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ANTIMICROBIAL RESISTANCE SURVEILLANCE PROGRAM

Antimicrobial Resistance Surveillance Program 2024 Annual Report

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Editorial Information

This edition of the ARSP Annual Report was published through the efforts of the Antimicrobial Resistance Surveillance Reference Laboratory (ARSRL) under the supervision of **Dr. Sonia B. Sia**, Head of the ARSRL and Chair of the Committee on Antimicrobial Resistance Surveillance Program and **Dr. Pamela T. Tan**, Deputy Department Head of the ARSRL.

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TABLE OF CONTENTS

List of Figures

List of Tables

Preface

Sonia B. Sia, MD

Chair, Committee on Antimicrobial Resistance Surveillance Program
Department Head, Antimicrobial Resistance Surveillance Reference Laboratory
Department of Health

In this 37th installment of the Antimicrobial Resistance Surveillance Program (ARSP), the Antimicrobial Resistance Surveillance Reference Laboratory (ARSRL) reaffirms its strong commitment to the mandate of providing AMR surveillance data to contribute to the Department of Health's efforts to promote rational drug use. The data contained in this report offer insight to the state and trends of AMR among select bacterial infections in the country as well as the emergence of new resistant phenotypes.

In the 2024 ARSP Annual Report, key shifts were made in the surveillance data analysis and AMR data presentation - from isolate-based to sample-based analysis and the presentation of AMR data in the context of infections occurring among patients. The presentation of AMR data as ***proportion of infections with resistance to tested antibiotics*** is premised on the assumptions that the submitted microbiological data truly relates to an episode of illness based on diagnostic criteria and that the growth of a pathogen in selected specimens is as a proxy for patient infection in associated anatomical sites. Although premised on assumptions and fully recognizing the challenge of distinguishing between colonization, contamination, and true infection, it is the hope that with the above mentioned AMR data presentation, aside from serving as surveillance data which provides insight towards the evaluation of ongoing interventions against AMR at the national and possibly at health facility specific level, ARSP data may also draw more focus on patients who are affected by infectious diseases and are invariably the most vulnerable to AMR.

As in previous years, the ARSP 2024 data shows that, resistance rates for *S. pneumoniae* and *H. influenzae* infections remained relatively low to most antibiotics. The same is observed among *Salmonella* Typhi infections as well as diarrheal diseases caused by *Shigella sp.* and *V. cholerae*. Further, no gonorrhea infections were noted to be resistant to ceftriaxone, cefixime nor azithromycin. On the other hand, high MDR and possible XDR rates were observed in gram-negative bacterial infections with ESBL positivity rates for *E. coli* and *K. pneumoniae* of 26.66 % and 30.99% respectively.

There was a noted persistence of sporadic occurrence of emerging resistant infections such as meropenem resistant *S. pneumonia*, vancomycin- and linezolid-resistant *Enterococcus faecalis*, and colistin resistant *E. coli, K. pneumoniae, P. aeruginosa*, and *A. baumannii* infections. There was, likewise, a notable increase in the number of amoxicillin-clavulanic acid-resistant *H. influenzae* infections.

The ARSP 2024 report provides data on the monitoring of the AMR indicators of the Sustainable Development Goal (SDG). On indicator 3.d: proportion of patients with MRSA bloodstream infections (BSI), ARSP data shows decreasing proportions of MRSA BSIs, from 47.1% in 2020 to 38.4% in 2024. On the other hand,

on indicator 3d: proportion of patients with BSIs caused by *E. coli* resistant to third-generation cephalosporins, ARSP data showed an increase in the proportion from rates in 2020 to the proportions in 2024.

For 2024, using a standard sampling protocol, ARSP conducted surveys of select resistant pathogens, namely methicillin-resistant *Staphylococcus aureus*, vancomycin-resistant *Enterococcus faecalis*, carbapenem & extended spectrum cephalosphorin resistant *Escherichia coli* & *Klebsiella pneumoniae*, and carbapenem resistant *Acinetobacter baumannii* & *Pseudomonas aeruginosa*. Aside from determining the occurrence and geographic distribution of these resistant bacteria, whole genome sequencing (WGS) of the isolates were performed to determine the population dynamics and elucidate the AMR mechanisms among these isolates. Understanding the mechanism conferring resistance may inform possible treatment options and infection control measures to mitigate the spread of these isolates. For example, the detection of carbapenemase genes *blaNDM-1, blaNDM-5*, and *blaNDM-7* as seen in over 90% of carbapenem resistant *K. pneumoniae* (CRKP) isolates in the studied cohort potentially limits the use of current β-lactamase inhibitors such as avibactam or vaborbactam among such infections. Further, among infections caused by *E. coli* and *K. pneumoniae*, the detection of mobile *mcr1.1* genes conferring resistance to colistin among our local isolates indicates the need for timely detection of these genes and the observance of appropriate containment measures to mitigate the spread of colistin resistance among the Enterobacterales . With directed continuous conduct of the survey, we look forward to enhanced timely understanding of the AMR mechanisms, i.e. whether driven by mobile genetic elements, chromosomal mutations, or through introduction or importation of new strains.

