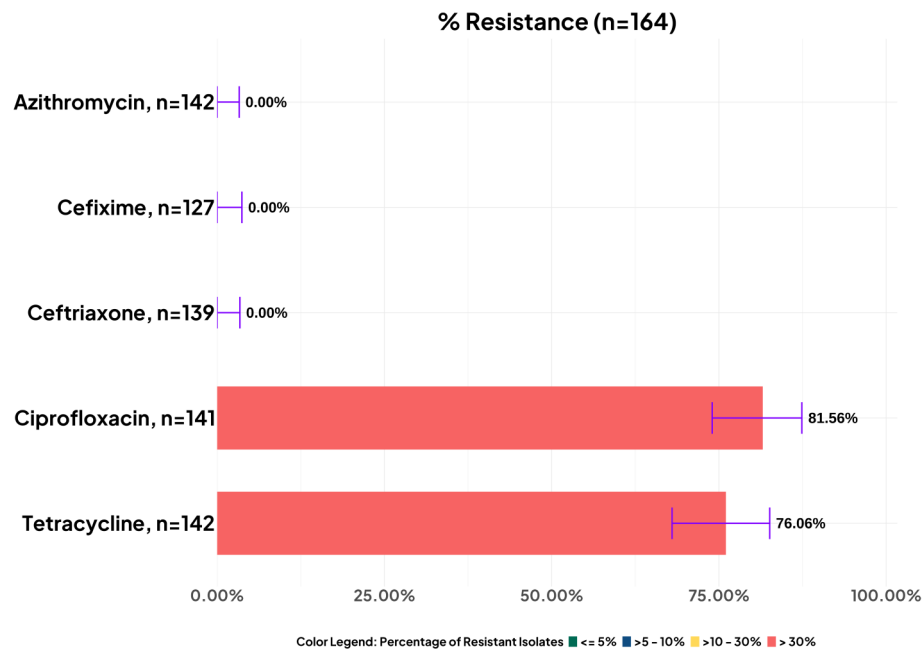


Figure 44. Proportion of all *N. gonorrhoeae* infections resistant to tested antibiotics, DOH-ARSP, 2025 (n=164)

Figure 45 shows the multi-year analysis of antibiotic resistance rates in gonococcal infections across the different antibiotics tested. No confirmed resistance to ceftriaxone and azithromycin have been reported over the past four years. All changes in yearly resistance rates were not statistically significant except for tetracycline (p=0.0000).

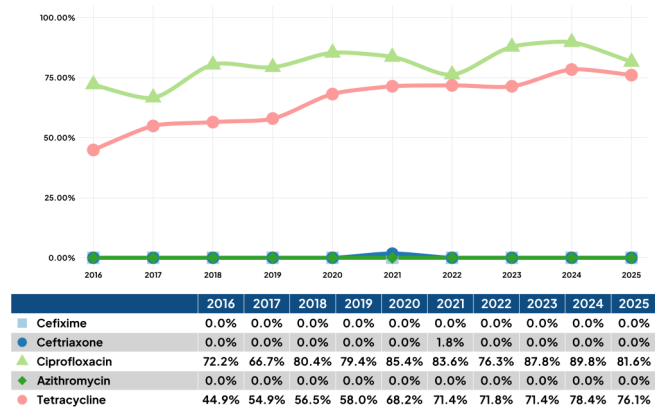


Figure 45. Yearly resistance rates of all *N. gonorrhoeae* infections, DOH-ARSP, 2025

Surveillance data for *Neisseria gonorrhoeae* resistant to ceftriaxone was reported for 2025, showing 0.0% resistance (n=164) as it has been in 2024. The EGASP (Enhanced Gonococcal Antimicrobial Susceptibility Programme), sustained under ARSP, contributes in generating more robust data for future monitoring cycles. The current rate, while based on a very small sample, is below the 1% PNAP-AMR 2024-2028 target. Scale-up of gonorrhea surveillance remains an essential priority.

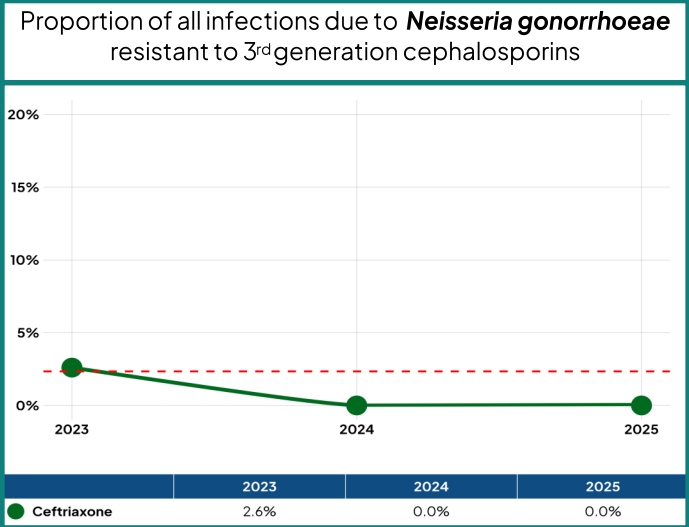
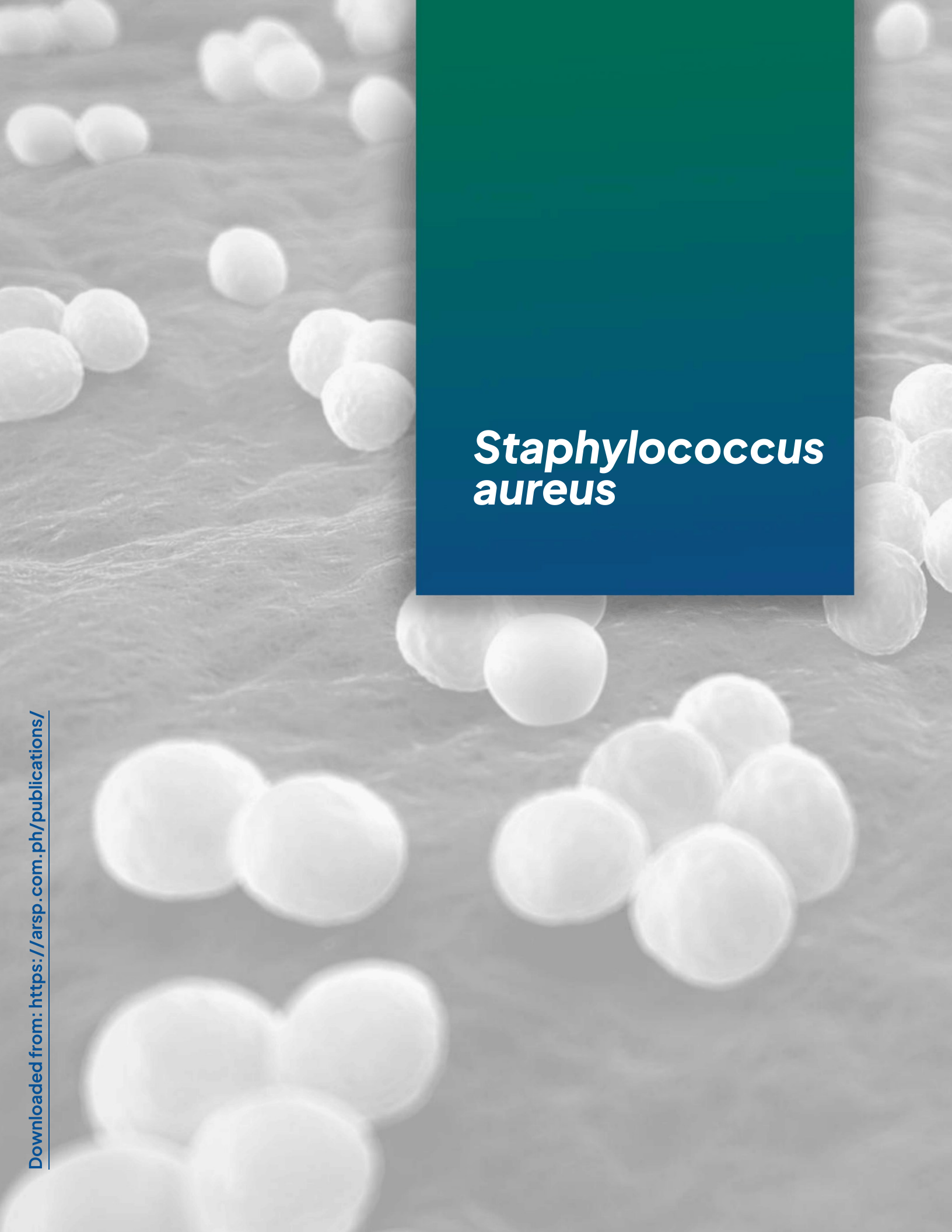


Figure 46. Yearly resistance rates of all *N. gonorrhoeae* infections, DOH-ARSP, 2023-2025

A scanning electron micrograph (SEM) of Staphylococcus aureus bacteria. The image shows numerous spherical, Gram-positive cocci. Some bacteria are isolated, while others are in small clusters or chains. The background is a textured, greyish surface, likely a substrate or a slide. A dark blue rectangular box is overlaid on the right side of the image, containing the text 'Staphylococcus aureus' in white.

Staphylococcus aureus

Staphylococcus aureus

Staphylococcus aureus pathogenesis relies on a potent array of toxins and enzymes that contribute to the development of infections ranging from localized skin and soft tissue infections to systemic sepsis. It remains a major global health burden, causing significant morbidity, mortality, and healthcare costs. In the Philippines, the epidemiology reflects a high clinical burden, particularly in bloodstream and surgical site infections across all age groups. A significant downward trend in oxacillin resistance among *Staphylococcus aureus* isolates has been observed over the past five years

There were **9,346** *Staphylococcus aureus* infections reported for 2025. A 7.84% increase in *S. aureus* infections was observed from 2024. The majority of *S. aureus* infections came from surveillance sites in Luzon (62.25%) Mindanao (23.15%), and Visayas (14.59%) (**Figure 47**).

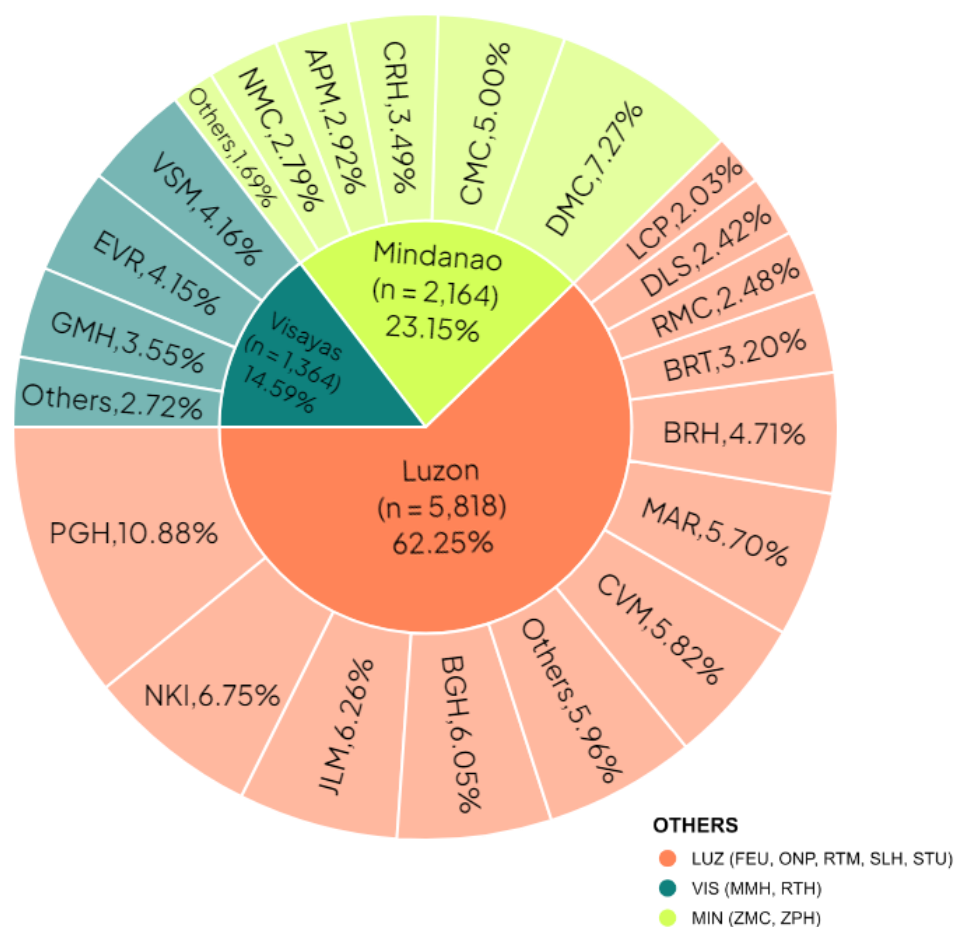


Figure 47. Distribution of *S. aureus* infections, DOH-ARSP, 2025 (n=9,346)

More than half of the infections (54.63%) were observed among male patients and most were from aged 20-64 years old (65.32%). As shown in **Figure 48**, most infections were isolated from skin and soft tissue (43.19%) and blood specimens (23.71%). The majority of the *S. aureus* infections (77.13%) were community-associated.

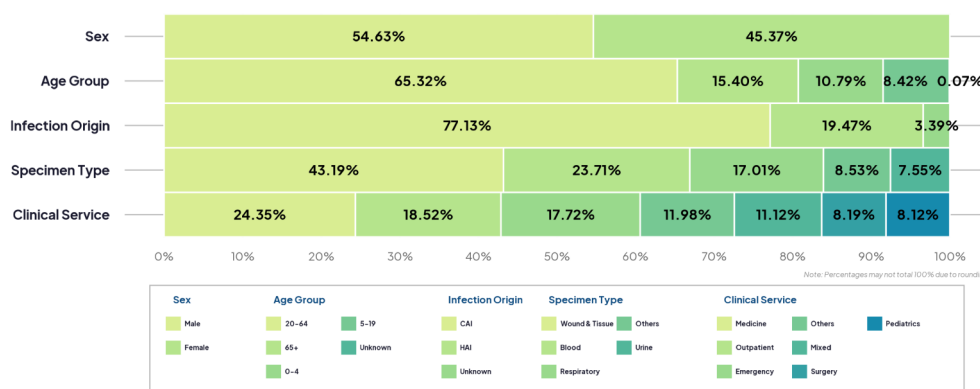


Figure 48. Patient characteristics in *S. aureus* infections, DOH-ARSP, 2025 (n=9,346)

All types of Infections

Figure 49 shows the resistance rates of isolates from all *S. aureus* infections. Oxacillin resistance was 35.26%, while co-trimoxazole resistance was 36.81%. Clindamycin resistance was observed in 11.30% of isolates, including those with inducible clindamycin resistance. Resistance rates for vancomycin and linezolid were recorded at 1.19% and 0.71%, respectively, however, these phenotypes were not confirmed by the reference laboratory.

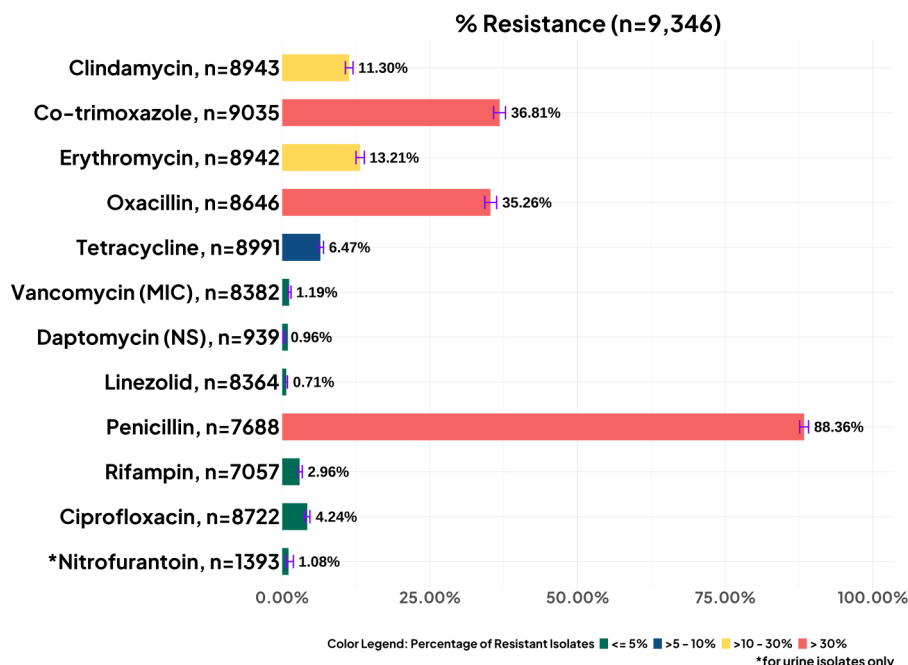


Figure 49. Proportion of all *S. aureus* infections resistant to tested antibiotics, DOH-ARSP, 2025 (n=9,346)

The resistance rates of all *S. aureus* infections are shown in **Figure 50**. Over the past decade, statistically significant changes in resistance rates were observed for oxacillin ($p=0.0000$), vancomycin ($p=0.0001$), and co-trimoxazole ($p=0.0000$), with oxacillin resistance showing a steadily decreasing trend.

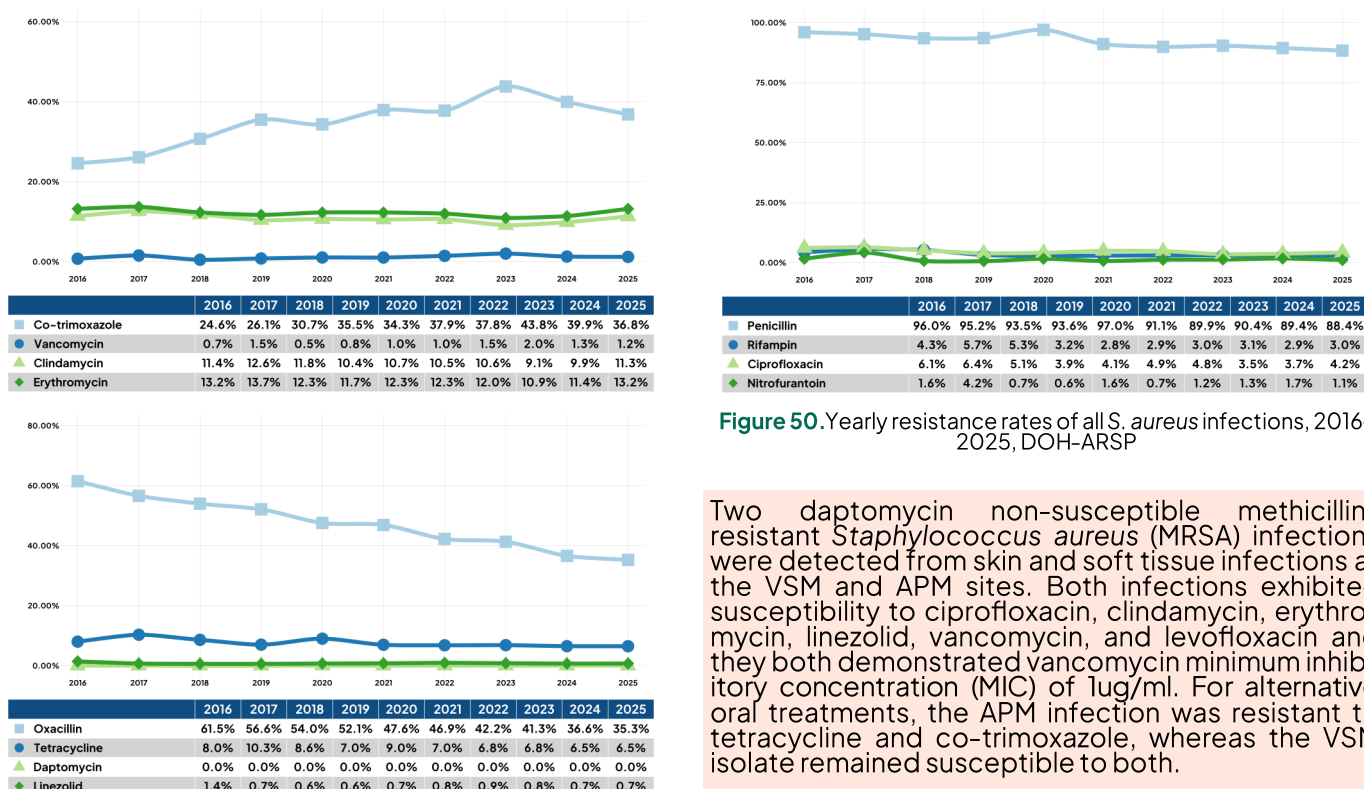


Figure 50. Yearly resistance rates of all *S. aureus* infections, 2016-2025, DOH-ARSP

Two daptomycin non-susceptible methicillin-resistant *Staphylococcus aureus* (MRSA) infections were detected from skin and soft tissue infections at the VSM and APM sites. Both infections exhibited susceptibility to ciprofloxacin, clindamycin, erythromycin, linezolid, vancomycin, and levofloxacin and they both demonstrated vancomycin minimum inhibitory concentration (MIC) of 1µg/ml. For alternative oral treatments, the APM infection was resistant to tetracycline and co-trimoxazole, whereas the VSM isolate remained susceptible to both.

Bloodstream Infections

Figure 51 shows the resistance rates of *S. aureus* bloodstream infections. The oxacillin resistance rate was at 37.53%, while clindamycin, ciprofloxacin resistance rates were at 12.83% and 5.59% respectively. Low resistance rate was observed on vancomycin, linezolid, and daptomycin at 0.97%, 0.58 %, 1.59%, respectively.

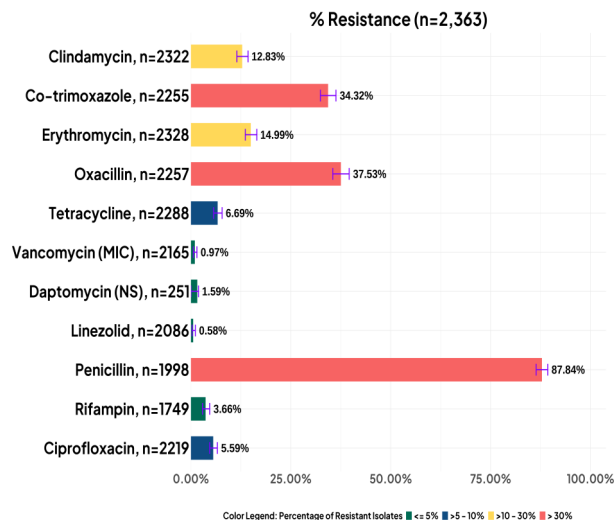


Figure 51. Proportion of *S. aureus* bloodstream infections resistant to tested antibiotics, DOH-ARSP, 2025 (n=2,363)

Figure 52 illustrates the resistance rates of *S. aureus* bloodstream infections over a 10-year period. The resistance rates in bloodstream infections were generally similar to those observed across all infection types. Notably, multiyear analysis results showed that the changes in resistance rates for co-trimoxazole (p=0.000), erythromycin (0.0062), oxacillin (p= 0.0000), penicillin (p= 0.0000) and gentamicin (0.0011).

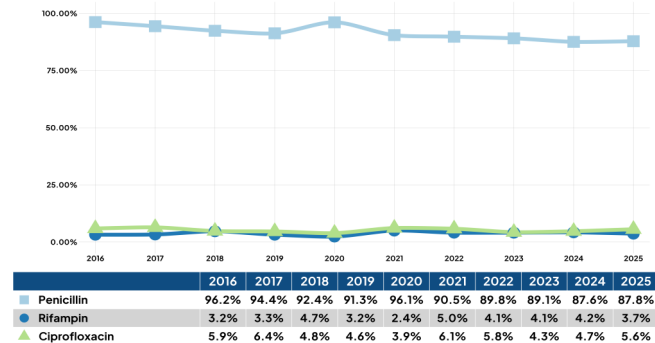
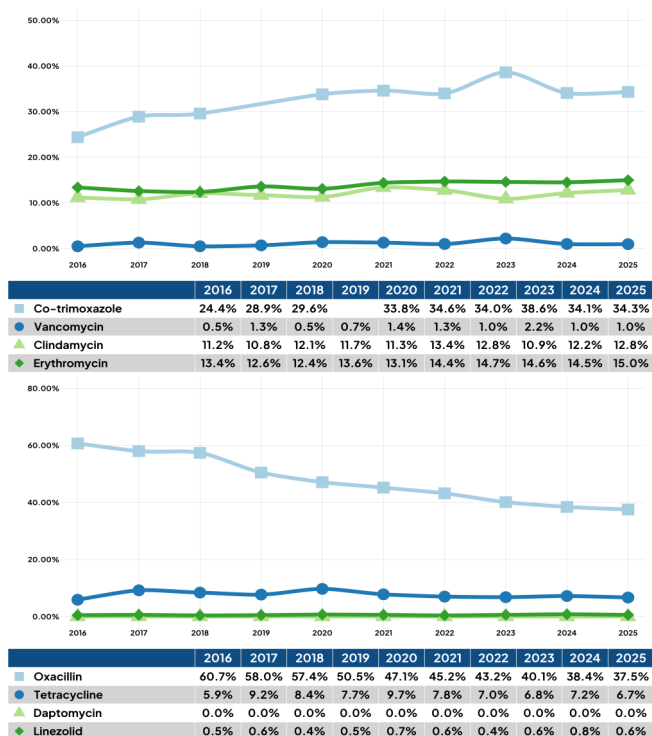


Figure 52. Yearly resistance rates of *S. aureus* bloodstream infections, 2016-2025, DOH-ARSP

Skin and Soft Tissue Infections

Resistance to oxacillin was at 30.98%, clindamycin at 9.16% and erythromycin at 10.22%. Vancomycin and linezolid resistance were less than 1%. *S. aureus* skin and soft tissue infections demonstrated comparatively lower resistance rates than all *S. aureus* infections.

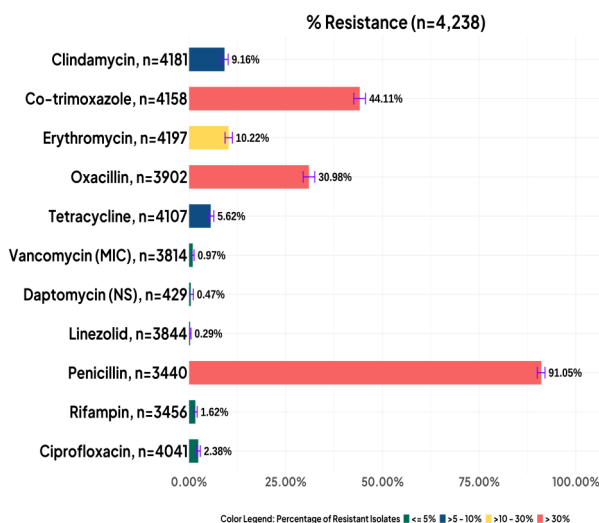
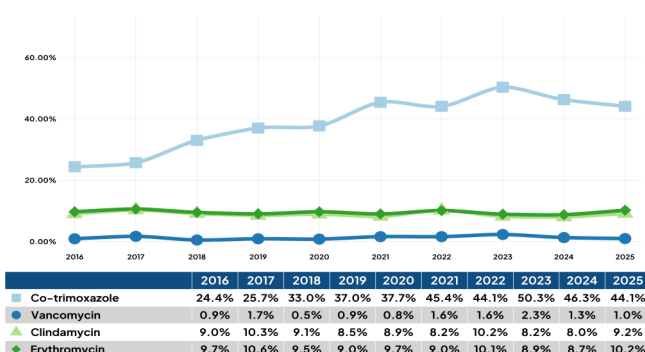


Figure 53. Proportion of *S. aureus* skin and soft tissue infections resistant to tested antibiotics, DOH-ARSP, 2025 (n=4,305)

Figure 54 shows the multi-year analysis of *S. aureus* skin and soft tissue infections. Oxacillin resistance (p=0.000) showed a decreasing trend over the last ten years, while multi-year analysis showed that the differences noted across years were significant for all antibiotics.



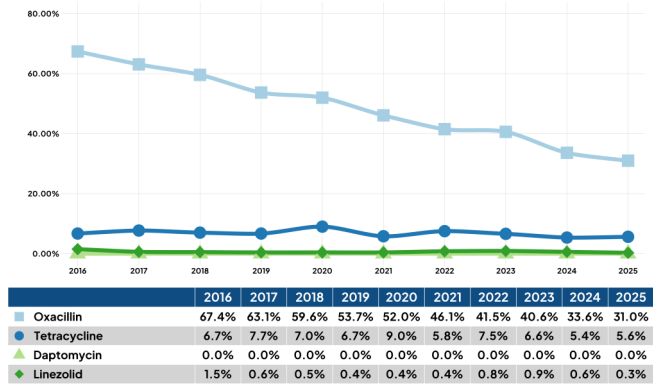
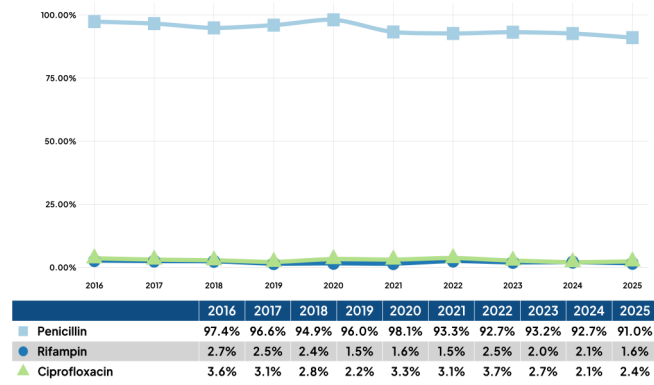


Figure 54. Yearly resistance rates of *S. aureus* skin and soft tissue infections, 2016–2025.DOH-ARSP



Global antimicrobial resistance (AMR) is monitored via Sustainable Development Goal (SDG) indicator 3.d.2, which measures the proportion of bloodstream infections (BSIs) caused by methicillin-resistant *Staphylococcus aureus* (MRSA). As a major cause of both healthcare-associated and community-acquired infections worldwide, MRSA presents a persistent clinical challenge. Its sustained prevalence in systemic infections directly impacts patient mortality, morbidity, and overall healthcare costs.

Figure 55 demonstrates a downward trend in MRSA resistance rates among bloodstream isolates, falling from 45.2% in 2021 to 37.5% in 2025. Serving as a proxy for MRSA BSIs, the ARSP data suggest measurable progress in the Philippines toward achieving this SDG target.

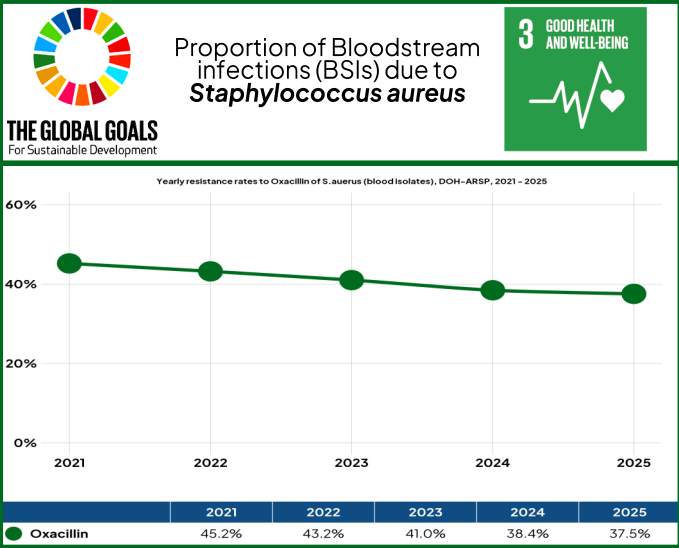


Figure 55. Yearly resistance rates of *S. aureus* bloodstream infections, DOH-ARSP, 2021–2025

The current monitoring of *Staphylococcus aureus* bloodstream infection resistance rates shows a decreasing trend, indicating progress toward the PNAP 2024–2028. Target 3 goal of reducing Methicillin-resistant *S. aureus* (MRSA) infections by 10%. The resistance rate has decreased by 6.43% since 2023, reflecting improvements in MRSA prevention and control efforts.

Despite this progress, the achieved reduction remains below the established target. However, the continued downward trend in resistance rates demonstrates positive movement toward the goal and highlights the need to sustain and strengthen the implementation of an integrated, comprehensive, and sustainable national program to address antimicrobial resistance.

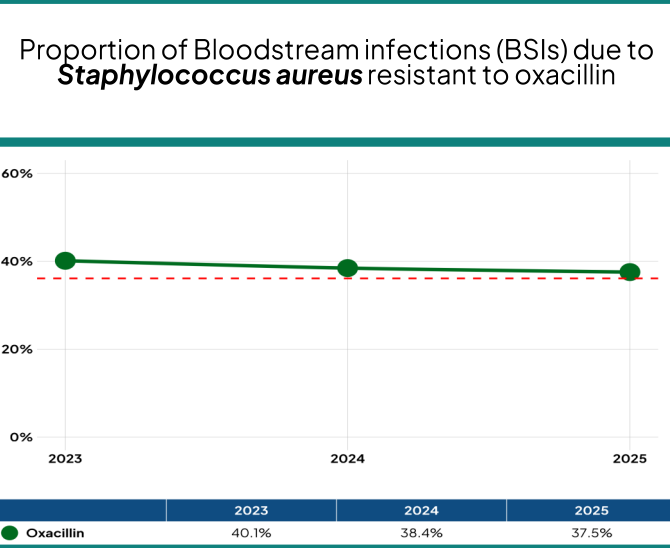


Figure 56. Yearly resistance rates of *S. aureus* bloodstream infections, DOH-ARSP, 2023–2025

A scanning electron micrograph (SEM) of Staphylococcus aureus bacteria. The image shows numerous spherical, Gram-positive cocci arranged in various clusters and chains. The bacteria are set against a background of fine, fibrous structures, likely part of a biofilm or extracellular matrix. A dark teal rectangular box is overlaid on the right side of the image, containing the title text.

Methicillin-resistant
***Staphylococcus
aureus***

Methicillin-resistant *Staphylococcus aureus*

There were **2,601** methicillin-resistant *S. aureus* (MRSA) infections reported for 2025. This is an 11.07% decrease from what was reported in 2024. The top contributing surveillance sites are CMC (8.84%), DMC (8.65%) and JLM (8.38%). Luzon contributed the majority of infections (50.60%) followed by Mindanao (32.41%) and Visayas (16.99%).

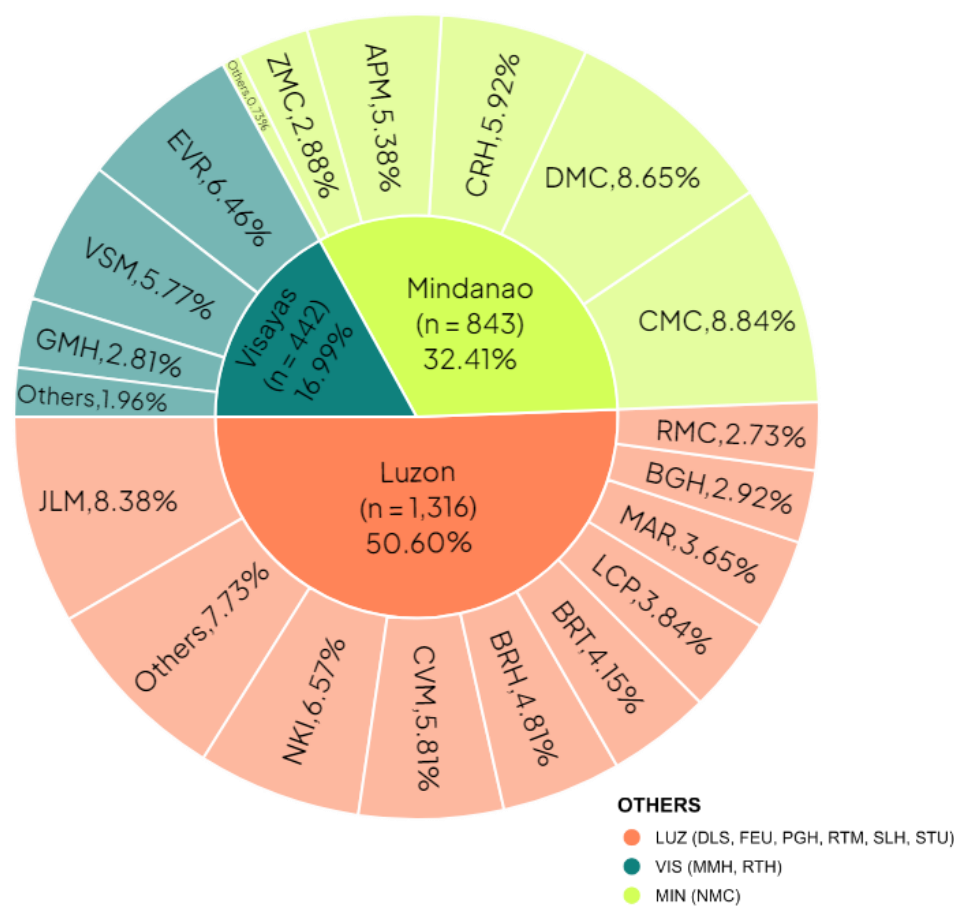


Figure 57. Distribution of MRSA infections, DOH-ARSP, 2025 (n=2,601)

Figure 58 shows that over half of the infections were observed among male patients (54.98%), and many (61.86%) occurring in the 20-64 age group. Many are skin and soft tissue (36.72%) and blood (27.18%) infections.

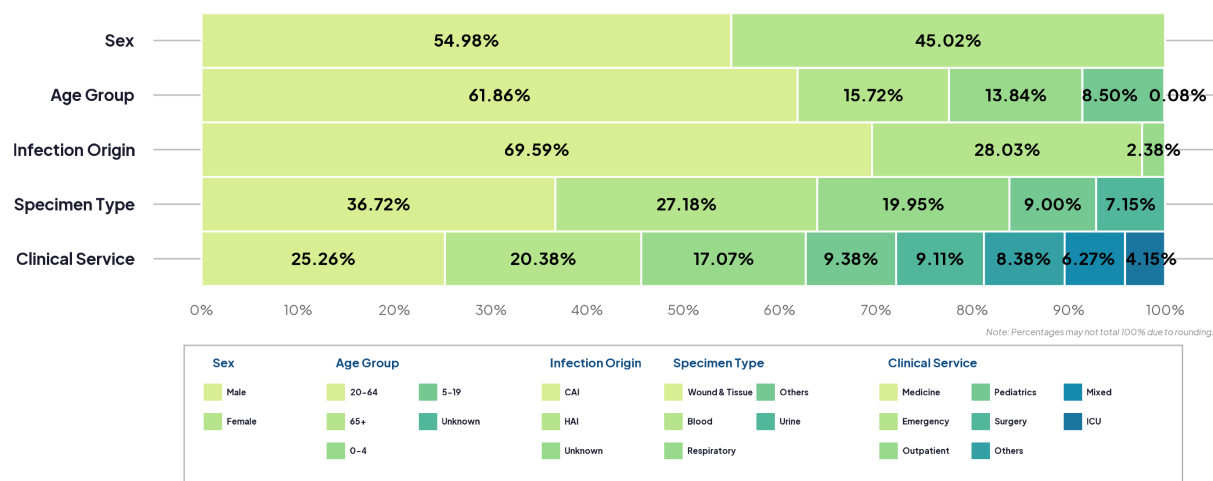
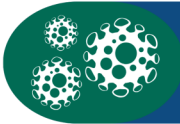


Figure 58. Patient characteristics in MRSA infections, DOH-ARSP, 2025 (n=2,601)



All types of Infections

Figure 59 shows the resistance rates of all MRSA infections. Resistance to clindamycin was at 21.5% and erythromycin at 24.74%, while resistance to vancomycin was at 2.77% and linezolid at 1.73%. Resistance rates to tetracycline, rifampicin, ciprofloxacin and nitrofurantoin were all less than 10%. Resistance to co-trimoxazole was still notably high at 46.39%. It was noted that from 2024, the increase in resistance rates for vancomycin ($p=0.2033$) and co-trimoxazole ($p=0.3594$) were found to be not statistically significant.

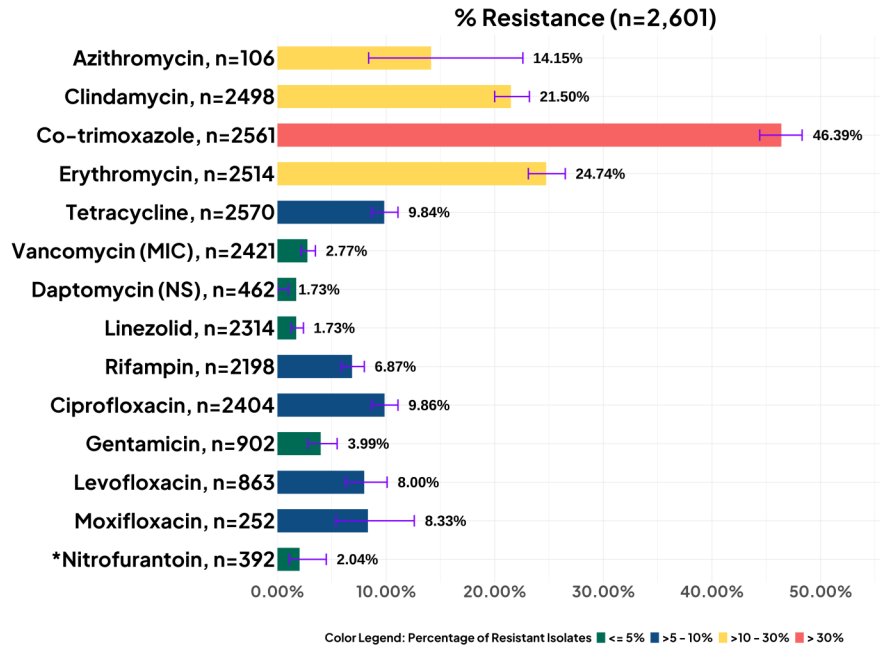


Figure 59. Proportion of all MRSA infections resistant to tested antibiotics, DOH-ARSP, 2025 (n=2,601)

Figure 60 illustrates the multiple year analysis and shows the changes in antibiotic resistance rates in MRSA infections over the past decade. The changes in resistance rates over the years were statistically significant for co-trimoxazole ($p=0.0000$), vancomycin ($p=0.0000$), erythromycin ($p=0.0000$), clindamycin ($p=0.0000$), erythromycin ($p=0.0000$) and ciprofloxacin ($p=0.0000$).

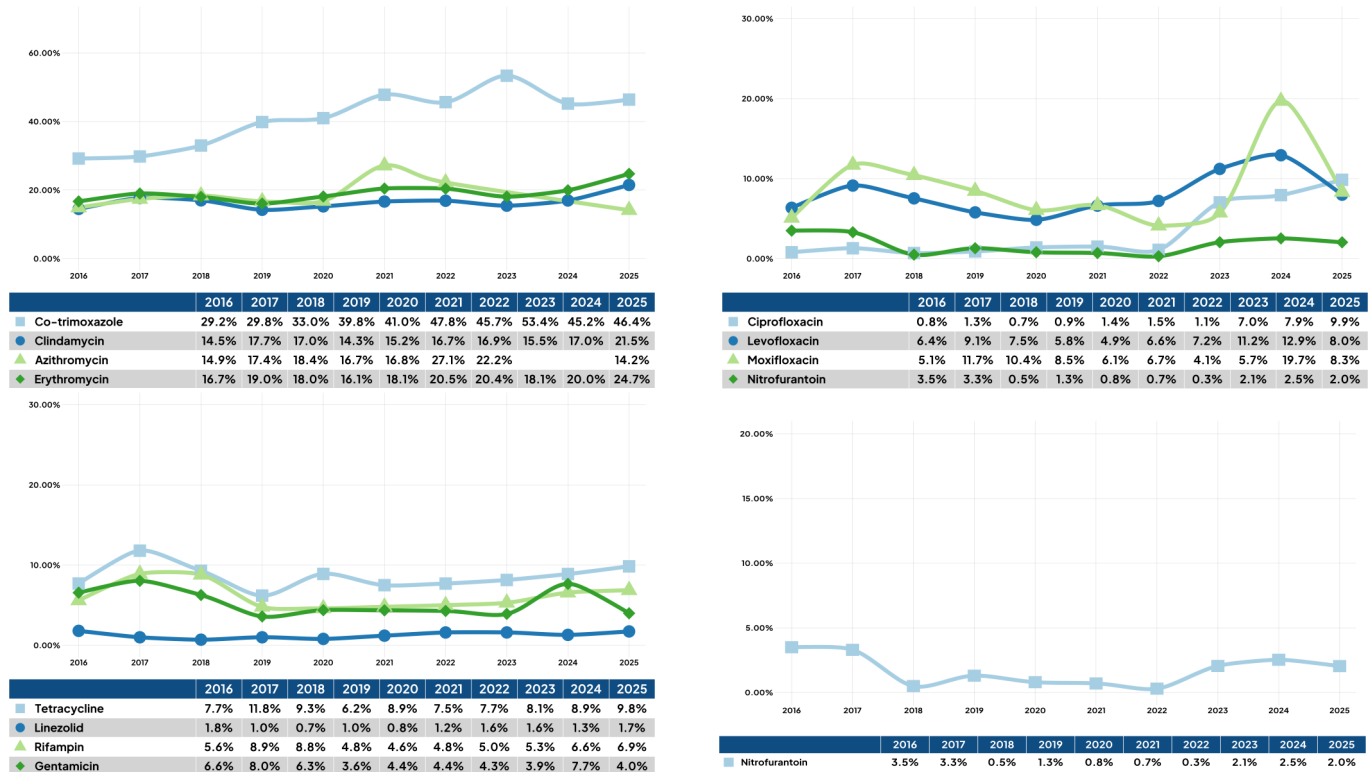


Figure 60. Yearly resistance rates of MRSA infection, 2016–2025, DOH-ARSP

Bloodstream Infections

Figure 61 illustrates that MRSA bloodstream infections exhibited slightly higher resistance rates to daptomycin, clindamycin, gentamicin, and ciprofloxacin compared to the resistance rates observed in all specimens.

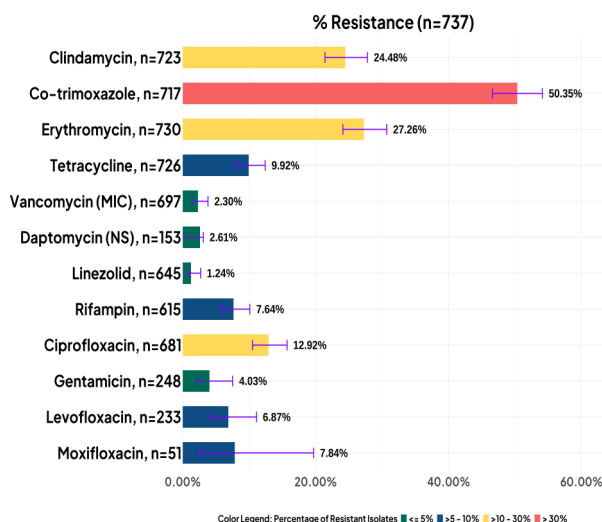


Figure 61. Proportion of MRSA bloodstream infections resistant to tested antibiotics, DOH-ARSP, 2025 (n=737)

Skin and Soft Tissue Infections

Figure 62 shows resistance rates of MRSA skin and soft tissue infections, resistance to erythromycin was at 18.63%, clindamycin at 15.93% and tetracycline at 7.44%. Resistance rates to rifampin and ciprofloxacin were 3.44% and 5.00% respectively while resistance rates to vancomycin and linezolid were at 2.13% and 0.57% respectively. Compared to overall MRSA infection rates, MRSA skin and soft tissue infections exhibited slightly lower resistance rates across most tested antibiotics.

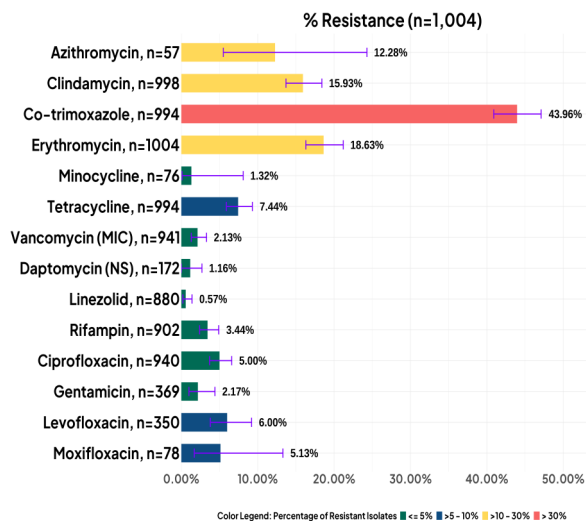


Figure 62. Proportion of MRSA skin and soft tissue infections with resistance to tested antibiotics, DOH-ARSP, 2025 (n=1,004)

Healthcare-associated Infections

Figure 63 shows the resistance rates for all presumptive healthcare-associated MRSA infections. Notably, resistance rates to critical last-line therapies remained low, with vancomycin at 2.79% and linezolid at 1.10%. Among other treatment options, ciprofloxacin and clindamycin exhibited resistance rates of 12.30% and 24.35%, respectively, while erythromycin resistance was recorded at 27.39%. Lower resistance rates were observed for tetracycline (10.22%) and rifampin (9.91%).

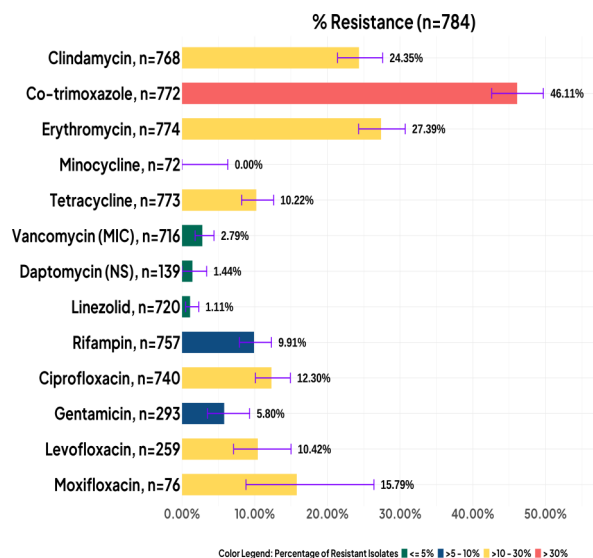


Figure 63. Proportion of all presumptive healthcare-associated MRSA infections with resistance to tested antibiotics, DOH-ARSP, 2025 (n=784)

A scanning electron micrograph (SEM) of Staphylococcus aureus bacteria. The image shows numerous spherical, Gram-positive cocci arranged in various clusters and chains. The bacteria are set against a background of fine, fibrous structures, likely part of a biofilm or extracellular matrix. A dark teal rectangular box is overlaid on the right side of the image, containing the text.

Methicillin-susceptible
***Staphylococcus
aureus***

Methicillin-susceptible *Staphylococcus aureus*

There were **6,562** methicillin-susceptible *S. aureus* (MSSA) infections reported in 2025. This is a 19.40% increase from what was reported in 2024. The highest contributors were PGH (14.96%), BGH (7.77%) and NKI (7.27%) (**Figure 64**). Based on island group distribution, the highest was Luzon with 66.31%, followed by Mindanao at 19.81%, and Visayas at 13.88%.