The enhancements in the 2024 ARSP report provides more focused perspective and more granular surveillance data. As such, it is our wish that this report may significantly contribute to the collective efforts towards addressing AMR in the country.

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Executive Summary

Streptococcus pneumoniae

The cumulative resistance rate of *S. pneumoniae* infections to penicillin, based on the non-meningitis breakpoint, remains low at 1.44% (n=478). This indicates that penicillin continues to be an effective treatment option for non-meningitis infections. Resistance to alternative antibiotics also remains below 10%: ceftriaxone (non-meningitis breakpoint) at 1.62%, ceftriaxone (meningitis breakpoint) at 1.95%, clindamycin at 6.46%, and levofloxacin at 0.70%. Compared with 2023, the overall resistance rates for all *S. pneumoniae* infections are higher in 2024, with the exceptions of penicillin, co-trimoxazole, and meropenem.

Haemophilus influenzae

In 2024, *H. influenzae* had an ampicillin resistance rate of 13.95%, an increase from 11.9% in 2023. These infections largely remained susceptible to ceftriaxone, with a non-susceptibility rate of 0% in 2024. Resistance to ampicillin/sulbactam was 8.7%, and to amoxicillin/clavulanic acid was 7.35%. No resistance to azithromycin or levofloxacin.

Salmonella enterica serovar Typhi

The resistance rates of *S.* Typhi infections to ciprofloxacin, imipenem, meropenem were at 1.37%, 0% and 1.52% respectively. Resistance to co-trimoxazole was at 1.12% while no resistance to azithromycin was reported. The yearly resistance rates of all *S.* Typhi infections to all antibiotics used for treatment have remained low in the past ten years. However, significant changes were observed for co-trimoxazole (p= 0.0135) and azithromycin (p=0.0121).

Non-typhoidal *Salmonella* species

As in the past years, resistance of non-typhoidal *Salmonella* to ampicillin (38.23%), co-trimoxazole (14.06%) and azithromycin (22.22%) are higher compared to that of *Salmonella* Typhi. Resistance to ciprofloxacin remained within the 9-12% range in the past seven years with 2024 resistance at 9.8%.

Shigella species

Ampicillin and co-trimoxazole resistance rates have remained elevated, exceeding 50% over the past ten years. Proportion of *Shigella* diarrheal diseases resistant to ceftriaxone decreased in 2024 but the proportion resistant to ciprofloxacin increased. Azithromycin, however, has maintained a resistance rate of less than 10% over the same five-year period.

Vibrio cholerae

Tetracycline resistance was observed at 1.43% in 2024, with rates remaining 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.

Neisseria gonorrhea

Antimicrobial resistance (AMR) in *Neisseria gonorrhoeae* is a global public health concern, particularly threatening ceftriaxone, the last recommended first-line treatment for gonorrhea. Despite reports of third-generation cephalosporin resistance in some Southeast Asian countries [1], no local resistance to ceftriaxone or alternative regimens (cefixime and azithromycin) was reported in 2024. Conversely, resistance to tetracycline and ciprofloxacin remained notably high in 2024, at 78.40% (n=125) and 89.76% (n=127) respectively.

Staphylococcus aureus

Antimicrobial resistance (AMR) indicator 3.d of the Sustainable Development Goals (SDG) tracks the proportion of patients with MRSA bloodstream infections (BSI). MRSA remains a major global health burden, causing significant morbidity, mortality, and healthcare costs. A continued decrease in oxacillin resistance among *Staphylococcus aureus* has been observed over the past five years, with rates from 47.5% in 2020 to 36.6% in 2024. Erythromycin resistance was observed at 11.4% and clindamycin at 9.86%, with both rates appearing stable for the past ten years. Although there were re-ported vancomycin resistance in 2024, this was not con-firmed in the reference laboratory.

Enterococcus species

Resistance rates for *E. faecalis* were observed at 13.08% for penicillin and 8.40% for ampicillin. High-level gentamicin and streptomycin resistance were both at 17.17% and 16.58%, respectively. Vancomycin resistance was recorded at 2.76% and linezolid at 2.65%. Compared to 2023, a statistically significant decrease was noted in resistance rates for penicillin and vancomycin, while a statistically significant increase was observed for ampicillin.

In 2024, *Enterococcus faecium* continued to exhibit high antibiotic resistance, with rates of 22.54% for vancomycin and 50.43% for nitrofurantoin.

Escherichia coli

The global concern on third-generation cephalosporin-resistant Enterobacterales (3GCRE) is reflected in persistently high local resistance rates. In 2024, resistance to these critical antibiotics remained above 30% over the past five years, with cefotaxime at 38.5% and ceftriaxone at 37.6% in 2024. This elevated resistance, particularly in bloodstream infections, is a critical indicator within the SDG monitoring framework. It poses a significant concern, especially in pediatric populations, where the increasing prevalence of 3GCRE in neonatal sepsis is alarming due to its association with higher morbidity and mortality, particularly in LMICs [2]. Such high local resistance rates need targeted policies and interventions to address the limited treatment options and this emerging threat.

Klebsiella pneumoniae

Local ESBL rates for *K. pneumoniae* increased to 30.99% in 2024 from 31.80% in 2023. Similar to *E. coli*, resistance to most third-generation cephalosporins is high (over 40%), with piperacillin-tazobactam at 37.36% and fluoroquinolones over 25%. Carbapenem resistance among *K. pneumoniae* infections also remained elevated in 2024, with rates exceeding 15%. Carbapenem-resistant *K. pneumoniae* (CRKP) poses a major public health threat, strongly associated with high mortality, particularly in immunocompromised and critically ill patients. Treatment options for CRKP infections are severely limited, often relying on last-resort drugs like colistin and tigecycline.

Pseudomonas aeruginosa

P. aeruginosa showed MDR rates of 22.7% and XDR rates of 15.6%. These figures represent a decrease from 2023 rates of 28.3% and 18.2%, respectively. Resistance to ceftazidime was 15.37% and to piperacillin/tazobactam was 18.78%. A statistically significant decrease was noted in the resistance rates for cefepime and tobramycin. Aztreonam resistance was 14.0%, and fluoroquinolone resistance exceeded 10%. Carbapenem resistance was reported at 16.63% for imipenem and 13.92% for meropenem.

Acinetobacter baumannii

Acinetobacter baumannii poses a significant global threat due to its virulence, extensive resistance, and limited treatment options. In 2024, *A. baumannii* infections showed resistance above 40% for most antibiotics [2]. Key antibiotics like cefepime (49.4%), meropenem (49.2%), ampicillin-sulbactam (42.0%), fluoroquinolones, and co-trimoxazole all had resistance rates over 40% resulting to limited treatment options locally. Colistin resistance remained very low at 0.44%, and minocycline at 5.34%.

Sources:

[1] Ouk, V., Heng, L. S., Virak, M., Deng, S., Lahra, M. M., Frankson, R., Kreisel, K., McDonald, R., Escher, M., Unemo, M., Wi, T., Maatouk, I., Penh, P., Fensham, V., Kersh, E., Cavailler, P., Mundade, Y., van Hal, S. J., Kundu, R. L., … Nishijima, T. (2024). High prevalence of ceftriaxone-resistant and XDR *neisseria gonorrhoeae* in several cities of Cambodia, 2022–23: Who enhanced gonococcal antimicrobial surveillance programme (EGASP). JAC-Antimicrobial Resistance, 6(2). https://doi.org/10.1093/jacamr/dlae053

[2] WHO bacterial priority pathogens list 2024: Bacterial pathogens of public health importance, to guide research, development, and strategies to prevent and control antimicrobial resistance. (2024). World Health Organization.

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** 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 2024-2023[2] reiterates the importance of surveillance as it identifies the strengthening of surveillance and laboratory capacity as among its key strategies.

The culture and susceptibility reports submitted into the program were noted to have increased by 3.79% compared with the reports submitted in 2023. This difference must be kept in mind in the interpretation of the surveillance data in the past two years.

Historically, the ARSP Annual Report has presented Philippine antimicrobial resistance data using an isolate-based approach. To enhance the clinical utility of its data, ARSP has incorporated several recommendations from the 2023 World Health Organization's (WHO) Global Antimicrobial Resistance and Use Surveillance System (GLASS) module, prompting a shift towards sample-based data. Isolate-based data, a subset of sample-based reporting, specifically tracks laboratory-confirmed infections from defined target pathogens under surveillance, revealing the proportion of patients with antimicrobial-resistant infections. Sample-based data, however, provides a broader overview of all pathogens identified in collected specimens. This offers valuable insight into the **frequency of infection** and resistance patterns within the tested population, categorized by **specimen type**.

Sample-based data covers the entire patient population with suspected infections from whom clinical specimens were collected. This includes patients with laboratory-confirmed target pathogen infections, those with no microbial growth, and positive samples with the growth of other organisms (both pathogens and commensals). Consequently, this approach facilitates the calculation of frequency of infection for different pathogens.