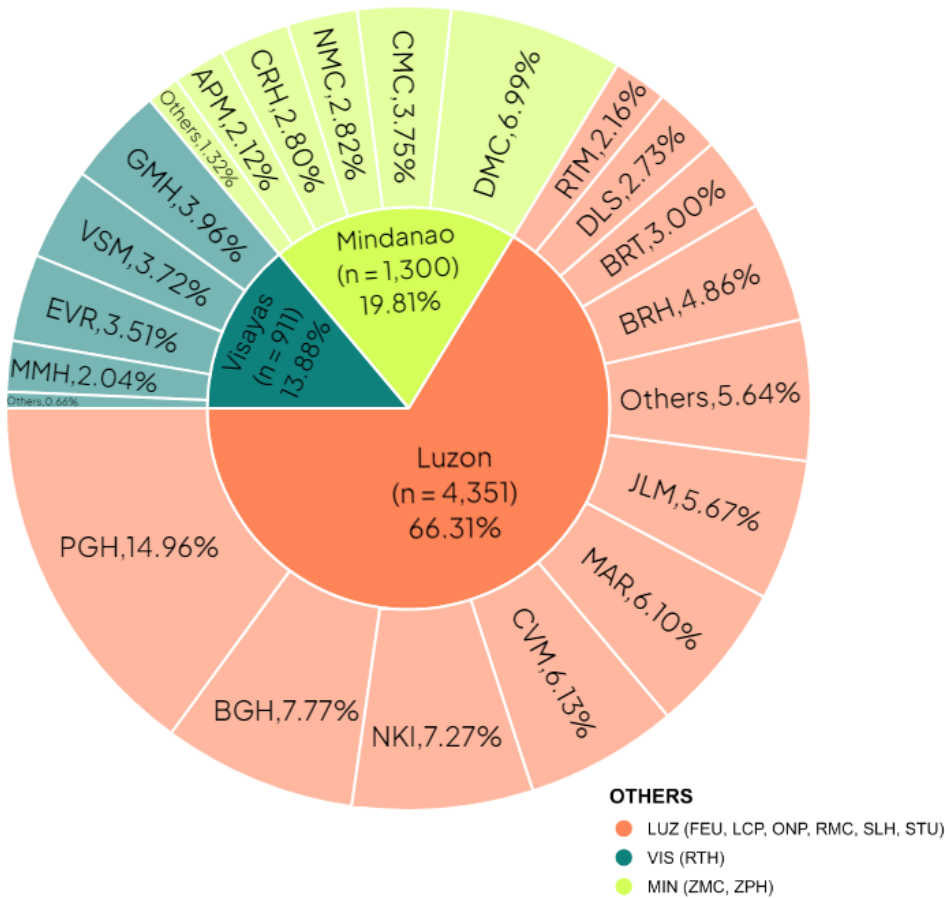


Figure 64. Distribution of MSSA infections, DOH-ARSP, 2025 (n=6,562)

A little more than half (54.65%) of infections were from male patients and many (67.19%) were from the 20-64 age group. Many of the infections were from skin and soft tissues 45.57%.

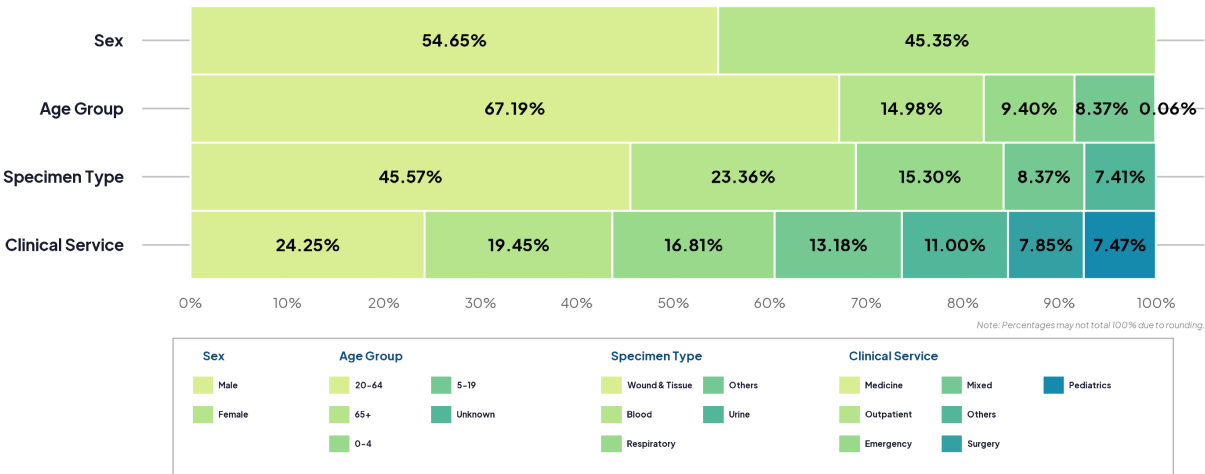
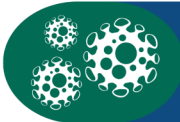


Figure 65. Patient characteristics in MSSA infections, DOH-ARSP, 2025 (n=6,552)



All types of Infections

Resistance to tested antibiotics among all types of MSSA infections are shown in **Figure 66**. Likewise, resistance to MSSA SSTI and BSIs are shown in **Figures 67** and **68**, respectively. Resistance to first line antibiotics other than oxacillin such as erythromycin, clindamycin, and tetracycline ranged from 5-9%.

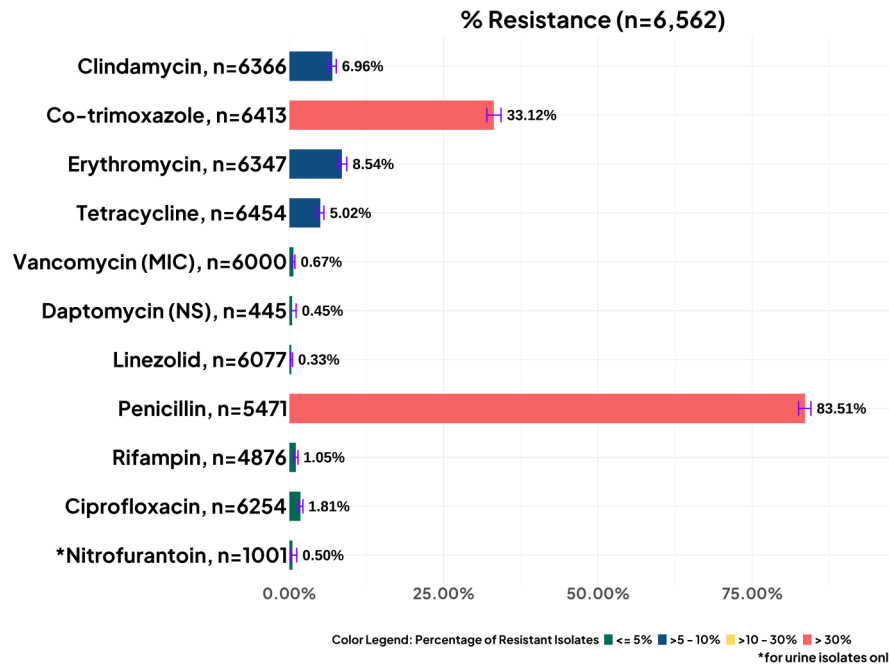


Figure 66. Proportion of all MSSA infections resistant to tested antibiotics, DOH-ARSP, 2025 (n=6,562)



Bloodstream Infections

Figure 67 shows the proportion of MSSA bloodstream infections. Resistance to penicillin, co-trimoxazole, erythromycin and clindamycin were 82.09%, 26.90%, 9.37% and 7.70% respectively.

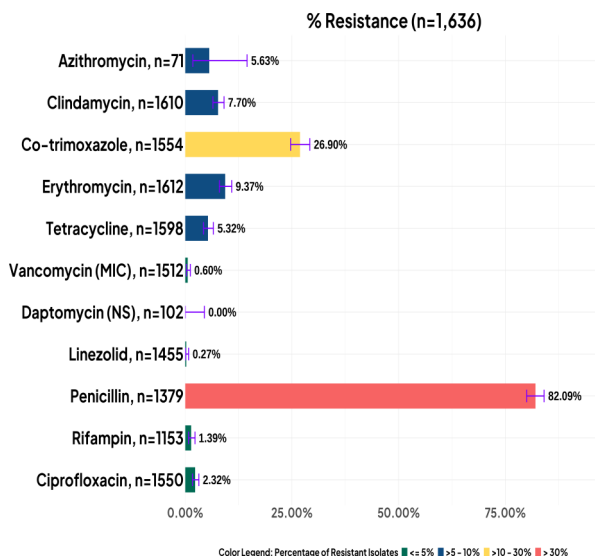


Figure 67. Proportion of all MSSA bloodstream infections with resistance to tested antibiotics, DOH-ARSP, 2025 (n=1,636)



Skin and Soft Tissue Infections

Figure 68 shows the proportion of MSSA skin and soft tissue infections. Resistance to penicillin, co-trimoxazole, erythromycin and clindamycin were 87.85%, 44.47%, 7.27% and 6.32% respectively.

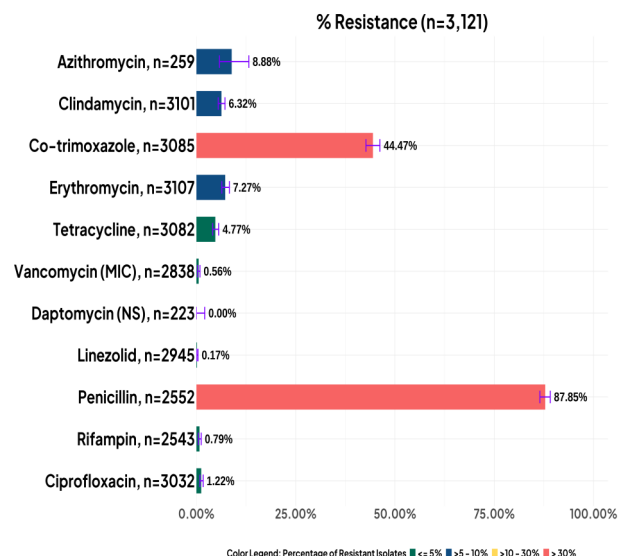
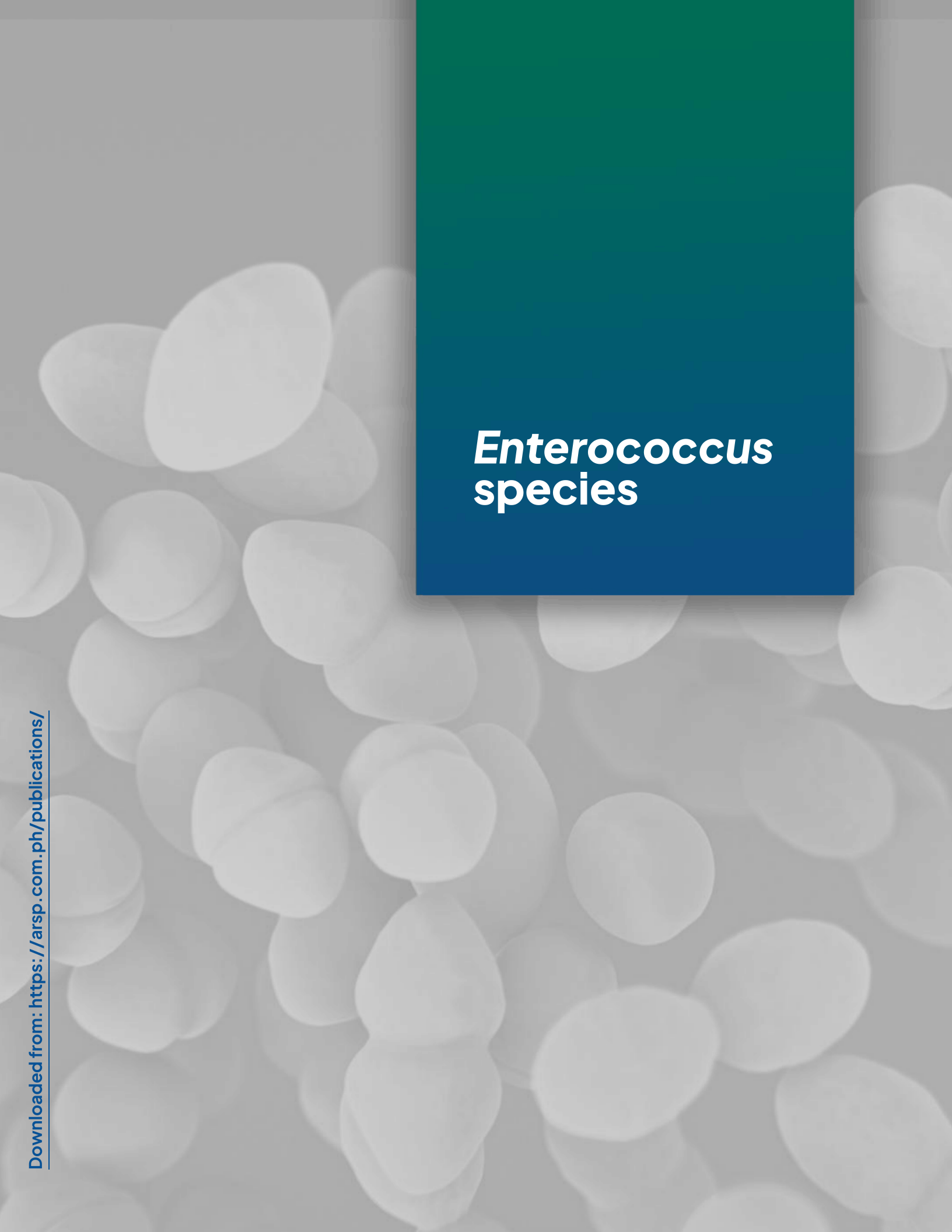


Figure 68. Proportion of all MSSA skin and soft tissue infections with resistance to tested antibiotics, DOH-ARSP, 2025 (n=3,121)

The background of the slide is a grayscale micrograph showing numerous oval-shaped, translucent cells, likely Enterococcus species, scattered across the field of view. A solid teal rectangular box is positioned in the upper right corner, containing the title text.

***Enterococcus* species**

Enterococcus faecalis

Enterococcus faecalis is a common cause of urinary tract infections, bacteremia, endocarditis, and intra-abdominal infections, particularly in hospitalized patients. It is intrinsically tolerant to many antibiotics including ceftriaxone, cefotaxime, low-level aminoglycosides. In the Philippines, *E. faecalis* generally remains susceptible to ampicillin. However, resistance to fluoroquinolones and high-level aminoglycosides has been reported. Vancomycin resistance is less common in *E. faecalis* than in *E. faecium*, but continued monitoring is necessary.

A total of **4,017** *E. faecalis* infections were reported for 2025. The highest number of reported cases were observed in PGH (14.12%), DMC (11.40%), and NKI (9.83%) (Figure 69). Based on island group distribution, the highest was Luzon with 60.42% followed by Mindanao at 23.77%, and Visayas at 15.81%.

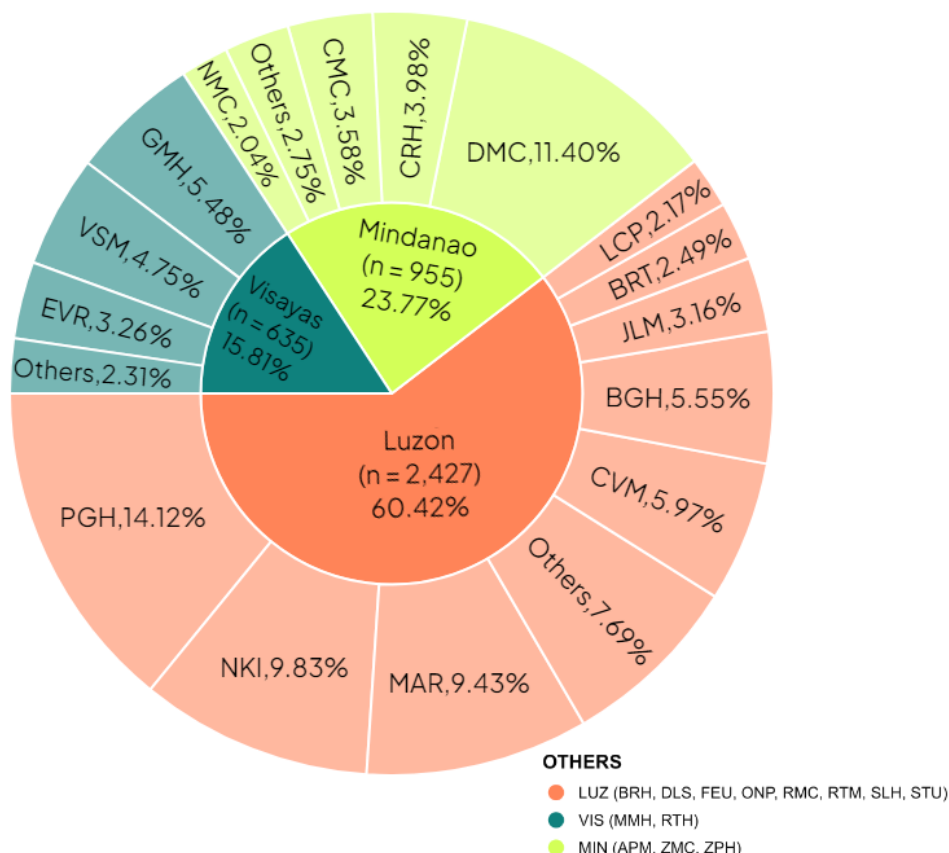


Figure 69. Distribution of *E. faecalis* infections, DOH-ARSP, 2025 (n=4,017)

More than half (54.42%) of the infections were from female patients and from the 20–64 age group (64.80%). Urinary infections are the most common type of *E. faecalis* infections, accounting for 50.56%.

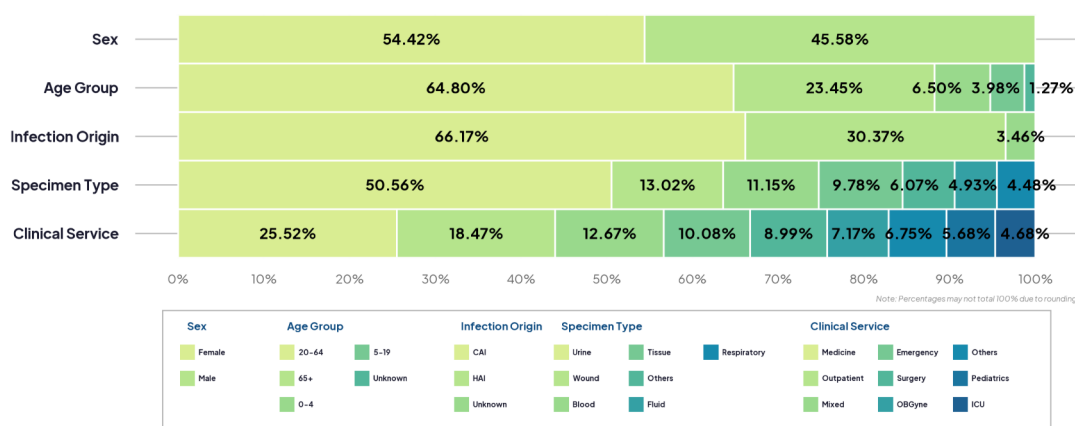


Figure 70. Patient characteristics in *E. faecalis* infections, DOH-ARSP, 2025 (n=4,017)

All types of Infections

The resistance rates of all *E. faecalis* infections to penicillin was at 16.36% and 6.77% to ampicillin. Resistance to vancomycin, and linezolid were at 2.03%, and 3.68% respectively. Resistance against high-level gentamicin and high-level streptomycin were at 25.84% and 22.94%, respectively. From 2024, there was a decrease in resistance rates of ampicillin and vancomycin, while an increase was observed in linezolid, penicillin, high-level gentamicin, high-level streptomycin and nitrofurantoin. These changes were found to be statistically significant.

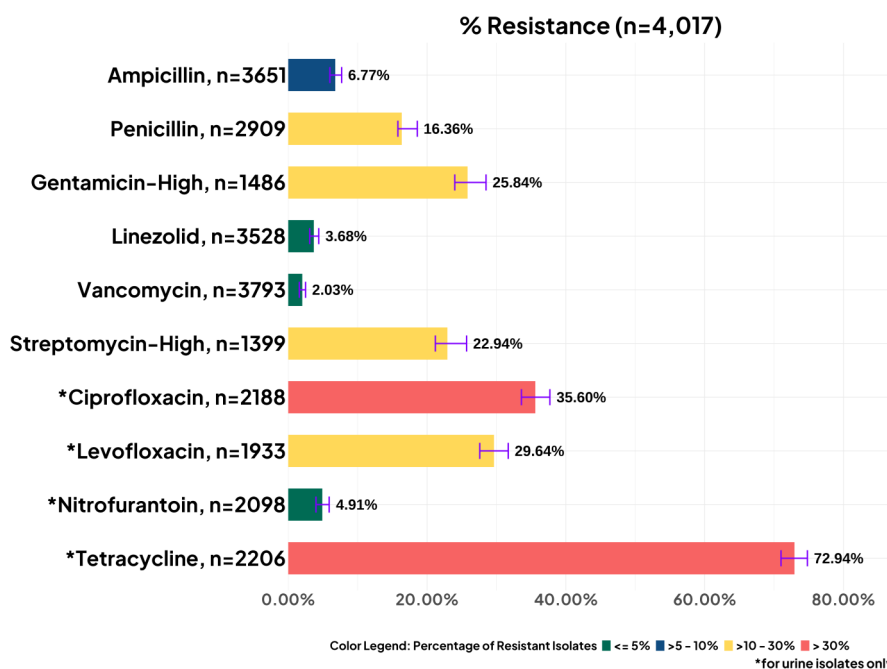


Figure 71. Proportion of all *E. faecalis* infections resistant to tested antibiotics, DOH-ARSP, 2025 (n=4,017)

Figure 72 shows antimicrobial resistance trends from 2016 to 2025. Resistance to ampicillin gradually decreased from 13.1% in 2016 to 6.8% in 2025, while penicillin remained relatively stable, with a slight increase from 12.8% to 16.4%. high-level gentamicin resistance fluctuated over time but declined from a peak of 31.7% in 2018 to 25.8% in 2025. Vancomycin resistance remained low throughout the period despite a temporary increase in 2021. Linezolid consistently showed low resistance rates, whereas high-level streptomycin, ciprofloxacin, and levofloxacin exhibited moderate to high resistance levels, with ciprofloxacin and levofloxacin remaining elevated in 2025. Tetracycline resistance stayed consistently high at over 70% across the decade, while nitrofurantoin maintained low resistance rates below 5%.

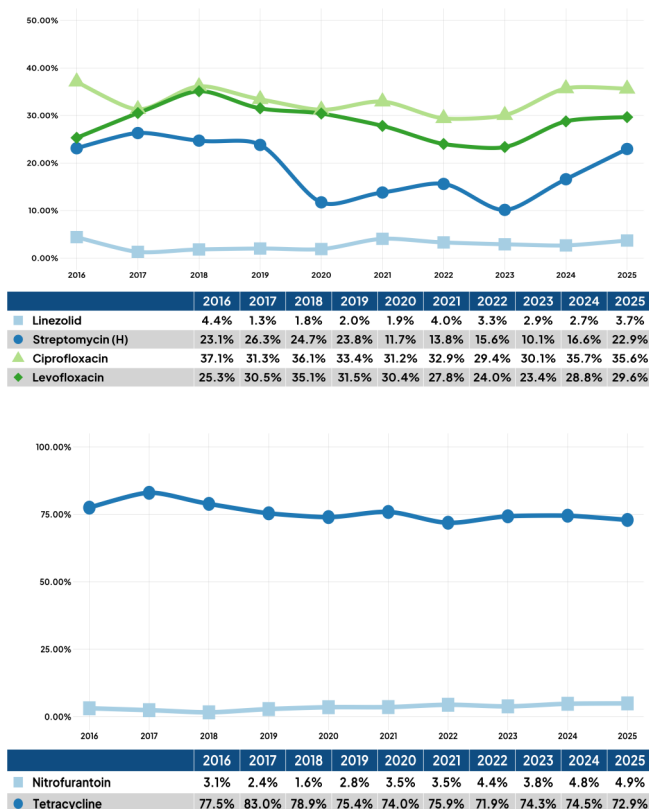
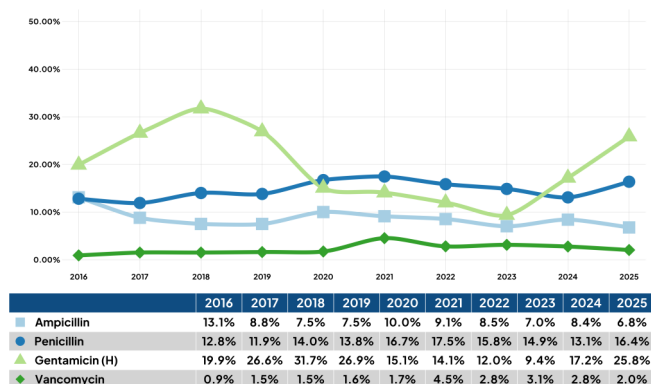


Figure 72. Yearly resistance rates of all *E. faecalis* infections

In 2025, a vancomycin-resistant *Enterococcus faecalis* urinary tract infection was detected from a 45-year old female patient in NKI. The infection was found to be resistant to other antibiotics including tetracycline, ciprofloxacin, high-level gentamicin and high-level streptomycin however susceptible to linezolid, nitrofurantoin and penicillin.

Figure 73 shows the geographic distribution for all *E. faecalis* infections with resistance to linezolid and vancomycin. For both antibiotics, resistance rates usually range from 1-20%.

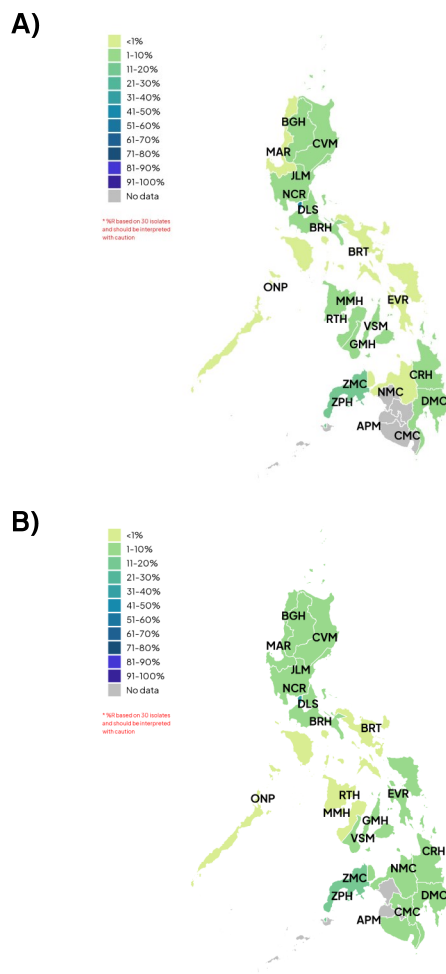


Figure 73. Resistance maps of *E. faecalis* infections for (A) Linezolid and, and (B) Vancomycin



Figure 74 shows the resistance rates of *E. faecalis* bloodstream infections. Resistance to penicillin was at 17.11%, ampicillin at 7.14%, vancomycin at 1.11% and linezolid at 4.48%. Resistance against high-level gentamicin was at 26.98% and high-level streptomycin at 23.78%. Compared to the overall resistance rates of *E. faecalis* across all samples, resistance rates among blood isolates were generally higher.

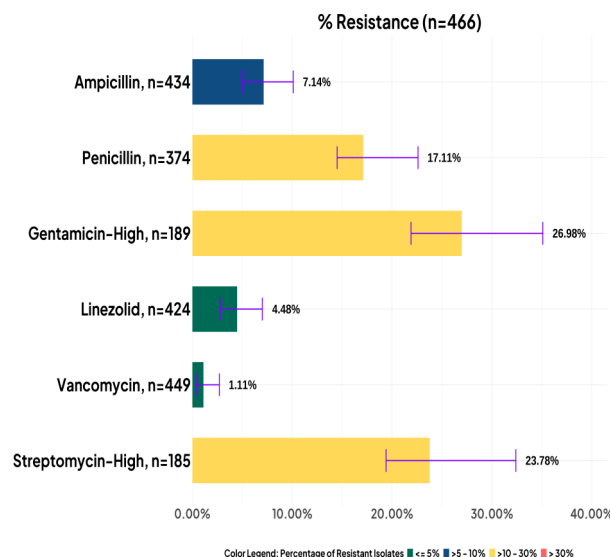


Figure 74. Proportion of *E. faecalis* bloodstream infections with resistance to tested antibiotics, DOH-ARSP, 2025 (n=466)

Figure 75 shows a fluctuating resistance rate of antibiotics tested against *E. faecalis* bloodstream infections. The changes over the years were significant for ampicillin, linezolid, penicillin, vancomycin and high-level gentamicin and high level streptomycin.

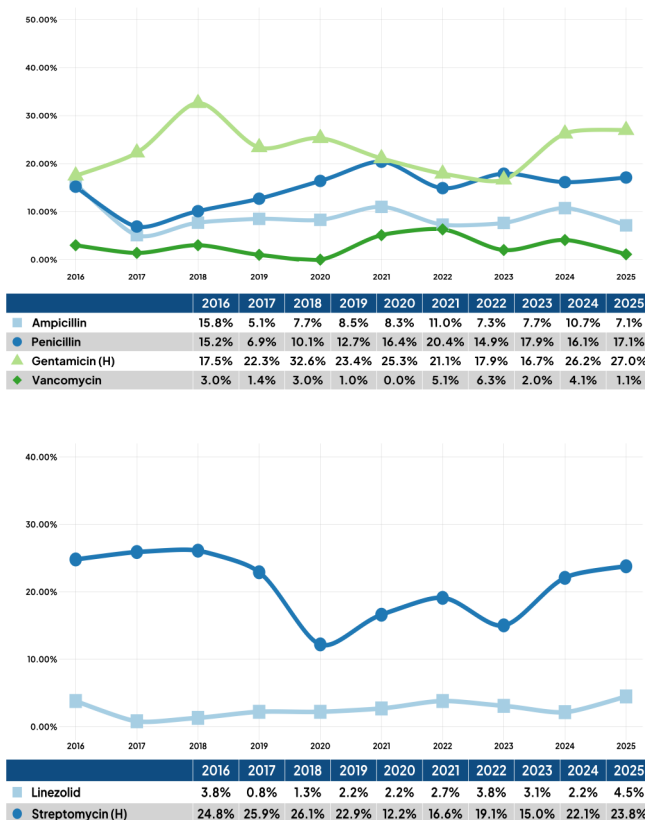


Figure 75. Yearly resistance rates of *E. faecalis* bloodstream infections, ARSP-DOH, 2025

Urinary Tract Infections

Figure 76 shows that *E. faecalis* urinary tract infections showed resistance to penicillin at 20.25% and ampicillin at 6.64%. Resistance rates for the following treatment options remained under 5%: nitrofurantoin, vancomycin, and linezolid.

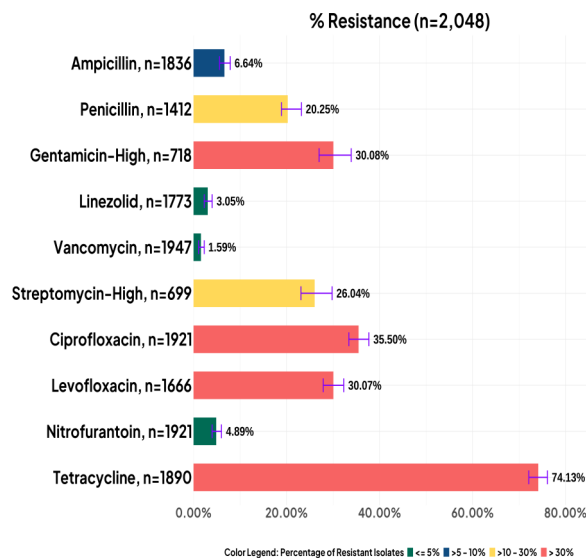


Figure 76. Proportion of *E. faecalis* urinary tract infections with resistance to tested antibiotics, DOH-ARSP, 2025 (n=2,048)

Figure 77 showed data from 2016–2025, *Enterococcus faecalis* isolated from urinary tract infections remained highly susceptible to vancomycin, linezolid, nitrofurantoin, and ampicillin, with resistance rates remaining low in 2025 (1.6%, 3.0%, 4.9%, and 6.6%, respectively). In contrast, tetracycline showed persistently high resistance throughout the decade (74.1% in 2025), while ciprofloxacin and levofloxacin maintained moderate resistance levels ranging from 30.8%–41.3%. Notably, high-level gentamicin and streptomycin resistance increased markedly in recent years, rising from their lowest levels in 2023 to 30.1% and 26.0%, respectively, in 2025.

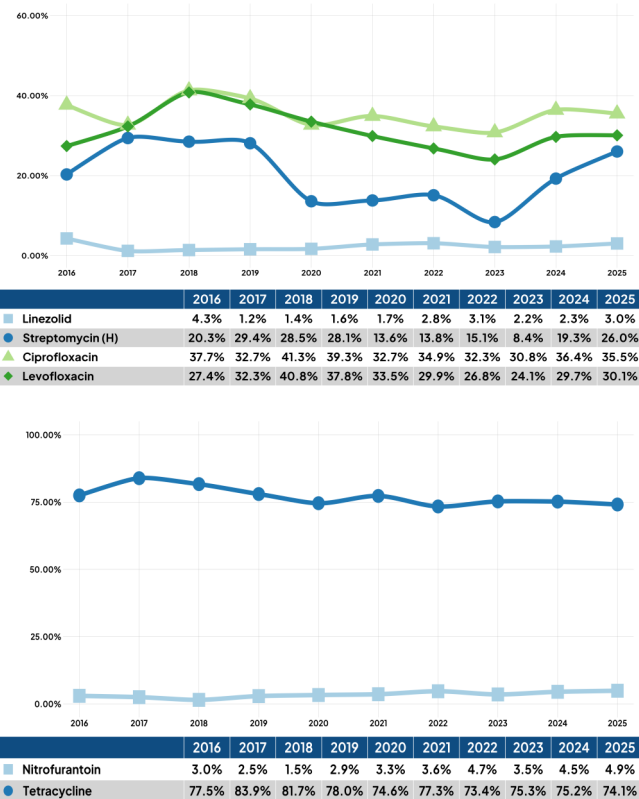
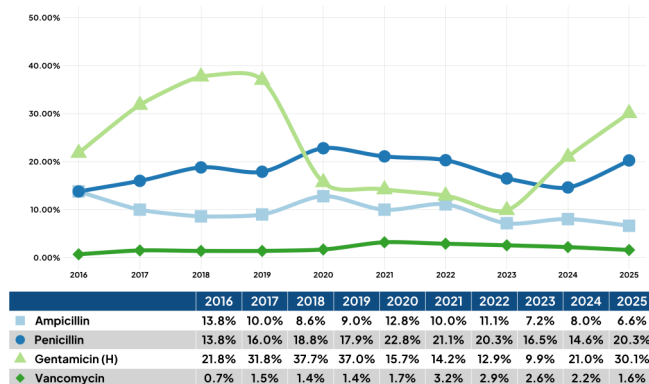


Figure 77. Yearly resistance rates of *E. faecalis* urinary tract infection, DOH-ARSP, 2025

Enterococcus faecium

Enterococcus faecium is a significant cause of healthcare-associated infections, including bloodstream and urinary tract infections. Compared to *E. faecalis*, *E. faecium* exhibits higher resistance levels, presenting greater therapeutic challenges. In the Philippines, high resistance to ampicillin and fluoroquinolones is well documented, and vancomycin-resistant *Enterococcus* (VRE) has been reported, particularly in tertiary hospitals. Consequently, treatment options are frequently limited to linezolid or daptomycin.

A total of **2,397** *E. faecium* infections were reported for 2025. The highest reported cases are in DMC (20.48%), PGH (17.19%) and MAR (7.68%) (Figure 78). Based on island group distribution, Luzon contributed the highest percentage of *E. faecium* infections with 48.44% followed by Mindanao at 33.29% and Visayas at 18.27%.

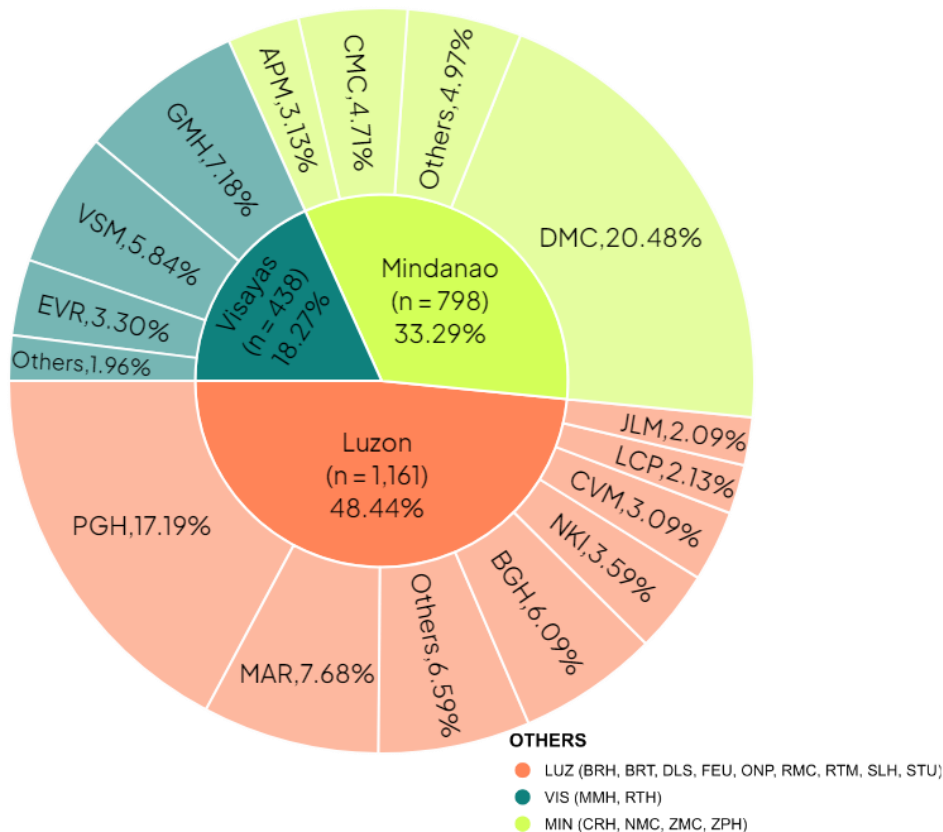


Figure 78. Distribution of *E. faecium* infections, DOH-ARSP, 2025 (n=2,397)

More than half (57.78%) of the infections were from female patients and the 20–64 age group (51.73%). Urine specimens are the most common source of *E. faecium* infections at 62.08%.

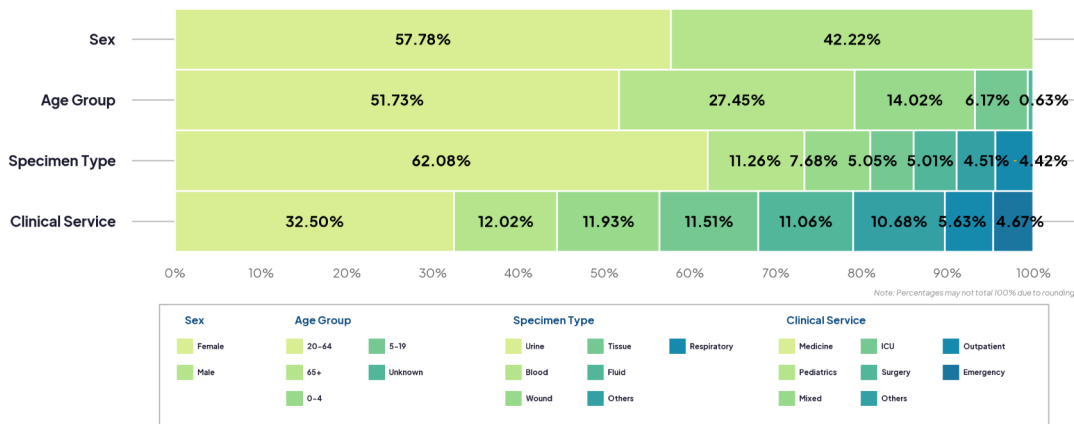


Figure 79. Patient characteristics in *E. faecium* infections, DOH-ARSP, 2025 (n=2,397)

All types of Infections

Figure 80 shows the resistance rates for all *E. faecium* infections. Resistance was highest for penicillin (90.79%) and ampicillin (87.42%). Moderate to high-level resistance to gentamicin and streptomycin was reported at 56.92% and 31.15%, respectively. For the reserve antibiotics, resistance rates were 20.95% for vancomycin and a minimal 0.96% for linezolid

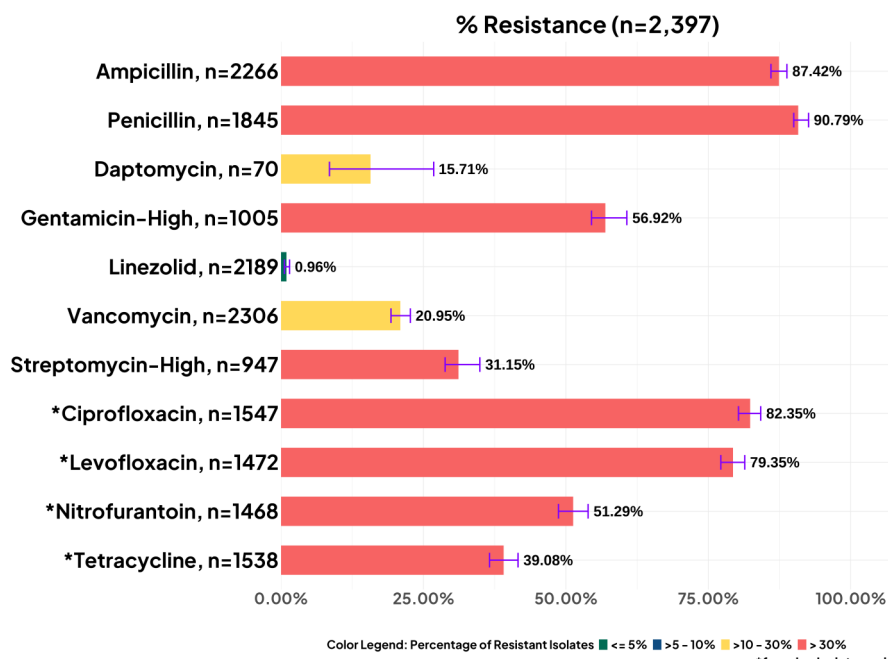


Figure 80. Proportion of all *E. faecium* infections with resistance to tested antibiotics, DOH-ARSP, 2025 (n=2,397)

Resistance to penicillin ($p=0.000$) and ampicillin ($p=0.000$) have remained consistently high over the past ten years (**Figure 81**). The decreasing trend seen in vancomycin resistance was noted to be statistically significant ($p=0.000$). Conversely, an increasing trend for nitrofurantoin ($p=0.000$) resistance has been observed over the past decade, while resistance rates to gentamicin-high level ($p=0.0000$) and streptomycin high-level ($p=0.000$) were observed to be fluctuating. Daptomycin resistance was at 15.71% in 2025 from 0% in 2024, however, this change was found to be not significant ($p=0.2219$).

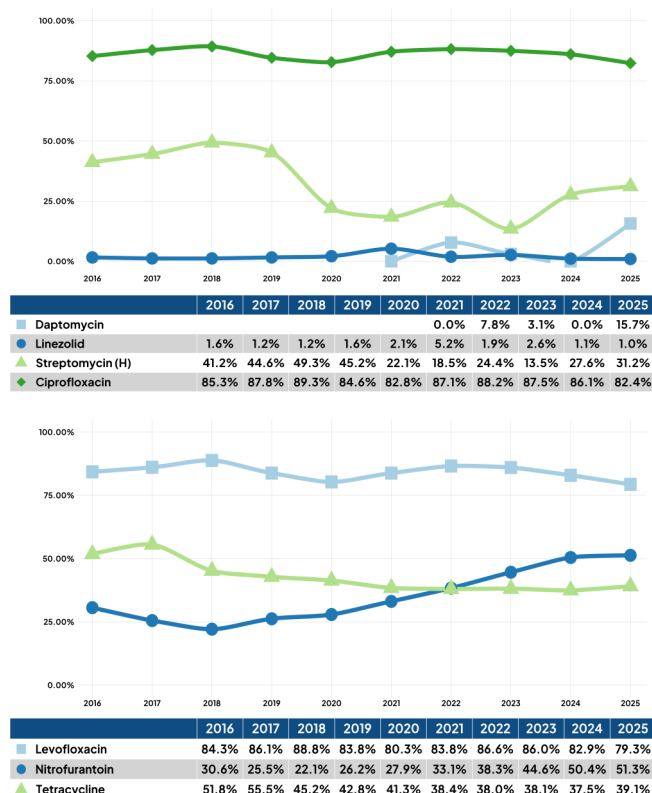
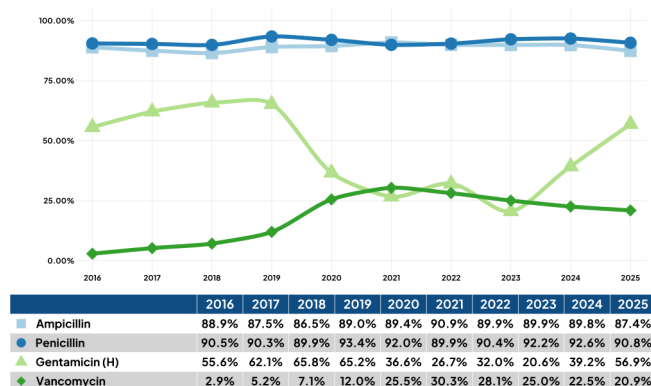


Figure 81. Yearly resistance rates of all *E. faecium* infections

Figure 82-A shows the geographical distribution of vancomycin-resistant *E. faecium* isolates across the country. Higher rates were observed among surveillance sites from Luzon and Visayas. Figure 82-B shows the geographical distribution of linezolid resistant *E. faecium* isolates across the country. Linezolid resistance rates across surveillance sites remained relatively low, ranging from 1% to 20%.

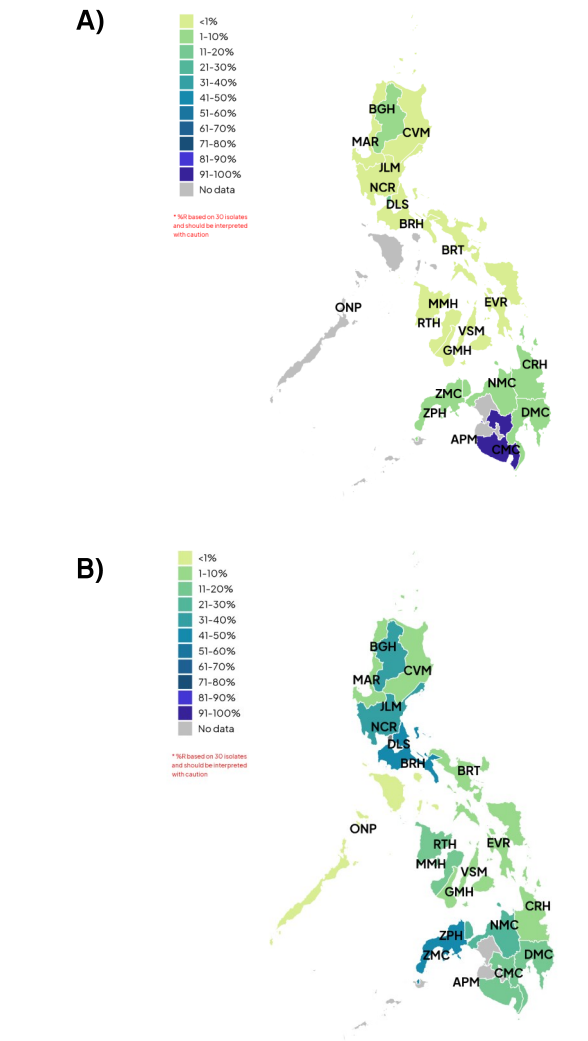


Figure 82. Resistance maps of *E. faecium* infections for (A) Linezolid, and (B) Vancomycin



Figure 83 illustrates the resistance rates of *E. faecium* bloodstream infections. Vancomycin resistance was at 26.28%, and linezolid at 1.42%. High-level gentamicin resistance was observed at 57.50%, while high-level streptomycin resistance was at 31.58%. These resistance rates for *E. faecium* bloodstream infections are similar to those observed across all *E. faecium* infections.

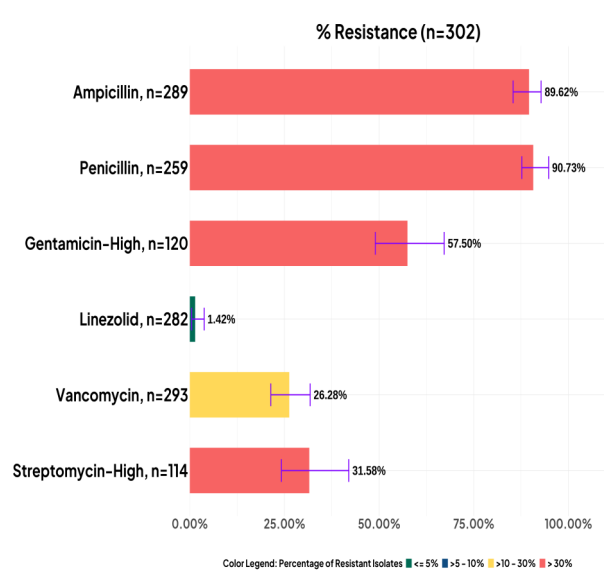


Figure 83. Proportion of *E. faecium* bloodstream infections with resistance to tested antibiotics, DOH-ARSP, 2025 (n=302)

Figure 84 shows the multi-year analysis of the resistance rates of *E. faecium* bloodstream infections: ampicillin (p=0.000), penicillin (p=0.000), vancomycin (p=0.000), linezolid (p=0.000), daptomycin (p=0.000), high-level gentamycin (p=0.000) and streptomycin (p=0.000).