This approach also considers **the growth of a pathogen in selected specimens as a proxy for patient infection in associated anatomical sites**, provided the submitted microbiological data truly relates to an episode of illness based on diagnostic criteria.

With this, instead of reporting resistance rates of isolates, we will be reporting the resistance rates of pathogens as the disease associated with the specimen type. We also included the prevalence of an infection type of the specific pathogen per surveillance site, to give insight on the frequency of the pathogens in the 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.

To maximize the clinical utility of surveillance data from lower respiratory samples, a subset analysis was conducted for relevant **lower respiratory tract infection (LRTI) pathogens** (*S. pneumoniae, H. influenzae, K. pneumoniae*, and *P. aeruginosa*). Secondary bacterial pneumonia is a recognized complication of post-viral influenza-like illness. Similarly, during the COVID-19 pandemic, a considerable number of hospitalized SARS-CoV-2 patients experienced significant morbidity and mortality associated with secondary bacterial infections.

Additionally, sentinel sites are now referred to as surveillance sites.

SURVEILLANCE, TESTING METHODS, DATA ANALYSIS & LIMITATIONS

The Surveillance

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

Currently participating in the program are 24 surveillance sites, and 2 gonococcal surveillance sites, representing 16 out of the 17 regions of the country (**Figure 1** & **Table 1**).