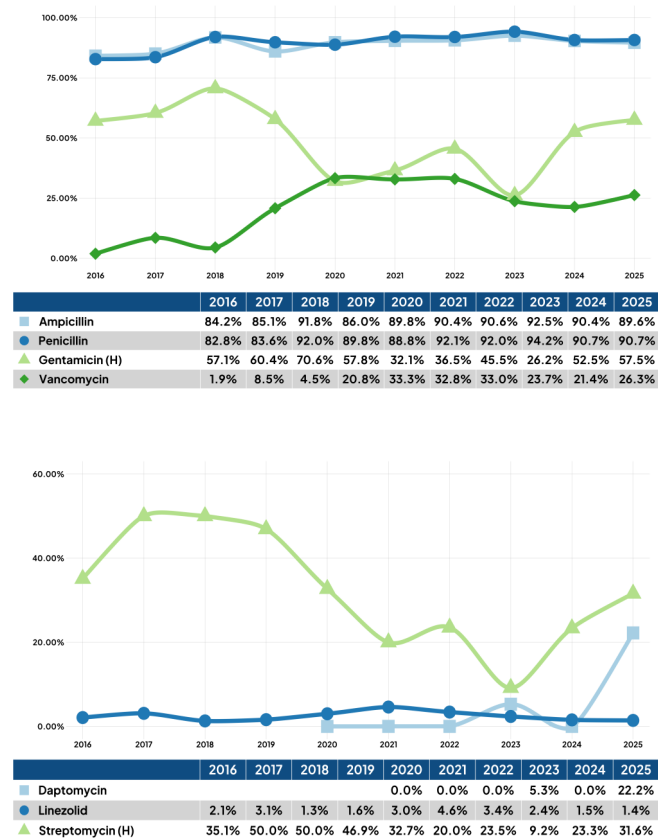


Figure 84. Yearly resistance rates of *E. faecium* bloodstream infections, DOH-ARSP, 2025

Urinary Tract Infections

Figure 85 illustrates the resistance rates of *E. faecium* urinary tract infections. High resistance rates were noted for nitrofurantoin (51.96%) and vancomycin (19.30%). While linezolid resistance remained low at 0.65%, high-level gentamicin and high-level streptomycin resistance rates were slightly higher compared to overall *E. faecium* infections. For other antibiotics, the resistance rates for *E. faecium* urinary tract infections were relatively similar to those observed across all *E. faecium* infections.

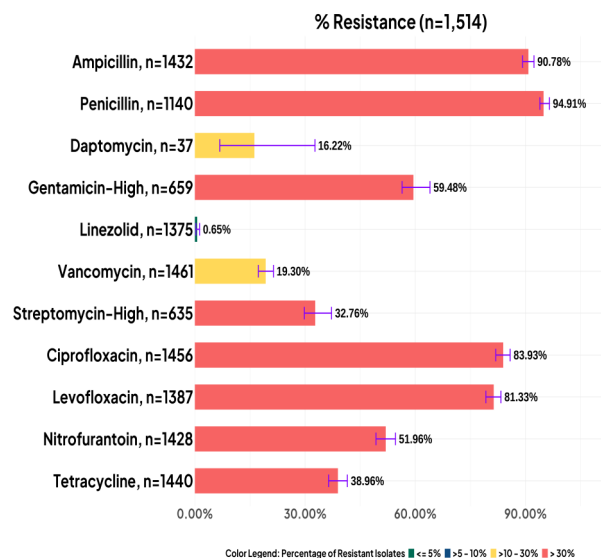


Figure 85. Proportion of *E. faecium* urinary tract infections with resistance to tested antibiotics, DOH-ARSP, 2025 (n=1,514)

A steady increase in nitrofurantoin resistance has been observed in *E. faecium* urinary tract infections over the past 10 years (**Figure 86**). Similar multi-year trends were also observed for *E. faecium* blood-stream infections. Yearly resistance rates of *E. faecium* urinary tract infections.

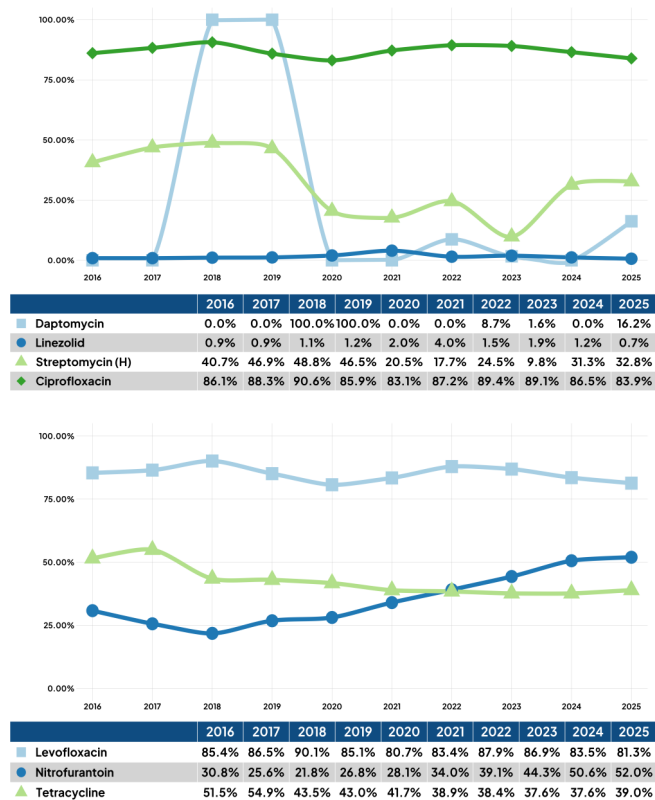
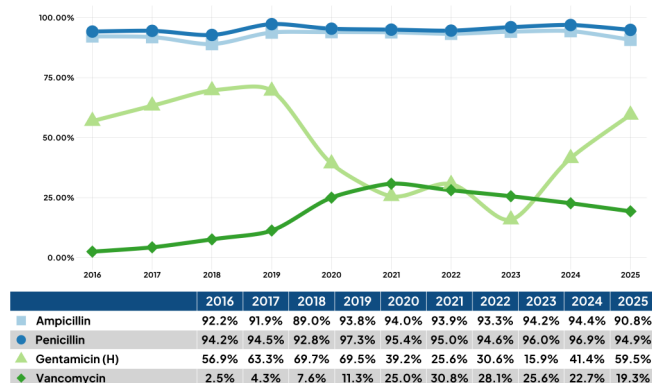
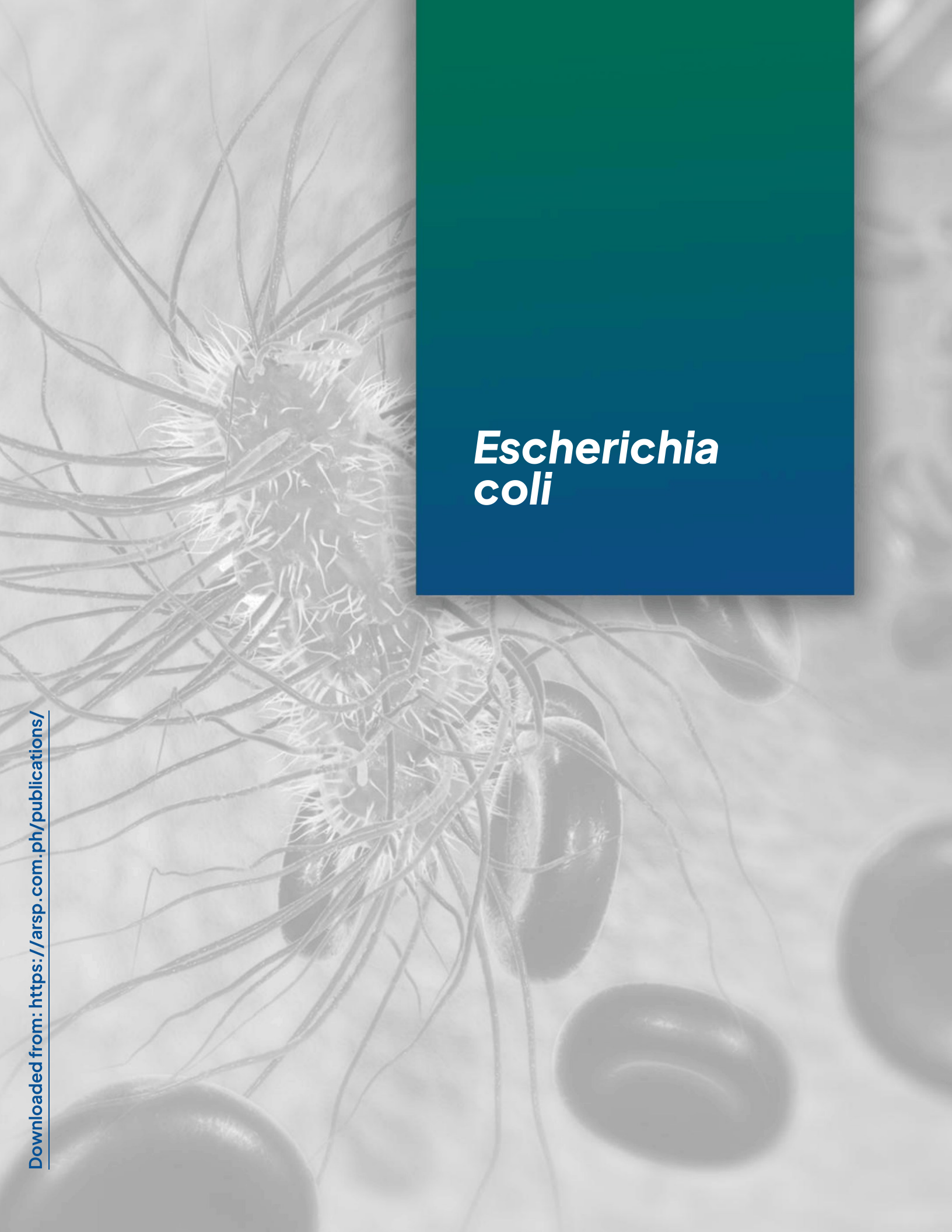


Figure 86. Yearly resistance rates of *E. faecium* urinary tract infections, DOH-ARSP 2025



Escherichia coli

Escherichia coli

Escherichia coli is the second most frequently isolated organism in the ARSP and remains the leading bacterial cause of urinary tract infections as well as a significant contributor to bloodstream infections, wound infections, and other clinical syndromes. Globally, drug-resistant *E. coli* is among the leading causes of AMR-attributable mortality; both third-generation cephalosporin-resistant and carbapenem-resistant *E. coli* are classified as critical priority pathogens in the 2024 WHO Bacterial Priority Pathogens List.

A total of **17,248** *E. coli* infections were reported and analyzed in 2025, this was a 15.81% increase from the previous year. Based on island group distribution, 57.91% were from Luzon, 24.89% from Mindanao and 17.20% from Visayas. PGH (12.88%) contributed to the most number of isolates followed by DMC (9.46%) and NKI (7.60%).

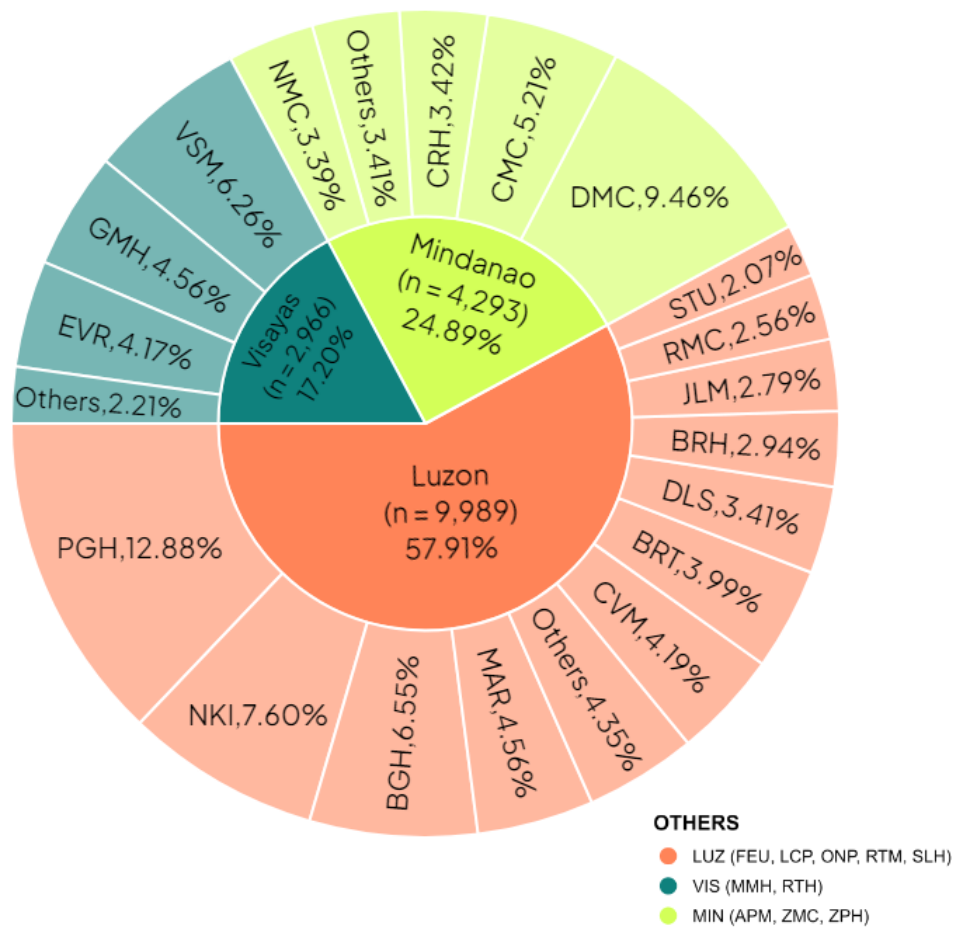


Figure 87. Distribution of *E. coli* infections, DOH-ARSP, 2025 (n=17,248)

More than half (64.37%) of the infections were from female patients and most (62.13%) were from 20-64 years old. More than half (75.59%) were presumptive community acquired infections.

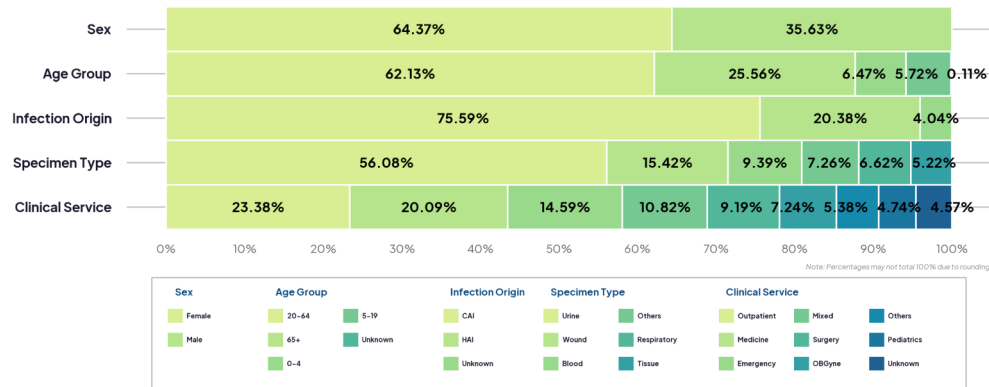


Figure 88. Patient characteristics in *E. coli* infections, DOH-ARSP, 2025 (n=17,248)



All types of Infections

As shown in **Figure 89**, the cumulative resistance rates for all *E. coli* isolates demonstrated elevated resistance across multiple antibiotic classes. Resistance was highest among narrow-spectrum penicillins, specifically ampicillin at 77.70%. Notably, significant resistance was observed among third-generation cephalosporins—cefotaxime (40.33%) and ceftriaxone (38.99%)—which is often indicative of ESBL production. Resistance also remained elevated among oral step-down options, including the fluoroquinolones (ciprofloxacin at 45.90% and levofloxacin at 42.60%) and co-trimoxazole (49.84%). Conversely, carbapenem resistance remained comparatively low—with imipenem at 10.88%, meropenem at 10.16%, and ertapenem at 9.92%—though these rates have been steadily increasing over the years. Resistance to novel β -lactam/ β -lactamase inhibitors was notable, with resistance rates of 28.02% for ceftazidime-avibactam and 12.59% for ceftolozane-tazobactam. While resistance to imipenem-relebactam was recorded at 60.00%, this elevated rate is attributable to a small sample size of tested isolates yielding a disproportionately high percentage of resistant strains.

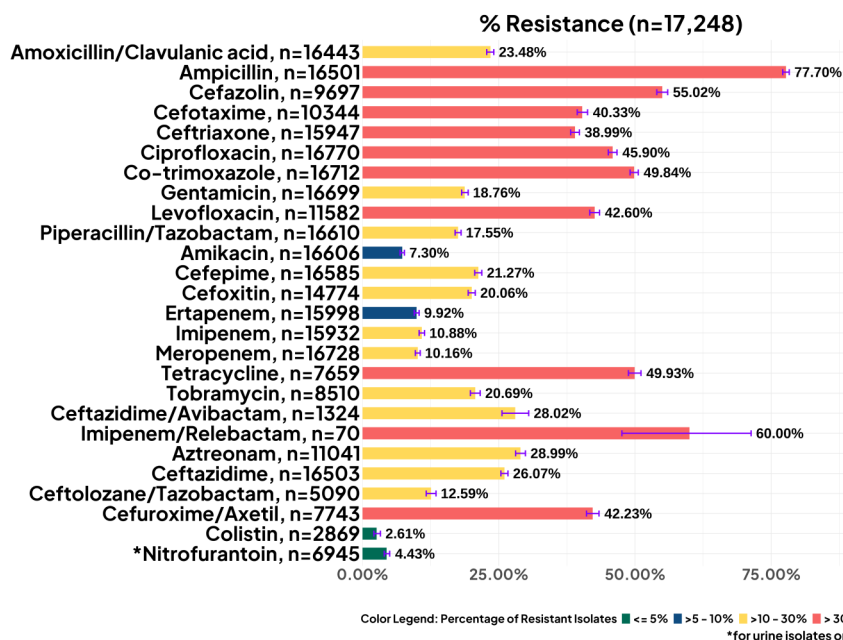
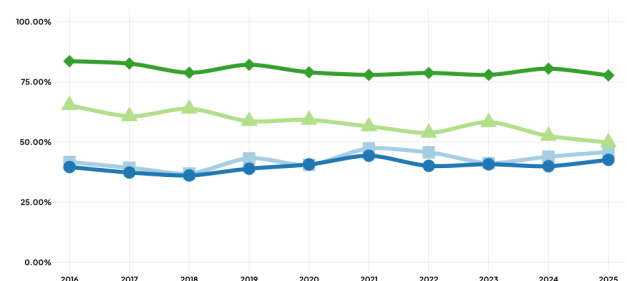
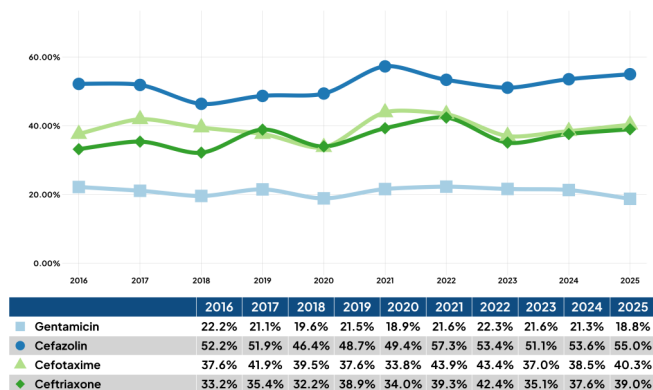
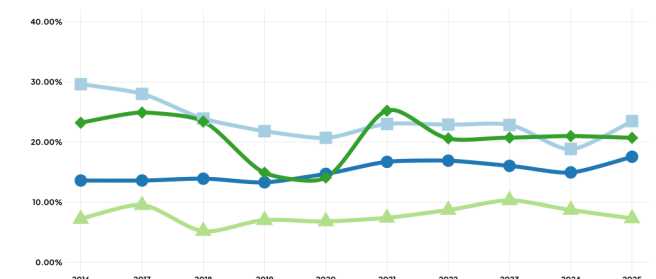


Figure 89. Proportion of all *E. coli* infections with resistance tested to antibiotics, DOH-ARSP, 2025 (n=17,248)

Between 2016 and 2025, yearly resistance rates of all *E. coli* infections demonstrated statistically significant changes across all tested antibiotics over time. Resistance trends showed persistent resistance in ampicillin at 77.7%–83.6% and co-trimoxazole at 49.8%–65.3%. Resistance to cephalosporins and fluoroquinolones were relatively stable at moderate-to-high levels cefazolin ~50%; ceftriaxone and ciprofloxacin 32%–47%). Colistin resistance was higher in 2021 (23.0%) and 2022 (13.2%) 28, although these figures were based on limited sample sizes and may reflect post-pandemic reporting changes.



	2016	2017	2018	2019	2020	2021	2022	2023	2024	2025
Ciprofloxacin	41.7%	39.3%	36.9%	43.2%	40.5%	47.3%	45.7%	41.2%	43.9%	45.9%
Levofloxacin	39.6%	37.3%	36.1%	38.9%	40.7%	44.3%	40.1%	40.7%	39.9%	42.6%
Co-trimoxazole	65.3%	60.7%	63.8%	58.7%	59.2%	56.5%	53.8%	58.2%	52.5%	49.8%
Ampicillin	83.6%	82.6%	78.8%	82.1%	79.0%	77.9%	78.7%	77.9%	80.5%	77.7%



	2016	2017	2018	2019	2020	2021	2022	2023	2024	2025
Amoxicillin/Clavulanic Acid	29.6%	28.0%	23.9%	21.8%	20.7%	23.0%	22.9%	22.8%	18.8%	23.5%
Piperacillin/Tazobactam	13.6%	13.6%	13.9%	13.3%	14.7%	16.7%	16.9%	16.0%	14.9%	17.5%
Amikacin	7.2%	9.5%	5.2%	7.0%	6.8%	7.4%	8.7%	10.3%	8.7%	7.3%
Tobramycin	23.2%	24.9%	23.4%	14.9%	14.1%	25.2%	20.6%	20.7%	21.0%	20.7%

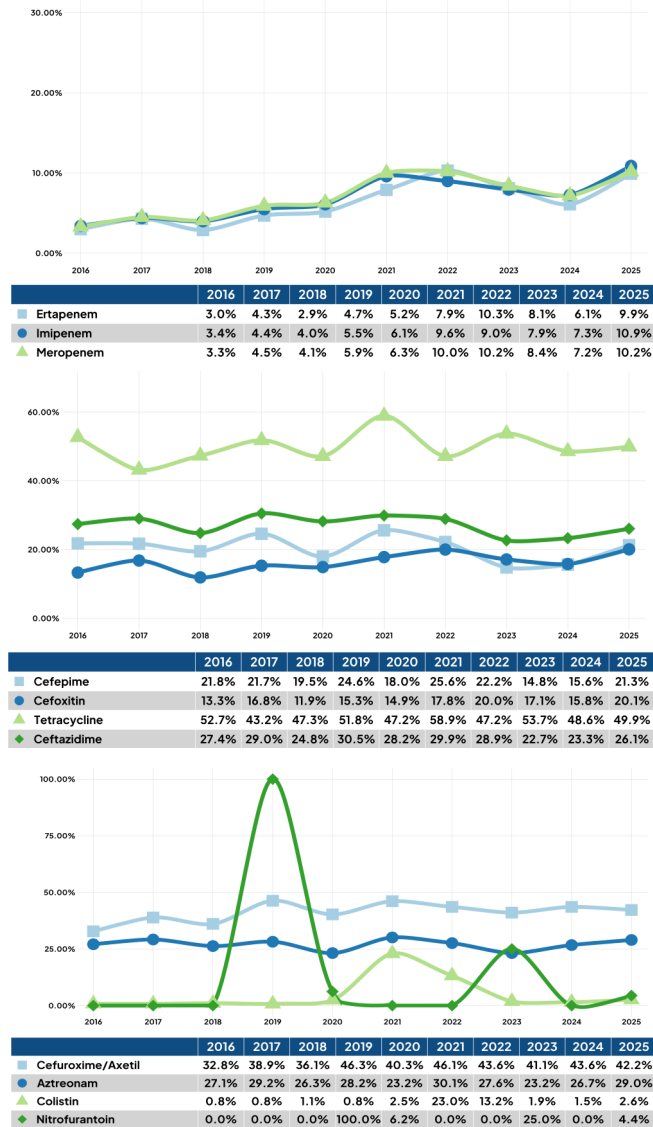


Figure 90. Yearly resistance rates of all *E. coli* infections, DOH-ARSP, 2025

Figure 91 illustrates the geographic distribution of resistance rates among all *E. coli* infections to carbapenems across the administrative regions represented by the surveillance sites. Resistance rates to imipenem, ertapenem, and meropenem across most regions remain relatively contained, ranging predominantly between 1% and 20%.

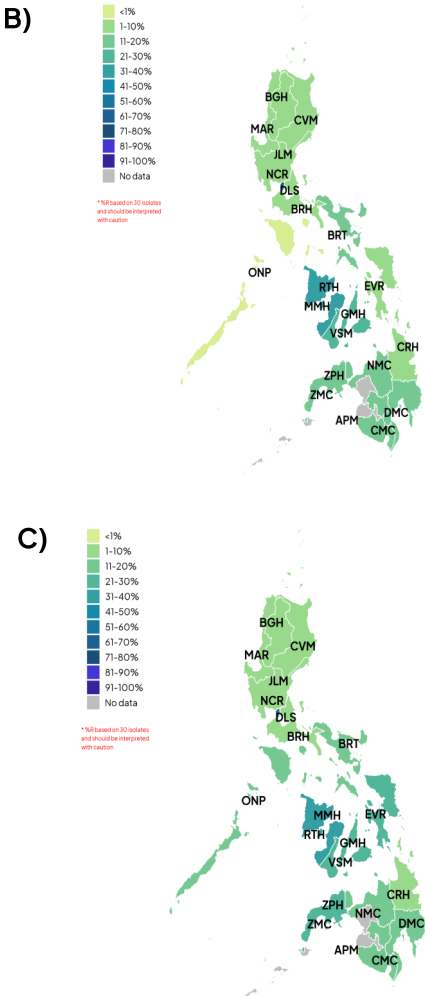
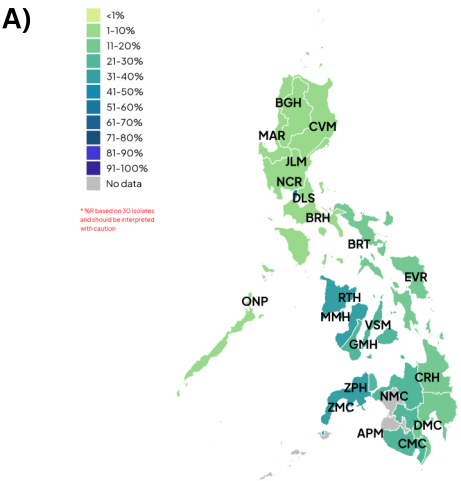


Figure 91. Resistance maps of *E. coli* infections for (A) Imipenem, (B) Ertapenem and (C) Meropenem

Bloodstream Infections

The figure presents the resistance rates for *E. coli* bloodstream infections. The highest resistance rate was observed for ampicillin at 79.00%. For first-generation cephalosporins, cefazolin resistance was recorded at 57.65%. Third-generation cephalosporin resistance reached 42.95% for cefotaxime and 39.32% for ceftriaxone. Fluoroquinolone resistance rates were 44.77% for ciprofloxacin and 42.83% for levofloxacin. Variations were noted among the aminoglycosides, where tobramycin (21.64%) and gentamicin (21.15%) showed moderate resistance, while amikacin resistance remained low at 8.29%. Carbapenem resistance was comparatively lower across ertapenem (10.87%), imipenem (11.26%), and meropenem (10.91%). Among novel combinations, the resistance rate for ceftazidime/avibactam was 37.50%.

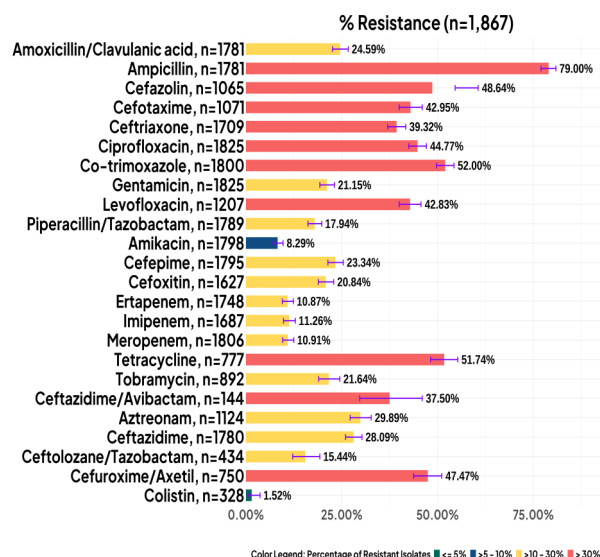


Figure 92. Proportion of *E. coli* bloodstream infections with resistance to tested antibiotics, DOH-ARSP, 2025 (n=1,867)

The yearly resistance rates of *E. coli* bloodstream infections shown in **Figure 93** demonstrate significant increases in resistance rates of meropenem ($p=0.0000$), imipenem ($p=0.0000$), ertapenem ($p=0.0000$) and piperacillin-tazobactam ($p=0.0000$). A significant steady decrease in resistance was shown for co-trimoxazole ($p=0.0000$). While there is no significant difference in the resistance rates for cefotaxime ($p=0.1921$) and aztreonam ($p=0.6862$).

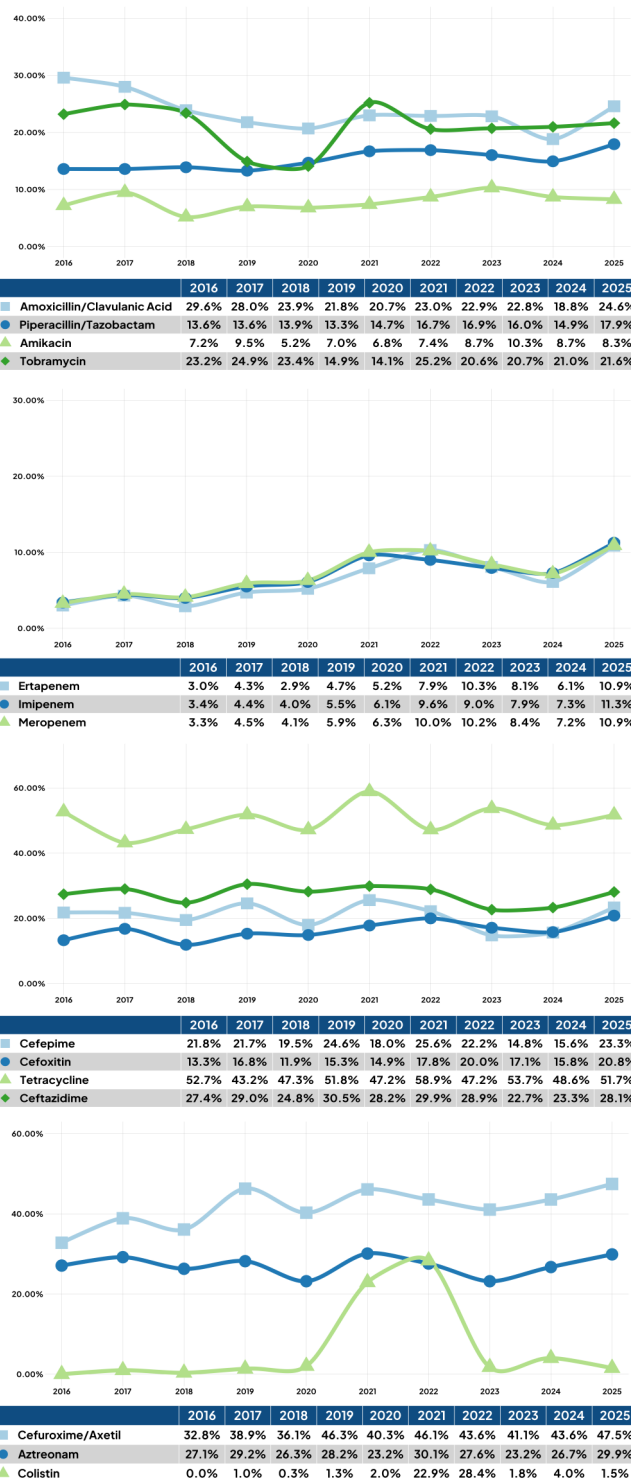
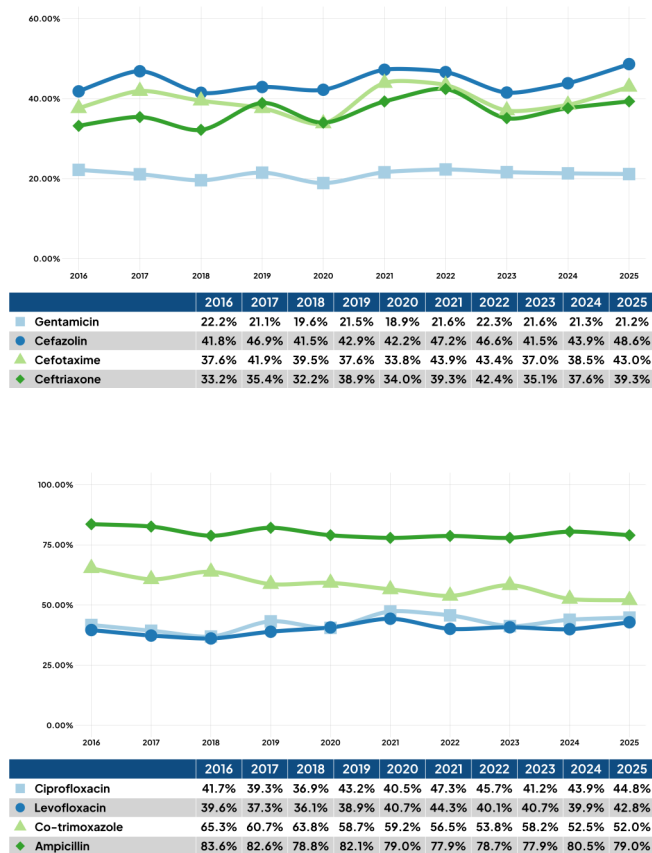


Figure 93. Yearly resistance rates of *E. coli* bloodstream infections, DOH-ARSP, 2025

Urinary Tract Infections

Figure 94 shows the resistance rates of *E. coli* urinary tract infections among inpatients. For first-line oral and empiric options, resistance to co-trimoxazole was elevated at 53.72%, while oral cephalosporins also showed high resistance, with cefazolin at 61.84% and cefuroxime axetil at 55.56%. For parenteral treatment options initiated in hospitalized patients,

resistance to the third-generation cephalosporin ceftriaxone was recorded at 50.21%. Resistance to beta-lactam/beta-lactamase inhibitor combinations was lower, at 27.35% for amoxicillin/clavulanic acid and 23.21% for piperacillin/tazobactam. The fluoroquinolone ciprofloxacin showed a resistance rate of 51.07%. Amikacin demonstrated the lowest resistance rate at 10.09%.

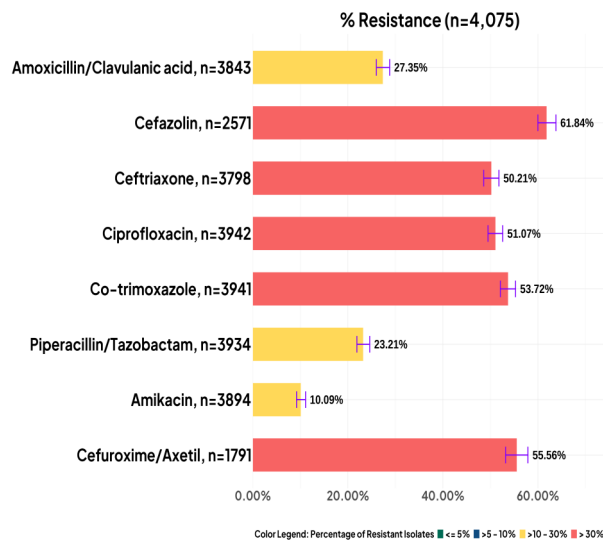


Figure 94. Proportion of *E. coli* urinary tract infections with resistance to tested antibiotics among inpatients, DOH-ARSP, 2025 (n=4,075)

Figure 95 shows the resistance rates of *E. coli* urinary tract infections among outpatients. For first-line, oral empiric UTI therapies, resistance to nitrofurantoin remained low at 3.92%, whereas resistance to co-trimoxazole was elevated at 45.23%. Among alternative oral classes, high resistance rates were observed for ciprofloxacin (46.18%) and oral cephalosporins, specifically cefazolin (46.98%) and cefuroxime axetil (33.98%). Conversely, the oral beta-lactamase inhibitor combination amoxicillin/clavulanic acid demonstrated moderate resistance at 14.79%. For parenteral agents often reserved for more complex cases, resistance to ceftriaxone was noted at 31.03%, while resistance rates remained low for piperacillin/tazobactam (9.60%) and amikacin (5.04%).

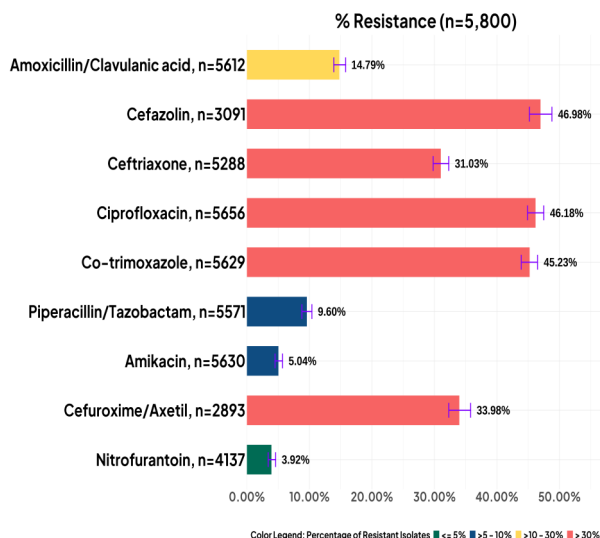


Figure 95. Proportion of *E. coli* urinary tract infections with resistance to tested antibiotics among outpatients, DOH-ARSP, 2025 (n=5,800)

The yearly resistance rates of *E. coli* urinary tract infections are shown in **Figure 96**. Changes in the resistance rates for most antibiotics were observed significant except for nitrofurantoin (p=0.1406).

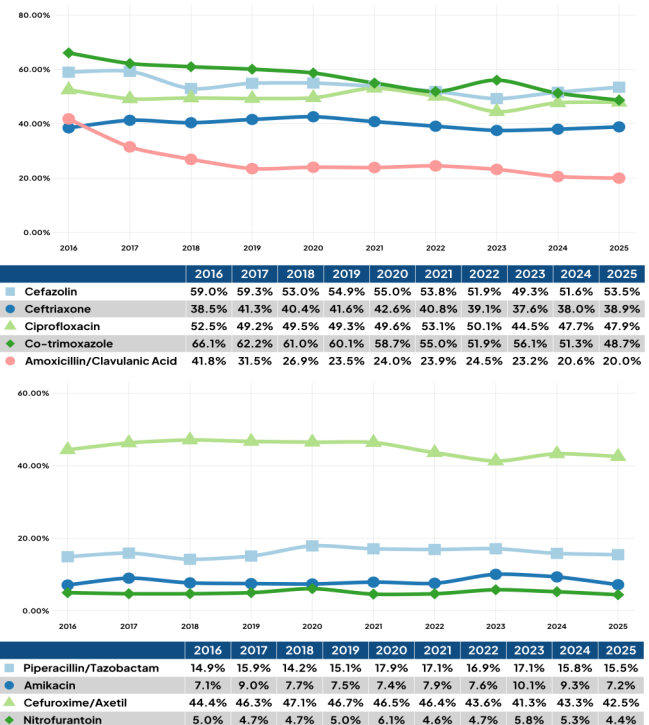


Figure 96. Yearly resistance rates of *E. coli* urinary tract infection, DOH-ARSP, 2025



Figure 97 illustrates the prevalence of carbapenem-resistant *E. coli* (CREC). High resistance rates were observed to almost all antibiotic agents ranging from 43.63% to 97.46%. Resistance remained moderate to low for amikacin (27.89%), nitrofurantoin (13.20%) and colistin (3.25%).

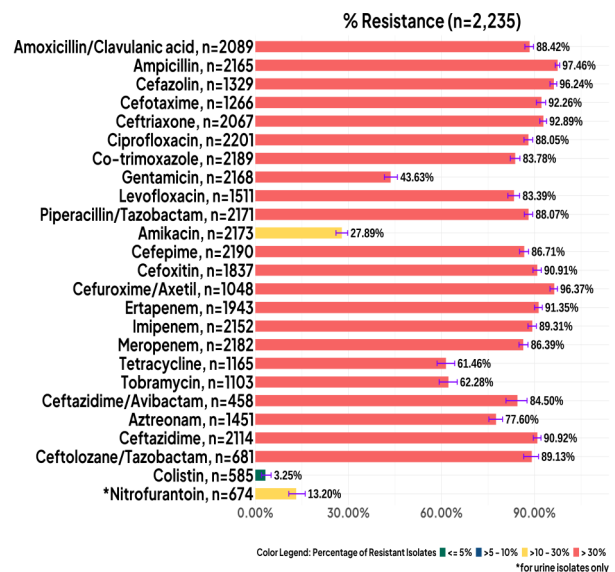


Figure 97. Proportion of carbapenem-resistant *E. coli* blood-stream infections, DOH-ARSP, 2025 (n=238)

Figure 98 for carbapenem-resistant *E. coli* blood-stream reveals high resistance rates for most agents. Highest resistance rates were observed for ampicillin (98.26%), cefazolin (98.11%), and cefuroxime/axetil (97.60%). Carbapenems and ceftazidime/avibactam resistance at over 87% while amikacin (26.96%) and colistin (2.27%) demonstrate considerably lower resistance rates.

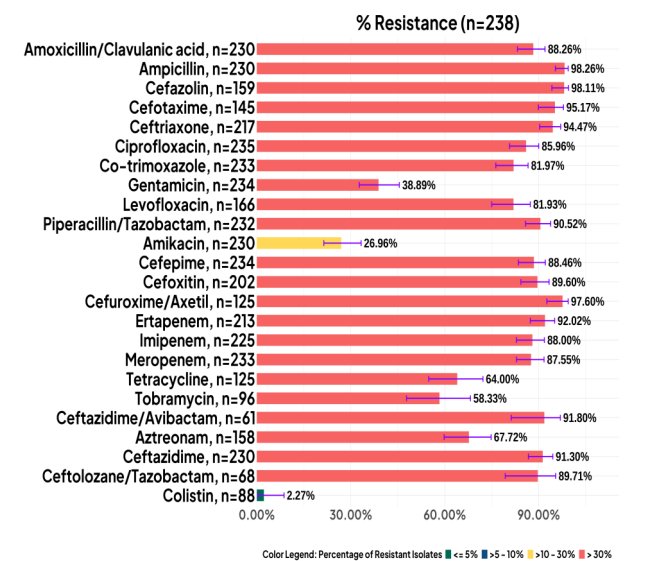


Figure 98. Proportion of carbapenem-resistant *E. coli* blood-stream infections, DOH-ARSP, 2025 (n=238)

From the subset of 2025 *E. coli* infections screened phenotypically for ESBL production (17,248), the positivity rate was at 33.68% details the yearly trend of phenotypic Extended-Spectrum Beta-Lactamase (ESBL) production among *E. coli* isolates from 2016 to 2025. Over this ten-year surveillance period, the ESBL(+) positivity rate demonstrated an overall upward trajectory, climbing from 19.4% in 2016 to a peak of 33.7% in 2025.

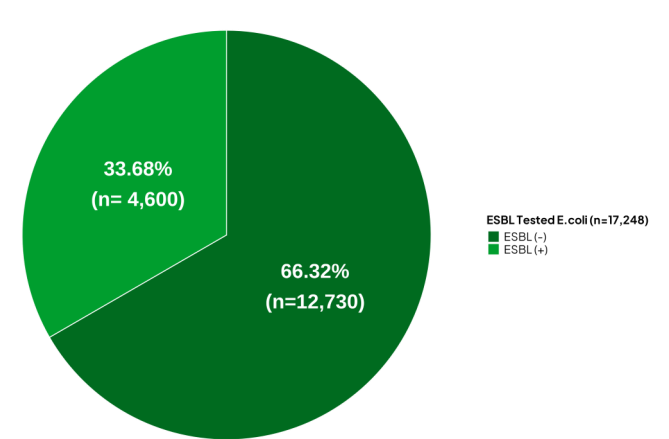


Figure 99. Percentage of ESBL positive and negative *E. coli* infections (n=17,248).

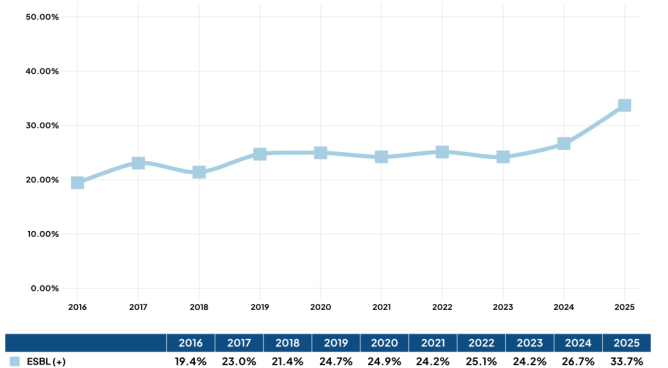


Figure 100. Yearly resistance rates of ESBL-positive *E. coli* infections, DOH-ARSP, 2016-2025

The proportion of bloodstream infections caused by third-generation cephalosporin-resistant (3GCR) *E. coli* remains a core AMR indicator within the SDG framework. This phenotype is characterized as a critical threat due to its high global incidence and resulting limited first-line therapeutic options.

Figure 101 illustrates that resistance rates for ceftriaxone and cefotaxime among *E. coli* bloodstream isolates have fluctuated over the past five years, with an upward trend observed from 2023 to 2025. Utilizing these bloodstream isolates as a proxy for the prevalence of resistant infections, ARSP data indicate that resistance levels continue to rise. These findings suggest that the Philippines may require the strengthening and implementation of targeted interventions to align with this SDG indicator.

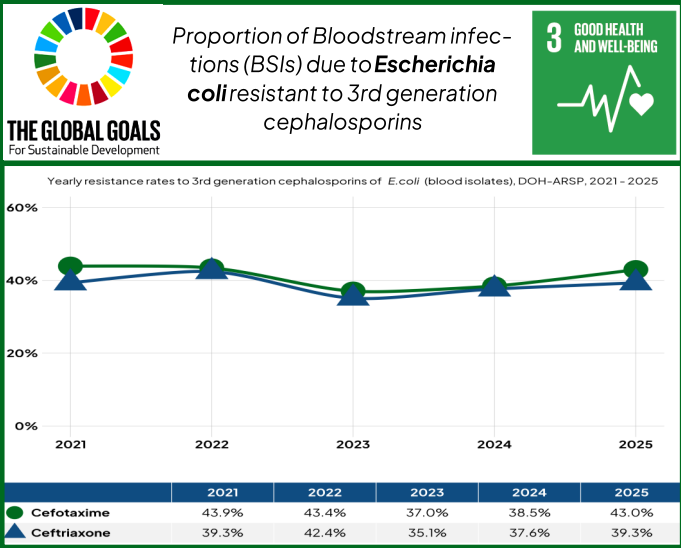


Figure 101. Yearly resistance rates of *E. coli* bloodstream infections, DOH-ARSP, 2021-2025

The figure displays the annual resistance rates of *Escherichia coli* bloodstream isolates to carbapenems and third-generation cephalosporins from 2023 to 2025, evaluated against the National Action Plan on Antimicrobial Resistance (NAP-AMR) target of a 10% reduction from the 2023 baseline. Third-generation cephalosporins did not reach the target reductions. Cefotaxime resistance increased from 37.0% in 2023 to 38.5% in 2024, and reached 43.0% in 2025, remaining above the 33.4% target threshold. Ceftriaxone resistance rose from 35.1% in 2023 to 37.6% in 2024, and 39.3% in 2025, compared to its 31.6% target.

For the carbapenems, ertapenem and meropenem met the 10% target in 2024, decreasing to 6.1% and 7.2%, respectively, while imipenem showed an 8.4% reduction to 7.3%. However, in 2025, resistance rates for all three carbapenems increased: meropenem and ertapenem rose to 10.9%, and imipenem rose to 11.3%, placing them above their respective target thresholds.

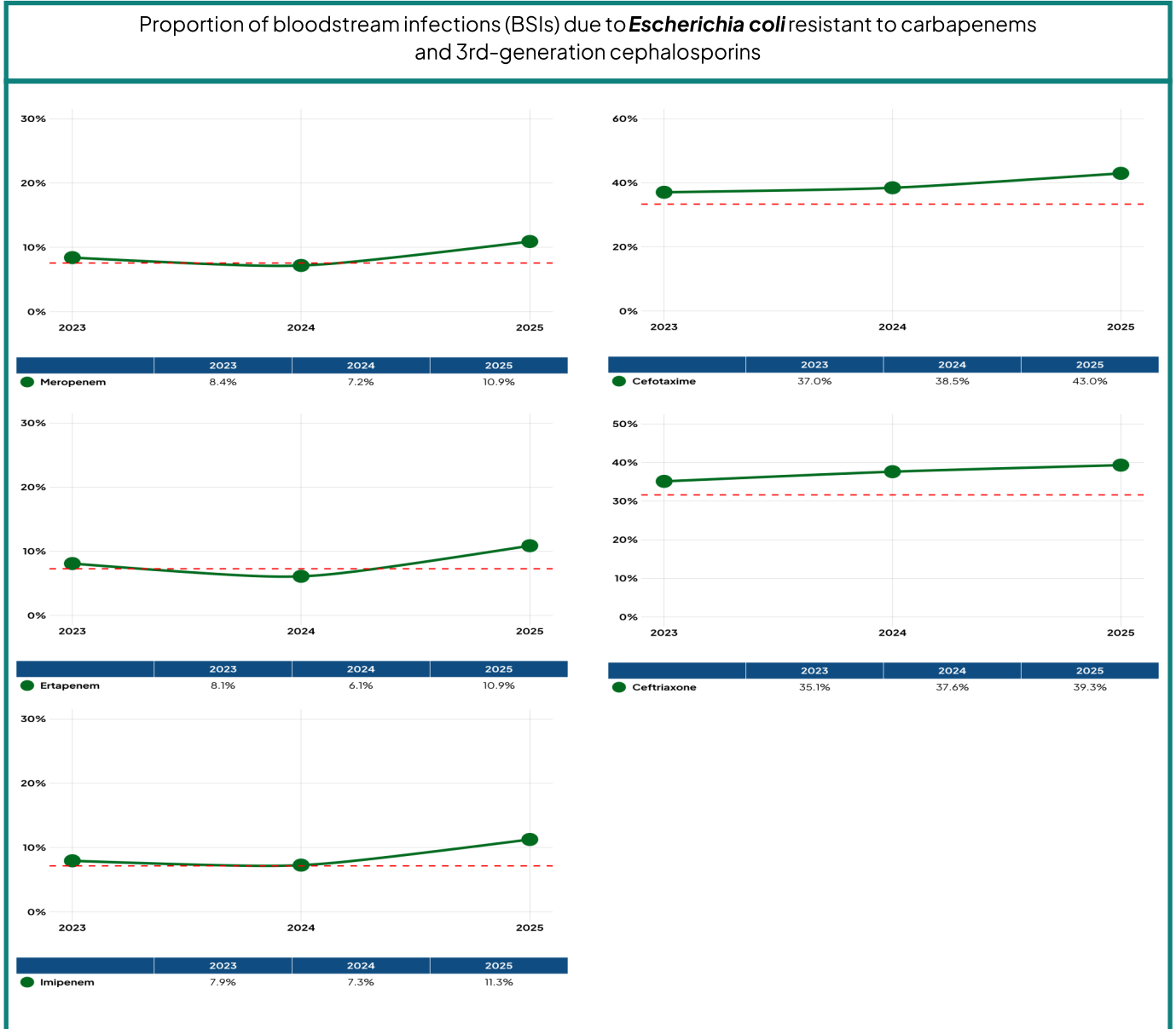


Figure 102. Yearly resistance rates of *E. coli* bloodstream infections, DOH-ARSP, 2023-2025



Isolate Characteristics and Distribution

The emergence of *Escherichia coli* exhibiting resistance to colistin and novel β -lactam/ β -lactamase inhibitor (BL/BLI) combinations represents a critical clinical challenge, given their role as important treatment options against multidrug-resistant infections. To characterize these emerging resistance, a total of 182 isolates were analyzed, categorized by their resistance to colistin (COL; n=1), ceftazidime-avibactam (CZA; n=134), imipenem-relebactam (IMR; n=26), and meropenem-vaborbactam (MEV; n=21) (Figure 103). Across all evaluated agents, isolates were predominantly recovered from females, ranging from 65.67% (n=88) in the CZA group to 100% (n=1) in the COL group, with the single COL isolate (100%) also originating from a female patient. The 25–64 age group consistently represented the largest group across all categories. Urine was the predominant specimen type for CZA, IMR, and MEV, whereas the sole COL isolate was recovered from blood. Furthermore, most infections were presumptive hospital-acquired (Figure 103).

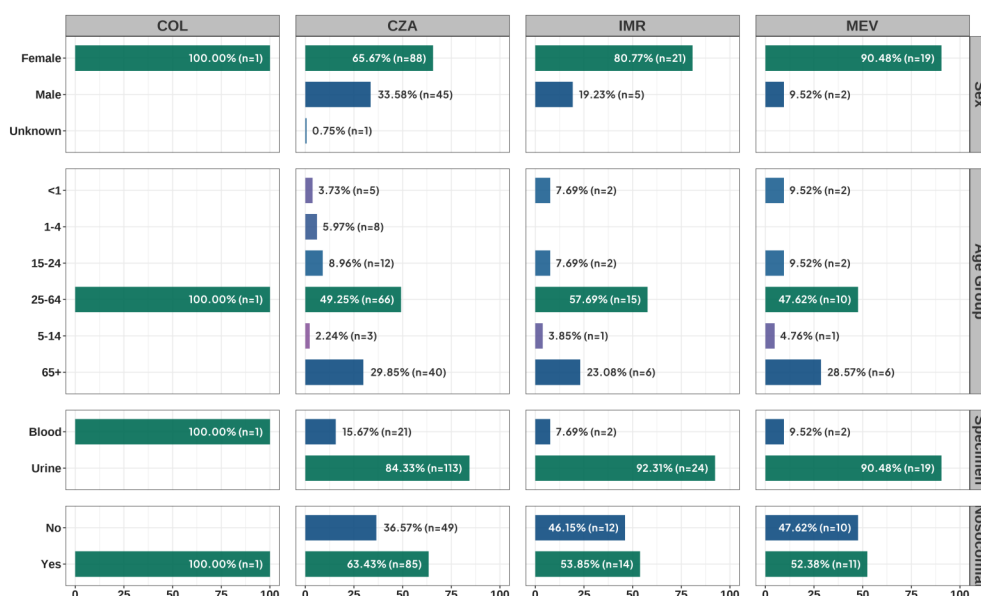


Figure 103. Patient demographics and clinical characteristics of *E. coli* isolates across key antibiotic agents

Colistin-resistant *E. coli*

In 2025, colistin resistance in *E. coli* remained exceptionally rare within the surveillance network, with only a single isolate (25ARS_VSM0114) identified from a blood specimen in Region VII (VSM). This specific isolate was identified as ST 1193, a high-risk clone belonging to phylogroup B2 and carrying the O75:H5 serotype. While the overall number of colistin-resistant *E. coli* remains low compared to other species^[1], the detection of ST 1193, a lineage frequently associated with extraintestinal infections and multidrug resistance, is a significant finding that requires continued monitoring.

Notably, genomic analysis of this isolate confirmed that no known *mcr* genes were detected. Absence of these mobile genetic elements suggests that the colistin-resistant phenotype in this ST 1193 strain is likely driven by chromosomal resistance mechanisms^[2]. High-resolution characterization is needed to further identify these hidden mechanisms and drivers.

Novel BL/BLI-resistant *E. coli*

E. coli emerging resistance to novel beta-lactam/beta-lactamase inhibitor combinations demonstrate a highly diverse genetic landscape with clonal expansion of specific high-risk lineages. ST 410 (phylogroup C) was identified as a widely disseminated high-risk clone, accounting for 25% (n = 34) of the novel BL/BLI-resistant and appearing across 13 surveillance sites in Luzon, Visayas, and Mindanao. This major lineage is followed in frequency by ST361 (phylogroup A, 18.4%) and ST8346 (phylogroup B1, 11%). Genomic serotype analysis further highlights this epidemiological diversity: ST410 is strongly associated with -H9, O8:H21, and O8:H9 antigen profiles, while ST361 is predominantly classified as O9:H30, and ST8346 is identified as O64:H10. This structural variation indicates that emerging BL/BLI resistance is not clonal, but is instead circulating within multiple distinct genetic backgrounds across the national network.

Table 8. Overall distribution and molecular typing summary of colistin and BL-BLI resistant *E. coli* isolates

Overall Isolates (N=137)	Surveillance Sites	ST	Serotypes	Phylogroups	Count
Colistin Resistant (n=1)	VSM	1193	O75:H5	B2	1 (100%)
Novel BLI Resistant (n=136)	APM, BRT, CMC, CRH, CVM, DMC, GMH, JLM, MAR, MMH, RTH, VSM, ZMC	410	-:H9, O8:H21, O8:H9	C	34 (25%)
	APM, CMC, CRH, DMC, GMH, MAR, NMC, PGH, VSM	361	O9:H30	A	25 (18.4%)
	APM, BRH, CMC, EVR, JLM, VSM, ZMC	8346	O64:H10	B1	15 (11%)
	BRH, CRH, DMC, GMH, VSM	448	O160:H8, O23:-, O23:H19, O8:H8	B1	12 (8.8%)
	APM, BRT, GMH, RMC, VSM	167	O89:H10, O89:H9	A	11 (8.1%)
	CRH, DMC, JLM, VSM	2083	-:H6	B1	5 (3.7%)
	BRT, MAR	2851	O100:H18	C	4 (2.9%)
	MAR	117	O161:H4	G	2 (1.5%)
	MMH, VSM	1193	O18:H5, O75:H5	B2	2 (1.5%)
	DMC	2772	O121:H7	B1	2 (1.5%)
	VSM	349	O166:H15, O86:H2	D	2 (1.5%)
	APM, DMC	405	O102:H6	D	2 (1.5%)
	BRT, DMC	-	-:H21, O2/O50:H18, O2/O50:H4, O89:H4	A, B2, C, D	7 (5.1%)
	BRT, CMC, CRH, DMC, JLM, MAR, VSM	156, 15639, 1722, 224, 2279, 354, 3580, 3640, 44, 646, 69, 88, 95	-:H12, -:H28, O1:H34, O15:H18, O153:H30, O166:H6, O2/O50:H4, O25:H4, O8:H12, O8:H19, O8:H23, O89:H10, O89:H4	A, B1, B2, C, D, F	13 (9.6%)

Table 9. Distribution of carbapenemase genes among colistin and BL-BLI resistant *E. coli* isolates

Category	Determinant / Mutation	Frequency (n)	Associated Resistance Profile	Region	Surveillance Site
Carbapenemases	<i>bla</i> NDM-5	75	CZA, CZT, IMR, MEV	I, IV-A, V, VI, VII, VIII, IX, X, XI, XII, XIII, BARMM, NCR	MAR, BRH, BRT, GMH, VSM, EVR, ZMC, NMC, DMC, CMC, CRH, APM, PGH, RMC, JLM
	<i>bla</i> NDM-7	26	CZA, CZT, IMR, MEV	I, III, V, VI, VII, X, XI, XIII, BARMM, NCR	MAR, RTH, BRT, MMH, VSM, NMC, DMC, CRH, APM, JLM
	<i>bla</i> NDM-1	19	CZA, CZT, IMR, MEV	VI, VII, XI, XII, XIII, BARMM, NCR	MMH, GMH, VSM, DMC, CMC, CRH, APM, JLM
	<i>bla</i> KPC-2	1	CZA, CZT	I	MAR
	<i>bla</i> NDM-6	1	CZA	V	BRT
	-	5	CZA, MEV, IMR	V, II, I, VII	BRT, CVM, MAR, VSM
PBP3 Mutations	<i>ftsI</i> _N337NYRIN	101	CZA	I, IV-A, V, VI, VII, VIII, IX, X, XI, XII, XIII, BARMM, NCR	MAR, BRH, BRT, GMH, VSM, EVR, ZMC, NMC, DMC, CMC, CRH, APM, PGH, RMC, JLM
	<i>ftsI</i> _I336IKYRI	6	CZA	XI, BARMM	DMC, APM
Porin Mutations	<i>ompC</i> _R195L	6	IMR, MEV	V, VII, XI, NCR	BRT, VSM, DMC, JLM
Colistin Genes	<i>mcr</i> genes	0	COL (No <i>mcr</i> detected in COL-R isolate)	VII	VSM

Table 9 reveals that the mechanisms driving resistance among these isolates are heavily dominated by the New Delhi metallo-beta-lactamase (NDM) family, specifically the *bla*NDM-5 (n=75), *bla*NDM-7 (n=26), and *bla*NDM-1 (n=19) variants. These carbapenemases frequently co-occur with specific chromosomal alterations. For instance, resistance to ceftazidime-avibactam (CZA) is structurally linked to the *ftsI_N337NYRIN* penicillin-binding protein 3 (PBP3) insertion, which was identified in 101 isolates across almost all surveillance sites. Resistance to the carbapenem/beta-lactamase inhibitor combinations imipenem-relebactam (IMR) and meropenem-vaborbactam (MEV) is notably associated with the *ompC_R195L* porin mutation. This porin alteration is primarily observed in isolates that already harbor *bla*NDM-5 or *bla*NDM-7. The presence of these outer-membrane porin mutations, particularly when combined with high-level NDM production in sentinel sites like DMC, VSM, and BRT, suggests a potent synergistic mechanism that simultaneously reduces drug entry and enzymatic inactivation [3].

The 2025 genomic surveillance data underscores a critical change in the landscape of multidrug-resistant *Escherichia coli* within the Philippines. The population has attained a high degree of genetic complexity, characterized by the convergence of mobile carbapenemase genes and stable chromosomal mutations, a combination that threatens to render novel beta-lactam/beta-lactamase inhibitor (BL/BLI) therapeutics increasingly ineffective. Specifically, the widespread dissemination of the high-risk ST410 lineage across Luzon, Visayas, and Mindanao represents a formidable public health threat, establishing a highly stable, national reservoir for NDM-mediated resistance. While colistin resistance remains rare overall, the identification of a non-*mcr*, chromosomally driven resistance phenotype in an emerging ST1193 blood isolate indicates an ongoing shift in available therapeutic options. These highlight the continuous utility of institutionalized, high-resolution genomic tracking alongside standard infection prevention and control protocols.

Reference

- [1] Vlad, M. A., Dan, M., Catana, A. N., Dumitriu, S., & Tuchilus, C. G. (2025). Trends in Colistin Resistance and Multidrug-Resistant Phenotypes Among Gram-Negative Bacilli: A Retrospective Analysis. *Molecules*, 30(14), 2950.
- [2] Rhouma, M., Beaudry, F., Thériault, W., & Letellier, A. (2016). Colistin in pig production: chemistry, mechanism of antibacterial action, microbial resistance emergence, and one health perspectives. *Frontiers in microbiology*, 7, 1789.
- [3] Simner, P. J., Bergman, Y., Conzemius, R., Jacobs, E., Tekle, T., Beisken, S., & Tamma, P. D. (2023, July). An NDM-producing *Escherichia coli* clinical isolate exhibiting resistance to cefiderocol and the combination of ceftazidime-avibactam and aztreonam: another step toward pan- β -lactam resistance. In *Open Forum Infectious Diseases* (Vol. 10, No. 7, p. ofad276). US: Oxford University Press.



Structured Genomic Survey

Isolate Characteristics and Distribution

One hundred and five (105) *Escherichia coli* isolates were included in whole-genome sequencing (WGS) analysis from 294 phenotypically tested isolates (189 carbapenem resistant, 105 carbapenem susceptible) submitted across 24 ARSP surveillance sites. Of the sequenced isolates, 95 (90.5%) were phenotypically resistant and 10 (9.5%) were susceptible to carbapenems. Specimens comprised blood (19 isolates) and urine (86 isolates). The 25–64 year age group accounted for the largest proportion of cases (49, 46.7%), followed by those aged 65 years and older (33, 31.4%); pediatric cases below 15 years of age totalled 15 (14.3%), including five neonates. Females outnumbered males (72, 69.2% versus 32, 30.8%). Presumptive nosocomial cases outnumbered community-onset cases (58, 55.2% versus 47, 44.8%).

Table 10. Patient demographics and clinical characteristics (n=105)

Specimen type distribution		Age group distribution		Sex distribution		Nosocomial (Yes/No)	
Specimen Type	Count	Age group	Count	Sex	Count	Nosocomial	Count
Blood	19	<1	5	Female	72	No	47
Urine	86	1-4	6	Male	32	Yes	58
		5-14	4	No data	1		
		15-24	8				
		25-64	49				
		65+	33				

Carbapenem Resistance Determinants

Of the 95 phenotypically resistant isolates, a carbapenem resistance gene was identified in 85 (89.5%). *bla*NDM-5 was the most common determinant (51 isolates, 60.0%), followed by *bla*NDM-7 (21, 24.7%) and *bla*NDM-1 (13, 15.3%). A single isolate (1.2%) from Region I carried *bla*KPC-2, the only non-NDM carbapenemase detected. No co-occurrence of two carbapenem resistance genes was found in any isolate, in contrast to the preceding year's structured survey [3] in which two ST410 isolates co-carried *bla*KPC-2 and *bla*NDM-5. No carbapenem resistance genes were detected in the 10 phenotypically susceptible isolates, indicating consistent genotype-phenotype agreement across the collection.

Regional distribution showed geographic variation. *bla*NDM-5 was the predominant allele in most regions, including BARMM (66.7%), Region I (71.4%), Region V (83.3%), Region VII (57.9%), Region XII (83.3%), and Region XIII (70.0%). In contrast, *bla*NDM-7 predominated in Region XI (54.5%) and Region VI (66.7%). The single *bla*KPC-2-carrying isolate in Region I co-occurred with NDM-type producers and did not appear to represent an established local cluster. The predominance of *bla*NDM-7 in Regions XI and VI, in contrast to *bla*NDM-5 elsewhere, suggests these regions may have distinct transmission histories.

Table 11. Distribution of carbapenemase genes among carba-R *E. coli* isolates by surveillance site and region, ARSP 2025 (n=85)

Surveillance Sites	Region	Count	NDM-1	NDM-5	NDM-7	KPC-2
APM	BARMM	9 (10.6%)	1 (11.1%)	6 (66.7%)	2 (22.2%)	0%
BRH	IV-A	2 (2.4%)	0%	2 (100.0%)	0%	0%
BRT	V	6 (7.1%)	0%	5 (83.3%)	1 (16.7%)	0%
CMC	XII	6 (7.1%)	1 (16.7%)	5 (83.3%)	0%	0%
CRH	XIII	10 (11.8%)	1 (10.0%)	7 (70.0%)	2 (20.0%)	0%
DMC	XI	11 (12.9%)	4 (36.4%)	1 (9.1%)	6 (54.5%)	0%
EVR	VIII	1 (1.2%)	0%	1 (100.0%)	0%	0%
GMH	VII	7 (8.2%)	1 (14.3%)	6 (85.7%)	0%	0%
JLM	III	4 (4.7%)	1 (25.0%)	2 (50.0%)	1 (25.0%)	0%
MAR	I	7 (8.2%)	0%	5 (71.4%)	2 (28.6%)	1 (14.3%)
MMH	VI	2 (2.4%)	1 (50.0%)	0%	1 (50.0%)	0%
NMC	X	3 (3.5%)	0%	2 (66.7%)	1 (33.3%)	0%
PGH	NCR	1 (1.2%)	0%	1 (100.0%)	0%	0%
RMC	NCR	1 (1.2%)	0%	1 (100.0%)	0%	0%
RTH	VI	1 (1.2%)	0%	0%	1 (100.0%)	0%
VSM	VII	12 (14.1%)	3 (25.0%)	5 (41.7%)	4 (33.3%)	0%
ZMC	IX	2 (2.4%)	0%	2 (100.0%)	0%	0%

Molecular Typing Analysis

WGS-based multi-locus sequence typing identified 28 distinct sequence types (STs) among 104 of the 105 isolates; one isolate could not be assigned to a known ST. Four lineages accounted for approximately 59% of the total. ST410 was the most common (21 isolates, 20.0%), belonging to phylogroup C with multiple serotypes mainly O8:H9, and was distributed across 11 surveillance sites (BARMM, Regions I, II, III, IX, V, VI, VII, XI, XII, and XIII). ST361 was second (18 isolates, 17.1%), with serotype O9:H30 and phylogroup A, detected across seven sites. ST8346 contributed 13 isolates (12.4%), with serotype O64:H10 (phylogroup B1) across seven sites, and ST167 contributed 10 isolates (9.5%) with serotype O89:H9 (phylogroup A) across five sites. Other recurring lineages included ST448 (6 isolates, 5.7%), ST2083 (5 isolates, 4.8%), and ST1193 (4 isolates, 3.8%). The remaining 17 isolates (16.2%) belonged to 16 different minor STs spanning multiple phylogroups. Comparison with carbapenem-resistant isolates from the 2024 structured survey^[3] identified several persisting lineages: ST410, ST361, ST167, ST448, ST2083, ST2851, ST10, and ST224 were detected in both years. ST8346, the third most common lineage in 2025 (13 isolates, 12.4%), was not identified in the preceding year's structured dataset, representing a notable change in lineage composition. ST5208, detected once in 2024, was not identified in the current collection.

Table 12. Overall distribution and molecular typing summary of *E. coli*

Surveillance Sites	Region	Sequence Type (ST)	Phylo group	Serotypes	Overall (N=105)	Phenotypic Resistant (n=95)	Phenotypic Susceptible (n=10)
APM, BRI, CMC, CRH, CVM, DMC, GMH, JLM, MAR, MMH, RTH, VSM, ZMC	BARMM, I, II, III, IX, V, VI, VII, XI, XII, XIII	410	C	-:H9(9), O8:H21(2), O8:H9(10)	21(20.0%)	21(22.1%)	0(0.0%)
APM, CMC, CRH, GMH, MAR, NMC, PGH, VSM	BARMM, I, NCR, VII, X, XII, XIII	361	A	O9:H30	18(17.1%)	18(18.9%)	0(0.0%)
APM, BRH, CMC, EVR, JLM, VSM, ZMC	BARMM, III, IV -A, IX, VII, VIII, XII	8346	B1	O64:H10	13(12.4%)	13(13.7%)	0(0.0%)
APM, BRI, CRH, GMH, RMC	BARMM, NCR, V, VII, XIII	167	A	O89:H10(1), O89:H9(9)	10(9.5%)	10(10.5%)	0(0.0%)
BRH, CRH, GMH, VSM	IV-A, VII, XIII	448	B1	O160:H8(1), O23:H19(5)	6(5.7%)	6(6.3%)	0(0.0%)
CRH, DMC, JLM, VSM	III, VII, XI, XIII	2083	B1	-:H6	5(4.8%)	5(5.3%)	0(0.0%)
MMH, VSM	VI, VII	1193	B2	O18:H5(2), O75:H5(2)	4(3.8%)	3(3.2%)	0(0.0%)
BRI, MAR	I, V	2851	C	O100:H18	3(2.9%)	3(3.2%)	0(0.0%)
MAR	I	117	G	O161:H4	2(1.9%)	2(2.1%)	0(0.0%)
CMC, MAR	I, XII	224	B1	O8:H23	2(1.9%)	1(1.1%)	0(0.0%)
DMC	XI	2772	B1	O121:H7	2(1.9%)	2(2.1%)	0(0.0%)
APM, DMC	BARMM, XI	405	D	O102:H6	2(1.9%)	2(2.1%)	0(0.0%)
BRI, CMC, CRH, DMC, FEU, JLM, MMH, VSM	III, NCR, V, VI, VII, XI, XII, XIII	Minor STs[*]	Diverse	O89:H4(2), -:H12(1), -:H28(1), O102:H51(1), O109:H8(1), O12:H4(1), O153:H30(1), O166:H6(1), O1:H34(1), O2/O50:H4(1), O25:H4(1), O75:H5(1), O83:H4(1), O86:H2(1), O8:H12(1), O8:H19(1)	10(9.5%)	10(10.5%)	0(0.0%)
BRI, CMC, FEU, MMH, VSM	NCR, V, VI, VII, XII	Sporadic Clones[**]	Diverse	O102:H51(1), O109:H8(1), O12:H4(1), O18:H5(1), O25:H4(1), O75:H5(1), O83:H4(1), O86:H2(1), O89:H4(1), O8:H23(1)	10(9.5%)	0(0.0%)	10(100.0%)

[*] Minor STs: Includes -, ST156, ST1722, ST224, ST354, ST3580, ST3640, ST646, ST88, and ST95.

[**] Sporadic Clones (exclusively susceptible): Includes ST10, ST101, ST1193, ST131, ST224, ST349, ST429, ST44, ST550, and ST770.

Phylogenetic Analysis

The core-genome SNP phylogeny of the 105 isolates showed high genomic diversity, with pairwise SNP distances ranging from 0 to 120,508 SNPs (median ~21,000 SNPs). Isolates partitioned broadly into lineage-specific clusters consistent with ST assignments, reflecting the genomic diversity expected across 28 distinct sequence types.

The four dominant lineages formed discrete clusters in the phylogeny. ST410 (21 isolates, phylogroup C) showed the broadest intra-lineage diversity, with heterogeneous carbapenem resistance gene profiles (*bla*NDM-5, *n*=7; *bla*NDM-7, *n*=10; *bla*NDM-1, *n*=2) and multiple co-circulating serotypes (O8:H9, O8:H21, -:H9). ST361 (18 isolates, phylogroup A, O9:H30) formed a cohesive cluster; all isolates shared a single serotype and predominantly carried *bla*NDM-5 (12/18). ST8346 (13 isolates, phylogroup B1, O64:H10) was the most genetically conserved of the four, with a uniform serotype and exclusive *bla*NDM-5 carriage across all 13 isolates. ST167 (10 isolates, phylogroup A, O89:H9) also formed a discrete cluster, predominantly carrying *bla*NDM-5 (7/10).

The 10 phenotypically susceptible isolates did not cluster within the four dominant resistant lineages; all belonged to minor STs and branched separately in the phylogeny. This distribution indicates that susceptibility in this collection reflects the genomic background of the isolates rather than the absence of acquired resistance genes within an otherwise resistant lineage.

Several closely related isolate pairs (≤ 20 SNPs) were identified across geographically distinct surveillance sites within the same ST. Inter-facility transmission dynamics are examined in further detail in the companion emerging pathogen analysis.

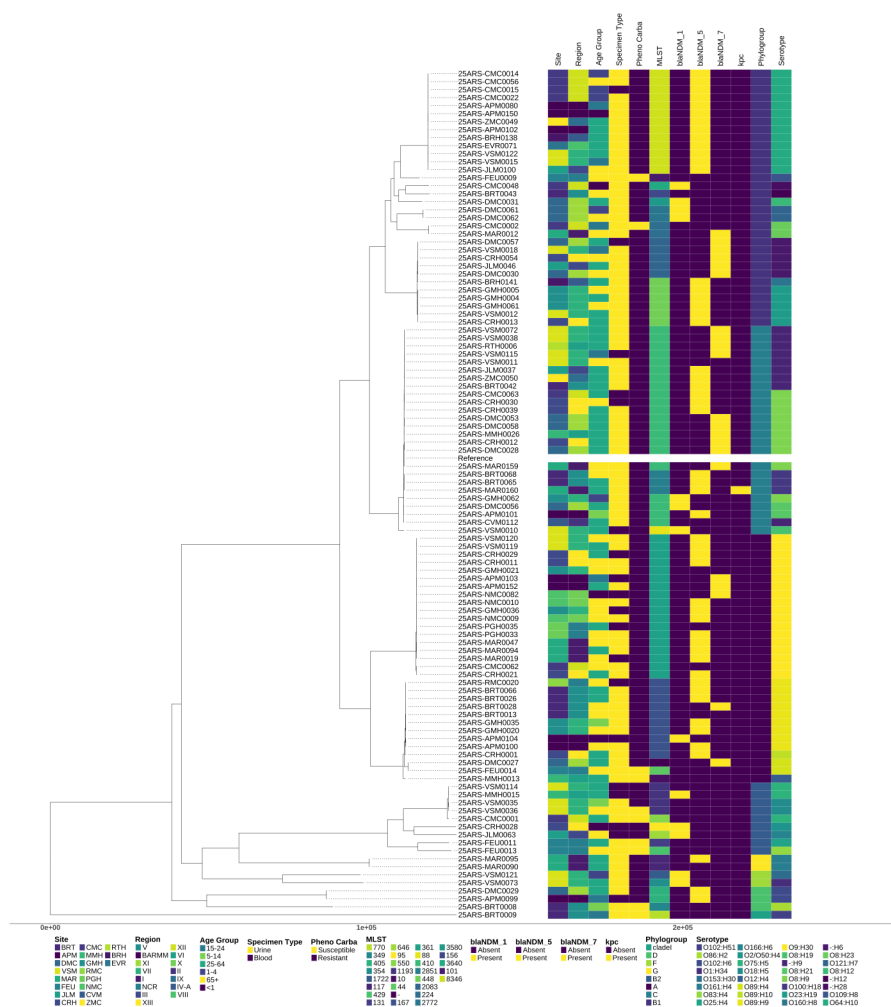


Figure 104. Phylogenetic tree of the carbapenem resistant *E. coli* isolates

Figure 105 presents the two-year comparison maps (2024–2025) illustrating the geographic distribution and frequency of major carbapenemase genes, NDM-1, NDM-5, NDM-7, and KPC-2, detected among *Escherichia coli* isolates across ARSP surveillance sites. NDM-5 is identified as the high-frequency carbapenemase, with total detections more than tripling from 16 isolates in 2024 to 51 in 2025. While the MAR site was the primary cluster in 2024 with 9 isolates, by 2025, NDM-5 reached the high detection category (6 or more isolates) at new sites including CRH (*n*=7), APM (*n*=6), and GMH (*n*=6). NDM-7 is classified as an intermediate-frequency gene, growing from 6 to 21 total isolates, with DMC reporting the highest site-specific detection for this gene in 2025 (*n*=6). NDM-1 demonstrates an emerging-frequency pattern, increasing from only 2 isolates in 2024 to 13 in 2025, with DMC (*n*=4) and VSM (*n*=3) exhibiting moderate detection (2–5 isolates). Conversely, KPC-2 remains rare and localized, recovered exclusively from the MAR site with 2 isolates in 2024 and a single isolate in 2025. Overall, the maps reveal that detection levels at individual surveillance sites are transitioning from primarily single isolates in 2024 to more frequent moderate and high detection categories in 2025.

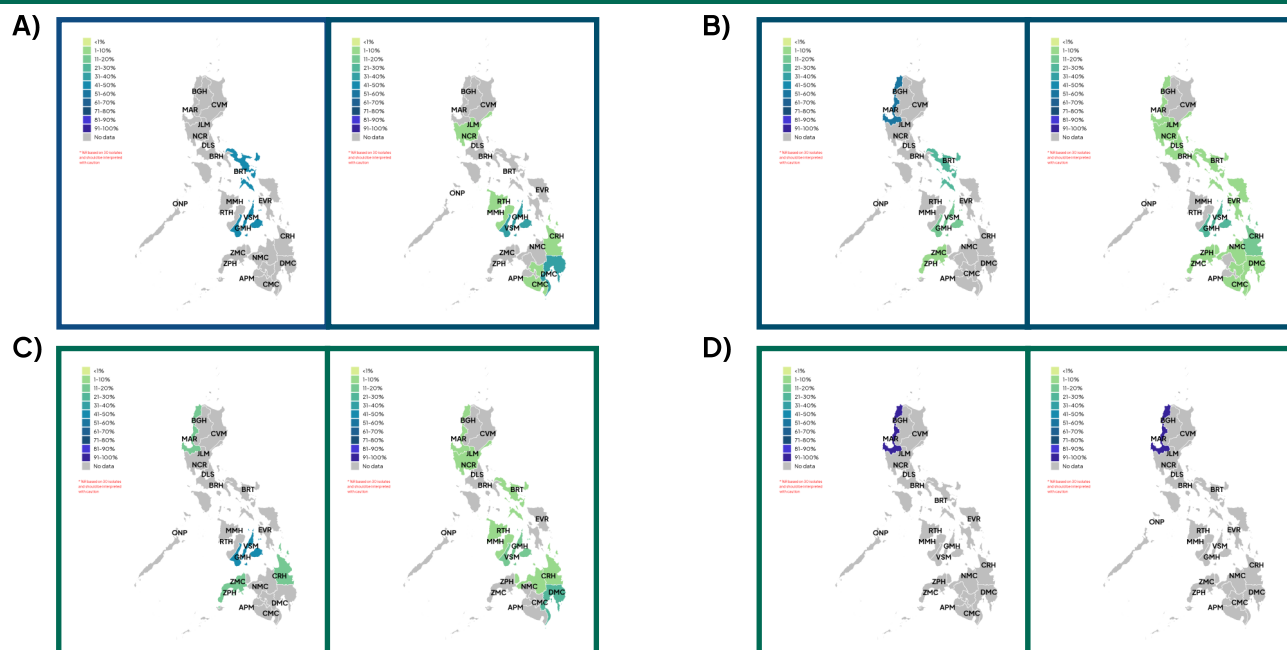


Figure 105. Two-year comparison maps of Carbapenemase genes detected among *E. coli* isolates from ARSP surveillance sites for (A) NDM-1, (B) NDM-5, (C) NDM-7, and (D) KPC-2

Discussion

NDM-type metallo- β -lactamases accounted for all but one of the carbapenem resistance determinants in this survey. *bla*NDM-5 was the most prevalent allele (60.0%), consistent with findings from prior ARSP surveillance cycles [3] and with its emergence as the dominant NDM variant in *E. coli* globally [1,2]. NDM enzymes hydrolyse nearly all β -lactam classes, including carbapenems, leaving treatment options limited to aztreonam, polymyxins, and tigecycline—agents that are themselves often compromised by co-resistance genes commonly found alongside NDM determinants [2]. Its detection across both the dominant high-risk lineages and multiple minor STs suggests its dissemination is not confined to a single clonal lineage.

Ten of the 95 (10.5%) phenotypically resistant isolates had no carbapenem resistance gene detected by WGS-based analysis. The mechanism of carbapenem resistance in these isolates was not identified and warrants further characterisation.

ST410, the most prevalent clone in this survey (21 isolates, 20.0%), is an internationally recognised multi-drug-resistant lineage with established links to carbapenem resistance dissemination globally [1]. Together with ST361 (18 isolates, 17.1%), these two clones accounted for approximately 37% of sequenced isolates. ST410 showed the widest geographic distribution, detected across 11 surveillance sites. Phylogenetic analysis confirmed the discrete clade structure of these lineages; the co-occurrence of *bla*NDM-1, *bla*NDM-5, and *bla*NDM-7 within the ST410 clade suggests that carbapenem resistance gene composition is not uniform within this lineage. The detection of ST410 and ST361 in both the 2024 and 2025 structured surveys [3] is consistent with these lineages being established in the sampled population. ST8346, absent from the 2024 structured dataset, accounted for 12.4% of isolates in 2025, indicating a shift in lineage composition that warrants continued monitoring.

The single *bla*KPC-2 isolate from Region I had no cross-site counterparts, and KPC-type enzymes do not appear established in the sampled population.

Conclusion

NDM-type metallo- β -lactamases, principally *bla*NDM-5, are the predominant mechanism of carbapenem resistance in *E. coli* across the ARSP surveillance network, indicating that NDM-producing *E. coli* is now well-established in local clinical settings with direct implications for empiric treatment, as carbapenems can no longer be assumed effective. ST410 and ST361 together accounted for approximately 37% of isolates and were detected across multiple dispersed surveillance sites, consistent with inter-facility clonal spread. Continued WGS-based surveillance is needed to monitor further changes in clone and gene distribution.

References

- [1] Xia C, et al. Epidemiological and genomic characteristics of global *bla*NDM-carrying *Escherichia coli*. *Ann Clin Microbiol Antimicrob.* 2024;23(1):58. <https://doi.org/10.1186/s12941-024-00719-x>
- [2] Wu W, et al. NDM metallo- β -lactamases and their bacterial producers in health care settings. *Clin Microbiol Rev.* 2019;32(2):e00115-18. <https://doi.org/10.1128/CMR.00115-18>
- [3] Antimicrobial Resistance Surveillance Program. 2024 Annual Report. Manila, Philippines: Antimicrobial Resistance Surveillance Reference Laboratory, Research Institute for Tropical Medicine, Department of Health; 2025. ISSN 3082-589X. <https://www.arsp.com.ph/publications/>

A scanning electron micrograph (SEM) of Klebsiella pneumoniae bacteria. The bacteria are rod-shaped with a highly textured, wrinkled surface. They are arranged in a chain, with one bacterium in the foreground being more prominent than the others in the background. The background is a light gray, and there is a dark blue vertical bar on the right side of the image.

Klebsiella pneumoniae

Klebsiella pneumoniae

Klebsiella pneumoniae is a common Gram-negative bacterial pathogen that causes a wide range of infections, including pneumonia, bloodstream infections, urinary tract infections, wound infections, and neonatal sepsis. It is an important healthcare-associated pathogen, particularly in intensive care units, neonatal intensive care units, and among critically ill, elderly, immunocompromised, or device-associated patients. In the Philippines, a high prevalence of ESBL-producing strains has been reported. Carbapenem-resistant *Klebsiella pneumoniae* (CRKP) has also emerged, with resistance frequently spanning multiple antibiotic classes.

A total of 20,732 *K. pneumoniae* infections were reported in 2025. Based on island group distribution, more than half (54.46%) of the infections were from Luzon, 23.91% from Visayas and 21.64% from Mindanao. PGH (11.14%) contributed most to the number of infections followed by GMH (7.63%) and VSM (7.31%).

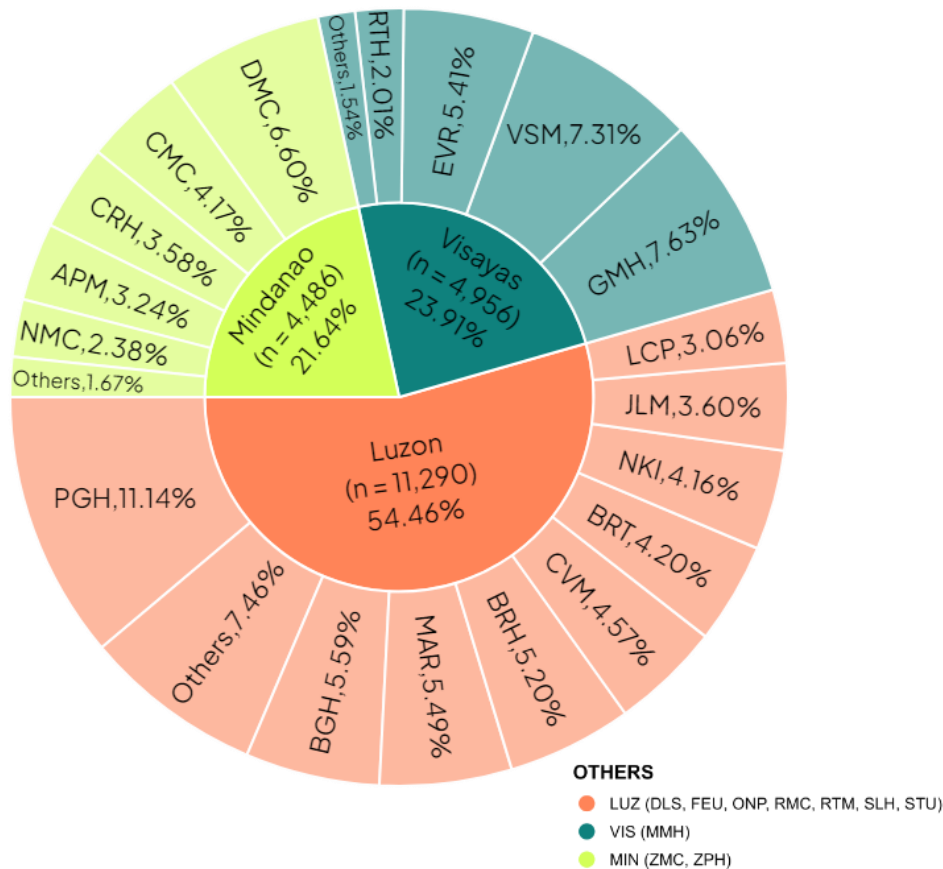


Figure 106. Distribution of *K. pneumoniae* infections, DOH-ARSP, 2025 (n=20,732)

More than half (59.63%) of the infections were from the 20-64 age group and most (53.73%) were from male patients. Many were respiratory infections (52.16%), with some urinary infections (19.68%) and wound infections (11.06%). Most (58.51%) of the infections were community acquired (Figure 107).

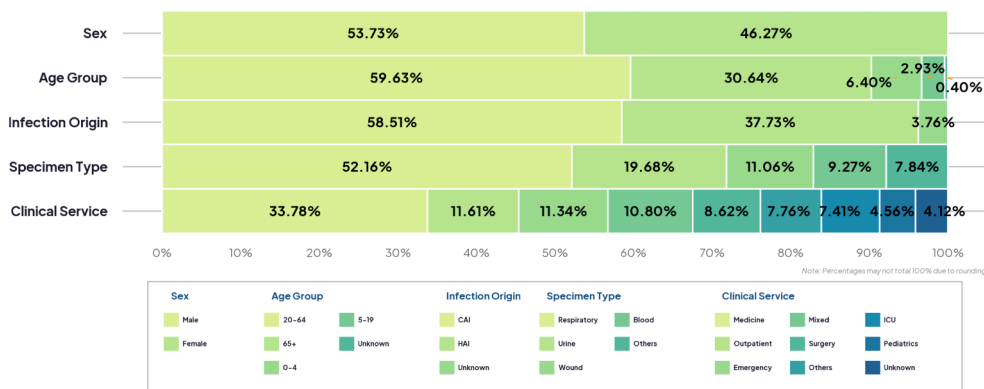


Figure 107. Patient characteristics in *K. pneumoniae* infections, DOH-ARSP, 2025 (n=17,248)

All types of Infections

Among all *K. pneumoniae* infections in 2025, resistance remained high to commonly used Gram-negative agents, including cefotaxime (46.74%), ceftriaxone (46.00%), ciprofloxacin (41.79%), co-trimoxazole (44.40%), and piperacillin/tazobactam (35.40%) (Figure 108).

Carbapenem resistance remained an important concern, with resistance to ertapenem, imipenem, and meropenem at 18.22%, 17.65%, and 17.41%, respectively. Amikacin showed comparatively lower resistance at 8.89%, while colistin resistance remained low at 4.00% (Figure 108).

For newer β -lactam/ β -lactamase inhibitor agents, resistance was 32.44% for ceftazidime/avibactam, 21.95% for ceftolozane/tazobactam, and 37.50% for imipenem/relebactam (Figure 108). These estimates should be interpreted with caution because testing for newer beta-lactam/ beta-lactamase inhibitor (BL/BLI) combinations agents is not yet widely available across surveillance sites, resulting in smaller testing numbers.

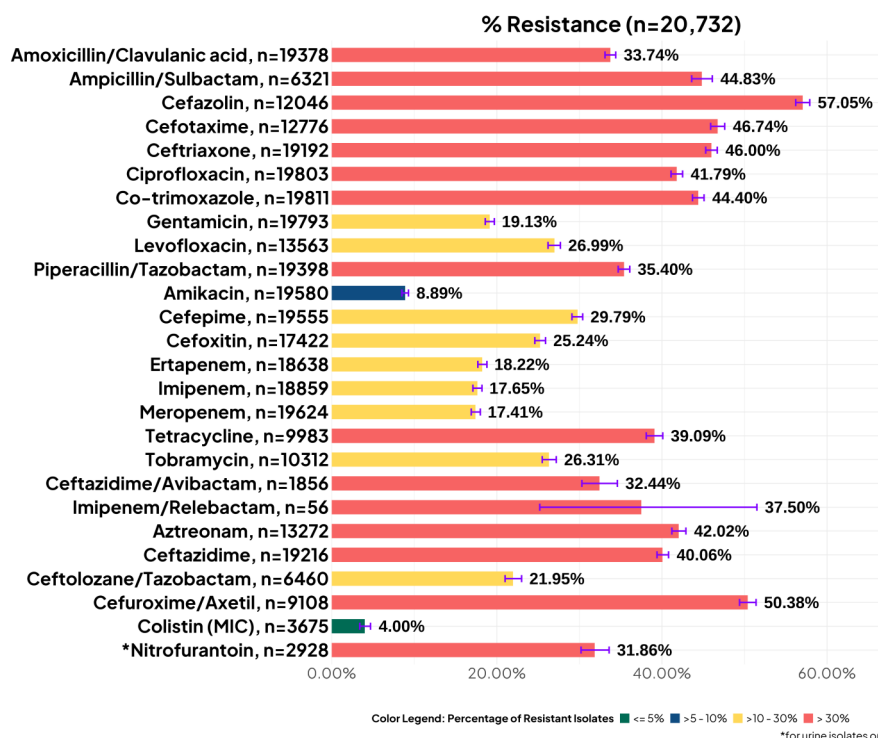
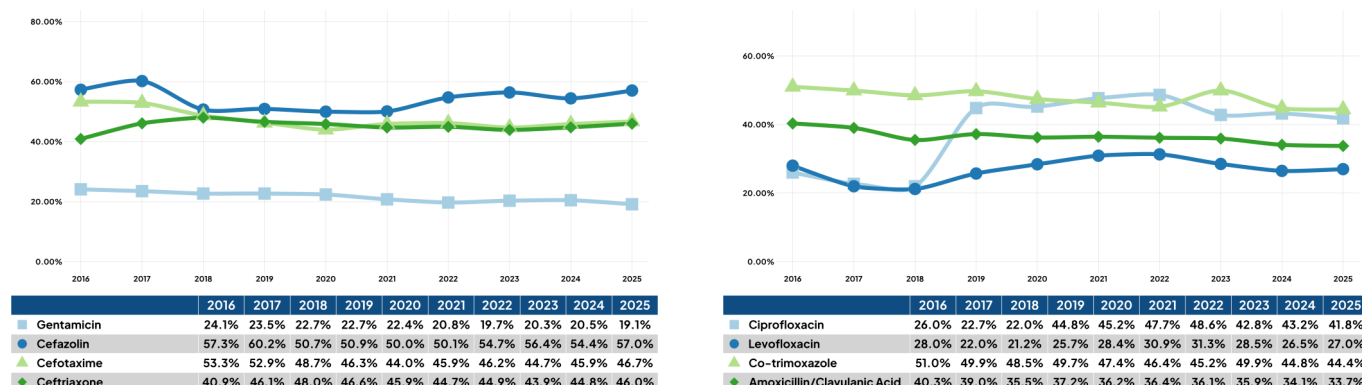


Figure 108. Proportion of all *K. pneumoniae* infections resistant to tested antibiotics, DOH-ARSP, 2025 (n=20,732)

From 2016 to 2025, *K. pneumoniae* showed persistently high resistance to several commonly used antibiotics, particularly cephalosporins, fluoroquinolones, co-trimoxazole, and ampicillin/sulbactam. Carbapenem resistance showed a gradual upward trend, with ertapenem, imipenem, and meropenem increasing from around 9%–12% in 2016 to about 17%–18% in 2025. Piperacillin/tazobactam resistance also increased over time, from 22.6% in 2016 to 35.4% in 2025, indicating reduced activity against *K. pneumoniae* infections. Among aminoglycosides, amikacin remained the most active agent, with resistance consistently below 11%, while tobramycin showed higher resistance. Colistin resistance remained low overall, although sudden increases were observed in 2021–2022, as it may partly reflect changes in testing practices and CLSI interpretive criteria for colistin (Figure 109).



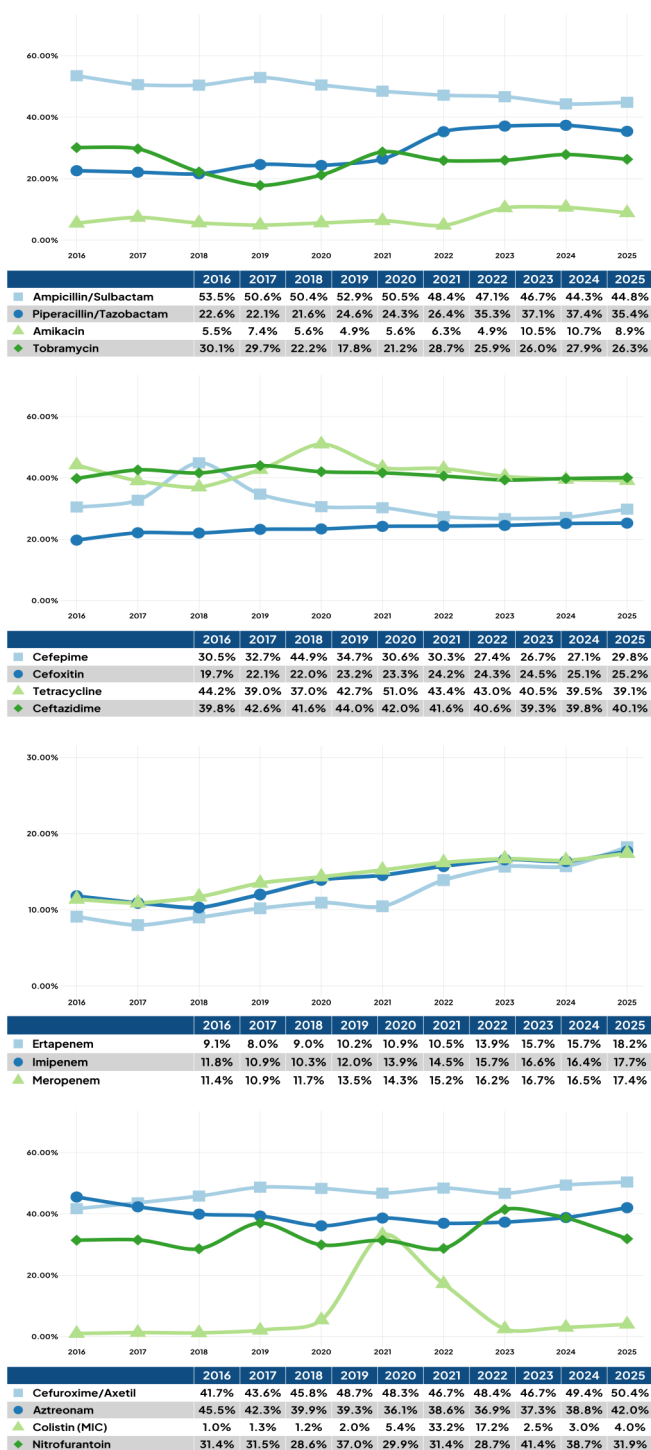


Figure 109. Yearly resistance rates of all *K. pneumoniae* infections, DOH-ARSP, 2025

Figures 110 shows the geographic distribution of resistance to imipenem, ertapenem, meropenem, and colistin among *K. pneumoniae* infections. Carbapenem resistance in *K. pneumoniae* was geographically widespread rather than confined to a single area, with higher rates observed in selected sites in Luzon, the Visayas, and Mindanao. Compared with the carbapenems, colistin resistance showed a more limited distribution, with most sites reporting resistance below 10% and only a few sites showing higher levels, particularly in selected areas of the Visayas and Mindanao. Site-level differences should be interpreted with caution, particularly for colistin, as estimates may be influenced by limited testing or small numbers of isolates tested in some sites.

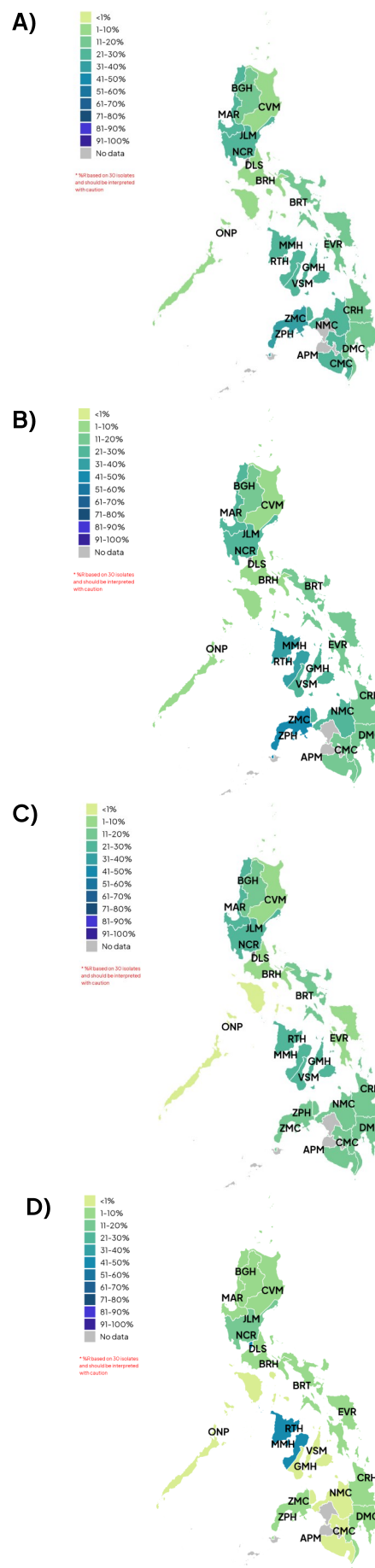


Figure 110. Resistance maps of *K. pneumoniae* infections for (A) Imipenem, (B) Ertapenem, (C) Meropenem, and (D) Colistin

Figures 111, 113, and 115 show the 2025 resistance profiles of *K. pneumoniae* bloodstream, lower respiratory tract, and urinary tract infections. Cephalosporin resistance remained consistently elevated across all three infection types. Resistance to cefotaxime and ceftriaxone was highest in bloodstream infections at 55.37% and 55.78%, followed by lower respiratory tract infections at 53.84% and 52.98%, and urinary tract infections at 50.24% and 49.44%, respectively. These findings are consistent with persistently high cephalosporin resistance observed from 2016 to 2025 (Figures 112, 114, and 116).

Carbapenem resistance showed an increasing trend over time and was highest in bloodstream and lower respiratory tract infections in 2025 (Figures 112, 114, and 115). Resistance to ertapenem, imipenem, and meropenem was approximately 27%–28% in bloodstream infections and 26%–27% in lower respiratory tract infections, compared with around 19%–20% in urinary tract infections.

For urinary tract infections, resistance to fluoroquinolones and co-trimoxazole remained elevated in 2025 despite gradual declines over time, with ciprofloxacin at 47.19%, levofloxacin at 35.06%, and co-trimoxazole at 47.33%; nitrofurantoin resistance was 33.52% (Figure 115).