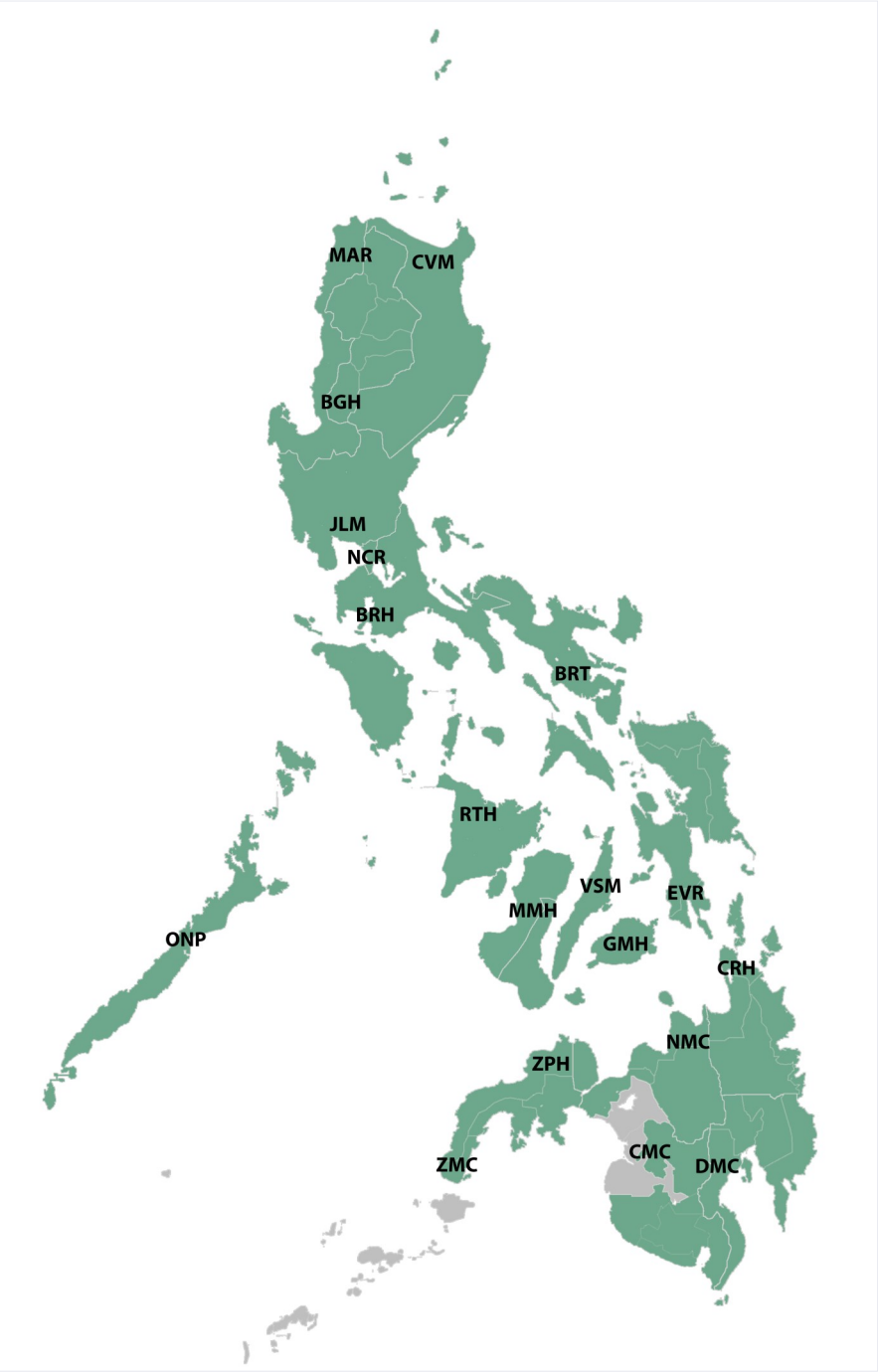


Figure 1. Regional representation in the ARSP 2024

Table 1. ARSP 2024 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 Far Eastern University Nicanor Reyes Medical Foundation Medical Center
Cordillera Administrative Region (CAR)	Baguio General Hospital and 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
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

Legend: CALABARZON: Cavite, Laguna, Batangas, Rizal, Quezon; MIMAROPA: Mindoro, Marinduque, Romblon, Palawan; SOCCSKSARGEN: South Cotabato, Cotabato, Sultan Kudarat, Sarangani, General Santos City.

The surveillance collects data on culture and antimicrobial susceptibility from its 24 surveillance sites and 2 gonococcal 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 culture and AST results 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, routine culture and AST results 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. gonorrhea* which requires manual AST methods. Serotyping using whole genome sequencing (WGS) for *S. pneumoniae, H. influenzae, Salmonellae, Shigellae* and *Vibrio cholera* was done for 2024.

Further, 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 Wizard® Genomic DNA Purification Kit (Promega, Madison, WI, USA), following the manufacturer's instructions. DNA quality and concentration were assessed using a Qubit fluorometer (Thermo Fisher Scientific, Waltham, MA, USA). Sequencing libraries were prepared using the Illumina DNA Prep Kit (Illumina, San Diego, CA, USA), and whole genome sequencing was performed on the Illumina MiSeq platform, generating 2 × 150 base pair (bp) 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.

Bioinformatics Methods

Short-read WGS data were processed with Bactopia v3.2.0. 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; Seroba v1.0.2 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 v4.0.19 (database 2024-12-18.1). A complete list of software and versions is 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 test. 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 2024 ARSP Data

Resistance data for 123,949 isolates were reported and analyzed for 2024. A 3.79% increase was observed when compared to the reported 119,398 isolates in 2023. **Table 2** shows that 23 of 24 sites had an increase in data submission for 2024.

The 2024 ARSP data were collected from all 24 sites and 1 *N. gonorrhoeae* surveillance site of the program which represents data from 16 of 17 regions in the Philippines. More than half (58.55%) of the reported infections were from Luzon, 21.84% from Mindanao and 19.61% from Visayas (**Figure 2**). More than half (51.06%) were among male patients and from the 20-64 age group (60.31%). The most common infections were respiratory (26.09%), blood (23.38%) and urine (21.98%). **Table 3** shows the most common isolates by specimen type.