Colistin resistance remained low at 3.96% in bloodstream infections and 5.71% in urinary tract infections (Figures 111 and 115). Overall, bloodstream and lower respiratory tract infections showed the highest resistance burden, particularly to carbapenems and piperacillin/tazobactam, while amikacin retained comparatively low resistance across infection types.

Bloodstream Infections

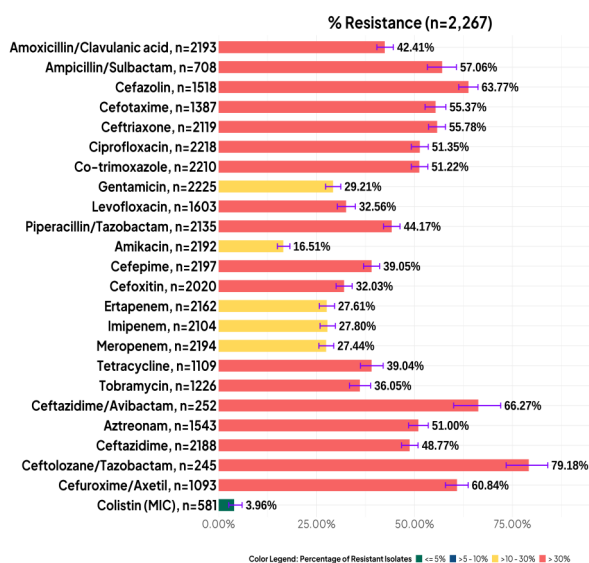
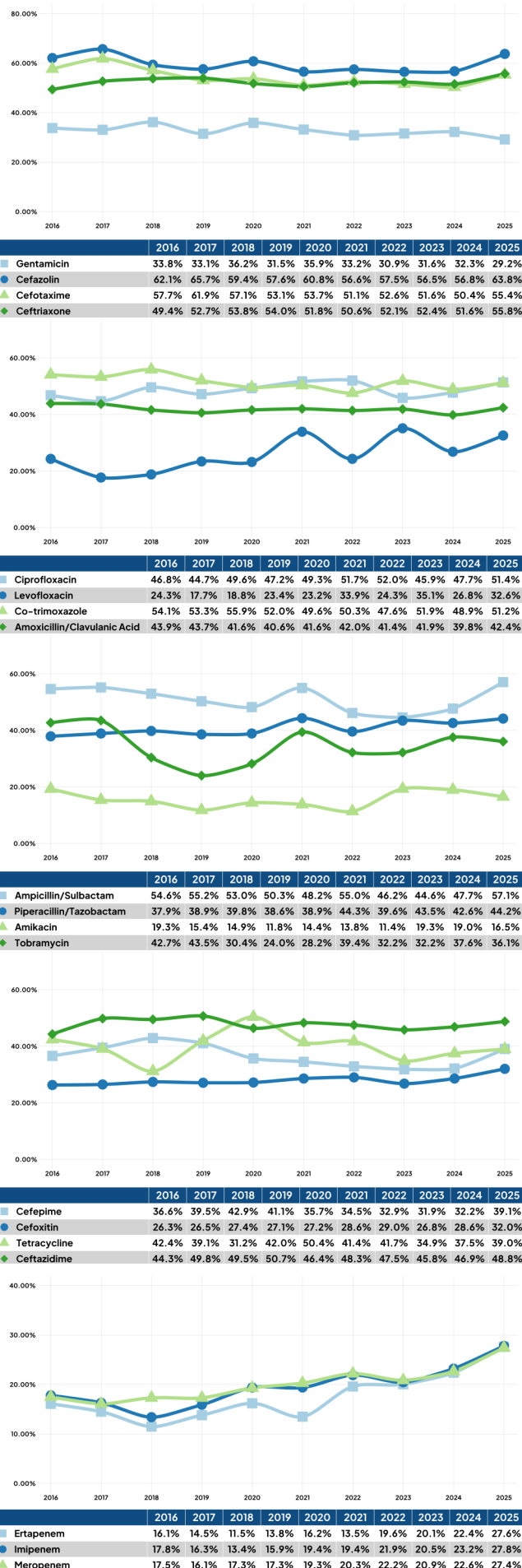


Figure 111. Proportion of *K. pneumoniae* bloodstream infections with resistance to tested antibiotics, DOH-ARSP, 2025 (n=2,267)



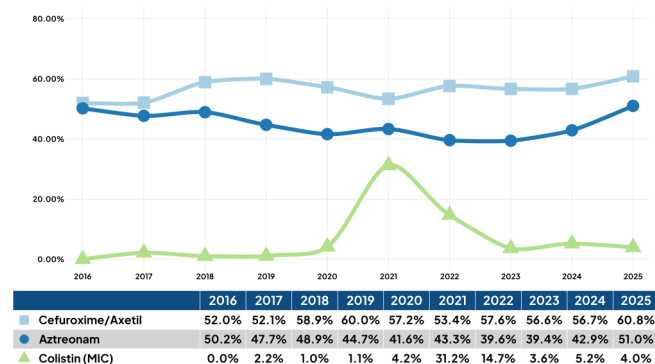
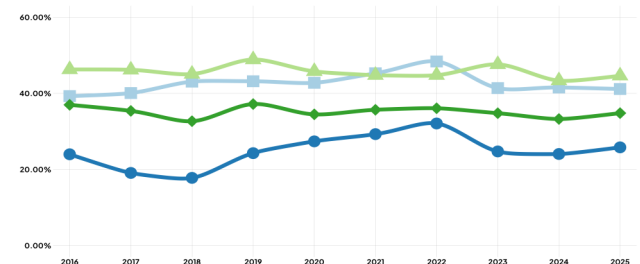
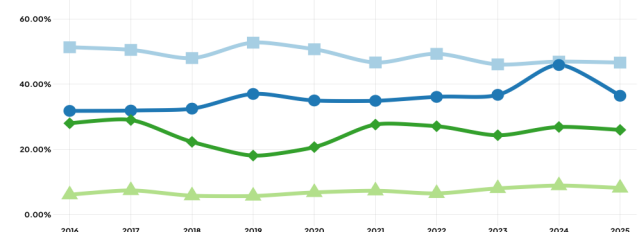


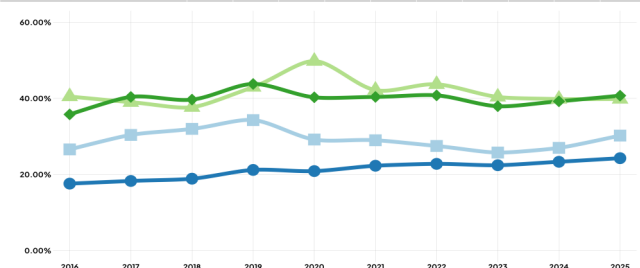
Figure 112. Yearly resistance rates of *K. pneumoniae* bloodstream infections, DOH-ARSP, 2025



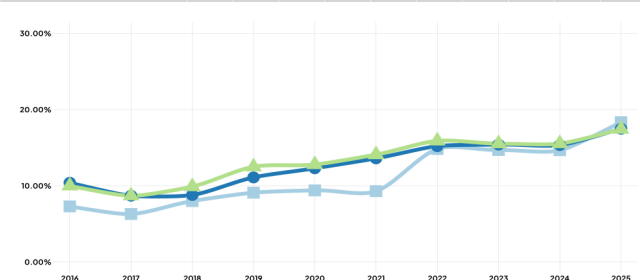
	2016	2017	2018	2019	2020	2021	2022	2023	2024	2025
Ciprofloxacin	39.3%	40.1%	43.1%	43.2%	42.8%	45.3%	48.4%	41.4%	41.6%	41.2%
Levofloxacin	24.0%	19.1%	17.8%	24.3%	27.4%	29.3%	32.1%	24.7%	24.1%	25.8%
Co-trimoxazole	46.3%	46.2%	45.1%	48.9%	45.8%	44.8%	47.6%	43.4%	44.6%	44.6%
Amoxicillin/Clavulanic Acid	37.0%	35.4%	32.7%	37.2%	34.5%	35.7%	36.1%	34.8%	33.3%	34.8%



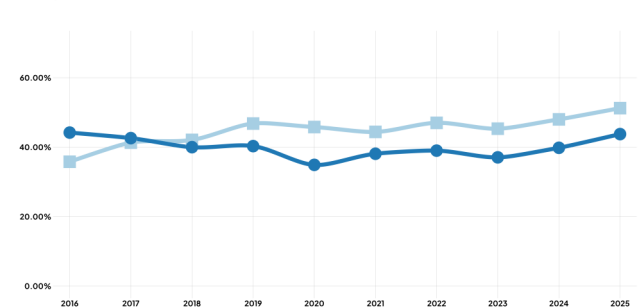
	2016	2017	2018	2019	2020	2021	2022	2023	2024	2025
Ampicillin/Sulbactam	51.3%	50.5%	48.0%	52.7%	50.7%	46.7%	49.3%	46.1%	46.9%	46.6%
Piperacillin/Tazobactam	31.8%	31.9%	32.5%	37.0%	35.0%	34.9%	36.1%	36.7%	45.9%	36.4%
Amikacin	6.1%	7.4%	5.8%	5.7%	6.8%	7.3%	6.5%	8.0%	8.9%	8.2%
Tobramycin	28.0%	29.0%	22.3%	18.1%	20.7%	27.6%	27.1%	24.3%	26.9%	26.0%



	2016	2017	2018	2019	2020	2021	2022	2023	2024	2025
Cefepime	26.6%	30.4%	32.0%	34.3%	29.2%	29.0%	27.5%	25.8%	27.0%	30.2%
Ceftazidime	17.6%	18.3%	18.9%	21.2%	20.9%	22.3%	22.8%	22.4%	23.3%	24.3%
Tobramycin	40.5%	39.0%	37.7%	43.0%	49.8%	42.2%	43.7%	40.4%	39.8%	39.8%
Meropenem	35.8%	40.4%	39.7%	43.8%	40.3%	40.4%	40.8%	37.9%	39.2%	40.7%



	2016	2017	2018	2019	2020	2021	2022	2023	2024	2025
Ertapenem	7.3%	6.3%	8.0%	9.1%	9.4%	9.3%	14.8%	14.7%	14.7%	18.4%
Imipenem	10.4%	8.7%	8.8%	11.1%	12.3%	13.6%	15.2%	15.4%	15.3%	17.5%
Meropenem	10.0%	8.7%	9.9%	12.5%	12.8%	14.1%	15.9%	15.5%	15.5%	17.4%



	2016	2017	2018	2019	2020	2021	2022	2023	2024	2025
Cefuroxime/Axetil	35.8%	41.3%	42.1%	46.8%	45.8%	44.4%	47.0%	45.3%	48.0%	51.2%
Aztreonam	44.2%	42.6%	40.0%	40.3%	34.9%	38.1%	39.0%	37.1%	39.8%	43.8%

Figure 114. Yearly resistance rates of *K. pneumoniae* respiratory infections, DOH-ARSP, 2025

Lower Respiratory Tract Infections

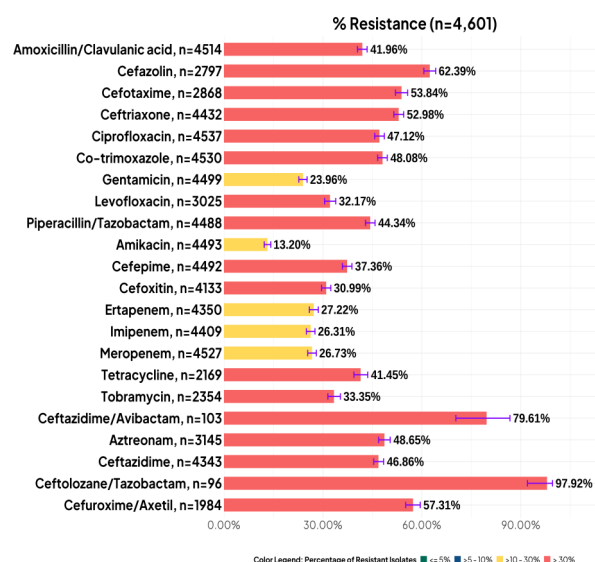
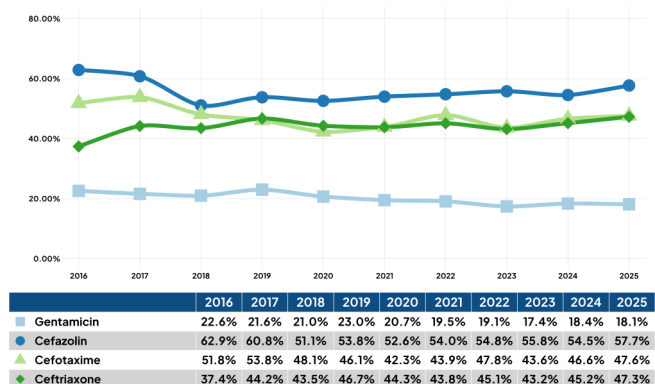


Figure 113. Proportion of *K. pneumoniae* lower respiratory tract infections with resistance to tested antibiotics DOH-ARSP, 2025 (n=4,601)



Urinary Tract Infections

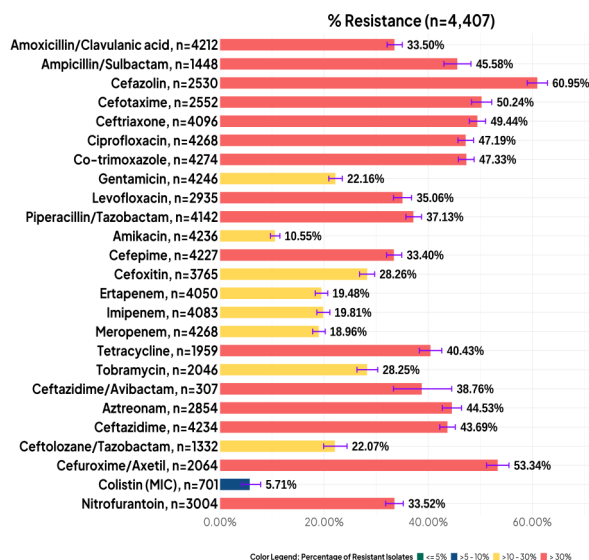
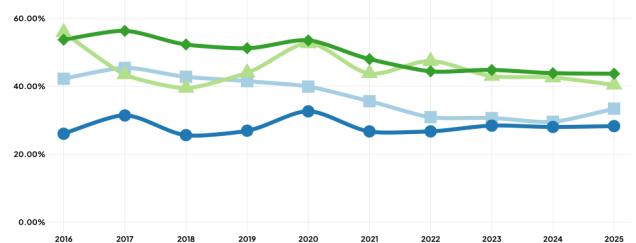
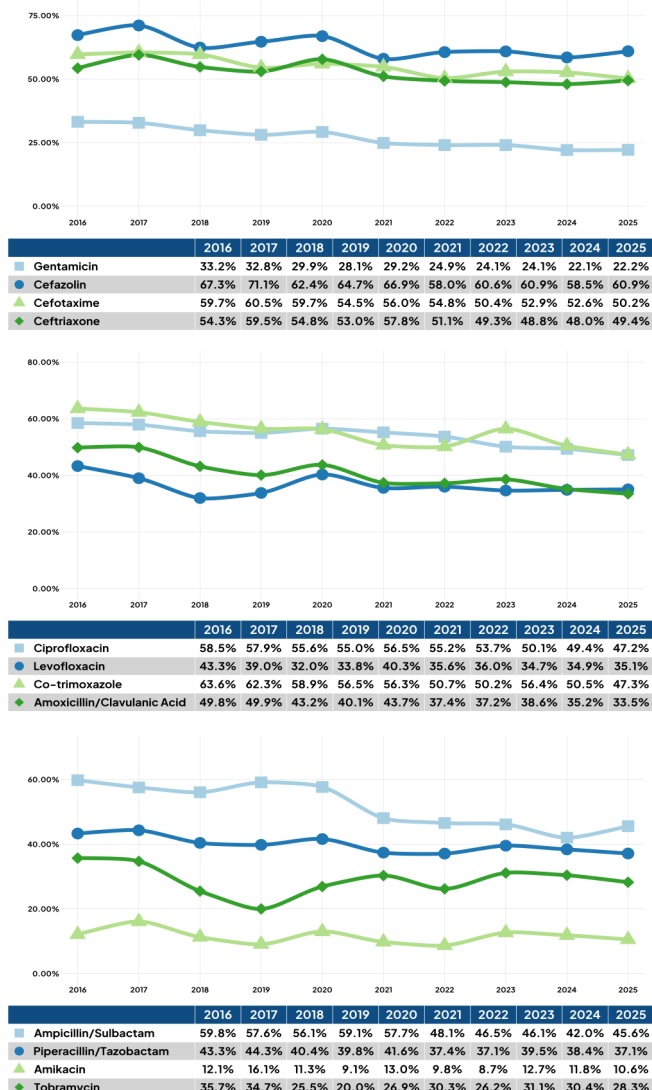
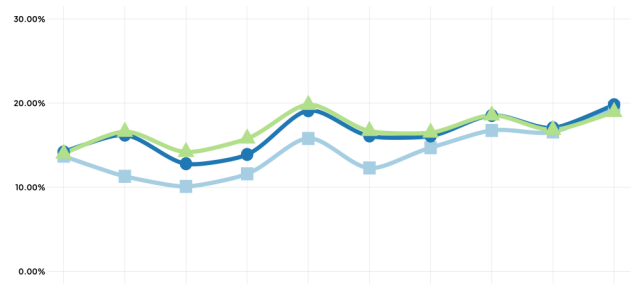


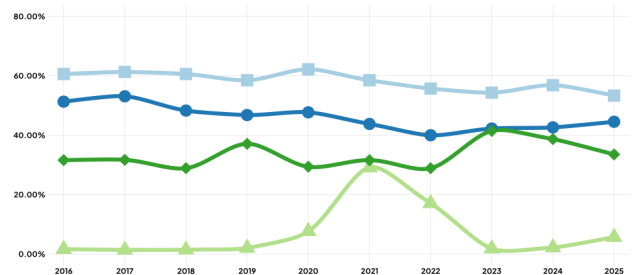
Figure 115. Proportion of *K. pneumoniae* urinary tract infections with resistance to tested antibiotics, DOH-ARSP, 2025 (n=4,407)



	2016	2017	2018	2019	2020	2021	2022	2023	2024	2025
Cefepime	42.2%	45.4%	42.8%	41.5%	39.9%	35.6%	30.9%	30.6%	29.5%	33.4%
Cefoxitin	26.0%	31.4%	25.6%	26.9%	32.6%	26.7%	26.7%	28.4%	28.0%	28.3%
Tetracycline	55.9%	43.5%	39.5%	44.0%	52.6%	43.8%	47.4%	43.0%	42.6%	40.4%
Ceftazidime	53.7%	56.3%	52.3%	51.2%	53.5%	48.0%	44.4%	44.8%	43.9%	43.7%



	2016	2017	2018	2019	2020	2021	2022	2023	2024	2025
Ertapenem	13.7%	11.3%	10.1%	11.6%	15.8%	12.3%	14.7%	16.8%	16.6%	19.5%
Imipenem	14.2%	16.2%	12.8%	13.9%	19.1%	16.1%	16.1%	18.5%	17.1%	19.8%
Meropenem	14.0%	16.6%	14.2%	15.8%	19.8%	16.7%	16.5%	18.5%	16.8%	19.0%



	2016	2017	2018	2019	2020	2021	2022	2023	2024	2025
Cefuroxime/Axetil	60.6%	61.3%	60.6%	58.5%	62.2%	58.5%	55.7%	54.3%	56.8%	53.3%
Aztreonam	51.3%	53.1%	48.3%	46.8%	47.7%	43.8%	40.0%	42.2%	42.6%	44.5%
Colistin (MIC)	1.7%	1.4%	1.4%	2.0%	7.7%	29.1%	17.1%	1.8%	2.2%	5.7%
Nitrofurantoin	31.6%	31.7%	28.9%	37.1%	29.4%	31.6%	28.9%	41.4%	38.7%	33.5%

Figure 116. Yearly resistance rates of *K. pneumoniae* urinary tract infections, DOH-ARSP, 2025

Healthcare-associated Infections

Figure 117 shows the resistance rates among presumptive healthcare-acquired *K. pneumoniae* isolates. High antibiotic resistance was observed overall, with the following rates: piperacillin-tazobactam (49.05%), ceftazidime (53.77%), cefotaxime (62.52%), levofloxacin (32.52%), ciprofloxacin (53.28%), meropenem (23.30%), imipenem (23.31%), gentamicin (26.65%), amikacin (12.07%), and aztreonam (54.95%). The newer β -lactam/ β -lactamase inhibitor combinations also showed increased resistance: ceftolozane-tazobactam (89.94%), and ceftazidime-avibactam (76.92%). However, these estimates should be interpreted with caution due to limited testing.

Carbapenem- resistant

Figure 119 shows the resistance profile of carbapenem-resistant *K. pneumoniae* infections in 2025. Resistance exceeded 88% for several beta-lactams, including cefazolin, cefotaxime, ceftriaxone, piperacillin/tazobactam, ertapenem, imipenem, meropenem, and ceftolozane-tazobactam. Colistin resistance was 6.76%. Interpretation of newer β -lactam/ β -lactamase inhibitor combinations should consider the carbapenemase gene detected, as expected activity differs by resistance mechanism; see the structured survey genomic results for carbapenem-resistant *K. pneumoniae* for further characterization (Page 91).

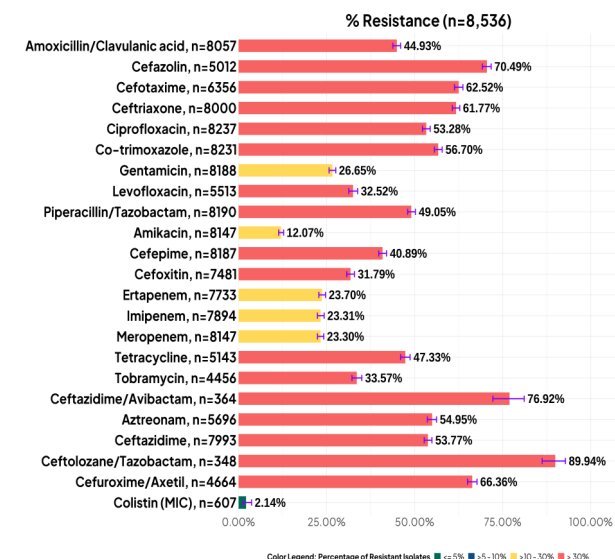


Figure 117. Proportion of presumptive healthcare-acquired *K. pneumoniae* infections with resistance to tested antibiotics, DOH-ARSP, 2025 (n=8,536)

Colistin - resistant

Figure 118 shows the resistance profile of colistin-resistant *K. pneumoniae* infections in 2025. Among colistin-resistant *K. pneumoniae*, resistance exceeded 30% for all antibiotics tested, with high co-resistance to cefazolin (86.13%), ceftriaxone (79.90%), cefotaxime (78.48%), ciprofloxacin (73.30%), and piperacillin/tazobactam (69.61%). Carbapenem resistance was also high, with ertapenem at 62.05%, imipenem at 59.20%, and meropenem at 59.33%. For further characterization, see the genomic surveillance report on colistin-resistant *K. pneumoniae* (Page 86).

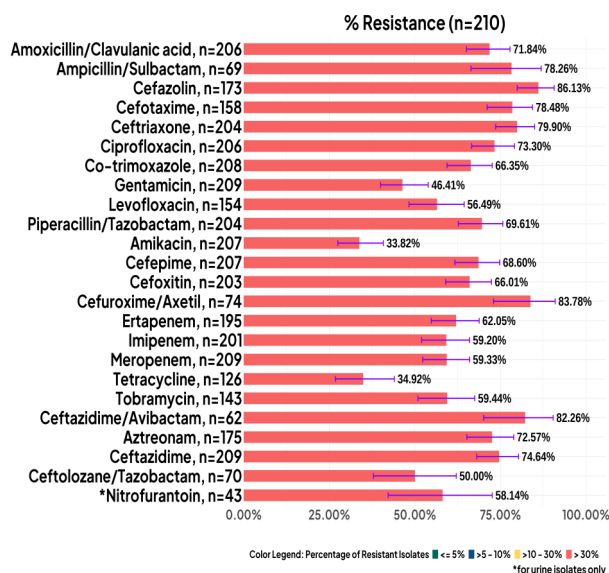


Figure 118. Proportion of all colistin-resistant *K. pneumoniae* infections, DOH-ARSP, 2025 (n=210)

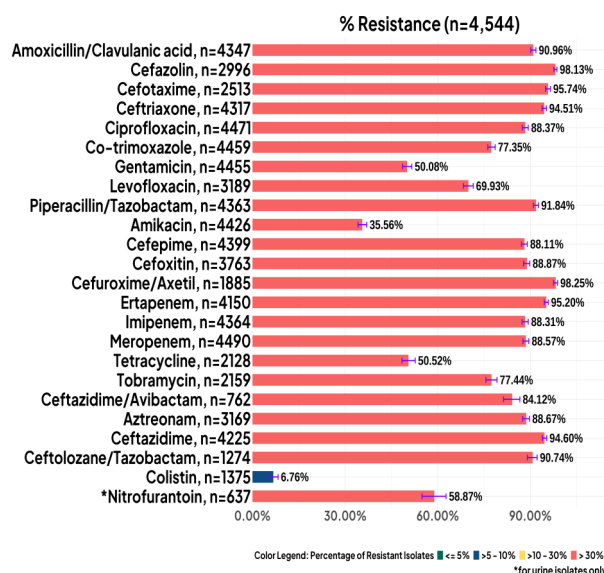


Figure 119. Proportion of all carbapenem-resistant *K. pneumoniae* infections, DOH-ARSP, 2025 (n=4,544)

From the subset of 2025 *K. pneumoniae* infections screened phenotypically for ESBL production (n=20,977), the positivity rate was at 31.41%. The yearly resistance rates of ESBL positive *K. pneumoniae* infections were observed to be fluctuating with a notable increase in rates in 2025.

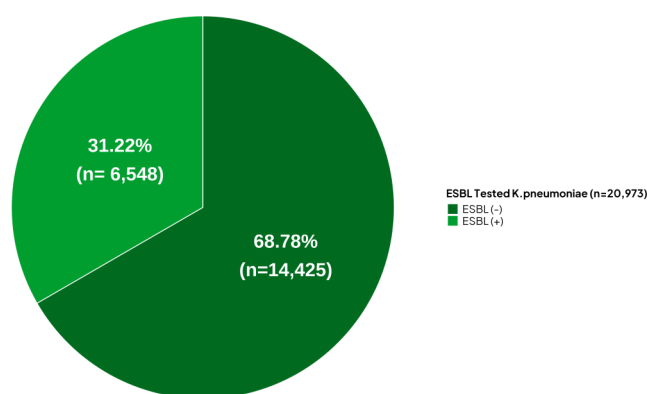


Figure 120. Percentage of ESBL positive and negative infections among tested *K. pneumoniae* infections.

ESBL production remains a critical resistance mechanism in *K. pneumoniae*, limiting the effectiveness of many β -lactam antibiotics and contributing to multidrug resistance. From 2016 to 2025, the proportion of ESBL-positive *K. pneumoniae* remained persistently high, ranging from 25.6% to 31.2%. Rates have remained above 30% since 2022, reaching 31.2% in 2025. Multi-year analysis showed that the overall changes in ESBL rates for ten years was not statistically significant.

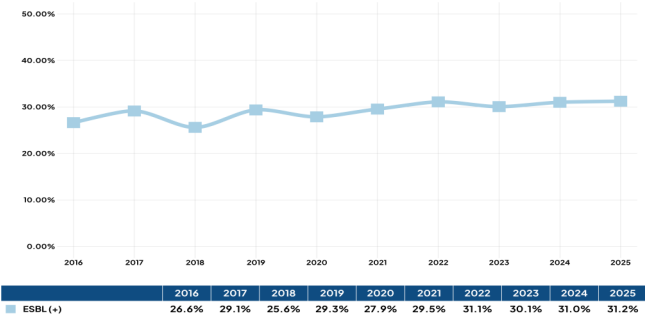


Figure 121. Yearly resistance rates of ESBL-positive *K. pneumoniae* infections, DOH-ARSP, 2016–2025

Under the NAP-AMR 2024–2028 target of a 10% reduction in carbapenem-resistant and third-generation cephalosporin-resistant *Enterobacterales*, *K. pneumoniae* bloodstream infections did not show progress in 2025 (Figure 121). Compared with the 2023 baseline, resistance increased for meropenem, imipenem, and ertapenem, reaching 27.44%, 27.80%, and 27.61%, respectively. Third-generation cephalosporin resistance also increased in 2025, with cefotaxime at 55.37% and ceftriaxone at 55.78%. Resistance rates continue to rise above the NAP-AMR target, this increasing trend necessitates adjustments to hospital infection control protocols and local antimicrobial stewardship programs.

Proportion of bloodstream infections (BSIs) due to *Klebsiella pneumoniae* resistant to carbapenems and 3rd-generation cephalosporins

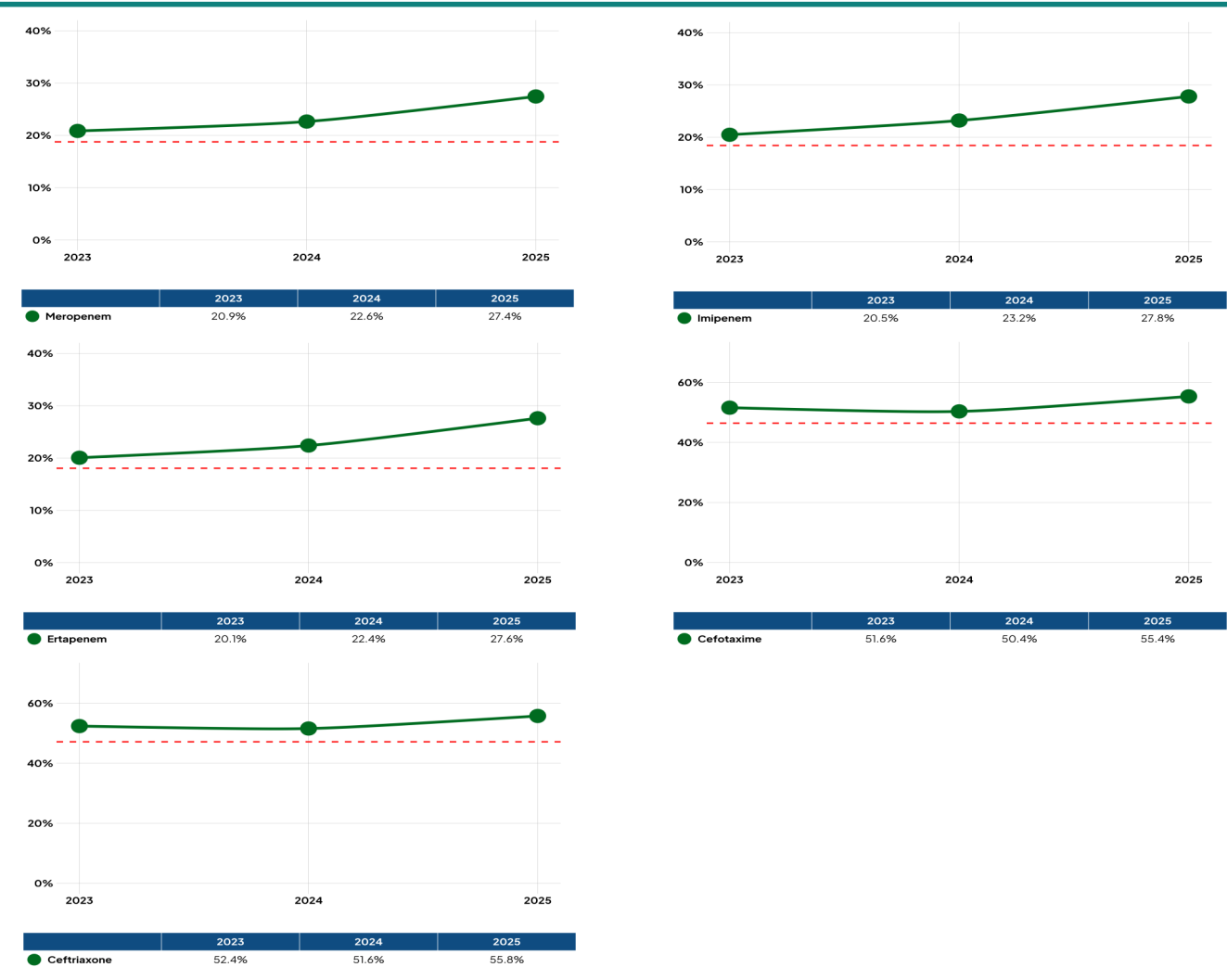


Figure 122. Yearly resistance rates of *K. pneumoniae* bloodstream infections, DOH-ARSP, 2023–2025



Isolate Characteristics and Distribution

Emerging resistance to colistin and newer β -lactam/ β -lactamase inhibitor combinations is a growing concern in *K. pneumoniae*, particularly because these agents are important treatment options for multidrug-resistant infections. **Figure 123** summarizes the characteristics of *K. pneumoniae* infections resistant to selected reserve or higher-tier agents: colistin (COL, n=18), ceftazidime-avibactam (CZA, n=230), imipenem-relebactam (IMR, n=14), and meropenem-vaborbactam (MEV, n=16).

By age group, resistance was most frequently observed among adults aged 25–64 years, particularly for COL and CZA, although resistant infections were also detected among children and older adults. Blood accounted for the largest proportion of resistant infections across the agents shown, especially for COL and CZA; all IMR- and MEV-resistant infections in the figure were from blood specimens.

Most resistant infections for COL, CZA, IMR, and MEV were classified as nosocomial.

Note: The findings for IMR and MEV should be interpreted with caution because testing for these agents has only recently started, resulting in smaller numbers of resistant infections.

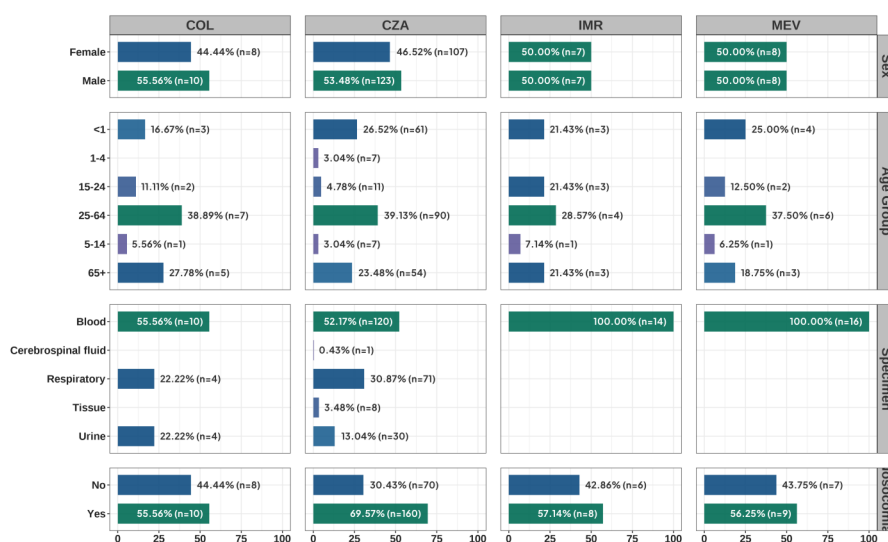


Figure 123. Patient demographics and clinical characteristics of *K. pneumoniae* isolates across key antibiotic agents

Colistin-resistant *K. pneumoniae*

In 2025, colistin-resistant *K. pneumoniae* remained genetically diverse, with multiple sequence types (STs) detected across surveillance sites. The most common STs were ST11 and ST147, each accounting for 22.2% of colistin-resistant isolates, followed by ST372 and ST437 at 16.7% each. Some STs reported among colistin-resistant *K. pneumoniae* in 2024, including ST147 and ST15, were again detected in 2025, while other STs differed between years. These year-to-year differences should be interpreted cautiously given the small number of colistin-resistant isolates.

Capsular typing also showed diversity among colistin-resistant *K. pneumoniae*. The most frequent KL types were KL15 and KL64, corresponding to ST11 and ST147, respectively. Other detected KL types included KL43, KL36, KL102, KL112, KL13, and KL35, indicating that colistin resistance was not confined to a single capsular background.

Colistin resistance mechanisms were heterogeneous. Chromosomal mutations associated with colistin resistance were detected, including *pmrB*_T157P, *mcrB*_Q30Ter, and *phoQ*_V24G, suggesting that resistance was not driven by a single mechanism. Plasmid-mediated colistin resistance was uncommon but notable. The 2023 ARSP report identified *mcr*-1-mediated colistin resistance in one *K. pneumoniae* isolate belonging to ST520. In 2025, *mcr*-1.1 was again detected in one isolate, this time in ST15 carrying NDM-5 and CTX-M-15. Although detected sporadically, the presence of *mcr* genes is important because plasmid-mediated colistin resistance has potential for horizontal transmission across strains and bacterial species.

Most colistin-resistant *K. pneumoniae* genomes in 2025 also carried carbapenemase genes, predominantly NDM-type carbapenemases, with additional detection of KPC-2 and OXA-48. Frequent co-occurrence with CTX-M-15 further indicates a multidrug-resistant background. *OmpK35* and *OmpK36* porin alterations were also detected in several genomes. Because these porins facilitate β -lactam entry into the bacterial

cell, their alteration may further reduce β -lactam susceptibility, particularly in genomes already carrying ESBL or carbapenemase genes.

Table 13. Overall distribution and molecular typing summary of colistin and BL-BLI resistant *K. pneumoniae* isolates

Overall Isolates (N=248)	Surveillance Sites	ST	K Loci	Count
Colistin Resistant (n=18)	BRT, EVR, MAR, MMH	ST11	KL15	4 (22.2%)
	CVM, DMC, GMH, STU	ST147	KL64	4 (22.2%)
	BRT	ST372	KL43	3 (16.7%)
	NKI	ST437	KL36	3 (16.7%)
	APM, BGH, JLM, ZMC	ST15, ST307, ST4023, ST6009	KL102, KL112, KL13, KL35	4 (22.2%)
Novel BLI Resistant (n=230)	APM, BGH, BRH, BRT, CMC, CRH, DMC, GMH, JLM, MMH, RTH, SLH, STU, VSM, ZMC	ST147	KL10(2), KL10(2), KL10(2), KL10(2)	40 (17.4%)
	APM, CMC, DMC, MAR, NKI	ST307	KL102	30 (13%)
	APM, CMC, CRH, DMC, MAR, ZMC	ST101	KL17	25 (10.9%)
	APM, BGH, BRT, JLM, MAR, MMH, NKI, RTH, STU, VSM	ST11	KL15	19 (8.3%)
	DMC, MAR	ST14	KL2	12 (5.2%)
	BGH, BRT, CMC, DMC, JLM, MAR, VSM	ST15	KL112(2), KL112(2), KL112(2)	10 (4.3%)
	RMC	ST515	KL62	6 (2.6%)
	MAR, NKI	ST437	KL36	5 (2.2%)
	CMC	ST101-ILV	KL17	3 (1.3%)
	MAR	ST25	KL2	3 (1.3%)
	APM, CMC, MAR	ST29	KL113(1), KL113(1)	3 (1.3%)
	MAR	ST39	KL23(1), KL23(1)	3 (1.3%)
	MAR	ST656	KL177	3 (1.3%)
	BGH, JLM	ST6851	KL10	3 (1.3%)
	APM, NMC	ST70	KL136	3 (1.3%)
	BGH, NKI	ST147-ILV	KL10(1), KL10(1)	2 (0.9%)
	BRT	ST1514	KL130	2 (0.9%)
	EVR, NKI	ST1535	KL114	2 (0.9%)
	DMC, VSM	ST17	KL147(1), KL147(1)	2 (0.9%)
	CRH, GMH	ST1887	KL121	2 (0.9%)
	DMC, GMH	ST219	KL114	2 (0.9%)
	MMH, NKI	ST231	KL51	2 (0.9%)
	MAR	ST268	KL20	2 (0.9%)
	APM, RMC	ST309	KL42	2 (0.9%)
	APM	ST327	KL39	2 (0.9%)
	GMH	ST6421-ILV	KL67	2 (0.9%)
	JLM	ST661	KL24	2 (0.9%)
	APM, BGH, BRH, CMC, CRH, CVM, DLS, DMC, EVR, GMH, JLM, MAR, MMH, NKI, RMC, RTH, SLH, STU, VSM	ST1109, ST1224-ILV, ST1229, ST1271, ST1308, ST1401, ST16, ST1715, ST1805, ST1838, ST196, ST197-ILV, ST20-ILV, ST22, ST23, ST2478, ST278, ST292, ST3132, ST334, ST337, ST353, ST372, ST3956, ST397, ST412, ST414, ST414-2LV, ST4226-2LV, ST476, ST4817-2LV, ST5616, ST5618-2LV, ST580, ST6403, ST6518, ST71, ST852	KL106(2), KL110(2), KL14(2), KL17(2), KL9(2), KL1, KL102, KL103, KL107, KL123, KL125, KL13, KL142, KL144, KL15, KL158, KL165, KL18, KL182, KL2, KL24, KL28, KL3, KL43, KL46, KL49, KL51, KL53, KL57, KL60, KL64, KL81, KL82	38 (16.5%)

Table14. MLST and Resistance Gene Profiles of 2025 colistinK. pneumoniae isolates

Site Code	Accession No	MLST	KL type	CARB	bla_esbl	mcr_gene	omp_mutations
APM	25ARS_APM0250	ST307	KL102	NDM-1	Not detected	Not detected	Not detected
BGH	25ARS_BGH0056	ST15	KL112	NDM-5	CTX-M-15	mcr-1.1	Not detected
BRT	25ARS_BRT0125	ST372	KL43	NDM-7	CTX-M-15	pmrB_T157P	Not detected
BRT	25ARS_BRT0126	ST372	KL43	NDM-7	CTX-M-15	pmrB_T157P	Not detected
BRT	25ARS_BRT0127	ST372	KL43	NDM-7	CTX-M-15	pmrB_T157P	Not detected
BRT	25ARS_BRT0150	ST11	KL15	KPC-2	CTX-M-15	Not detected	Not detected
CVM	25ARS_CVM0137	ST147	KL64	OXA-48	CTX-M-15	mcrB_Q30Ter	OmpK35:p.Arg6Ofs
DMC	25ARS_DMC0209	ST147	KL64	KPC-2;NDM-7	Not detected	Not detected	OmpK36:p.134_135insGlyAsp;OmpK35:p.Val88fs
EVR	25ARS_EVR0121	ST11	KL15	NDM-7	CTX-M-15	Not detected	Not detected
GMH	25ARS_GMH0007	ST147	KL64	NDM-7	CTX-M-15	pmrB_T157P	OmpK35:p.Ile120fs
JLM	25ARS_JLM0140	ST4023	KL13	Not detected	CTX-M-15	Not detected	Not detected
MAR	25ARS_MAR0120	ST11	KL15	KPC-2	CTX-M-15	Not detected	Not detected
MMH	25ARS_MMH0030	ST11	KL15	NDM-7	CTX-M-15	phoQ_V24G	Not detected
NKI	25ARS_NKI0004	ST437	KL36	NDM-5	CTX-M-15	Not detected	OmpK36:p.134_135insGlyAsp
NKI	25ARS_NKI0043	ST437	KL36	NDM-5	CTX-M-15	Not detected	OmpK36:p.134_135insGlyAsp
NKI	25ARS_NKI0112	ST437	KL36	Not detected	CTX-M-15	Not detected	OmpK36:p.134_135insGlyAsp
STU	25ARS_STU0072	ST147	KL64	NDM-7	CTX-M-15	Not detected	OmpK35:c.G1031del
ZMC	25ARS_ZMC0067	ST6009	KL35	NDM-1	CTX-M-15	Not detected	OmpK35:p.Ala229fs

Novel BL-BLI-resistant *K. pneumoniae*

Among novel BL-BLI-resistant *K. pneumoniae* isolates in 2025 (n=230), molecular typing showed a diverse distribution of sequence types (STs) across multiple surveillance sites. ST147 was the most frequently detected ST, accounting for 40 isolates (17.4%) and identified in 15 surveillance sites. This is notable because ST147 is a recognized global high-risk clone and has also been observed as a predominant clone among multidrug-resistant *K. pneumoniae* in the Philippines.

Other frequent sequence types included ST307 (30, 13.0%), ST101 (25, 10.9%), and ST11 (19, 8.3%), all of which are clinically important clones associated with antimicrobial resistance. The detection of these high-risk clones across multiple sites suggests wide dissemination of resistant *K. pneumoniae* lineages rather than occurrence in a single facility.

Capsular locus typing showed that KL10 was the most prominent capsular background, mainly associated with the dominant ST147 group. Other frequent KL backgrounds included KL102, KL17, KL15, KL2, and KL112, corresponding to other major resistant lineages. The presence of several KL types among novel BL-BLI-resistant isolates highlights the capsular diversity that should be considered in monitoring resistant *K. pneumoniae* lineages and potential vaccine coverage.

Carbapenemase gene analysis showed that resistance to newer BL-BLI agents was strongly associated

with NDM-type metallo- β -lactamases. The most frequent carbapenemase genes were *bla*NDM-1 (n=81), *bla*NDM-7 (n=69), and *bla*NDM-5 (n=66), while *bla*NDM-9 was detected less frequently (n=4). This predominance of NDM is clinically important because currently available BL-BLI combinations, including ceftazidime-avibactam, imipenem-relebactam, and meropenem-vaborbactam, do not reliably inhibit metallo- β -lactamases. These findings highlight the importance of carbapenemase gene identification when interpreting resistance to newer agents and considering treatment options.

Non-NDM carbapenemases were detected less frequently, including *bla*OXA-48 (n=4), *bla*KPC-2 (n=3), and *bla*OXA-232 (n=1). Eight CZA-resistant isolates had no carbapenemase gene detected, suggesting that other resistance mechanisms may be involved. The ESBL gene *bla*CTX-M-15 was widely detected (n=211), supporting a multidrug-resistant background. In addition, OmpK35 and OmpK36 porin alterations were detected among isolates resistant to imipenem-relebactam and meropenem-vaborbactam; these alterations may reduce β -lactam entry into the bacterial cell and further contribute to resistance when combined with ESBL or carbapenemase genes.

Table 15. Distribution of AMR determinants among BL/BLI-resistant *K. pneumoniae* isolates

Category	Determinant / Mutation	Frequency (n)	Associated Resistance Profile	Region	Surveillance Site
Carbapenemases	<i>bla</i> NDM-7	69	CZA, IMR, MEV	I, III, V, VI, VII, VIII, XI, XII, CAR, BARM, NCR	MAR, JLM, BRT, MMH, GMH, VSM, EVR, DMC, CMC, BGH, APM, STU, NKI
	<i>bla</i> NDM-5	66	CZA, IMR, MEV	I, III, IV-A, VII, IX, XI, XII, XIII, BARM, CAR, NCR	MAR, JLM, DLS, GMH, ZMC, DMC, CMC, CRH, APM, BGH, NKI, RMC, SLH
	<i>bla</i> NDM-1	81	CZA, IMR, MEV	I, II, III, V, VI, VII, VIII, X, XI, XII, XIII, BARM, CAR, NCR, IX	MAR, CVM, JLM, BRT, MMH, RTH, GMH, VSM, EVR, NMC, DMC, CMC, CRH, APM, BGH, NKI, RMC, SLH, STU, ZMC
	<i>bla</i> NDM-9	4	CZA, IMR, MEV	XI, BARM	DMC, APM
	<i>bla</i> OXA-48	4	CZA	II, BARM	CVM, APM
	<i>bla</i> KPC-2	3	CZA	I, V, VIII	MAR, BRT, EVR
	<i>bla</i> OXA-232	1	CZA	NCR	NKI
	-	8	CZA	BARM, IV-A, XII, XI, VII, VI	APM, BRH, CMC, DMC, GMH, RTH
ESBLs	<i>bla</i> CTX-M-15	211	CZA, IMR, MEV	All Regions	All Surveillance Sites
Porin Mutations	OmpK35:p.Trp133 fs	11	IMR, MEV	I, BARM, NCR	MAR, APM, STU
	OmpK35:p.Arg60 fs	6	IMR, MEV	II, III, IV-A, XI, NCR, V	CVM, JLM, BRH, DMC, STU, BRT
	OmpK35:c.G1031 del	12	IMR, MEV	VII, XI, NCR, VI	GMH, VSM, DMC, STU, MMH
	OmpK36:p.Ala183 fs	14	IMR, MEV	III, IV-A, VI, VII, VIII, XII, XIII, NCR	JLM, DLS, RTH, GMH, EVR, CMC, CRH, NKI
	OmpK35:p.Ala22 9fs	5	IMR, MEV	V, VII, IX, XIII	BRT, GMH, ZMC, CRH
	OmpK36:p.134_135insGlyAsp	7	IMR, MEV	I, XI, NCR	MAR, DMC, NKI

The emergence of resistance to colistin and novel beta-lactam/beta-lactamase inhibitor (BL-BLI) combinations in *K. pneumoniae* represents a significant clinical challenge, as these agents are often the last line of defense for multidrug-resistant infections. Our findings indicate that these resistant infections are predominantly nosocomial in nature and are frequently associated with bloodstream infections, particularly for ceftazidime-avibactam (CZA), imipenem-relebactam (IMR), and meropenem-vaborbactam (MEV). Resistance to colistin is characterized by high genetic heterogeneity, involving a mix of chromosomal mutations in genes such as *pmrB*, *mgtB*, and *phoQ*, and the sporadic but critical presence of the plasmid-mediated *mcr-1.1* gene, which carries the risk of horizontal transmission across bacterial species. The landscape of BL-BLI resistance is even more concerning, driven by the wide dissemination of global high-risk clones, most notably ST147, ST307, and ST101. The primary mechanism driving resistance to CZA, IMR, and MEV is the dominance of NDM-type metallo-beta-lactamases (specifically NDM-1, NDM-7, and NDM-5).

Because currently available inhibitors like avibactam, relebactam, and vaborbactam do not reliably inhibit these metallo-beta-lactamases, their prevalence severely limits effective treatment options [1]. Furthermore, the frequent co-occurrence of porin alterations in OmpK35 and OmpK36 likely exacerbates this resistance by restricting drug entry into the cell, especially when paired with a background of CTX-M-15 and carbapenemases [2]. In local contexts, previous molecular mapping has consistently highlighted that the blaNDM genotype represents the dominant carbapenemase background sustaining these clonal expansions in prominent Philippine surveillance sites [3].

In conclusion, the 2025 data underscores that resistance to reserve antibiotics in *K. pneumoniae* is established through the persistence and expansion of multiple high-risk lineages across the Philippines. The resistance profile is complex, involving a synergy between NDM-type carbapenemases and structural mutations in porins, which renders many newer therapeutic combinations ineffective. These findings highlight the urgent need for enhanced diagnostic capacity to identify specific carbapenemase genes in clinical settings, as this is essential for guiding appropriate therapy. Continued genomic surveillance is vital to monitor the potential horizontal spread of mcr genes and to track the movement of high-risk clones, ensuring that infection control strategies remain responsive to the evolving threat of multidrug-resistant *K. pneumoniae* [4].