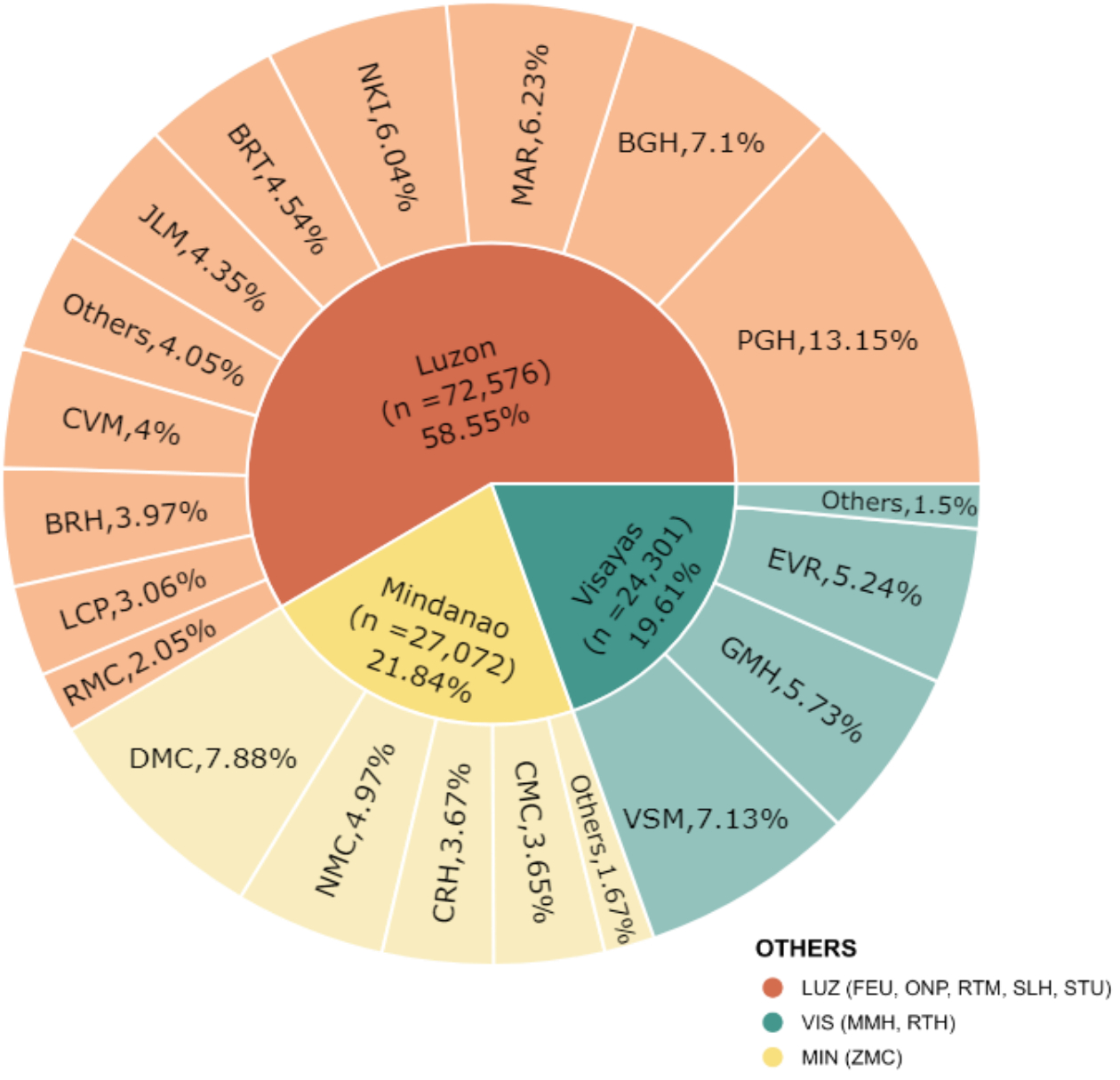


Figure 2. ARSP surveillance sites contribution , DOH-ARSP, 2024 (n=123,949)

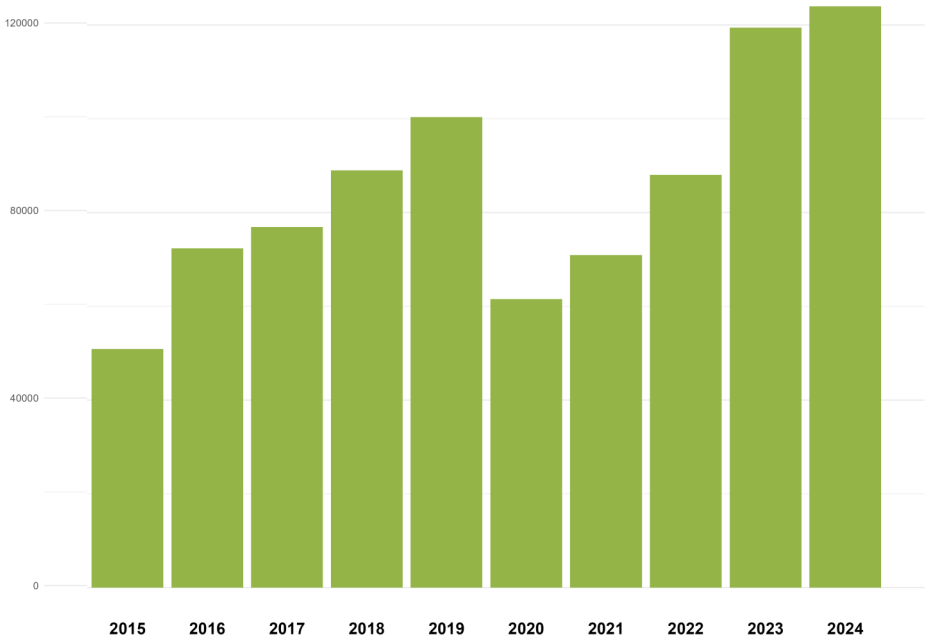


Figure 3. Number and percentage difference of isolates received from 2015-2024

Table 2. Surveillance sites isolate contribution, DOH-ARSP, 2015-2024

Surveillance Sites	2014	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024	Change %
BGH	2625	3214	4628	4842	5775	5234	2968	3191	5459	8385	8796	4.67
BRH	1022	1294	2075	2472	3133	3633	1569	1472	2570	3133	4924	36.37
BRT	1047	1251	1584	1640	1842	2521	1176	1903	3055	4131	5628	26.60
CMC	833	1300	1599	1704	2642	3181	2076	2021	3257	4509	4526	0.38
CRH	-	-	10	624	879	1205	1115	1314	2075	3885	4551	14.63
CVM	1223	1512	3473	4141	4276	5668	3782	2687	4207	6114	4961	-23.24
DMC	4062	5109	8058	8680	10762	12177	7412	8189	9135	11613	9767	-18.90
EVR	823	1514	1731	3303	3879	3874	4056	4584	5388	5793	6491	10.75
FEU	956	712	810	1201	1173	548		322	413	785	745	-5.37
GMH	1351	1807	1669	3153	3258	3957	2624	2818	4254	6752	7106	4.98
JLM	638	1266	2768	3261	3880	4824	3248	3753	5229	5890	5387	-9.34
LCP	2921	2905	3115	1367	3098	4433	2713	3205		3999	3799	-5.26
MAR	1706	1849	2759	3565	4293	4462	3581	3302	4492	6416	7718	16.87
MMH	2289	2940	2886	3133	3026	2539	1425	1140	1575	2062	1858	-10.98
NKI	2918	1455	5894	627	2959	4358		5571	5911	6719	7488	10.27
NMC	2416	2237	3105	2245	2961	3409	3735	2780	4868	6893	6160	-11.90
ONP	-	-	2	5	68	90	13	31	202	204	423	51.77
PGH	12471	11710	12860	14572	12808	13895	6818	9557	12343	15906	16304	2.44
RMC	320	1054	3252	3160	3241	2375	2027	1968	2298	2895	2545	-13.75
RTH	-	-	25	69	159	352	289	0	4	5	5	0.00
RTM	303	336	410	513	598	507	255	423	528	555	706	21.39
SLH	575	824	1410	2460	2044	2371	1019	1157	1060	1352	1468	7.90
STU	2002	1923	2275	2088	2184	2722	1419	1250	854	1122	1684	33.37
VSM	3951	3834	4803	6838	8714	10286	6886	6971	7596	8390	8841	5.10
ZMC	819	841	1142	1222	1346	1644	1192	1341	1276	1913	2068	7.50
ZPH	9	8	4	7	3	69	129	1	0	0	0	
TOTAL	**47280**	**50895**	**72347**	**76892**	**89001**	**100334**	**61527**	**70951**	**88049**	**119421**	**123949**	**35.63**

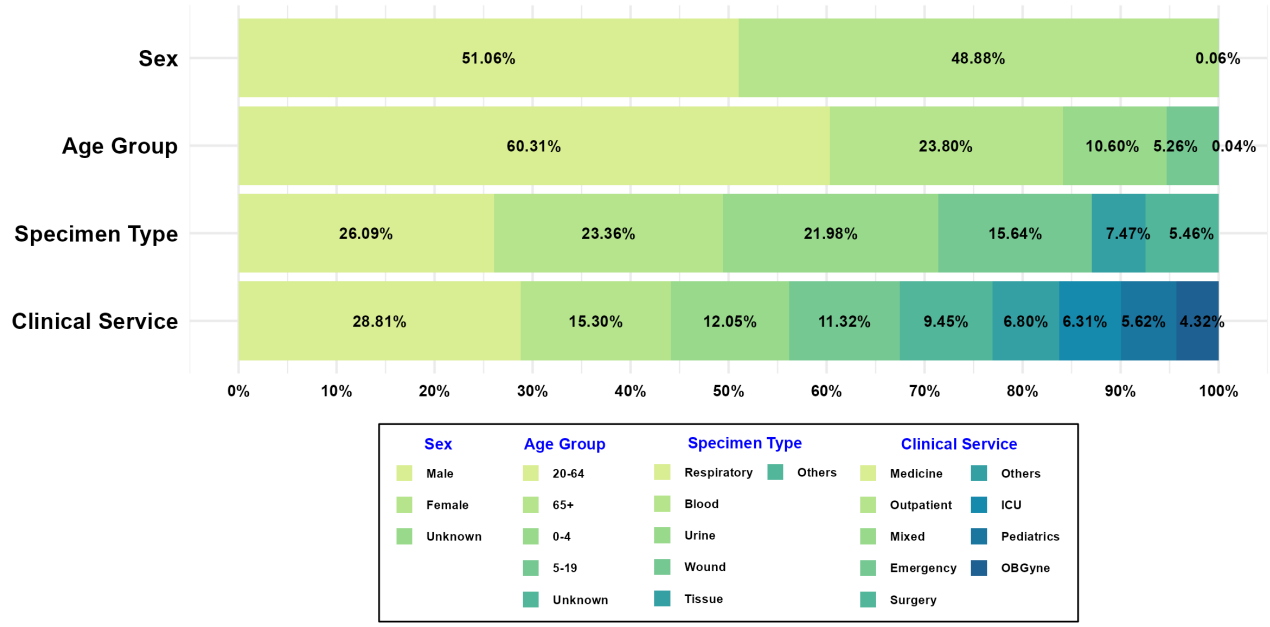


Figure 4. Patient characteristics of 2024 reported and analyzed bacterial infections, DOH-ARSP