References

[1] Hackel, M., Axelrad, J., Bradford, P., Nichols, W., & Sahm, D. (2016). Assessment of the in vitro activity of ceftazidime-avibactam against multidrug-resistant *Klebsiella* spp. collected in the INFORM global surveillance study, 2012 to 2014. *Antimicrobial Agents and Chemotherapy*, 60(8), 4629–4634. <https://doi.org/10.1128/aac.02841-15>

[2] Rodrigues, C., Desai, S., Passet, V., Gajjar, D., & Brisse, S. (2022). Genomic evolution of the globally disseminated multidrug-resistant *Klebsiella pneumoniae* clonal group 147. *Microbial Genomics*, 8(3), 000737. <https://doi.org/10.1099/mgen.0.000737>

[3] Antimicrobial Resistance Surveillance Program. 2024 Annual Report. Manila, Philippines: Antimicrobial Resistance Surveillance Reference Laboratory, Research Institute for Tropical Medicine, Department of Health; 2025. ISSN 3082-589X. <https://www.arsp.com.ph/publications/>

[4] Argimón, S., Masim, M. A. L., Gayeta, J. M., Lagrada, M. L., Macaranas, P. K. V., Cohen, V., ... & Carlos, C. C. (2020). Integrating whole-genome sequencing within the National Antimicrobial Resistance Surveillance Program in the Philippines. *Nature Communications*, 11(1), 1-12. <https://doi.org/10.1038/s41467-020-16322-5>



Structured Genomic Survey

Isolate Characteristics and Distribution

Two hundred and twenty-four (224) *Klebsiella pneumoniae* isolates were included in whole-genome sequencing (WGS) analysis from 379 phenotypically tested isolates (273 resistant, 106 susceptible) submitted across 22 ARSP surveillance sites. Of the sequenced isolates, 154 (68.75%) were phenotypically resistant and 70 (31.25%) were susceptible to carbapenems. Blood was the most frequent specimen source (175, 78.13%), followed by respiratory (35, 15.63%), urine (11, 4.9%) and tissue (3, 1.3%). . The 25–64 year age group accounted for the largest share of cases (92, 41.1%), followed by pediatric cases below 15 years of age totalled 69 (30.8%), including 52 neonates, aged 65 years and older (51, 22.8%) and 15–24 year age group (12, 5.4%). Females outnumbered males (108, 48.2% versus 116, 51.8%). Presumptive nosocomial cases were more common than community-onset cases (141, 62.9% versus 83, 37.1%).

Table 16. Patient demographics and clinical characteristics (n=224)

Specimen type distribution		Age group distribution		Sex distribution		Nosocomial (Yes/No)	
Specimen Type	Count	Age group	Count	Sex	Count	Nosocomial	Count
Blood	175	<1	52	Female	108	No	83
Respiratory	35	1-4	10	Male	116	Yes	141
Urine	11	5-14	7				
Tissue	3	15-24	12				
		25-64	92				
		65+	51				

Carbapenemase gene distribution among phenotypically resistant *K. pneumoniae* infections showed predominance of NDM-type carbapenemases across surveillance sites (Table 17). NDM-1, NDM-5, and NDM-7 were detected in multiple regions, indicating widespread circulation of NDM-associated carbapenem resistance. NDM-1 was frequent in APM, BGH, CMC, CRH, and NKI, while NDM-5 was more common in DMC, RMC, RTH, and VSM. NDM-7 was prominent in several sites, including EVR, MAR, MMH, STU, and BRH.

Non-NDM carbapenemases were less frequently detected. KPC-2 was identified in selected sites, including EVR, MAR, NKI, and SLH, while OXA-48 was detected in CVM only. Overall, the findings show that carbapenemase-mediated resistance in *K. pneumoniae* infections was largely driven by NDM-type enzymes, with site-level variation in the dominant NDM variant.

Table 17. Distribution of carbapenemase genes among carba-RK *K. pneumoniae* isolates by surveillance site and region, ARSP 2025 (n=147)

Surveillance Sites	Region	Count	NDM-5	NDM-1	NDM-7	OXA-48	KPC-2
APM	BARMM	5 (3.4%)	1 (20.0%)	4 (80.0%)	0%	0%	0%
BGH	CAR	10 (6.8%)	3 (30.0%)	5 (50.0%)	2 (20.0%)	0%	0%
BRH	IV-A	3 (2.0%)	1 (33.3%)	0%	2 (66.7%)	0%	0%
BRT	V	6 (4.1%)	2 (33.3%)	1 (16.7%)	3 (50.0%)	0%	0%
CMC	XII	9 (6.1%)	4 (44.4%)	5 (55.6%)	0%	0%	0%
CRH	XIII	12 (8.2%)	4 (33.3%)	8 (66.7%)	0%	0%	0%
CVM	II	2 (1.4%)	1 (50.0%)	0%	0%	1 (50.0%)	0%
DLS	IV-A	1 (0.7%)	0%	1 (100.0%)	0%	0%	0%
DMC	XI	12 (8.2%)	6 (50.0%)	4 (33.3%)	2 (16.7%)	0%	0%
EVR	VIII	7 (4.8%)	1 (14.3%)	0%	5 (71.4%)	0%	2 (28.6%)
GMH	VII	12 (8.2%)	5 (41.7%)	1 (8.3%)	6 (50.0%)	0%	0%
JLM	III	9 (6.1%)	3 (33.3%)	3 (33.3%)	3 (33.3%)	0%	0%
MAR	I	12 (8.2%)	1 (8.3%)	2 (16.7%)	7 (58.3%)	0%	2 (16.7%)
MMH	VI	5 (3.4%)	2 (40.0%)	0%	3 (60.0%)	0%	0%
NKI	NCR	13 (8.8%)	1 (7.7%)	6 (46.2%)	4 (30.8%)	0%	2 (15.4%)
NMC	X	1 (0.7%)	1 (100.0%)	0%	0%	0%	0%
RMC	NCR	8 (5.4%)	6 (75.0%)	2 (25.0%)	0%	0%	0%
RTH	VI	2 (1.4%)	2 (100.0%)	0%	0%	0%	0%
SLH	NCR	4 (2.7%)	2 (50.0%)	1 (25.0%)	0%	0%	1 (25.0%)
STU	NCR	5 (3.4%)	2 (40.0%)	0%	3 (60.0%)	0%	0%
VSM	VII	5 (3.4%)	3 (60.0%)	0%	2 (40.0%)	0%	0%
ZMC	IX	4 (2.7%)	2 (50.0%)	2 (50.0%)	0%	0%	0%

Table 18 shows the sequence type (ST) and serotype distribution of *K. pneumoniae* infections in 2025. The population was genetically diverse, with high-risk clones and multiple low-frequency sequence types detected across surveillance sites. ST147 was the most frequent sequence type, accounting for 35 infections (15.6%), and was more common among phenotypically resistant infections than susceptible infections.

ST101 was the second most frequent sequence type, accounting for 23 infections (10.3%). This represents a notable change from the previous year, when ST101 was detected in only one site and was not among the predominant clones. Other resistant lineages included ST11 and ST15, which were detected only among phenotypically resistant infections. ST39, previously associated with the MAR neonatal cluster, was again detected in MAR in 2025 but at lower frequency.

Serotype distribution varied by sequence type. ST147 was most frequently associated with KL64:OL2_a.1, while ST101 was mainly associated with KL17:OL2_a.1. Overall, the findings show continued genetic diversity, persistence of high-risk resistant clones, and increased detection of ST101 among *K. pneumoniae* bloodstream infections.

Table 18. Overall distribution and molecular typing summary of *K. pneumoniae*

Surveillance Sites	Region	Sequence Type (ST)	Serotypes	Overall (N=224)	Phenotypic Resistant (n=154)	Phenotypic Susceptible (n=70)
BGH, BRH, BRT, CMC, CRH, CVM, DMC, EVR, GMH, JLM, MMH, NKI, NMC, RTH, SLH, STU, VSM, ZMC	CAR, II, III, IV-A, IX, NCR, V, VI, VII, VIII, X, XI, XII, XIII	ST147	KL10:OL3 α/β (3), KL14:OL3 γ (5), KL39:OL3 γ (1), KL64:OL2 α .1(23), KL81:OL13(3)	35 (15.6%)	32 (20.8%)	3 (4.3%)
APM, BGH, CMC, CRH, DMC, MAR, SLH, STU, ZMC	BARMM, CAR, I, IX, NCR, XI, XII, XIII	ST101	KL106:OL2 α .2(2), KL17:OL2 α .1(19), KL2:OL2 α .2(2)	23 (10.3%)	18 (11.7%)	5 (7.1%)
BGH, EVR, JLM, MAR, MMH, NKI, RTH, STU, VSM	CAR, I, III, NCR, VI, VII, VIII	ST11	KL15:OL4	12 (5.4%)	12 (7.8%)	0 (0.0%)
BGH, BRT, CMC, DMC, JLM, MAR, VSM	CAR, I, III, V, VII, XI, XII	ST15	KL112:OL2 α .1(3), KL28:OL2 α .1(6), KL48:OL2 α .1(2)	11 (4.9%)	11 (7.1%)	0 (0.0%)
APM, BRT, CMC, GMH, NKI, STU, VSM	BARMM, NCR, V, VII, XII	ST307	KL102:OL2 α .2	9 (4.0%)	4 (2.6%)	5 (7.1%)
CVM, EVR, RMC	II, NCR, VIII	ST515	KL62:OL2 α .1	9 (4.0%)	6 (3.9%)	3 (4.3%)
DMC, GMH, MAR	I, VII, XI	ST14	KL16:OL2 α .1(1), KL2:OL2 α .1(4)	5 (2.2%)	3 (1.9%)	2 (2.9%)
NKI	NCR	ST437	KL36:OL4	4 (1.8%)	4 (2.6%)	0 (0.0%)
CMC	XII	ST101-1LV	KL17:OL2 α .1	3 (1.3%)	3 (1.9%)	0 (0.0%)
DMC, NMC	X, XI	ST2167	KL10:OL3 α/β	3 (1.3%)	0 (0.0%)	3 (4.3%)
CMC, EVR, STU	NCR, VIII, XII	ST260	KL1:OL2 α .2	3 (1.3%)	0 (0.0%)	3 (4.3%)
MAR	I	ST39	KL23:OL2 α .2(1), KL62:OL2 α .2(2)	3 (1.3%)	3 (1.9%)	0 (0.0%)
CMC, ZMC	IX, XII	ST45	KL20:OL3 α/β (1), KL43:OL2 α .1(2)	3 (1.3%)	0 (0.0%)	3 (4.3%)
BGH, NKI	CAR, NCR	ST147-1LV	KL10:OL3 α/β (1), KL64:OL2 α .1(1)	2 (0.9%)	2 (1.3%)	0 (0.0%)
BRT	V	ST1514	KL130:OL2 α .2	2 (0.9%)	0 (0.0%)	2 (2.9%)
CRH, GMH	VII, XIII	ST1887	KL121:OL5	2 (0.9%)	2 (1.3%)	0 (0.0%)
DMC, GMH	VII, XI	ST219	KL114:OL2 α .1	2 (0.9%)	2 (1.3%)	0 (0.0%)
CVM, JLM	II, III	ST23	KL1:OL2 α .2	2 (0.9%)	0 (0.0%)	2 (2.9%)
MAR, MMH	I, VI	ST268	KL20:OL2 α .1	2 (0.9%)	1 (0.6%)	1 (1.4%)
EVR, MMH	VI, VIII	ST278	KL136:OL2 α .2(1), KL15:OL4(1)	2 (0.9%)	1 (0.6%)	1 (1.4%)
BRH, CMC	IV-A, XII	ST29	KL19:OL2 α .2	2 (0.9%)	1 (0.6%)	1 (1.4%)
APM, RMC	BARMM, NCR	ST309	KL42:OL4	2 (0.9%)	2 (1.3%)	0 (0.0%)
CVM	II	ST35	KL16:OL2 α .1(1), KL62:OL2 α .1(1)	2 (0.9%)	0 (0.0%)	2 (2.9%)
ZMC	IX	ST6009	KL35:OL5	2 (0.9%)	2 (1.3%)	0 (0.0%)
GMH	VII	ST6421-1LV	KL67:OL2 α .2	2 (0.9%)	2 (1.3%)	0 (0.0%)
MAR	I	ST656	KL177:OL13	2 (0.9%)	2 (1.3%)	0 (0.0%)
JLM	III	ST661	KL24:OL2 α .1	2 (0.9%)	2 (1.3%)	0 (0.0%)
BGH, JLM	CAR, III	ST6851	KL10:OL3 α/β	2 (0.9%)	2 (1.3%)	0 (0.0%)
BGH, EVR	CAR, VIII	ST86	KL2:OL2 α .1	2 (0.9%)	0 (0.0%)	2 (2.9%)
Widespread Across Sites		Minor STs [*]		69 (30.8%)	40 (26.0%)	29 (41.4%)
		Sporadic Clones [**]	Sporadic Minor Serotypes	37 (16.5%)	0 (0.0%)	37 (52.9%)

[*] Minor STs: These are low-frequency strains found in the population, but a significant portion of them still express drug resistance.

Includes ST1224-1LV, ST1271, ST13, ST1308, ST1393, ST1401, ST1424, ST1535, ST16, ST17, ST1715, ST1731, ST1838, ST186, ST193, ST196, ST197-1LV, ST20, ST20-1LV, ST200, ST2039, ST2050, ST22, ST231, ST2385, ST2449, ST2543, ST2607, ST2793, ST292, ST3132, ST3140, ST33, ST334, ST337, ST353, ST356-3LV, ST3565, ST36, ST37, ST372, ST394, ST3956, ST397, ST412, ST414, ST414-2LV, ST420, ST4226-2LV, ST43, ST432, ST476, ST4813, ST4817-2LV, ST484-1LV, ST4845, ST495, ST502, ST5092, ST5239, ST5616, ST5618-2LV, ST60, ST6403, ST65, ST6518, ST70, ST71, and ST883.

[**] Sporadic Clones: These are "true singletons" or unique strains that popped up exactly once in the entire collection and are completely, 100% phenotypically susceptible (0% resistant).

Includes ST13, ST1393, ST1424, ST1514, ST1731, ST186, ST193, ST20, ST200, ST2039, ST2050, ST2385, ST2449, ST2543, ST2607, ST268, ST278, ST29, ST3140, ST33, ST356-3LV, ST3565, ST36, ST37, ST394, ST3956, ST420, ST43, ST432, ST4813, ST495, ST502, ST5239, ST60, ST65, ST71, and ST883.

Phylogenetic Analysis

Figure 124 shows the phylogenetic relatedness of ST147 *K. pneumoniae* genomes from the 2025 structured survey. Given its frequency and distribution across multiple surveillance sites, ST147 was further assessed through phylogenetic analysis to examine clustering and relatedness within this high-risk lineage. The tree is shown with accompanying metadata on site, region, age group, specimen type, phenotypic carbapenem resistance, serotype, and carbapenemase gene.

Phylogenetic Distribution of ST147 *K. pneumoniae* in 2025

The tree shows several small clusters of closely related ST147 genomes. Some clusters contained genomes from the same or nearby surveillance sites, suggesting possible local relatedness or repeated detection of similar ST147 sublineages within specific sites. For example, closely related genomes were observed among selected GMH and STU genomes, while other branches included genomes from different sites and regions, indicating broader dissemination of ST147 across the surveillance network.

Despite belonging to the same sequence type, the ST147 genomes were not confined to a single tight cluster. Instead, the tree showed multiple branches with different site, serotype, and carbapenemase gene profiles. KL64_{a.1} was the most frequent serotype background, but other KL/OL profiles were also present, indicating diversity within the ST147 lineage. The presence of different carbapenemase genes across related and separate branches suggests that resistance may be maintained through both clonal spread of resistant ST147 sublineages and acquisition of resistance genes in different genetic backgrounds.

Overall, the phylogenetic pattern indicates that ST147 *K. pneumoniae* was widely distributed and genetically diverse, with evidence of localized clustering in some sites and broader relatedness across regions.

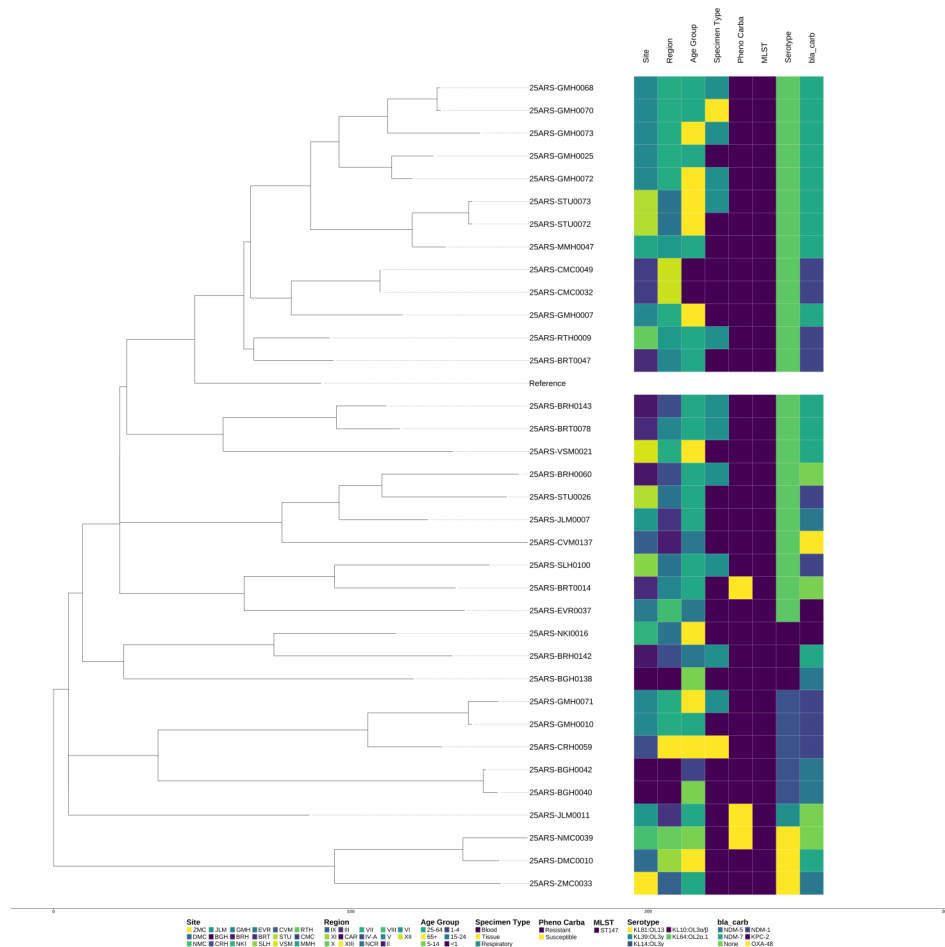


Figure 124. Phylogenetic tree of the carbapenem resistant *K. pneumoniae* isolates

Figure 125 shows the two-year comparison of carbapenemase genes detected among *K. pneumoniae* from ARSP surveillance sites. NDM-1 was detected across sites in Luzon, the Visayas, and Mindanao, with higher proportions observed in selected sites in the Visayas and Mindanao, including GMH, VSM, and CRH, and continued detection in several Luzon sites.

NDM-5 also showed geographic spread but with a different distribution pattern. Higher proportions were observed in selected northern Luzon sites, including BGH and MAR, while lower-level detection was seen

in other sites in Luzon and the Visayas. NDM-7 was detected in fewer areas than NDM-1 and NDM-5, but remained present in selected sites, including DMC in Mindanao and several sites in Luzon and the Visayas.

KPC-2 showed the most limited distribution among the carbapenemase genes shown. Detection was concentrated in selected sites, particularly in northern Luzon, including BGH and MAR, with additional detection in EVR. Overall, the maps indicate that carbapenemase-mediated resistance in *K. pneumoniae* was largely driven by geographically widespread NDM variants, while KPC-2 remained more localized. Site-level differences should be interpreted with caution, particularly for sites with fewer than 30 isolates tested.

OXA-48 was detected in only one surveillance site in the heat map, with detection concentrated in CVM in northern Luzon (Figure 126). Unlike the NDM variants, which were distributed across several sites and regions, OXA-48 showed a localized pattern in 2025. This finding should be interpreted with caution, particularly if based on a small number of tested isolates.

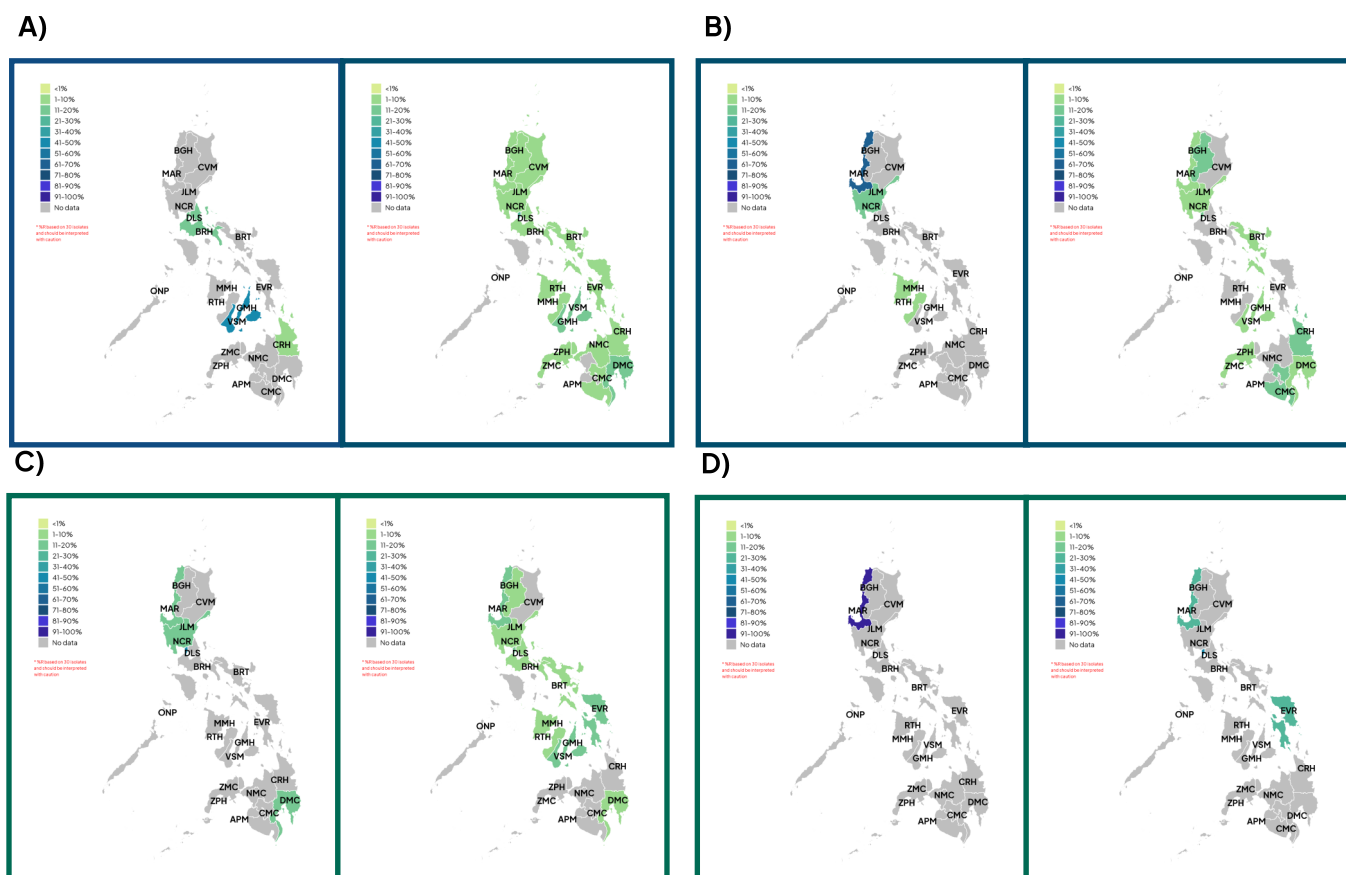


Figure 125. Two-year comparison maps of Carbapenemase genes detected among *K. pneumoniae* isolates from ARSP surveillance sites for (A) NDM-1, (B) NDM-5, (C) NDM-7, and (D) KPC-2

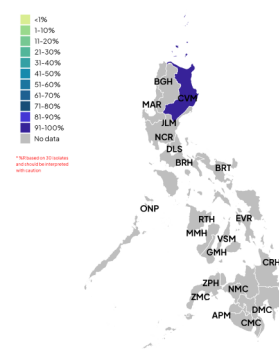


Figure 126. Carbapenemase genes maps detected among *K. pneumoniae* isolates from ARSP surveillance sites for OXA-48

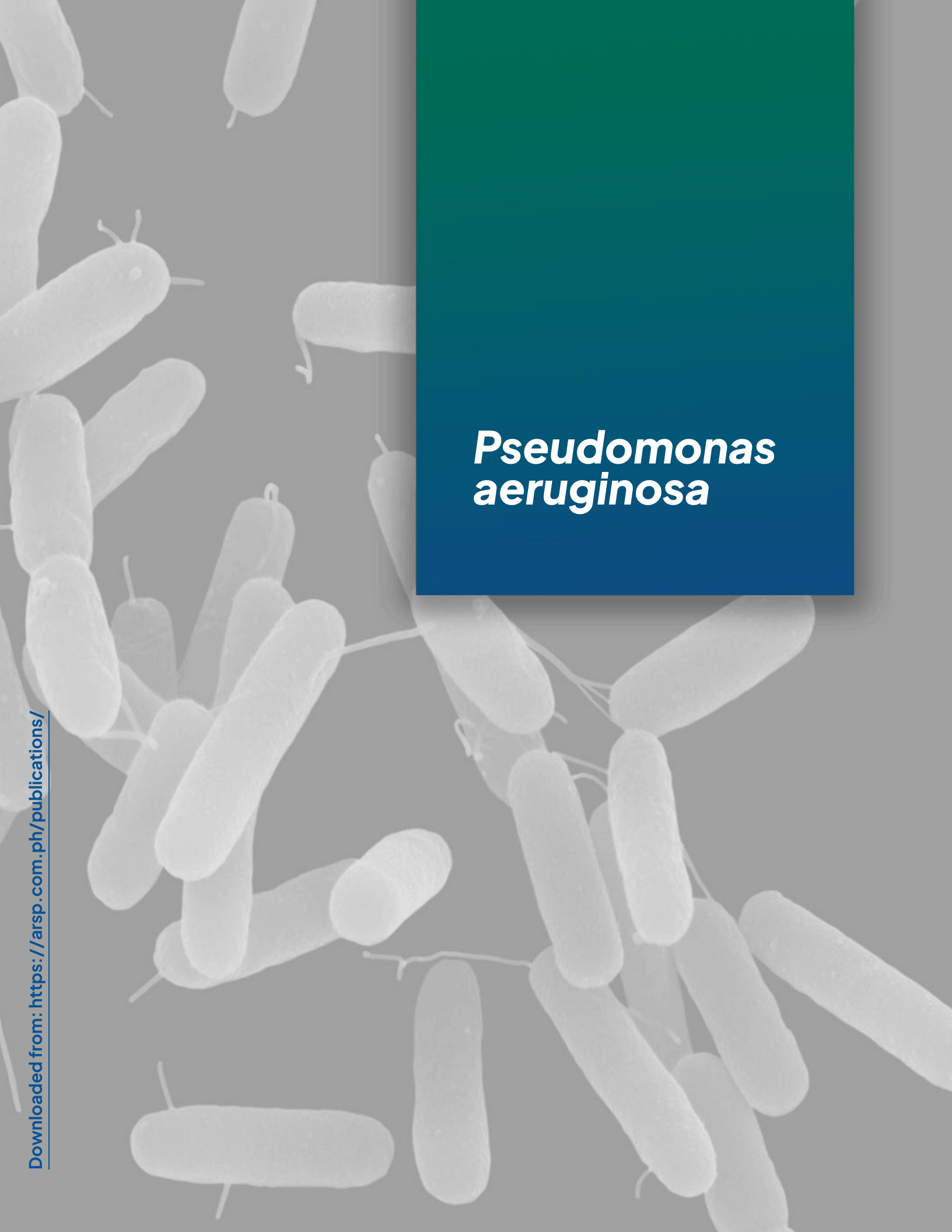
Discussion

The 2025 structured genomic survey of *Klebsiella pneumoniae* reveals a high burden of carbapenem resistance, with 68.75% of the sequenced isolates exhibiting phenotypic resistance. The clinical impact is particularly pronounced in bloodstream infections, which accounted for 78.13% of the cases, and the majority (62.9%) are associated with presumptive nosocomial transmission. A concerning demographic trend is the high prevalence among pediatric patients, particularly neonates, who represent a significant portion of the cases. This severe burden in vulnerable pediatric populations mirrors previous findings from the National Antimicrobial Resistance Surveillance Program (ARSP), where genomic sequencing unmasked large, previously undetected *K. pneumoniae* clonal outbreaks tightly concentrated within Philippine neonatal intensive care units [1]. Mechanistically, resistance is largely driven by the widespread dissemination of NDM-type carbapenemases. Variants such as NDM-1, NDM-5, and NDM-7 were detected across Luzon, the Visayas, and Mindanao, indicating a nationwide circulation of these genes. This dominance of the blaNDM genotype across multiple geographical regions aligns with recent local molecular assays confirming it as the primary driver of carbapenem resistance within prominent Philippine clinical sectors [2]. In contrast, non-NDM carbapenemases like KPC-2 and OXA-48 remain relatively localized, with KPC-2 concentrated in northern Luzon and EVR, and OXA-48 identified only at a single site (CVM).

The epidemiological landscape is further characterized by the dominance of high-risk clones. ST147 remains the most frequent sequence type (15.6%), showing significant diversity and evidence of both localized clustering and broader regional dissemination. This prominent role of ST147 is consistent with international surveillance data from platforms like Pathogenwatch, which highlighted that the molecular epidemiology of *K. pneumoniae* in the Philippines is uniquely dominated by this specific epidemic, drug-resistant lineage relative to other global contexts [3]. A notable shift in this survey is the rise of ST101, which increased from a single-site detection in previous years to become the second most frequent clone (10.3%). Other lineages, such as ST11 and ST15, were exclusively associated with resistant phenotypes, further emphasizing their role in maintaining the carbapenem-resistant population through a combination of clonal spread and the horizontal acquisition of resistance genes [4]. These findings highlight the critical need for continued genomic surveillance to monitor shifting clonal dynamics and to guide targeted infection control measures, especially in nosocomial settings and neonatal care units where the impact of these resistant pathogens is most severe.

References

- [1] Carlos, C. C., Masim, M. A. L., Lagrada, M. L., Gayeta, J. M., Macaranas, P. K. V., Sia, S. B., ... & Bernal, J. F. (2021). Genome sequencing identifies previously unrecognized *Klebsiella pneumoniae* outbreaks in neonatal intensive care units in the Philippines. *Clinical Infectious Diseases*, 73(Supplement_4), S316–S324. <https://doi.org/10.1093/cid/ciab776>
- [2] Antimicrobial Resistance Surveillance Program. 2024 Annual Report. Manila, Philippines: Antimicrobial Resistance Surveillance Reference Laboratory, Research Institute for Tropical Medicine, Department of Health; 2025. ISSN 3082-589X. <https://www.arsp.com.ph/publications/>
- [3] Argimón, S., David, S., Underwood, A., Abrudan, M., Wheeler, N. E., Kekre, M., ... & Aanensen, D. M. (2021). Rapid genomic characterization and global surveillance of *Klebsiella* using Pathogenwatch. *Clinical Infectious Diseases*, 73(Supplement_4), S325–S335. <https://doi.org/10.1093/cid/ciab784>
- [4] Argimón, S., Masim, M. A. L., Gayeta, J. M., Lagrada, M. L., Macaranas, P. K. V., Cohen, V., ... & Carlos, C. C. (2020). Integrating whole-genome sequencing within the National Antimicrobial Resistance Surveillance Program in the Philippines. *Nature Communications*, 11(1), 1–12. <https://doi.org/10.1038/s41467-020-16322-5>

A scanning electron micrograph (SEM) of numerous Pseudomonas aeruginosa bacteria. The bacteria are rod-shaped, with some showing flagella (fine hair-like structures) at one end. They are distributed across the frame, with a higher density in the lower half. A dark blue rectangular box is positioned in the upper right corner, containing the text 'Pseudomonas aeruginosa' in white.

Pseudomonas aeruginosa

Pseudomonas aeruginosa

Pseudomonas aeruginosa is a gram-negative, oxidase-positive rod known for its non-fermenting metabolism. Its pathogenesis is driven by biofilm formation and exotoxins, causing severe opportunistic infections in hospitalized patients. In the Philippines, it remains a top cause of healthcare-associated pneumonia and bacteremia. Resistance to carbapenems and ceftazidime persists at roughly 13–17%, complicating treatment in critical care. Consequently, the ARSP prioritizes strict infection control and stewardship to manage its high environmental resilience.

A total of **11,330** *Pseudomonas aeruginosa* infections were reported for 2025. Based on island group distribution, surveillance sites from Luzon contributed more than half (57.04%) of the infections, 22.91% from Visayas, and 20.04% from Mindanao. Large contributors of *P. aeruginosa* data were PGH (10.49%), DMC (8.12%) and VSM (8.08%).

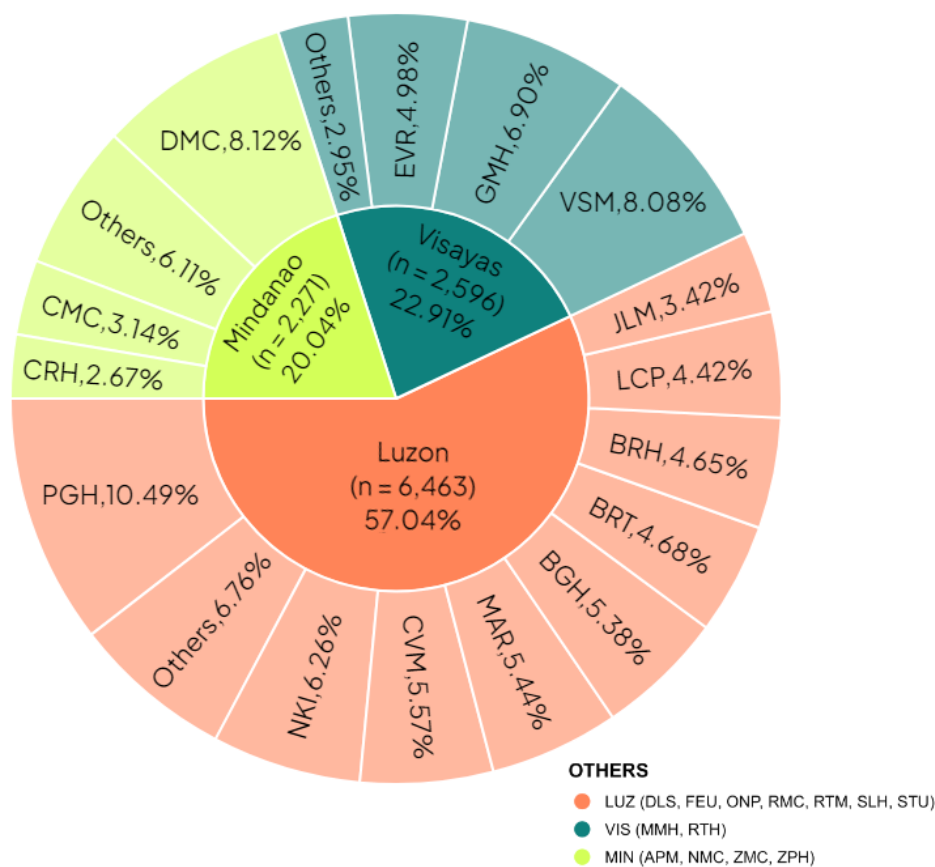


Figure 127. Distribution of *P. aeruginosa* infections, DOH-ARSP, 2025 (n=11,330)

Most (61.92%) *P. aeruginosa* infections were obtained from patients aged 20–64 years old and from male patients (57.03%). A large proportion of the infections were from respiratory (47.66%) and wound (17.05%) specimens. Additionally, more than half (57.69%) were community-acquired.

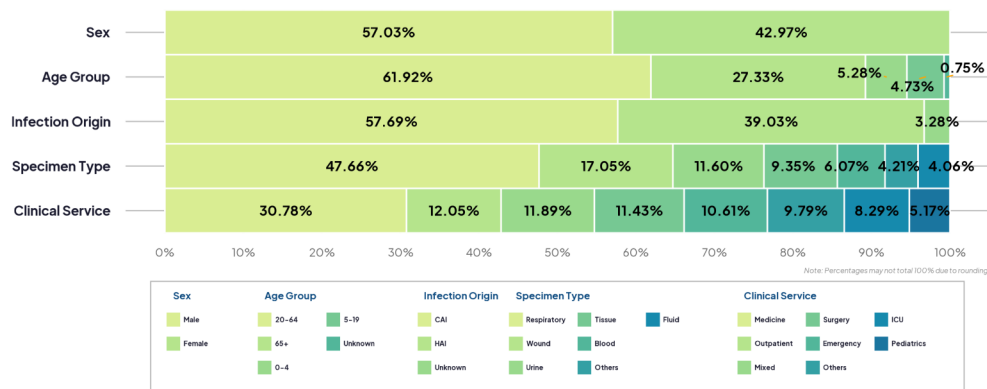


Figure 128. Patient characteristics in *P. aeruginosa* infections, DOH-ARSP, 2025 (n=11,330)

All types of Infections

Figure 129 shows the cumulative resistance rates of all *P. aeruginosa* infections in 2025. The resistance to cephalosporins are as follows: ceftazidime was at 15.80% and cefepime at 11.94%. Aztreonam resistance was at 11.89% and piperacillin-tazobactam at 19.73%. For fluoroquinolone resistance, levofloxacin and ciprofloxacin resistance were at 11.30% and 12.05% respectively. Due to the small number of specimens tested, the high resistance rates observed for novel beta-lactam/beta-lactamase inhibitor combinations should be interpreted with caution. This caveat applies to all *Pseudomonas aeruginosa* resistance data in this dataset imipenem-relebactam at 41.51%, ceftolozane-tazobactam 6.86% and ceftazidime-avibactam at 15.71%.

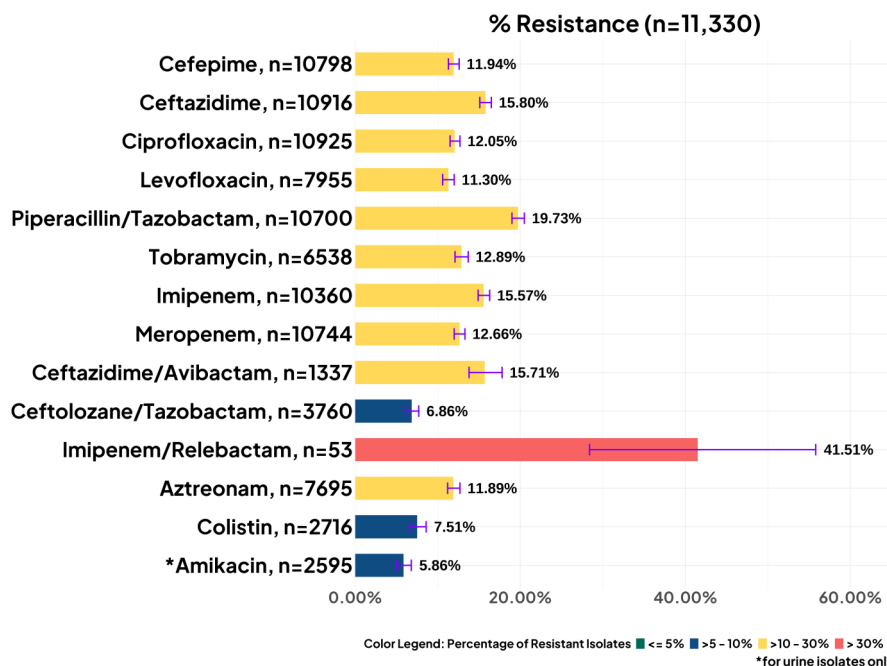


Figure 129. Proportion of all *P. aeruginosa* infections resistant to tested antibiotics, DOH-ARSP, 2025 (n=11,330)

From 2016–2025, *Pseudomonas aeruginosa* infection showed significantly decreasing resistance to cefepime ($p=0.0000$), ciprofloxacin ($p=0.0000$), levofloxacin ($p=0.0000$), and meropenem ($p=0.0000$). Colistin resistance increased significantly ($p=0.0000$), while piperacillin/tazobactam showed a non-significant upward trend ($p=0.0000$). Resistance patterns for ceftazidime, tobramycin, imipenem, and amikacin showed fluctuating resistance rates.

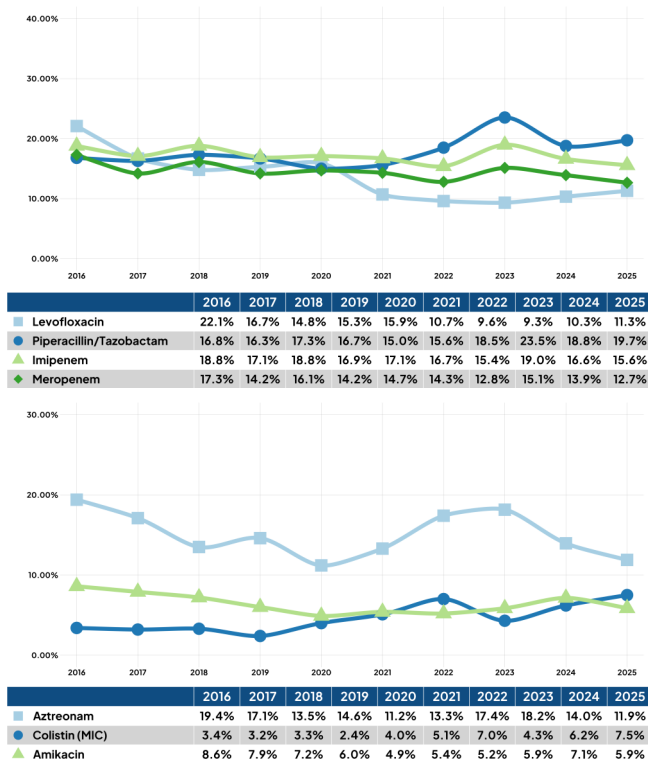
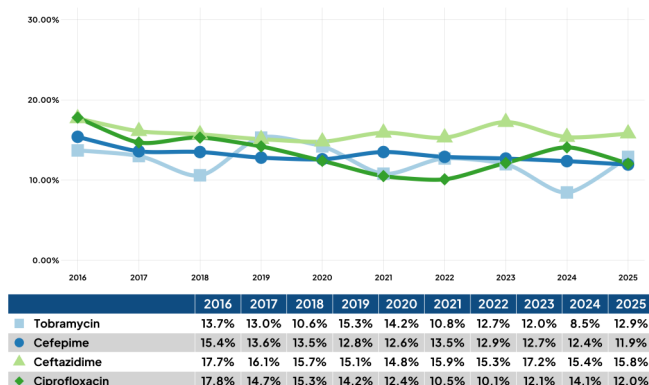


Figure 130. Yearly resistance rates of all *P. aeruginosa* infections, DOH-ARSP, 2025

Figure 131 shows the geographic distribution of carbapenem-resistant *P. aeruginosa* infections across sentinel sites. Imipenem and meropenem resistance were less than 30% in most of the sentinel sites.

The geographic distribution of carbapenem resistance among *Pseudomonas aeruginosa* infection isolates showed generally low to moderate imipenem and meropenem resistance rates across most Philippine sentinel sites, particularly in Luzon and the Visayas, where resistance commonly ranged from 11–30%. Higher resistance levels were concentrated in selected Mindanao sites, especially ZPH and ZMC, with GMH and VSM also showing elevated rates. Overall, meropenem resistance closely mirrored imipenem resistance, indicating localized carbapenem resistance hotspots in parts of Mindanao despite relatively preserved susceptibility nationwide.

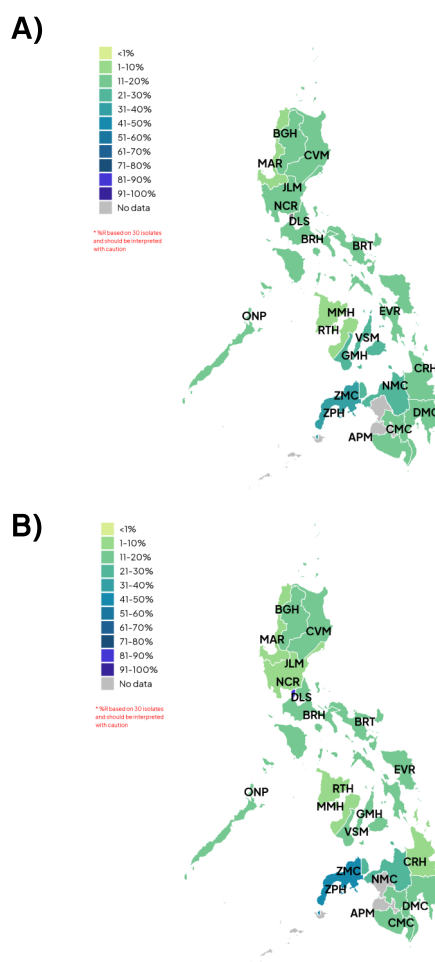


Figure 131. Resistance maps of *P. aeruginosa* infections for (A) Imipenem, and (B) Meropenem

Figure 132 shows the geographic distribution of colistin-resistant *P. aeruginosa* infections. It can be observed that most of the sentinel sites have resistance rates of less than 20%, while sentinel sites including MMH, RTH and EVR have resistance rates from 31–50%.

The geographic distribution of colistin resistance among *Pseudomonas aeruginosa* infection isolates showed generally low resistance rates across most Philippine sentinel sites, particularly in Luzon and Mindanao. However, selected Visayas centers, including EVR, MMH, and RTH, demonstrated elevated resistance levels reaching 31–50%. These findings suggest localized emergence of colistin resistance,

highlighting the importance of continued surveillance and antimicrobial stewardship.

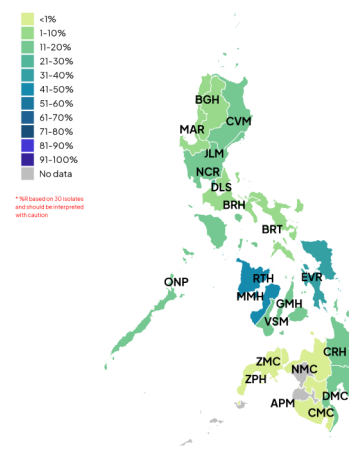


Figure 132. Resistance maps of *P. aeruginosa* infections for Colistin



Figure 133 shows the resistance rates of *P. aeruginosa* lower respiratory infections. Most of the resistance rates were below 30%. Resistance to ceftazidime and cefepime were 22.97% and 18.77% respectively. Resistance to ciprofloxacin and levofloxacin were 19.14% and 18.23% respectively. Carbapenem resistance showed resistance to meropenem at 24.01% and imipenem at 27.44%. Colistin resistance was at 7.10%. Resistance to the novel B-lactam/B-lactamase inhibitor combination antibiotics are as follows: ceftolozane-tazobactam at 11.61% and ceftazidime-avibactam at 31.22%.

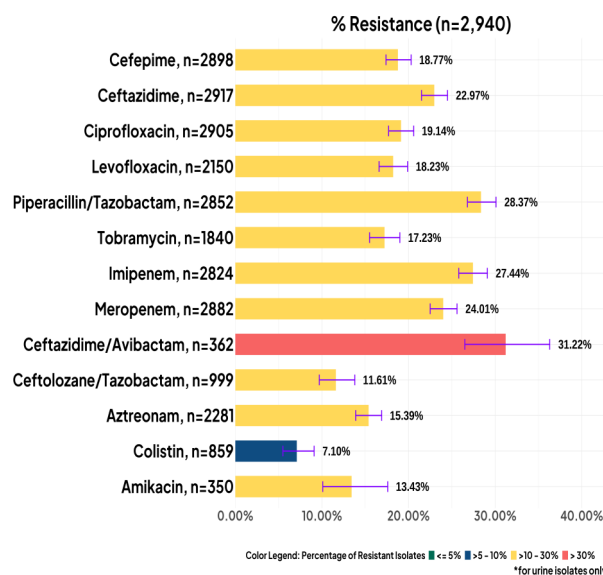


Figure 133. Proportion of *P. aeruginosa* lower respiratory tract infections with resistance to tested antibiotics, DOH-ARSP, 2025 (n=2,940)

Figure 134 shows the yearly resistance rates of *P. aeruginosa* lower respiratory tract infections. The multi-year analyses for these subsets likewise showed sta-

tistically significant changes in resistance rates for ciprofloxacin ($p=0.0001$), ceftipime ($p=0.000$) and tobramycin ($p=0.0002$).

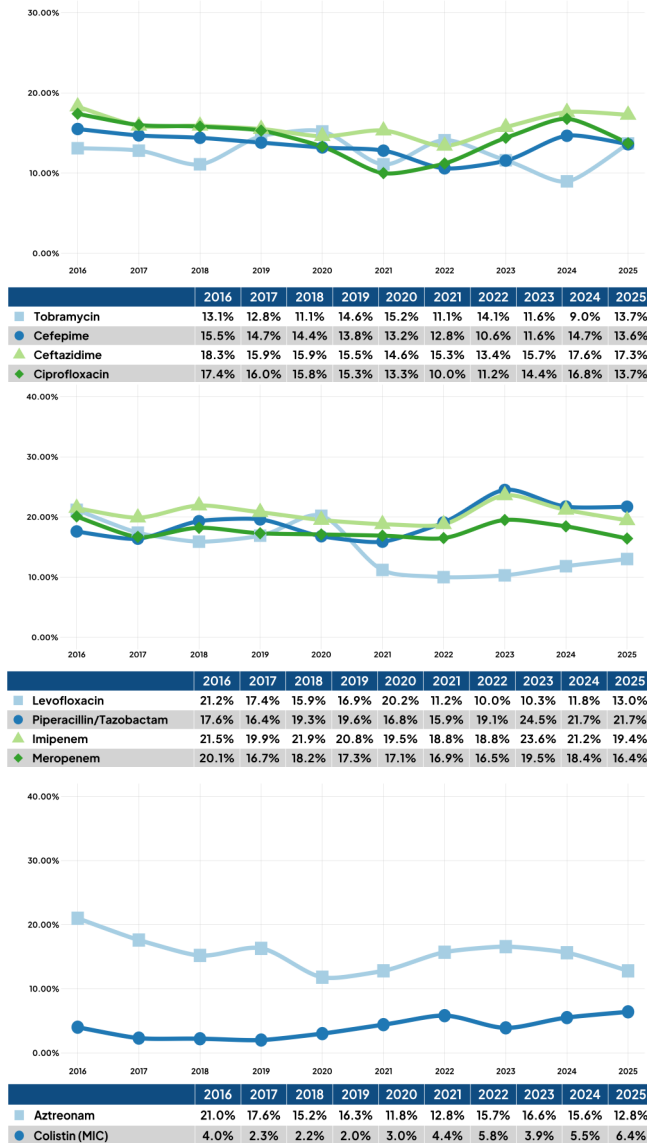


Figure 134. Yearly resistance rates of *P. aeruginosa* lower respiratory tract infections, DOH-ARSP, 2025



Bloodstream Infections

Figure 135 shows the resistance rates of *P. aeruginosa* bloodstream infections. Resistance to ceftazidime was at 13.93%, ceftipime at 10.01%, aztreonam at 11.80% and piperacillin-tazobactam at 16.89%. Resistance to fluoroquinolone were as follows: ciprofloxacin at 9.15% and levofloxacin at 7.72%. Resistance to carbapenems are as follows: meropenem at 11.44% and imipenem at 13.67%. Compared to the cumulative resistance rates across all infection types, bloodstream infections caused by *P. aeruginosa* have relatively lower resistance to all tested antibiotics. Among the novel β -lactam/ β -lactamase inhibitor combinations, resistance rates to ceftazidime-avibactam and ceftolozane-tazobactam were at 13.95% and 7.30% respectively.

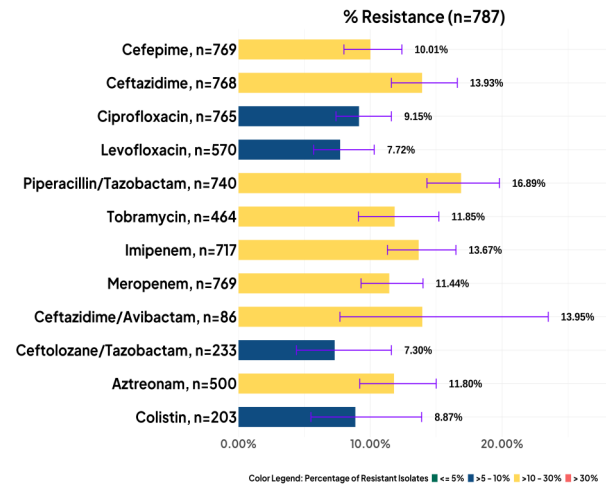


Figure 135. Proportion of *P. aeruginosa* bloodstream infections with resistance to tested antibiotics, DOH-ARSP, 2025 (n=787)

Figure 136 shows the yearly resistance rates of *P. aeruginosa* bloodstream infections. The multi-year analyses for these subsets likewise showed statistically significant changes in resistance rates for ciprofloxacin ($p=0.0000$), ceftipime ($p=0.0004$) and tobramycin ($p=0.0000$).

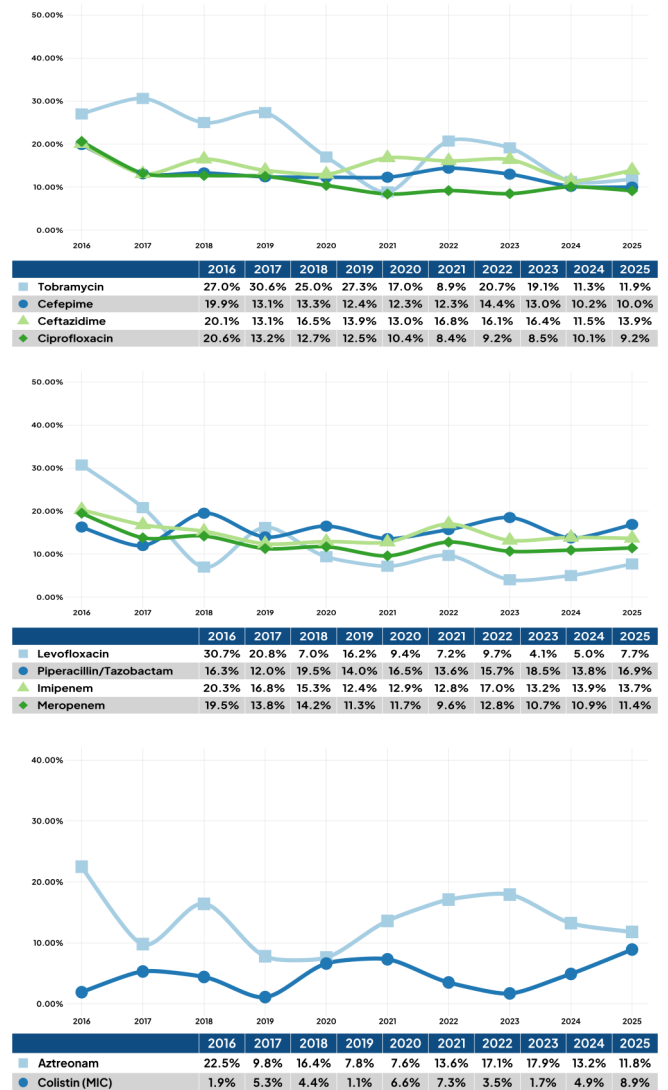


Figure 136. Yearly resistance rates of *P. aeruginosa* bloodstream infections, DOH-ARSP, 2025



Healthcare-associated Infections

The resistance rates of presumptive healthcare-associated *P. aeruginosa* infections are shown in **Figure 137**. Resistance to ceftazidime was at 21.96%, cefepime at 16.81%, aztreonam at 15.23%. Resistance to fluoroquinolones were as follows: ciprofloxacin (15.07%) and levofloxacin (14.20%).

Carbapenem resistance were as follows: meropenem (18.58%) and imipenem (21.39%). Among the novel β -lactam/ β -lactamase inhibitor combinations, imipenem/relebactam exhibited the highest resistance rate at 45.95% followed by ceftazidime/avibactam at 20.25% and ceftolozane/tazobactam at 8.83%. Caution is required in interpreting the resistance rates of the newer combination antibiotics due to the limited number of isolates tested.

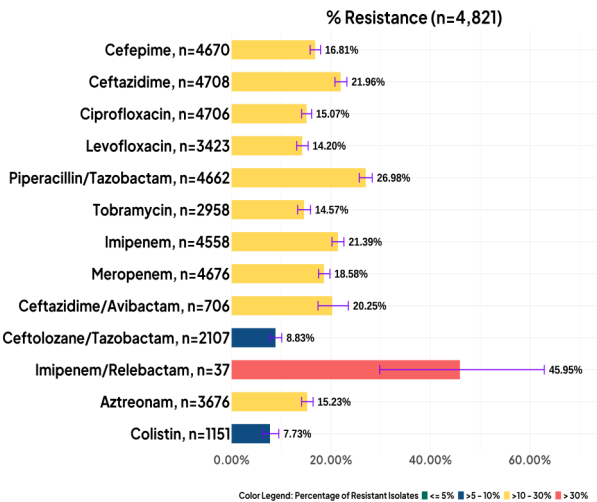


Figure 137. Proportion of *P. aeruginosa* isolates from presumptive healthcare-associated infections with resistance to tested antibiotics, DOH-ARSP, 2025 (n=4,821)



Emerging Resistance

Isolate Characteristics and Distribution

A total of 31 ceftazidime (CZA) -resistant infections and 3 imipenem/ relebactam (IMR)- resistant *Pseudomonas* infections were collected and sequenced in 2025. From these subsets, 18/31 CAZ-resistant isolates and all of 3 IMR-resistant were sequenced and analyzed included in the final dataset. **Figure 138** shows that there is an equal number of infections seen for both male and female patients, most (66.67%) were respiratory infections and were presumptive hospital-acquired. On the other hand, there were three IMR-resistant respiratory infections and all were presumptive nosocomial.

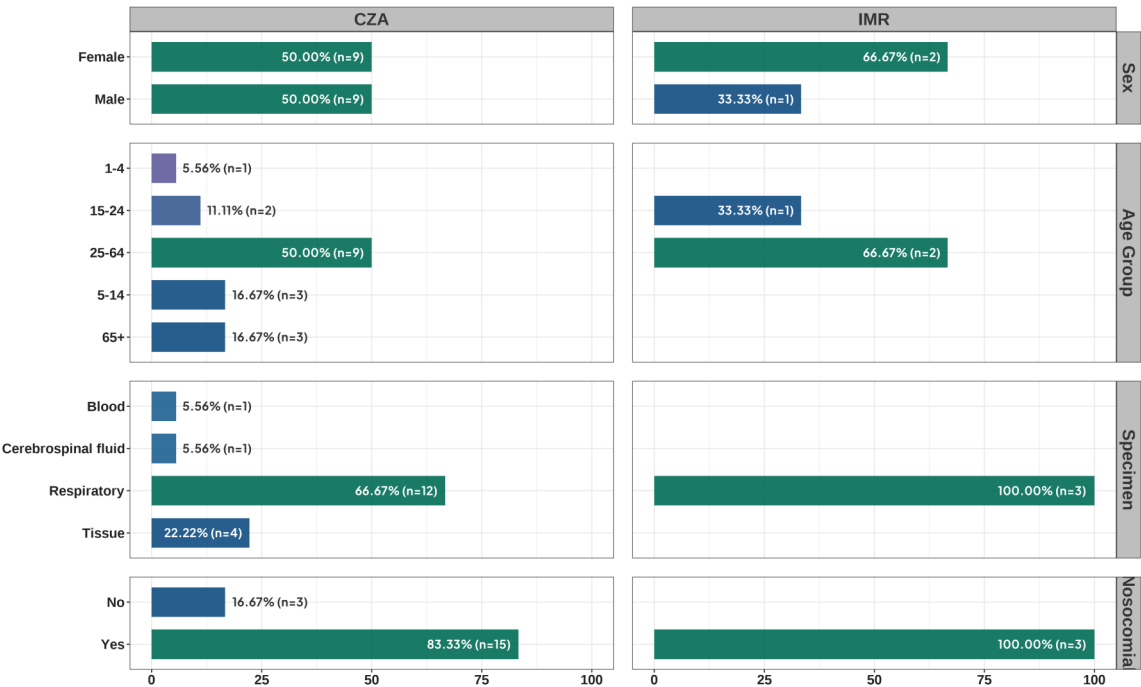


Figure 138. Patient demographics and clinical characteristics *P. aeruginosa* isolates across key antibiotic agents

Table 19 shows that there were 16 ceftazidime/ avibactam- resistant infections, and these are commonly seen in DMC and VSM. Most of these infections were ST 235 and belong to serogroup O11. ST235 has been reported across Asia, Europe, North America, South America, Africa, and Oceania and is considered the most geographically widespread *P. aeruginosa* high-risk clone. Most MBL-producing and XDR *P. aeruginosa* isolates identified globally belong to ST235 or a few other epidemic lineages^[1]. In CRH, a respiratory infection was seen on a 61-year old female in ICU. This infection was identified to be ST 245 and belongs to serogroup O5. There were two *Pseudomonas aeruginosa* infections from DMC which showed resistance to ceftazidime/ relebactam, imipenem/ relebactam and colistin. The first nosocomial infection was seen on a 75-year-old female followed by a respiratory infection detected from a 10-year old male. The latter was identified to be ST 773 and belongs to serogroup O11.

Table 19. Overall distribution and molecular typing summary of BL-BLI resistant *P. aeruginosa* isolates

Surveillance Sites	ST	Serogroups	Overall Isolates (N=29)	Novel BLI Isolates (n=19)
BRT, DMC, JLM, VSM	235	O11	8 (27.6%)	8 (42.1%)
APM, CRH	245	O5	3 (10.3%)	2 (10.5%)
BRT, DMC	773	O11	2 (6.9%)	2 (10.5%)
DMC, EVR, NKI, VSM	1684, 1974, 2850, 360, 389, 553	O11, O4, O5, O6, O7	16 (55.2%)	7 (36.8%)

Table 20 shows the distribution of carbapenemase genes among the sentinel sites. Ceftazidime/ avibactam-resistant *P. aeruginosa* isolates usually harbors the *blaIMP-26* and *blaVIM-2* genes. The metallo- β -lactamase genes *blaIMP-26* and *blaVIM-2* are important resistance determinants because avibactam lacks activity against class B β -lactamases. The gene *blaVIM-2* is the most widely disseminated VIM-type enzyme globally, whereas *blaIMP-26* has emerged as a notable regional carbapenemase in Southeast Asia, particularly in the Philippines and Vietnam, with strong association to the international high-risk clone ST235. The dissemination of these genes contributes substantially to the emergence of difficult to treat resistant *P. aeruginosa* in healthcare settings^[1]. One ceftazidime/avibactam-resistant isolate from JLM harbors both *blaNDM-1* and *blaVIM-2*. On the other hand, in this subset, infections from APM, DMC, NKI and VSM do not possess any resistance genes. Moreover, the two infections that showed resistance to both ceftazidime/ avibactam and imipenem/relebactam both harbors the *blaVIM-2* gene. While the single imipenem/ relebactam-resistant infection likewise possesses the *blaVIM-2* gene.

Table 20. Distribution of carbapenemase genes among BL-BLI resistant *P. aeruginosa* isolates

Phenotype	Surveillance Sites	IMP-26	VIM-2	NDM-1	NDM-1 & VIM-2	No genes detected
Ceftazidime-avibactam Resistant	BRT (2), DMC (5), EVR, JLM, VSM (3)	4	4	3	1	4
Ceftazidime-avibactam and Imipenem/relebactam -Resistant	DMC, VSM	0	2	0	0	0
Imipenem/relebactam-Resistant	CRH	0	1	0	0	0

Pathogen genomics plays a critical role in the identification and surveillance of high-risk *Pseudomonas aeruginosa* strains by providing detailed information on their genetic relatedness, antimicrobial resistance mechanisms, and transmission dynamics. Through whole-genome sequencing, resistance determinants such as *blaIMP-26* and *blaVIM-2* can be accurately detected, enabling the identification of ceftazidime/ avibactam-resistant isolates that may not be fully characterized by conventional laboratory methods. Genomic analysis also facilitates the detection of internationally disseminated high-risk clones such as ST235, allowing public health and healthcare institutions to distinguish between sporadic cases and outbreak-related transmission. Furthermore, continuous genomic surveillance supports early detection of emerging resistant lineages, informs infection prevention and control strategies, and provides evidence for targeted interventions to limit the spread of difficult-to-treat *P. aeruginosa* within and between healthcare facilities. Ultimately, pathogen genomics strengthens antimicrobial resistance surveillance by transforming laboratory data into actionable information for outbreak detection, monitoring, and response.

Reference

- [1] Horcajada JPMontero M Oliver A, Sorlí LLuque S, Gómez-Zorrilla S Benito N, Grau S. 2019. Epidemiology and Treatment of Multidrug-Resistant and Extensively Drug-Resistant *Pseudomonas aeruginosa* Infections. Clin Microbiol Rev 32:10.1128/cmr.00031-19. <https://doi.org/10.1128/cmr.00031-19>
- [2] Vezina, B., Morampalli, B.R., Nguyen, HA. et al. The rise and global spread of IMP carbapenemases (1996-2023): a genomic epidemiology study. Nat Commun 17, 183 (2026). <https://doi.org/10.1038/s41467-025-66874-7>



Isolate Characteristics and Distribution

A total of 18 (6.84%) carbapenem-resistant *P. aeruginosa* isolates passed the quality control and no carbapenem-susceptible isolates were included in the structured survey analysis. Most (77.7%) isolates were from respiratory specimens and from the 25-64 (44.44%) age group. Most (83.33%) infections were considered hospital-acquired (**Table 21**).

Table 21. Patient demographics and clinical characteristics (n=18)

Specimen type distribution		Age group distribution		Sex distribution		Nosocomial (Yes/No)	
Specimen Type	Count	Age group	Count	Sex	Count	Nosocomial	Count
Respiratory	14	1-4	1	Female	9	No	3
Blood	2	5-14	3	Male	9	Yes	15
Tissue	2	15-24	3				
		25-64	8				
		65+	3				

Carbapenem resistance determinants

Table 22 shows the distribution of carbapenemase genes among carba-R *P. aeruginosa* isolates by surveillance site and region. It was observed that half (n=9) of the isolates harbor carbapenemase genes including NDM-1, VIM-2 and IMP-26. This is consistent with the 2024 structured genomic survey for *Pseudomonas aeruginosa*, wherein these three genes were the most frequently identified together with the presence of mutations in the *oprD* gene. It can be seen that the three carbapenem-resistant isolates from DMC all harbored the IMP-26 gene, while the two isolates from BRT likewise shared a common gene (NDM-1). Notably, a single isolate from JLM harbors both NDM-1 and VIM-2. It can also be observed that isolates from other sentinel sites carry similar carbapenemase genes.

Table 22. Distribution of carbapenemase genes among carba-R *P. aeruginosa* isolates by surveillance site and region, ARSP 2025 (n=9)

Surveillance Sites	Region	Count	NDM-1	VIM-2	IMP-26	NDM-1 & VIM-2
BRT	V	2	2	0	0	0
CRH	XIII	1	0	1	0	0
DMC	XI	3	0	0	3	0
EVR	VIII	1	1	0	0	0
JLM	III	1	0	0	0	1
VSM	VII	1	0	1	0	0

Molecular Typing Analysis

The carbapenem-resistant isolates belonged to 12 sequence types and most of the isolates were identified as ST 235 (16.7%) and ST 773 (16.7%). There were four serotypes detected among the isolates, with O11 (55.55%) being the most prevalent followed by O6 (16.66%). **Table 23** shows that the STs and serotypes of isolates from a single sentinel site are different from each other and with no distinct pattern which may suggest the diversity of the organism in the sentinel sites. In 2024, ST 235 and ST155 were likewise the predominating sequence type, however, the latter was not apparent in 2025. *Pseudomonas aeruginosa* ST235 is one of the most important and widely disseminated high-risk clones of *P. aeruginosa* associated with healthcare-associated infections worldwide. It is strongly linked to multidrug-resistant (MDR) and extensively drug-resistant (XDR) phenotypes and has been reported across Europe, Asia, the Americas, Africa, and the Philippines.

Table 23. Overall distribution and molecular typing summary of *P. aeruginosa*

Surveillance Sites	Region	ST	Serotype	Overall (N=18)	Carbapenem-Resistant (n=18)	Carbapenem-Susceptible (n=0)
BRT, JLM, VSM	III, V, VII	235	O11	3 (16.7%)	3 (16.7%)	0 (0.0%)
BRT, DMC, EVR	V, VIII, XI	773	O11	3 (16.7%)	3 (16.7%)	0 (0.0%)
APM, CRH	BARMM, XIII	245	O5	2 (11.1%)	2 (11.1%)	0 (0.0%)
DMC, EVR, GMH, NKI	NCR, VII, VIII, XI	-, 110, 1684, 2850, 357, 389, 3960, 4500, 553, 620	O11(4), O6 (3), O4(2), O5	10 (55.6%)	10 (55.6%)	0 (0.0%)

Phylogenetic Analysis

Figure 139 shows six clusters of isolates detected from the network analysis conducted. There were three clusters with lone isolates from EVR, which may suggest the high diversity of this organism in this sentinel site. Moreover, there were two clusters having three isolates linked to each other by minimal SNPs differences. The first cluster involving isolates from JLM, BRT, and VSM have SNPs differences ranging from 150-272, while the second cluster among DMC, BRT and EVR have SNPs differences from 150-187. The minimal differences among these clusters suggest that isolates are likely closely related.

Figure 140 shows the phylogenetic analysis of *Pseudomonas aeruginosa* isolates, which likewise shows the clusters previously described above. Isolates included in the first cluster (JLM-BRT-VSM) were all from respiratory specimens, harbors *bla*IMP gene, identified as ST 235 and found to be serotype O11. On the other hand, all isolates in the second cluster (EVR-DMC-BRT) were likewise from respiratory isolates, uniformly harbors *bla*VIM gene, identified as serotype 773 noted to be serotype O11.

structured_pae_2025 — Cluster Network at ≤ 29228 SNPs

6 clusters | 3 isolates linked

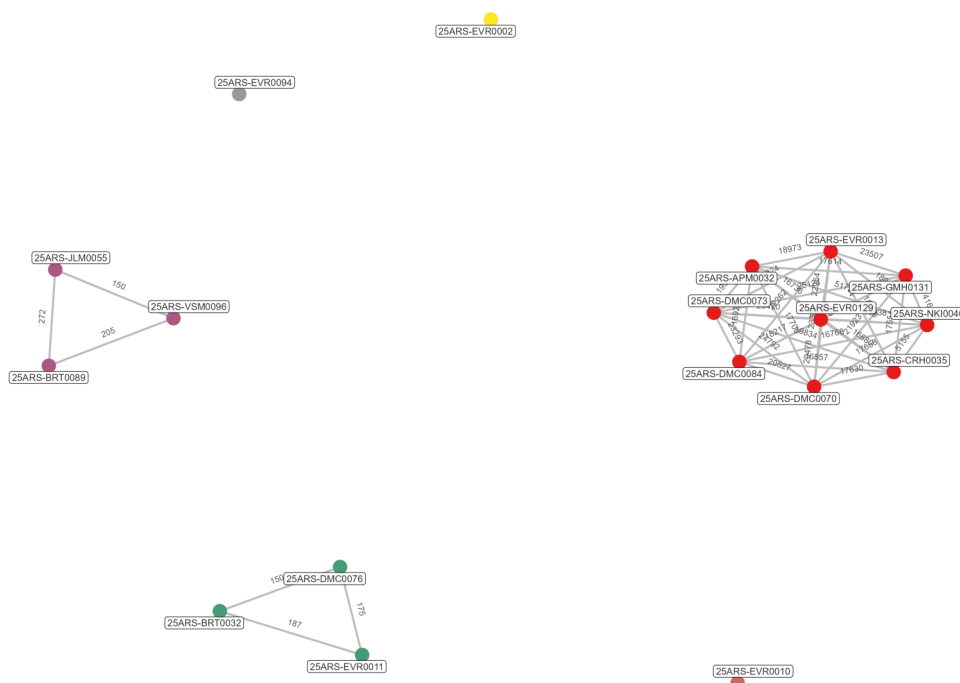


Figure 139. Cluster network analysis of *Pseudomonas aeruginosa* isolates at ≤ 29,228 Single Nucleotide Polymorphisms (SNPs)

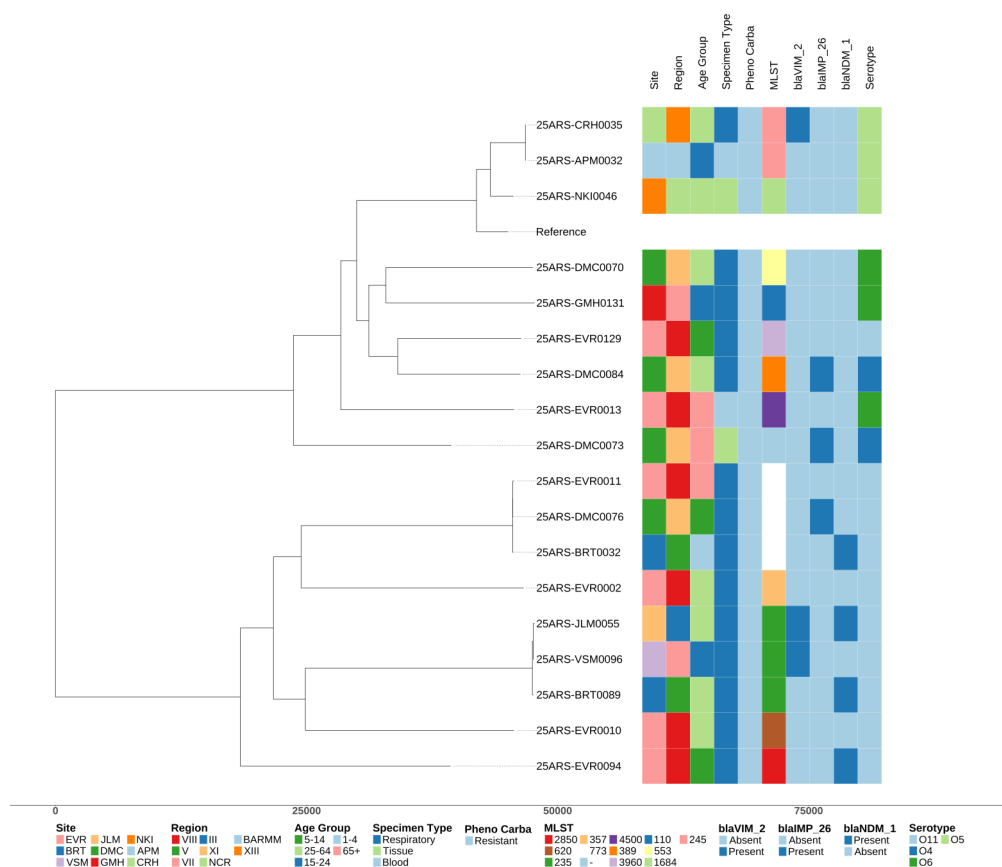


Figure 140. Phylogenetic tree of the carbapenem resistant *P. aeruginosa* isolates

Discussion

This genomic survey highlights that the two main drivers of carbapenem resistance among *Pseudomonas aeruginosa* isolates were *bla*NDM and *bla*IMP. This is consistent with the 2024 structured survey results done by ARSP^[1]. The *bla*NDM and *bla*IMP genes encode for New Delhi metallo-beta-lactamase (NDM) and imipenemase (IMP), which are prominent types of carbapenemase enzymes. These enzymes utilize a zinc-dependent active site to efficiently hydrolyze the beta-lactam ring that forms the core structure of carbapenem antibiotics. By breaking this critical ring structure, the enzymes successfully deactivate the antibiotics before they can disrupt bacterial cell wall synthesis^[2]. It can be observed that nine (50%) of the sequenced isolates do not have any gene that confers resistance to carbapenems. The mechanism of carbapenem resistance in these isolates were not thoroughly described in this report and requires in depth analyses. However, several studies would explain that the phenomenon can be due to mutational resistance—specifically driven by the downregulation/loss of the OprD porin, often acting synergistically with the upregulation of MexAB–OprM efflux pumps or derepressed AmpC beta-lactamase^[3].

Genomic surveillance plays a critical role in mitigating carbapenem resistance in *Pseudomonas aeruginosa* by offering precise insights into underlying resistance pathways, including mutational changes in oprD porins or the acquisition of mobile carbapenemase genes. This level of genetic resolution allows public health authorities to track the emergence and transmission of high-risk clones, such as ST235, across healthcare facilities and global regions, thereby enabling swift outbreak identification and containment. Additionally, genomic frameworks standardize data comparability across international laboratories, driving a more cohesive global response to this escalating pathogen. Ultimately, mapping these genetic foundations is vital for designing targeted clinical interventions and guiding the development of novel antibiotics.

Reference:

- [1] Antimicrobial Resistance Surveillance Program. 2024 Annual Report. Manila, Philippines: Antimicrobial Resistance Surveillance Reference Laboratory, Research Institute for Tropical Medicine, Department of Health; 2025. ISSN 3082-589X. <https://www.arsp.com.ph/publications/>
- [2] Queenan, A. M., & Bush, K. (2007). Carbapenemases: the versatile beta-lactamases. *Clinical Microbiology Reviews*, 20(3), 440–458.
- [3] Rodriguez-Martinez, J. M., Poirel, L., & Nordmann, P. (2009). Molecular epidemiology and mechanisms of carbapenem resistance in *Pseudomonas aeruginosa*. *Antimicrobial Agents and Chemotherapy*, 53(11), 4783–4788.

A scanning electron micrograph (SEM) of Acinetobacter baumannii cells. The image shows three distinct cells. Two cells in the upper left are oval-shaped and covered in fine, hair-like pili. A third cell in the lower right is more irregular and elongated, also showing a textured surface with pili. The background is a uniform light gray.

Acinetobacter baumannii

Acinetobacter baumannii

Acinetobacter baumannii is a notable healthcare-associated pathogen that is responsible for severe nosocomial infections, especially in intensive care units (ICUs). This organism exhibits a remarkable ability to acquire or develop resistance to nearly all antimicrobial agents available for treatment, leading to a high prevalence of multidrug-resistant (MDR) and extensively drug-resistant (XDR) phenotypes. As the proportion of carbapenem-resistant strains rises, the efficacy of this last-resort therapeutic option diminishes. Nevertheless, these resistant strains often remain susceptible to colistin and/or tigecycline.

The ARSP has reported carbapenem resistance rates exceeding 50%, indicating the prevalence of high-risk international clones and the contribution of horizontal gene transfer to carbapenem resistance in *A. baumannii* infections within healthcare settings. These epidemic strains can endure within hospital environments, surviving on medical equipment and environmental surfaces near colonized patients. As a result, the persistence of these strains significantly restricts effective empiric treatment options and necessitates the implementation of targeted infection control measures.

A total of **8,207** *A. baumannii* infections were reported for 2025. Based on island group distribution, more than half of the infections originated from Luzon (50.72%), followed by the Visayas (24.86%) and Mindanao (24.42%). The largest contributors were PGH (12.45%), DMC (8.96%) and GMH (8.65%).

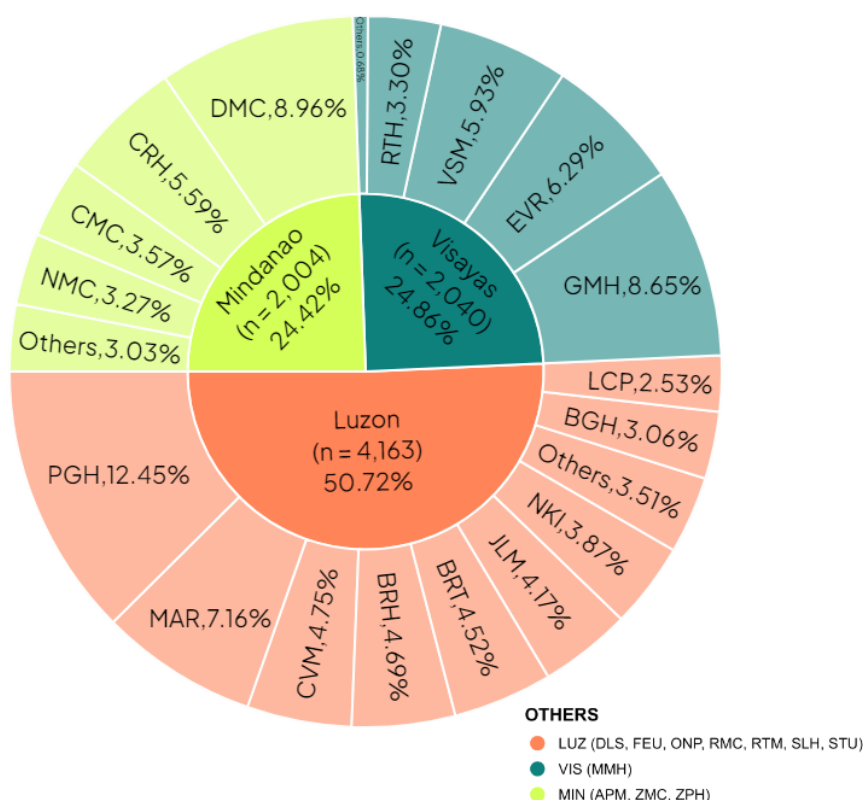


Figure 141. Distribution of *A. baumannii* infections, DOH-ARSP, 2025 (n= 8,207)

More than half of *A. baumannii* infections were among males (54.81%) and the 20–64 age group (58.27%). Most (61.83%) are respiratory infections and 48.93% are presumptively community acquired while 47.30% are healthcare associated infections.

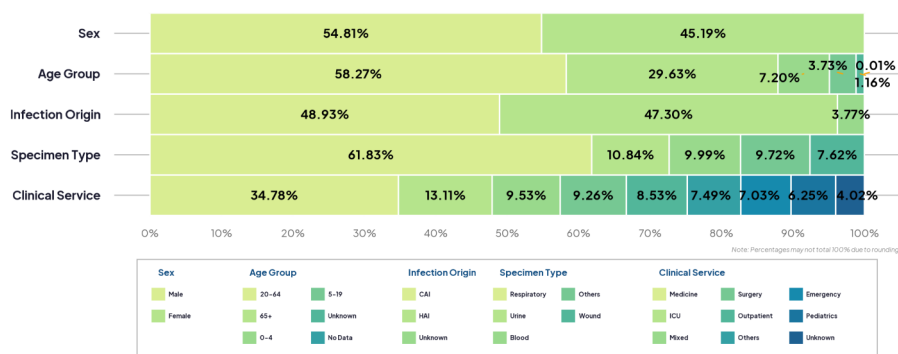


Figure 142. Patient characteristics of *A. baumannii* infections, DOH-ARSP, 2025 (n= 8,207)

All types of Infections

Figure 143 shows the resistance rates of all *A. baumannii* infections for 2025. Many were resistant to beta-lactam/beta-lactamase inhibitor combination antibiotics: ampicillin-sulbactam (43.32%) and piperacillin-tazobactam (45.66%), carbapenems: imipenem (45.42%) and meropenem (44.23%), cephalosporins: cefepime (45.18%) and ceftazidime (44.17%); and aminoglycosides: tobramycin (39.98%). Resistance to minocycline was at 19.27% and colistin at 3.52%.

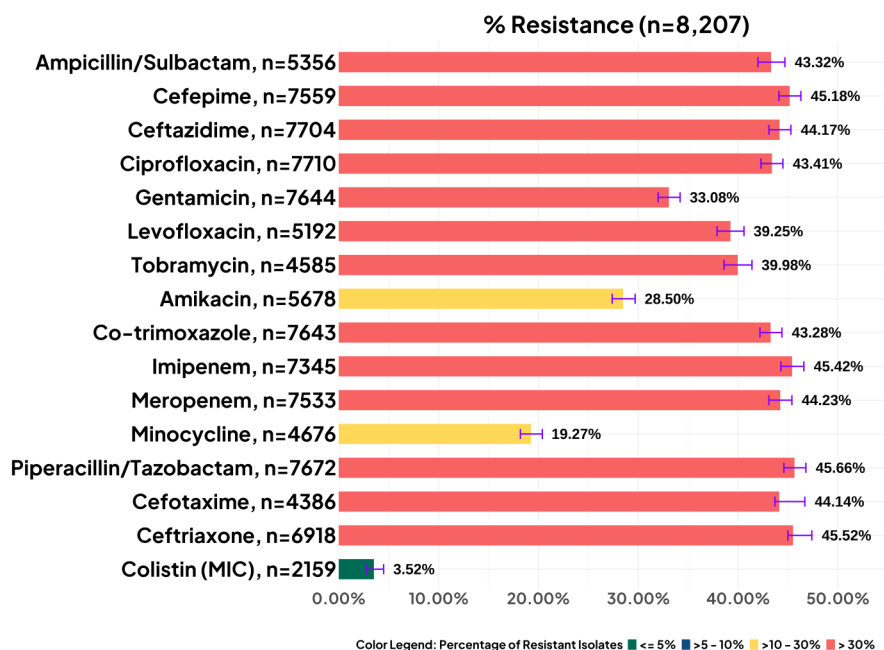
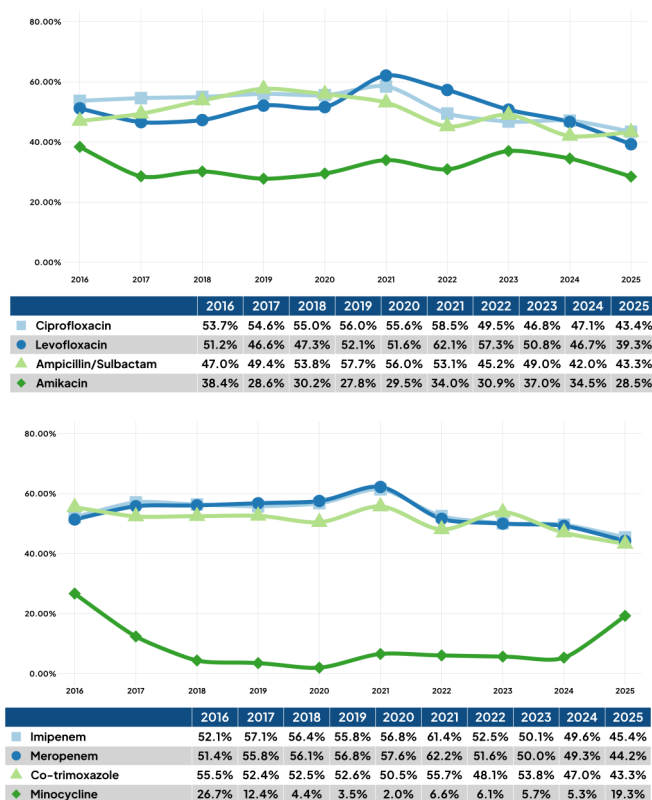
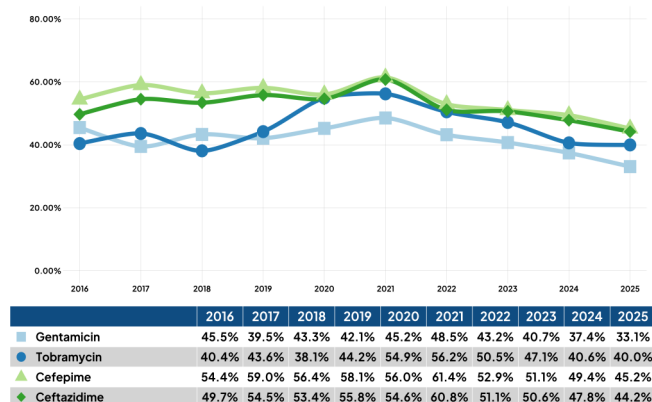


Figure 143. Proportion of all *A. baumannii* infections resistance to tested antibiotics, DOH-ARSP, 2025 (n=8, 207)

Figure 144 shows the yearly resistance rates of all *A. baumannii* infections. The decreasing patterns observed for most antibiotic classes are statistically significant. Specifically, significant decreases were recorded for meropenem ($p=0.0000$), imipenem ($p=0.0000$), amikacin ($p=0.0000$), gentamicin ($p=0.0002$), co-trimoxazole ($p=0.0000$), levofloxacin ($p=0.0085$), and tobramycin ($p=0.0000$). Colistin ($p=0.0000$) demonstrated significant increase, rising from 0.4% in 2024 to 3.5% in 2025. No significant differences in resistance were observed for ampicillin-sulbactam ($p=0.0812$), ceftazidime ($p=0.6172$), and minocycline ($p=0.1916$).



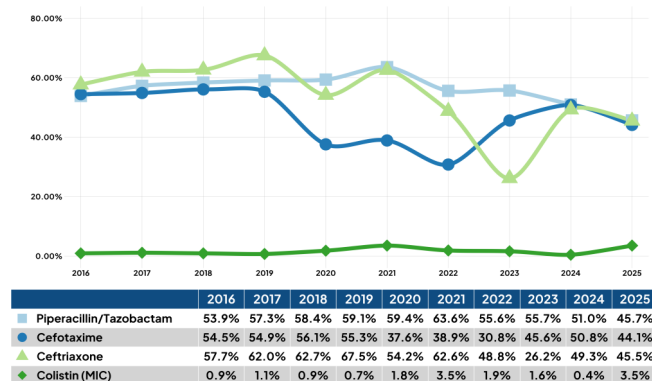


Figure 144. Yearly resistance rates for all *A. baumannii* infections, DOH-ARSP, 2025

Figure 145 illustrates the regional resistance rates of *A. baumannii* infections for imipenem and meropenem across the Philippines. High carbapenem resistance was observed across all three major island groups, with several surveillance sites reporting resistance levels in the 71-80% and 81-90%. Specifically, surveillance sites in Luzon (including BGH, JLM and MAR), the Visayas (VSM and GMH), and Mindanao (DMC and NMC) exhibited the highest intensities of resistance for both imipenem and meropenem.

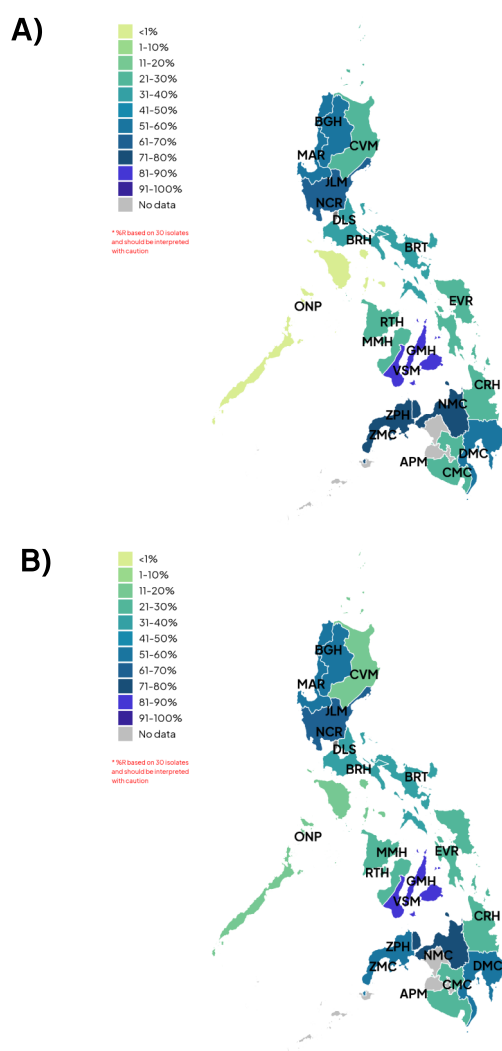


Figure 145. Resistance maps of *A. baumannii* infections for (A) Imipenem, and (B) Meropenem

Bloodstream Infections

Figure 146 shows the resistance rates of *A. baumannii* bloodstream infections. Most of the resistance rates to tested antibiotics were above 40%. Many were resistant to beta-lactam/beta-lactamase inhibitor combination antibiotics: ampicillin-sulbactam (41.76%) and piperacillin-tazobactam (43.06%). Resistance to carbapenem antibiotics, meropenem and imipenem were 38.79% and 40.52% respectively. Resistance to cephalosporin antibiotics, cefepime and ceftazidime were at 41.20% and 40.60% respectively.

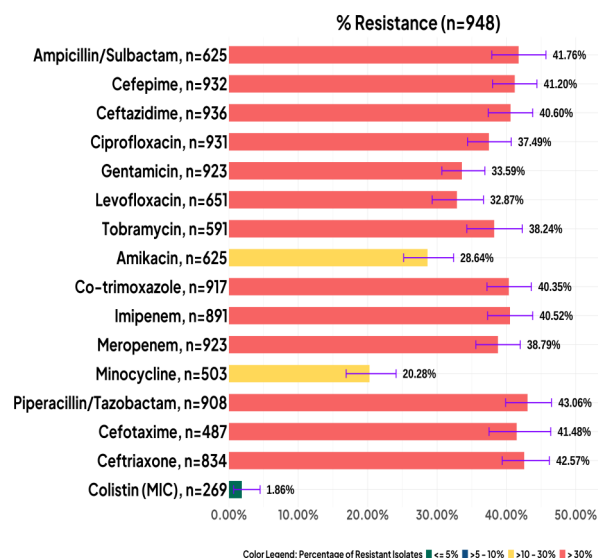
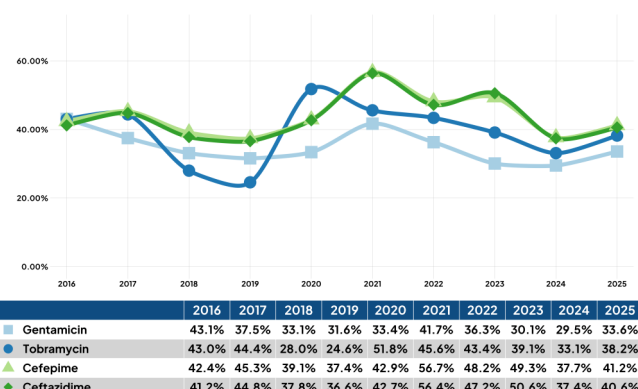


Figure 146. Proportion of *A. baumannii* bloodstream infections with resistance to tested antibiotics, DOH-ARSP, 2025 (n=948)

Figure 147 shows the yearly resistance rates of *A. baumannii* bloodstream infections. The increasing trend seen among the following antibiotics were all statistically significant: ampicillin-sulbactam, gentamicin, tobramycin, cefepime, ceftazidime, ceftriaxone, ciprofloxacin, colistin, co-trimoxazole, imipenem, meropenem, and minocycline. While decreasing resistance rates were observed for amikacin ($p=0.0000$) and cefotaxime ($p=0.0000$).



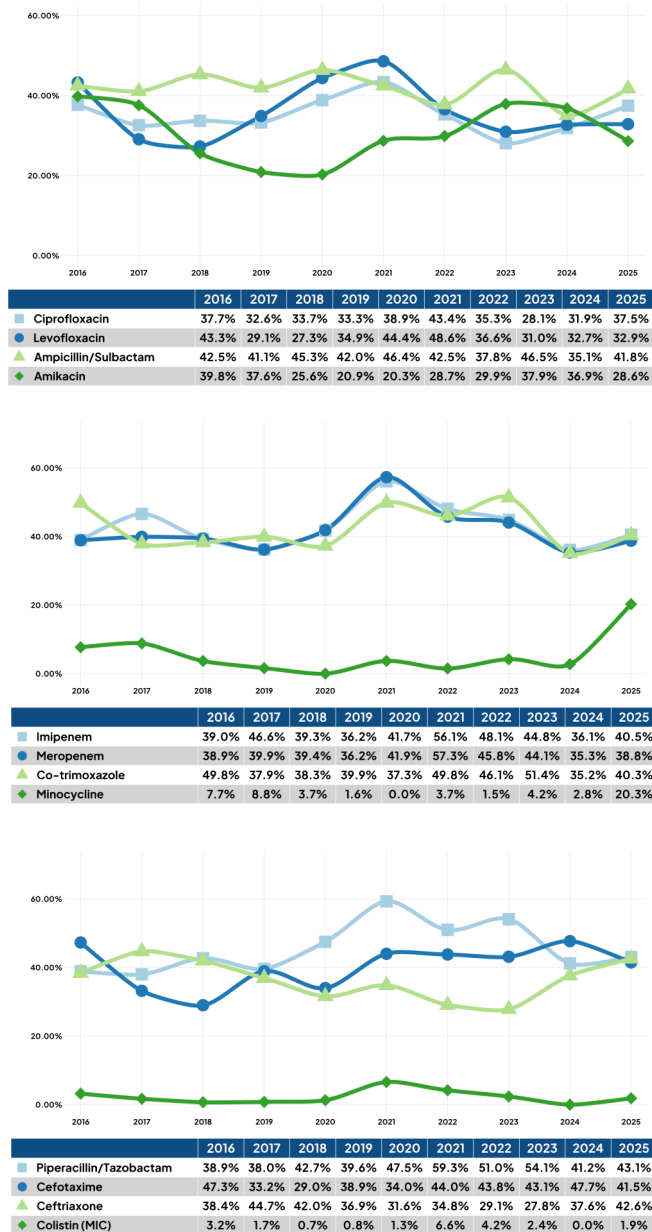


Figure 147. Yearly resistance rates of *A. baumannii* blood-stream infections, DOH-ARSP, 2025



Lower Respiratory Tract Infections

Figure 148 shows the resistance rates of *A. baumannii* lower respiratory tract infections. Most of the resistance rates were above 50%. The highest resistance was observed for piperacillin-tazobactam (70.99%) followed by ceftriaxone (70.12%) and imipenem (70.04%). Minocycline resistance was at 28.79%.

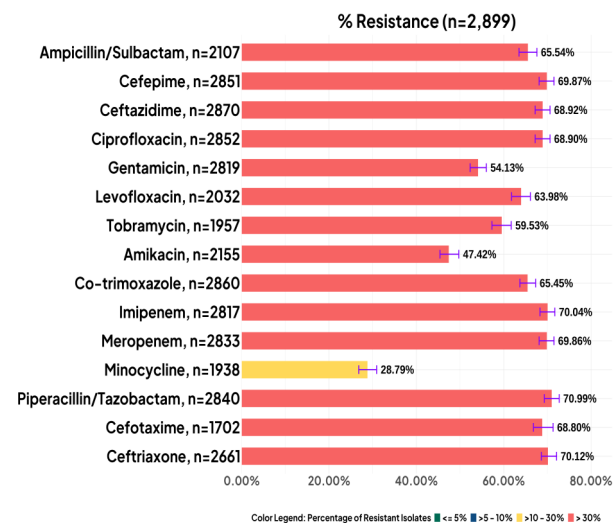
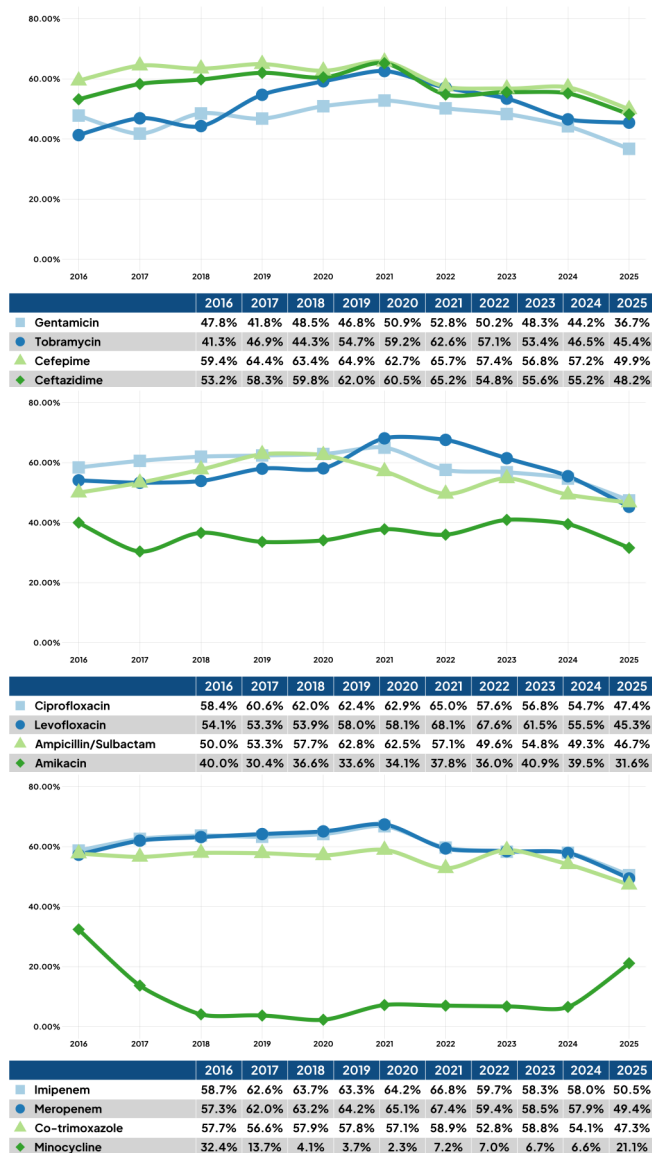


Figure 148. Proportion of *A. baumannii* lower respiratory tract infections with resistance to tested antibiotics, DOH-ARSP, 2025 (n=2,899)

Figure 149 shows the yearly resistance rates for *A. baumannii* respiratory infections. The observed decreasing trend for ciprofloxacin ($p=0.0000$), levofloxacin ($p=0.0085$), tobramycin ($p=0.0000$) and gentamicin ($p=0.0002$) were all significant.



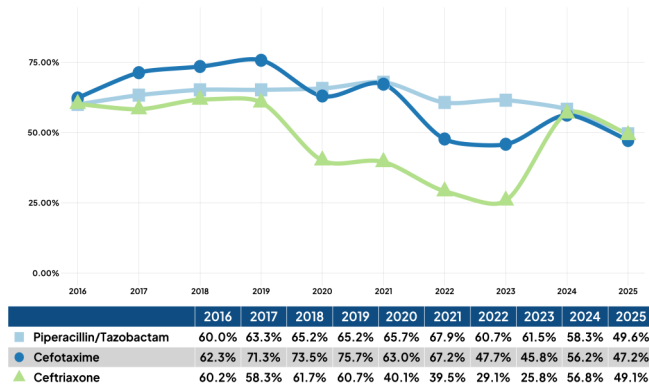


Figure 149. Yearly resistance rates of *A. baumannii* respiratory infections with resistance to tested antibiotics, DOH-ARSP, 2025

Healthcare-associated Infections

Acinetobacter baumannii is a prevalent pathogen in healthcare-associated infections, often presenting significant therapeutic challenges due to multidrug resistance (MDR). As shown in **Figure 150**, MDR *A. baumannii* presumptive healthcare-acquired infections exhibited high resistance rates to standard treatments, including ampicillin-sulbactam (49.61%)

and piperacillin-tazobactam (54.86%). Resistance reached 52.57% for ceftazidime and 53.87% for cefepime, while carbapenem resistance was 54.33% for imipenem and 53.68% for meropenem. Among alternative choices, resistance rates were lower, with minocycline at 19.89% and colistin at 4.55%.

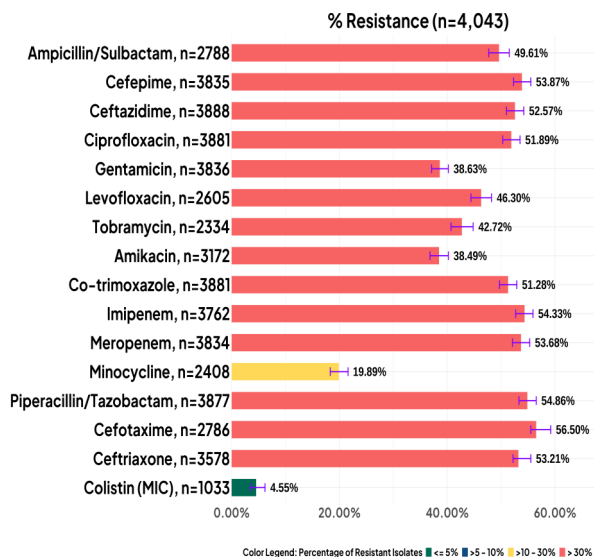


Figure 150. Proportion of presumptive healthcare-associated *A. baumannii* infections with resistance to tested antibiotics, DOH-ARSP, 2025 (n=4,043)

Target 2 of the National Action Plan for Antimicrobial Resistance (NAP-AMR) 2024–2028 aims for a 10% reduction in healthcare-associated infections caused by carbapenem-resistant *Acinetobacter baumannii* (CRAB). For bloodstream infections, the 2023 baseline resistance rates were at 44.08% for meropenem and 44.83% for imipenem. In 2024, surveillance data showed a decrease in resistance to 35.33% and 36.09%, respectively, representing nearly 20% reductions for both agents. However, the 2025 data reflects an increase in resistance rates to 38.79% for meropenem and 40.52% for imipenem. Based on the 10% reduction target, the threshold for meropenem is 39.67%, and for imipenem, it is 40.35%. Consequently, as of 2025, the 12.00% reduction for meropenem continues to meet the target objective, while the 9.61% reduction for imipenem places the current rate slightly above the intended target threshold. Overall, the data indicates an encouraging net downtrend from the baseline, though additional years of surveillance are necessary to confirm if these reductions will establish a consistent and stable trajectory.

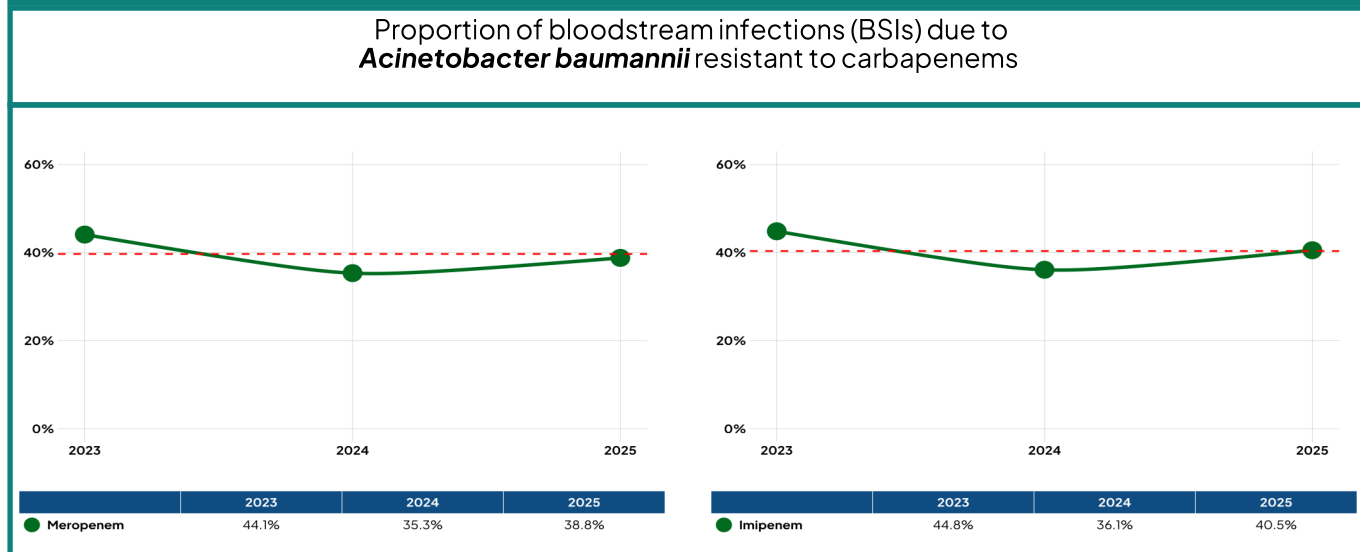


Figure 151. Yearly resistance rates of *A. baumannii* bloodstream infections, DOH-ARSP, 2023-2025



Emerging Resistance

Isolate Characteristics and Distribution

Acinetobacter baumannii is recognized as a clinically significant pathogen causing a wide spectrum of healthcare-acquired infections, especially the most vulnerable patients. In 2025, a detailed characterization was performed on a subset of 23 colistin-resistant isolates. These isolates were predominantly recovered from females (56.52%) and patients in the 65+ age group (47.83%). 78.26% of these were identified as presumptive healthcare-associated infections (HAI) and 86.96% originating from respiratory specimens.

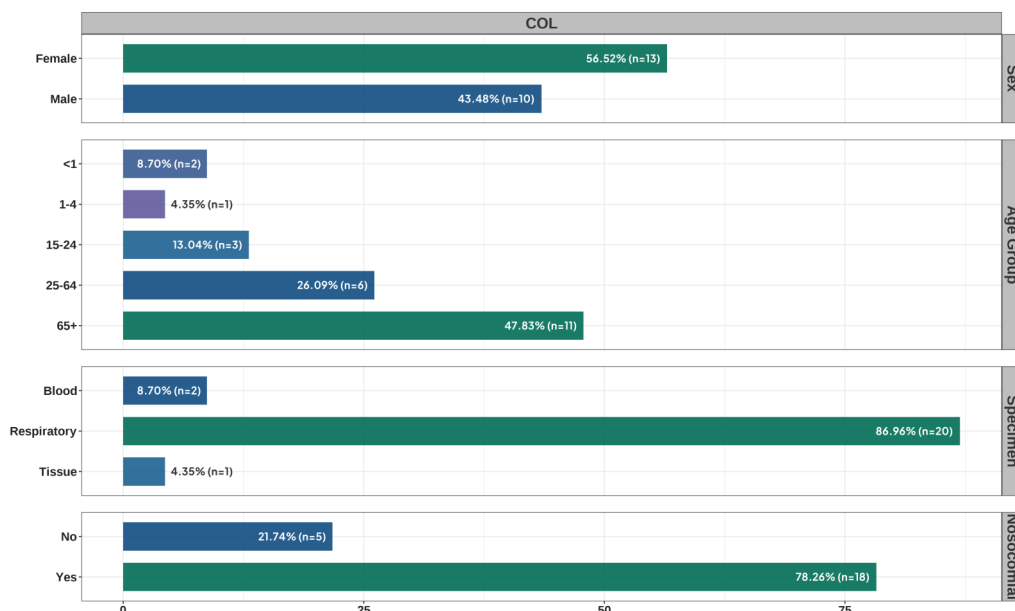


Figure 152. Patient demographics and clinical characteristics of colistin-resistant *A. baumannii* isolates, n=23

Table 24 shows that the majority of the infections belonged to ST2 and were characterized by the OCL1 capsule type. ST2, which corresponds to international clone II, is recognized as the most successful and geographically widespread high-risk clone of *A. baumannii* globally and in the Philippines^[1]. Meanwhile, in CRH, a nosocomial respiratory infection was detected in a 61-year old female admitted to the ICU. This infection was identified as ST164 and belonged to the OCL5 capsule type.

There were multiple healthcare-associated infections from DMC that demonstrated phenotypic resistance to colistin, all of which harbored the *pmrB*_A138T mutation and belonged to the OCL1 capsule type. One such nosocomial infection was seen in a 60-year-old female in the ICU2, followed by invasive infections in the neonatal and pediatric units, including a 16-day-old male with a bloodstream infection in the neonatal isolation ward and a 3-month-old male with a respiratory infection in the PCU3. These findings underscore the persistence of the stable ST2 epidemic lineage alongside the emergence of diverse sequence types carrying potent resistance markers within critical care environments.

Table 24. Overall distribution and molecular typing summary of colistin resistant *A. baumannii* isolate

Surveillance Sites	ST	Capsule Types	Overall Isolates (N=23)	Colistin Resistant (n=23)
DLS, DMC, GMH, MAR, NMC	2	OCL1	17 (73.9%)	17 (73.9%)
BRT, GMH	-	OCL1	4 (17.4%)	4 (17.4%)
CRH, JLM	164, 78	OCL1, OCL5	2 (8.7%)	2 (8.7%)

There was a complete absence of plasmid-mediated colistin resistance genes (**Table 25**). Instead, colistin resistance among the 23 genomically characterized isolates was driven primarily by chromosomal mutations. Specifically, the *pmrB*_A138T mutation was identified in 78.26% (n=18) of the resistant isolates. Within the *pmrCAB* operon, these amino acid modifications alter the PmrB sensor kinase, leading to downstream lipid A modification. This reduces the negative charge of the outer membrane and decreases colistin binding affinity^[2]. Crucially, this *pmrB*_A138T mutation was consistently detected in ST2 isolates across multiple surveillance sites (DLS, DMC, GMH, MAR, NMC), signaling its role as a stable national vehicle for chromoso-

mal colistin resistance. Conversely, 21.74% (n=5) of the resistant isolates harbored no known mutations within this operon. This divergence demonstrates that while pmrB-mediated chromosomal modification remains the dominant pathway for the established ST2 clone, alternative or novel resistance mechanisms are likely active within emerging lineages like ST164 and ST78^[2,3], necessitating further high-resolution characterization to identify these hidden mechanisms and drivers.

Table 25. Distribution of colistin resistant *A. baumannii* isolates

Phenotype	Surveillance Sites	mcr genes	pmrB_A138T mutation	No mutation
Colistin-resistant	DLS, DMC, GMH, MAR, NMC	0	18	0
	BRT, CRH, JLM	0	0	5

Reference

- [1] Wang, X., Xu, T., Zhang, Y., Qiu, Y., Liang, Y., Feng, J., ... & Ren, L. (2026). Comparative genomic and resistance characterization of ST2 and ST164 carbapenem-resistant *Acinetobacter baumannii* from hospital environments and clinical specimens. *Microbial Genomics*, 12(4), 001679.
- [2] Novović, K., & Jovčić, B. (2023). Colistin resistance in *Acinetobacter baumannii*: molecular mechanisms and epidemiology. *Antibiotics*, 12(3), 516.
- [3] Da Silva, G. J., & Domingues, S. (2017). Interplay between colistin resistance, virulence and fitness in *Acinetobacter baumannii*. *Antibiotics*, 6(4), 28.
- [4] Zafer, M. M., Hussein, A. F., Al-Agamy, M. H., Radwan, H. H., & Hamed, S. M. (2023). Retained colistin susceptibility in clinical *Acinetobacter baumannii* isolates with multiple mutations in pmrCAB and lpxACD operons. *Frontiers in cellular and infection microbiology*, 13, 1229473.



Structured Genomic Survey

Isolate Characteristics and Distribution

A total of 23 carbapenem-resistant *Acinetobacter baumannii* isolates met the stringent quality control criteria and were included in the structured survey analysis. The analyzed isolates were collected across eight surveillance sites and were predominantly healthcare-associated. Over half of the source patients were female (56.5%) and aged 25–64 years (26.09%) (Table 26).

Table 26. Patient demographics and clinical characteristics (n=23)

Specimen type distribution		Age group distribution		Sex distribution		Nosocomial (Yes/No)	
Specimen Type	Count	Age group	Count	Sex	Count	Nosocomial	Count
Respiratory	20	<1	2	Female	13	No	5
Blood	2	1-4	1	Male	10	Yes	18
Tissue	1	15-24	3				
		25-64	6				
		65+	11				

Carbapenem Resistance Determinants

The distribution of carbapenemase genes among the 23 characterized isolates (Table 27) reveals that blaOXA-23 remains the driver of resistance, detected in 95.7% (n=22) of the isolates. This is consistent with

the 2024 surveillance data. Concurrently, a notable shift is evident in the expansion of *bla*NDM-1, which was detected in 21.7% of the 2025 isolates. While *bla*NDM-1 detection was sporadic and confined to Luzon, current data indicates its emergence in new regions, including CRH (Region XIII) and JLM (Region III), often co-occurring with *bla*OXA-23 at sites such as BRT (Region V).

Table 27. Distribution of carbapenemase genes among carba-R *A. baumannii* isolates by surveillance site and region, ARSP 2025 (n=23)

Surveillance Sites	Region	Count	NDM-1	OXA-23
BRT	V	3	3	3
CRH	XIII	1	1	0
DLS	IV-A	1	0	1
DMC	XI	6	0	6
GMH	VII	7	0	7
JLM	III	1	1	1
MAR	I	3	0	3
NMC	X	1	0	1

Molecular Typing Analysis

ST2, which corresponds to the high-risk International Clone II, accounts for 73.9% of the carbapenem-resistant isolates. This clone demonstrated dissemination across five regions. Within the stable ST2 backbone, the molecular typing analysis revealed a significant capsular heterogeneity, with five unique capsule (KL) types detected KL2 (n=6), KL3 (n=7), KL49 (n=1), KL73 (n=1), and KL9 (n=2). All 17 ST2 isolates shared the OCL1 lipooligosaccharide outer-core locus, a hallmark of this epidemic lineage. Emerging lineages ST78 and ST164 were also identified, collectively representing 8.7% of the collection and originating from Regions III and XIII, respectively. Additionally, 17.4% of the isolates from surveillance sites BRT and GMH remained uncategorized by standard MLST but were characterized by the KL3:OCL1 and KL37:OCL1 capsule types (Table 28).

Table 28. Overall distribution and molecular typing summary of *Acinetobacter baumannii*

Surveillance Sites	Region	ST	Capsule Type	Overall (N=23)	Carbapenem-Resistant (n=23)	Carbapenem-Susceptible (n=0)
DLS, DMC, GMH, MAR, NMC	I, IV-A, VII, X, XI	2	KL2:OCL1(6), KL3:OCL1(7), KL49:OCL1(1), KL73:OCL1(1), KL9:OCL1(2)	17 (73.9%)	17 (73.9%)	0%
BRT, GMH	V, VII	-	KL3:OCL1(1), KL37:OCL1(3)	4 (17.4%)	4 (17.4%)	0%
CRH, JLM	III, XIII	164, 78	KL37:OCL1, KL47:OCL5	2 (8.7%)	2 (8.7%)	0%

Phylogenetic Analysis

The phylogenetic analysis of carbapenem-resistant *A. baumannii* (CRAB) as shown in Figure 153, reveals a prominent and relatively homogenous cluster composed entirely of ST2 isolates. This dominant cluster is defined by the presence of the *bla*OXA-23 carbapenemase gene. The phylogenetic tree also highlights the divergence of minor and emerging lineages that contribute to the country's complex resistance landscape. A distinct branch is formed by the ST164 isolate (25ARS-CRH0073) from Region XIII, which is unique for possessing the OCL5 outer core and carrying *bla*NDM-1 alone, lacking the otherwise ubiquitous *bla*OXA-23. Another divergent lineage includes the ST78 isolate (25ARS-JLM0130) from Region III, which harbors both *bla*OXA-23 and *bla*NDM-1. Furthermore, a separate cluster of uncategorized isolates from BRT (Region V) is visible, all sharing the KL37:OCL1 capsule type and primarily driven by *bla*NDM-1. This high-resolution visualization confirms that while the ST2/OXA-23 lineage is the stable dominant clone, the Philippine CRAB population is actively diversifying through the regional emergence of distinct sequence types carrying potent metallo- β -lactamase genes.

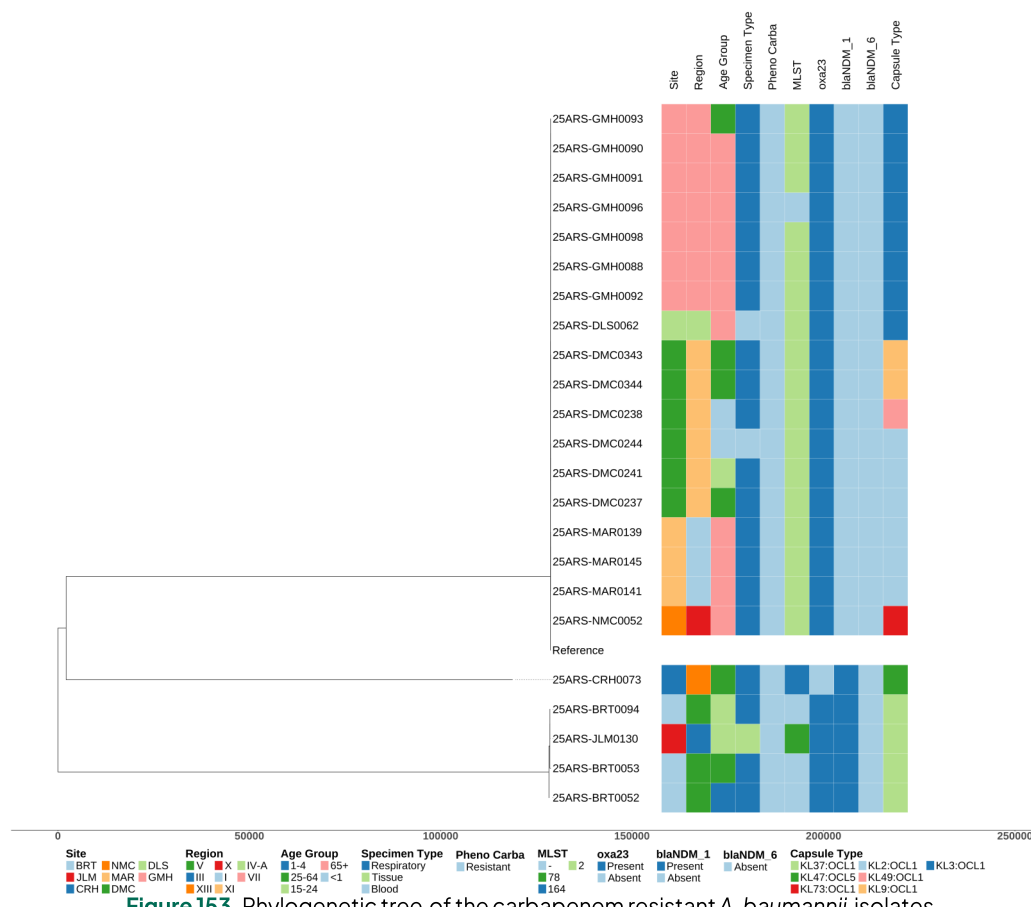


Figure 153. Phylogenetic tree of the carbapenem resistant *A. baumannii* isolates

Figure 154 shows the two-year comparison maps of carbapenemase genes detected among *A. baumannii* isolates from ARSP surveillance sites for (A) NDM-1, and (B) OXA-23

(A) blaNDM-1 Distribution

During 2024, blaNDM-1 was detected sporadically, with a total of 9 isolates identified across four surveillance sites (BGH, BRH, BRT and ZMC). Detections were primarily concentrated in Luzon, with the highest detection observed in BRH (55.6%). By 2025, blaNDM-1 shifted to 5 isolates across three (3) surveillance sites. Detection at BRT reached 60%, while new detection emerged in CRH and JLM.

(B) blaOXA-23 Distribution

blaOXA-23 served as the predominant carbapenemase, identified in 30 isolates across ten different surveillance sites. Detection frequently falls within low to moderate categories, with DMC (31-40%) being the largest contributor. In 2025, a total of 22 isolates carrying blaOXA-23 across seven surveillance sites were recorded. While the gene remains widely distributed, its presence becomes more localized to specific sites with GMH and DMC at 31-40% and 21-30%, respectively.

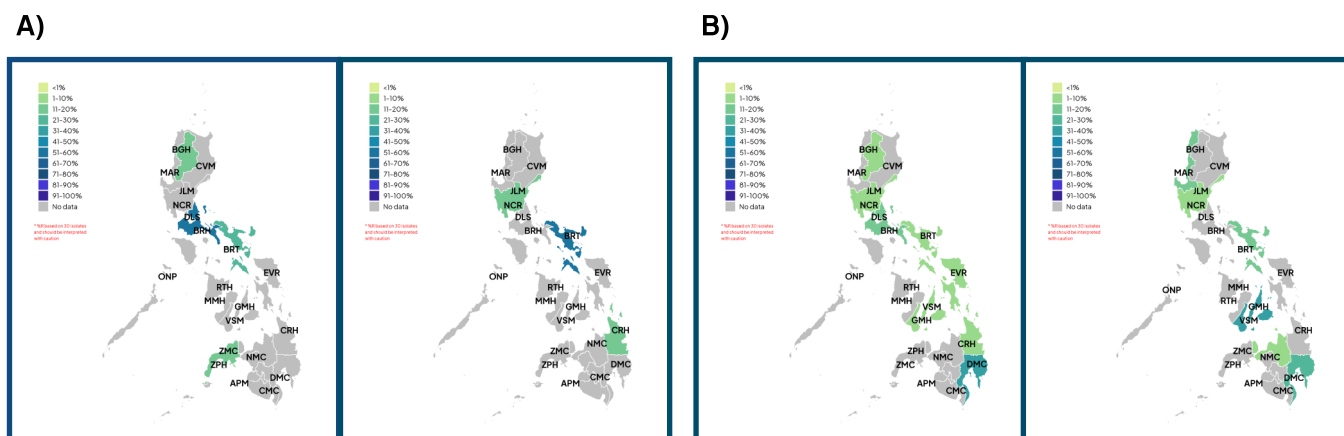


Figure 154. Two-year comparison maps of Carbapenemase genes detected among *A. baumannii* isolates from ARSP surveillance sites for (A) NDM-1, and (B) OXA-23

Discussion

In this structured genomic survey, complete concordance was observed between phenotypic carbapenem resistance and molecular predictions across the 23 characterized isolates, with *bla*OXA-23 and *bla*NDM-1 identified as the primary resistance determinants. Consistent with established national and global patterns, the carriage of *bla*OXA-23 remains the predominant driver of carbapenem-resistant *Acinetobacter baumannii* (CRAB) in the Philippines. This class D oxacillinase functions by efficiently hydrolyzing carbapenems, and its ubiquitous baseline distribution has historically driven the high endemicity of CRAB across the Philippines^[1]. The expanding detection of *bla*NDM-1 which is frequently co-occurring with *bla*OXA-23 at surveillance sites such as BRT and JLM indicates an increasing diversity in resistance profiles. Because NDM-type metallo- β -lactamases utilize a zinc-dependent catalytic mechanism to disrupt the β -lactam ring, they are fundamentally uninhibited by currently available clinical β -lactamase inhibitors, severely restricting therapeutic options to a narrow index of reserve agent. Furthermore, the detection of *bla*NDM-1 within emerging sequence types like ST164 and ST78, alongside unclassified regional clusters, suggests that this high-risk metallo- β -lactamase is actively disseminating across diverse genetic backgrounds. This inter-lineage mobilization is likely facilitated by horizontal gene transfer ^[2,3].

The broader epidemiological landscape for *A. baumannii* remains anchored by the persistence of ST2. ST2 represents the globally disseminated International Clone II (IC2), a highly successful lineage uniquely adapted to survive long periods of desiccation on healthcare surfaces and harbor massive multidrug resistance (MDR) cassettes^[4]. While its wide national distribution reflects sustained clonal expansion, the pathogen concurrently exhibits profound genomic variation. The integration of *bla*NDM-1 into specific subsets of ST2 isolates, coupled with pronounced capsular heterogeneity (KL2, KL3, KL9, KL49, and KL73 types) alongside a conserved OCL1 lipooligosaccharide outer-core locus, underscores an active state of localized micro-evolution. This phenotypic and structural diversity is mediated by recombination events at the hypervariable K-locus, allowing the bacteria to constantly alter its surface polysaccharides to evade host immune responses while maintaining its underlying metabolic fitness. Together, these findings illustrate two parallel evolutionary characteristics of the circulating strains: the structural stability of the foundational ST2 lineage and its continuous genetic diversification. This dual trajectory reinforces the vital utility of ongoing high-resolution genomic surveillance within the national sentinel network to monitor these crucial epidemiological shifts.

References

- [1] Antimicrobial Resistance Surveillance Program. 2024 Annual Report. Manila, Philippines: Antimicrobial Resistance Surveillance Reference Laboratory, Research Institute for Tropical Medicine, Department of Health; 2025. ISSN 3082-589X. <https://www.arsp.com.ph/publications/>
- [2] Joshi, P.R., Acharya, M., Kakshapati, T. et al. Co-existence of *bla*OXA-23 and *bla*NDM-1 genes of *Acinetobacter baumannii* isolated from Nepal: antimicrobial resistance and clinical significance. *Antimicrob Resist Infect Control* 6, 21 (2017). <https://doi.org/10.1186/s13756-017-0180-5>
- [3] Ramadan, R. A., et al. (2021). Genomic Characterization of Extensively Drug-Resistant NDM-Producing *Acinetobacter baumannii* Clinical Isolates With the Emergence of Novel *bla*ADC-257. *Frontiers in Microbiology*.
- [4] Palmieri, M., D'Andrea, M. M., Pelegrin, A. C., Perrot, N., Mirande, C., Blanc, B., ... & van Belkum, A. (2020). Abundance of colistin-resistant, OXA-23-and ArmA-producing *Acinetobacter baumannii* belonging to international clone 2 in Greece. *Frontiers in microbiology*, 11, 668.

Multidrug Resistant *Escherichia coli*, *Klebsiella pneumoniae*, *Pseudomonas aeruginosa*, and *Acinetobacter baumannii*

Table 29. Multidrug-resistant, extremely drug-resistant and pan-drug resistant bacteria in an international expert proposal interim standard definitions for acquired resistance

Term	Definition
MDR Multidrug-resitant	Acquired non-susceptibility to at least one agent in three or more antimicrobial categories
XDR Extensively drug-resitant	Non-susceptibility to at least one agent in all but two or fewer antimicrobial categories
PDR Pandrug-resitant	Non-susceptibility to all agents in all antimicorbial categories

Table 30. MDR and Possible XDRE. coli, K. pneumoniae, A. baumannii and P. aeruginosa infections, DOH-ARSP, 2025.

	Number of isolates tested	Percentage MDR	Percentage Possible XDR
<i>Escherichia coli</i>			
All isolates	17,248	68.1%	14.2%
Blood isolates	1,867	69.6%	13.8%
<i>Klebsiella pneumoniae</i>			
All isolates	20,973	56.9%	27.2%
Blood isolates	2,267	63.7%	37.0%
<i>Acinetobacter baumannii</i>			
All isolates	8,207	46.8%	39.6%
Blood isolates	948	45.8%	38.1%
<i>Pseudomonas aeruginosa</i>			
All isolates	11,330	21.5%	14.8%
Blood isolates	787	19.3%	13.6%

More than half of the 2025 bloodstream infections caused by *E. coli* and *K. pneumoniae* were MDR (68.3% and 63.7%, respectively). Additionally, possible XDR strains were found in 13.6% of *E. coli* and 37.0% of *K. pneumoniae* BSIs.

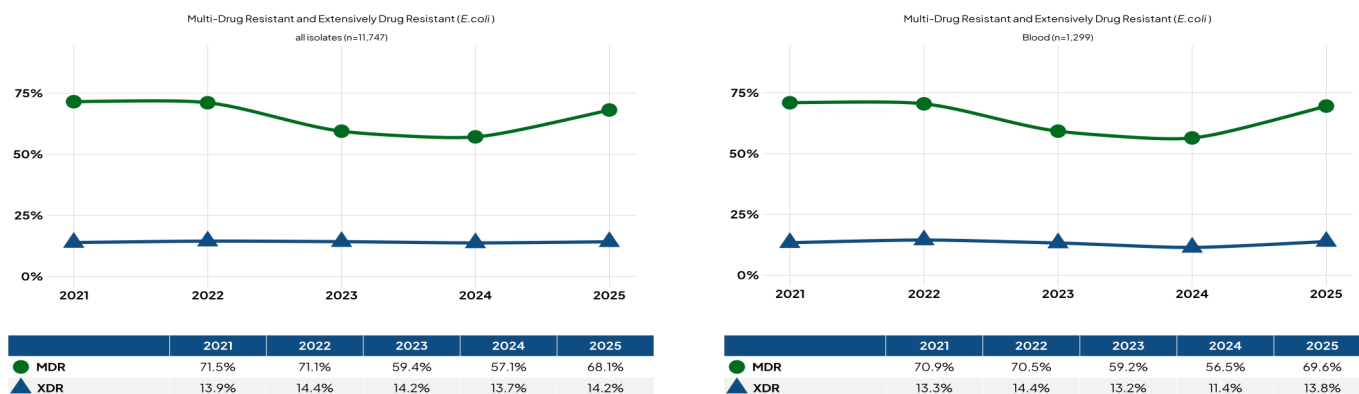


Figure 155. Yearly percentage of MDR and XDR *E. coli* infections from all specimens and blood specimens, DOH-ARSP, 2023-2025

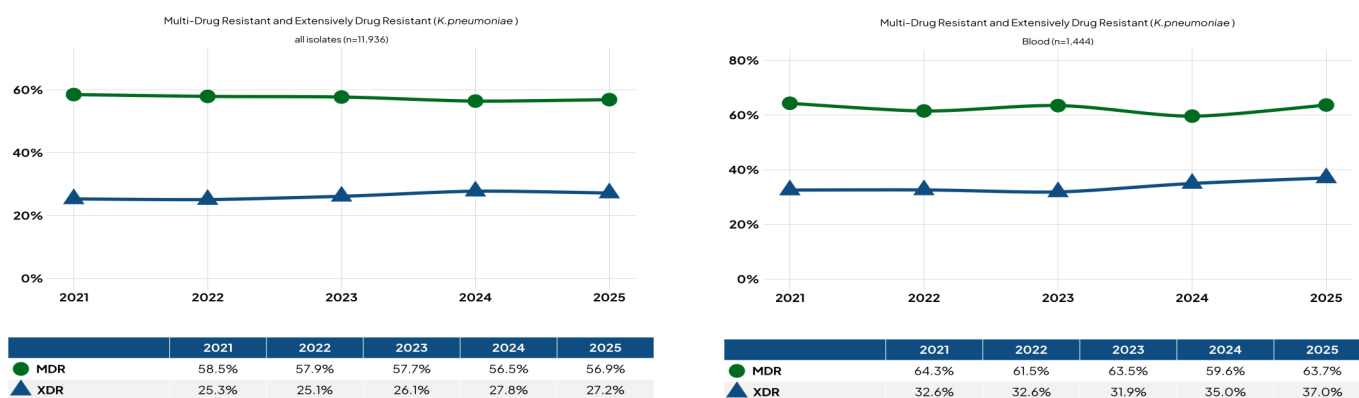


Figure 156. Yearly percentage of MDR and XDR *K. pneumoniae* infections from all specimens and blood specimens, DOH-ARSP, 2023-2025

Many of the *P. aeruginosa* BSIs are MDR (19.3%) and possible XDR (13.6%). It appears that this trend had persisted in the past decade.

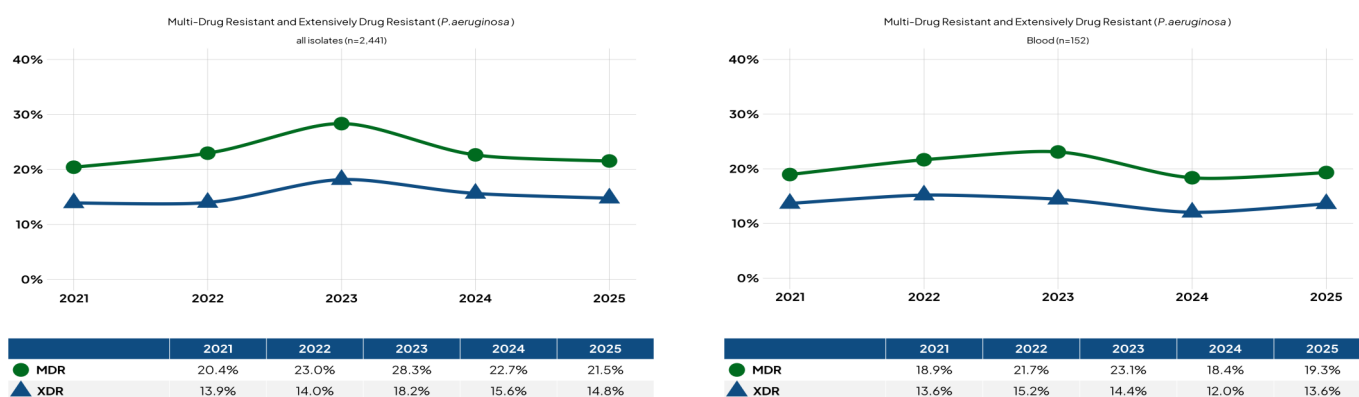


Figure 157. Yearly percentage of MDR and XDR *P. aeruginosa* infections from all and bloodstream infections, DOH-ARSP, 2016-2025

Phenotypic resistance data underscores that *Acinetobacter baumannii* is frequently resistant to multiple antibiotic classes. This is reflected in the high burden of BSIs caused by this pathogen, with 45.8% classified as MDR and 38.1% identified as potentially XDR.

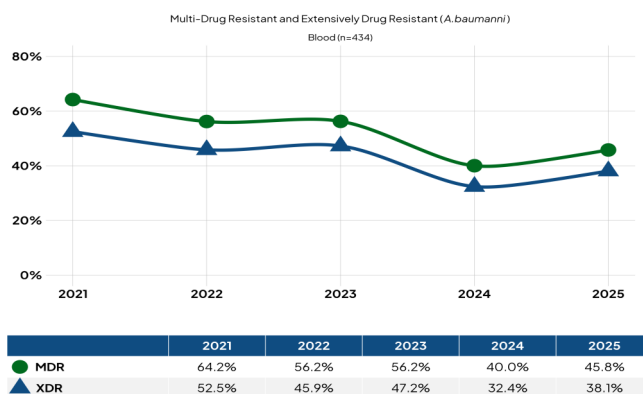
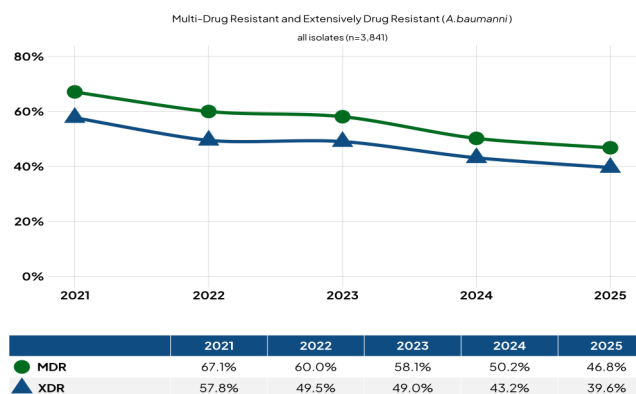


Figure 158. Yearly percentage of MDR and XDR *A. baumannii* infections from all specimens and blood specimens, DOH-ARSP, 2016–2025

Conclusions and Recommendations

Ongoing surveillance of antimicrobial resistance (AMR) among priority bacterial pathogens is critical for preventing disease and informing appropriate treatment strategies. Based on the 2025 data, the following recommendations are proposed:

Streptococcus pneumoniae

1. Penicillin continues to be an effective therapeutic option for non-meningeal *S. pneumoniae* infections, provided its use is supported by ongoing local susceptibility monitoring and surveillance data.
2. In pneumococcal meningitis, increasing penicillin resistance based on meningitis-specific breakpoints highlights the importance of susceptibility-guided therapy.
3. Third-generation cephalosporins have maintained stable, low resistance rates over the past five years, supporting their role in empiric regimens pending susceptibility results.
4. Integrating pneumococcal serotyping into AMR surveillance—complemented by genomic sequencing where feasible—is critical to evaluating the long-term efficacy of the national vaccination strategy and monitoring AMR pathogen dynamics.

Haemophilus influenzae

1. Ampicillin resistance has remained relatively stable, fluctuating between 7.3% and 13.9% over the past ten years, which continues to limit its utility for empiric treatment.
2. Third-generation cephalosporins have maintained low resistance (<5%) over the past decade, supporting their role as the preferred treatment for invasive *H. influenzae* infections.
3. Amoxicillin-clavulanic acid and ampicillin-sulbactam generally remain viable treatment options for non-invasive infections, with resistance rates consistently below 10%.

***Salmonella enterica* serovar Typhi**

1. When selecting empiric antibiotic therapy for enteric fever, clinicians should carefully consider the patient's travel history alongside local antibiotic resistance patterns should be carefully considered.
2. Locally, no multidrug-resistant (MDR) *Salmonella* infections were detected and resistance rates to most tested antibiotics remained below 10% in the past ten years.

Non-typhoidal *Salmonella* species

1. The 2025 findings indicate that while ciprofloxacin resistance in non-typhoidal *Salmonella* remains stable within established historical trends, resistance to other critical therapeutic agents—particularly ceftriaxone and ampicillin—is pronounced and outpaces rates seen in *Salmonella* Typhi, underscoring the need for tailored empiric prescribing guidelines.

Shigella

1. Due to increasing AMR, particularly to fluoroquinolones, ceftriaxone, and co-trimoxazole, susceptibility testing remains vital to guide targeted treatment for *Shigella* infections.
2. Because azithromycin resistance has remained consistently below 10% over the past five years, it represents a viable first-line option for patients eligible for oral therapy.
3. Enhancing surveillance by encouraging clinicians to routinely submit stool specimens for culture is essential to monitor dynamic resistance trends.

Vibrio cholerae

1. Ultimately, while co-trimoxazole resistance exhibited a statistically significant increase in 2025, azithromycin, tetracycline, and chloramphenicol demonstrated complete susceptibility with 0% resistance rates.
2. This underscores the continued long-term efficacy of these second-line options, particularly tetracycline, which has consistently maintained a resistance profile under 7% for the past ten years.

Neisseria gonorrhea

1. Ceftriaxone remains the recommended empiric first-line monotherapy for gonococcal infections; however, the sharp rise in resistance and emergence of extensively drug-resistant (XDR) strains across neighboring Southeast Asian nations makes enhanced surveillance urgent.
2. To effectively track these shifting patterns, clinicians should be urged to prioritize culture collection, and surveillance networks must ensure the rapid referral of isolates to the reference laboratory for confirmatory and genomic testing.

Staphylococcus aureus

1. Despite a statistically significant decrease in MRSA rates from 2016 to 2025, high rates of oxacillin resistance contin-

ue to limit its effectiveness as an empiric treatment option for *S. aureus* infections.

2. Given the relatively lower resistance rates to clindamycin and macrolides, these antibiotics could be considered as viable treatment options.
3. The judicious use of reserve antibiotics, such as vancomycin and linezolid, remains critical to preserving their long-term efficacy against *S. aureus*.

***Enterococcus* sp.**

1. *Enterococcus faecalis*: Remains largely susceptible to nitrofurantoin (<5%), but risks are emerging due to a notable increase in high-level gentamicin resistance (25.84%) and the isolated detection of a vancomycin-resistant strain.
2. *Enterococcus faecium*: Shows a favorable, statistically significant long-term decline in vancomycin resistance, contrasted by a significant increase in nitrofurantoin resistance and fluctuating aminoglycoside susceptibility.
3. Linezolid resistance remains clinically negligible (<1%) and stable, serving as a reliable salvage therapy for resistant enterococcal infections.

Escherichia coli

1. *E. coli* isolates display high, stable resistance to fluoroquinolones and third-generation cephalosporins (~39%–46%), driven by a rising ESBL rate (33.68%) that demands scaled antimicrobial stewardship to meet national SDG targets.
2. Carbapenems (~10% resistance) and colistin (<2%) continue to maintain relatively stable efficacy profiles compared to standard frontline therapeutics.
3. Initial resistance rates for novel combinations—such as ceftazidime-avibactam (28.02%) and imipenem-relebactam (60%)—were moderate to high, a phenotype primarily driven by the production of carbapenemases, particularly New Delhi metallo-beta-lactamases (NDMs).

Klebsiella pneumoniae

1. *K. pneumoniae* isolates show persistently high resistance to first-line treatment options, cephalosporins, fluoroquinolones, and beta-lactamase inhibitors, compounded by a gradual ten-year increase in carbapenem resistance (17%–18% in 2025) and piperacillin-tazobactam resistance (increasing to 35.4%).
2. The ESBL rate has remained stable above 30% since 2022 (reaching 31.2% in 2025); however, multi-year analysis indicates that this decade-long trend is not statistically significant.
3. While colistin maintains low overall resistance at 4%, novel combinations—such as ceftazidime-avibactam (32.44%) and imipenem-relebactam (37.5%)—already display relatively high resistance rates, suggesting the widespread circulation of challenging carbapenemase mechanisms.

Pseudomonas aeruginosa

1. *P. aeruginosa* isolates demonstrated statistically significant drops in resistance across multiple therapeutic classes in 2025, including cefepime (down to 11.9%), fluoroquinolones (down to 12.0%), and meropenem (down to 12.7%).
2. In contrast to the declining trends of other first-line therapies, piperacillin-tazobactam resistance remained relatively stable, showing a minor, statistically non-significant upward shift to 19.7%.
3. Resistance rates for novel combinations varied widely—ranging from a low of 6.86% for ceftolozane-tazobactam to a high of 41.51% for imipenem-relebactam—though these initial metrics warrant cautious interpretation due to limited sample sizes.

Acinetobacter baumannii

1. *A. baumannii* isolates maintained elevated resistance rates exceeding 43% across nearly all major antimicrobial classes in 2025, including cefepime, piperacillin-tazobactam, meropenem, and ampicillin-sulbactam.
2. Despite high resistance rates, multi-year analysis reveals statistically significant downward trends in resistance over time for core agents, most notably carbapenems (meropenem, imipenem), aminoglycosides (amikacin, gentamicin, tobramycin), co-trimoxazole, and levofloxacin.
3. Conversely, a critical public health concern emerged with a sharp, statistically significant spike in colistin resistance, which increased from 0.4% in 2024 to 3.5% in 2025 (p=0.0000), signaling a threat to last-line therapeutic options.

Program Future Directions

1. Pursue ARSP expansion through gradual addition of surveillance sites taking into consideration feasibility, geographical location and hospital types.
2. Expand susceptibility testing across all surveillance sites for newer combination antibiotics such as ceftazidime-avibactam, ceftolozane-tazobactam, meropenem-vaborbactam, cefiderocol among gram-negative rods.
3. Implement structured genomic surveys among the common pathogens of public health importance as part of the continuous and expanded integration of whole genome sequencing (WGS) into ARSP.
4. Pursue fostering of continued growth of technical expertise and skills in molecular diagnostics and bio-informatics within the reference laboratory through training, collaborative projects and advocacy for requisite fund and resource requirements.
5. The detection of case clustering in ARSP surveillance sites will be improved through the application of WHONET SaTScan analysis to ARSP data, contingent upon encouraging prompt data transfer from these sites. Furthermore, the investigation of potential outbreaks within these surveillance sites will be enhanced via close coordination with their respective Infection Prevention Committees and the implementation of WGS.
6. Actively engage in the One Health approach to AMR surveillance together with relevant stakeholders from the food chain and environmental sectors including pursuing implementation of the Tricycle Project.
7. Actively contribute and participate in the implementation of the Philippine Action Plan to Combat Antimicrobial Resistance.
8. Advocate for and implement relevant AMR surveys, studies and researches to inform the policies towards the control and prevention of the emergence and spread of AMR in the country.
9. Generate more relevant collaborative and investigator-initiated research. Continue to ensure the high quality of surveillance data through active capacity building of surveillance sites and reference laboratory staff, robust efforts to improve facilities, and safeguard availability of resources, equipment and services.
10. Incorporate the technology of geographic information system and mapping in surveillance.
11. Pursue ISO 15189 and ISO 17043 accreditation for the reference laboratory.
12. Explore in-vitro diagnostic product development informed by genomic data of local resistant pathogens.

References

- [1] World Health Organization. (2023, November 21). Antimicrobial resistance. <https://www.who.int/news-room/fact-sheets/detail/antimicrobial-resistance>.
- [2] Department of Health (Philippines). (2024). Philippine National Action Plan on Antimicrobial Resistance 2024–2028. Manila: Department of Health.
- [3] World Health Organization. (2025). Global antimicrobial resistance and use surveillance system (GLASS) report 2025. Geneva: World Health Organization. <https://www.who.int/publications/i/item/WHO-GLASS-2025>
- [4] Clinical and Laboratory Standards Institute. (2025). Performance Standards for Antimicrobial Susceptibility Testing (35th ed., CLSI supplement M100). Wayne, PA: Clinical and Laboratory Standards Institute.
- [5] World Health Organization. (2024). WHO bacterial priority pathogens list, 2024: Bacterial pathogens of public health importance, to guide research, development and strategies to prevent and control antimicrobial resistance. <https://iris.who.int/items/dbc5f2d3-6d22-46d3-881c-b5e8c119d6c4>
- [6] Murray, P. R., Rosenthal, K. S., & Pfaller, M. A. (Eds.). (2023). Manual of clinical microbiology (13th ed.). American Society for Microbiology Press.
- [7] O'Brien TF, Stelling JM. WHONET: An information system for monitoring antimicrobial resistance. Emerg Infect Dis. 1995;1:66. doi: 10.3201/eid0102.950209.
- [8] Petit, R. A. & Read, T. D. Bactopia: a Flexible Pipeline for Complete Analysis of Bacterial Genomes. mSystems 5, (2020)
- [9] Seemann T Shovill: De novo assembly pipeline for Illumina paired reads (GitHub)
- [10] Bankevich A, Nurk S, Antipov D, Gurevich AA, Dvorkin M, Kulikov AS, Lesin VM, Nikolenko SI, Pham S, Pribelski AD, Pyshkin AV, Sirotkin AV, Vyahhi N, Tesler G, Alekseyev MA, Pevzner PA SPAdes: a new genome assembly algorithm and its applications to single-cell sequencing. Journal of computational biology 19.5 455–477 (2012)
- [11] Seemann T Prokka: rapid prokaryotic genome annotation Bioinformatics 30, 2068–2069 (2014)
- [12] Ondov BD, Starrett GJ, Sappington A, Kostic A, Koren S, Buck CB, Phillippy AM Mash Screen: high-throughput sequence containment estimation for genome discovery Genome Biol 20, 232 (2019)
- [13] Seemann T mlst: scan contig files against PubMLST typing schemes (GitHub)
- [14] Jolley KA, Bray JE, Maiden MCJ Open-access bacterial population genomics: BIGSdb software, the PubMLST.org website and their applications. Wellcome Open Res 3, 124 (2018)
- [15] Feldgarden, M., Brover, V., Gonzalez-Escalona, N. et al (2021) . AMRFinderPlus and the Reference Gene Catalog facilitate examination of the genomic links among antimicrobial resistance, stress response, and virulence. Sci Rep 11, 12728. <https://doi.org/10.1038/s41598-021-91456->
- [16] Beghain J, Bridier-Nahmias A, Le Nagard H, Denamur E, Clermont O. ClermonTyping: an easy-to-use and accurate in silico method for Escherichia genus strain phylotyping. Microbial Genomics, 4(7), e000192. (2018)
- [17] Laing C, Bessonov K, Sung S, La Rose C ECTyper – In silico prediction of Escherichia coli serotype (GitHub)
- [18] Epping L, van Tonder AJ, Gladstone RA, The Global Pneumococcal Sequencing Consortium, Bentley SD, Page AJ, Keane JA SeroBA: rapid high-throughput serotyping of Streptococcus pneumoniae from whole genome sequence data. Microbial Genomics, 4(7) (2018)

- [19] Lam MMC, Wick RR, Watts, SC, Cerdeira LT, Wyres KL, Holt KE A genomic surveillance framework and genotyping tool for *Klebsiella pneumoniae* and its related species complex. Nat Commun 12, 4188 (2021)
- [20] Petit III RA pasty: *in silico* serogrouping of *Pseudomonas aeruginosa* isolates (GitHub)
- [21] Yoshida CE, Kruczkiewicz P, Laing CR, Lingohr EJ, Gannon VPJ, Nash JHE, Taboada EN The *Salmonella* In Silico Typing Resource (SISTR): An Open Web-Accessible Tool for Rapidly Typing and Subtyping Draft *Salmonella* Genome Assemblies. PloS One, 11(1), e0147101. (2016)
- [22] Sanchez-Herrero JF, and Sullivan M spaTyper: Staphylococcal protein A (spa) characterization pipeline. Zenodo. (2020)
- [23] Petit III RA, Read TD *Staphylococcus aureus* viewed from the perspective of 40,000+ genomes. PeerJ 6, e5261 (2018)
- [24] Wyres, K.L., Cahill, S.M., Holt, K.E., Hall, R.M., Kenyon, J.J., 2020. Identification of *Acinetobacter baumannii* loci for capsular polysaccharide (KL) and lipooligosaccharide outer core (OCL) synthesis in genome assemblies using curated reference databases compatible with Kaptive. Microbial Genomics 6. <https://doi.org/10.1099/mgen.0.000339>
- [25] Seemann T Snippy: fast bacterial variant calling from NGS reads (GitHub)
- [26] Seemann T snp-dists - Pairwise SNP distance matrix from a FASTA sequence alignment. (GitHub)
- [27] B.Q. Minh, H.A. Schmidt, O. Chernomor, D. Schrempf, M.D. Woodhams, A. von Haeseler, R. Lanfear (2020) IQ-TREE 2: New models and efficient methods for phylogenetic inference in the genomic era. Mol. Biol. Evol., 37:1530-1534. <https://doi.org/10.1093/molbev/msaa015>
- [28] Harry C. H. Hung, Narender Kumar, Victoria Dyster, Corin Yeats, Benjamin Metcalf, Yuan Li, Paulina A. Hawkins, Lesley McGee, Stephen D. Bentley, and Stephanie W. Lo. GPS Pipeline: portable, scalable genomic pipeline for *Streptococcus pneumoniae* surveillance from Global Pneumococcal Sequencing Project. Nat Commun 16, 8345 (2025). <https://doi.org/10.1038/s41467-025-64018-5>

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