Table 3. Most common pathogen among bacteriologically confirmed infections, DOH-ARSP, 2024 (N= 123, 949)

	Rank	Organism
Bloodstream Infections	1	*Staphylococcus aureus ss. aureus*
	2	*Klebsiella pneumoniae ss. pneumoniae*
	3	*Escherichia coli*
Cerebrospinal Fluid Infections	1	*Pseudomonas sp.*
	2	*Acinetobacter baumannii*
	3	*Klebsiella pneumoniae ss. pneumoniae*
Respiratory Tract Infections	1	*Klebsiella pneumoniae ss. pneumoniae*
	2	*Pseudomonas aeruginosa*
	3	*Acinetobacter baumannii*
Diarrheal Disease	1	*Salmonella sp.*
	2	*Vibrio cholerae*
	3	*Shigella sp.*
Urinary Tract Infection	1	*Escherichia coli*
	2	*Klebsiella pneumoniae ss. pneumoniae*
	3	*Staphylococcus, coagulase negative*
Skin and Soft Tissue Infection	1	*Staphylococcus aureus ss. aureus*
	2	*Escherichia coli*
	3	*Klebsiella pneumoniae ss. pneumoniae*

Streptococcus pneumoniae

There were **478** *Streptococcus pneumoniae* infections reported for 2024.

478
infections

Surveillance sites located in Luzon contributed most (63.18%) of the reported infections, 20.92% from Mindanao and 15.90% from Visayas (Figure 5).

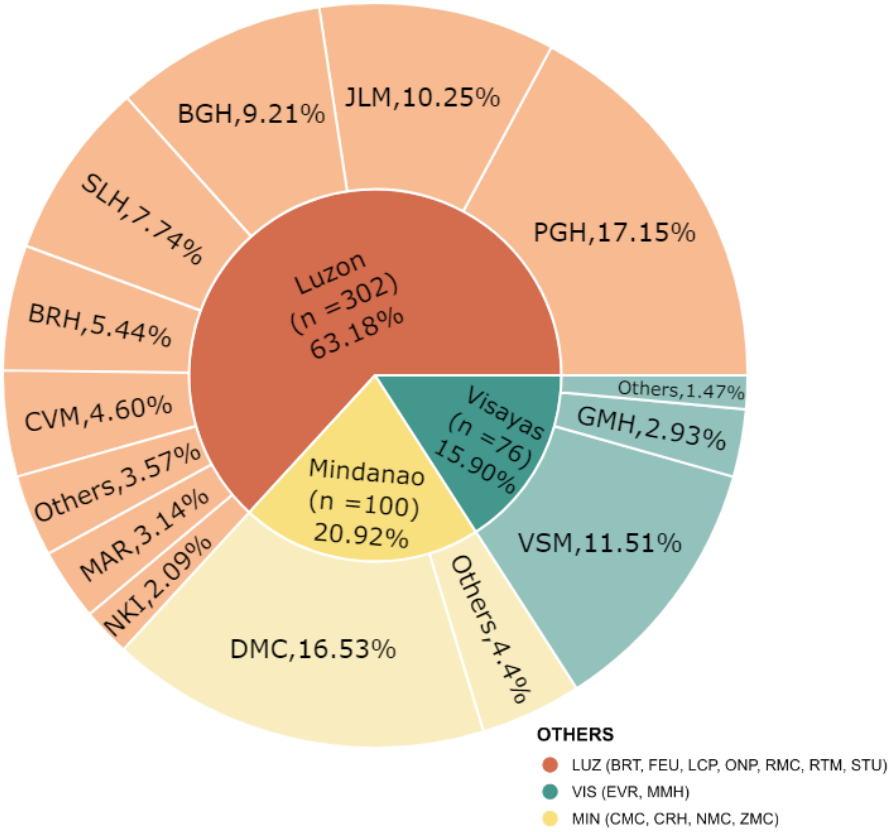


Figure 5. Distribution of *S. pneumoniae* infections, DOH-ARSP, 2024 (n=478)

Figure 6 illustrates that most (60.67%) infections came from male patients and most (55.02%) were from aged 20-64 years old. Most common types of infections were respiratory (68.62%) and bloodstream (25.10%).

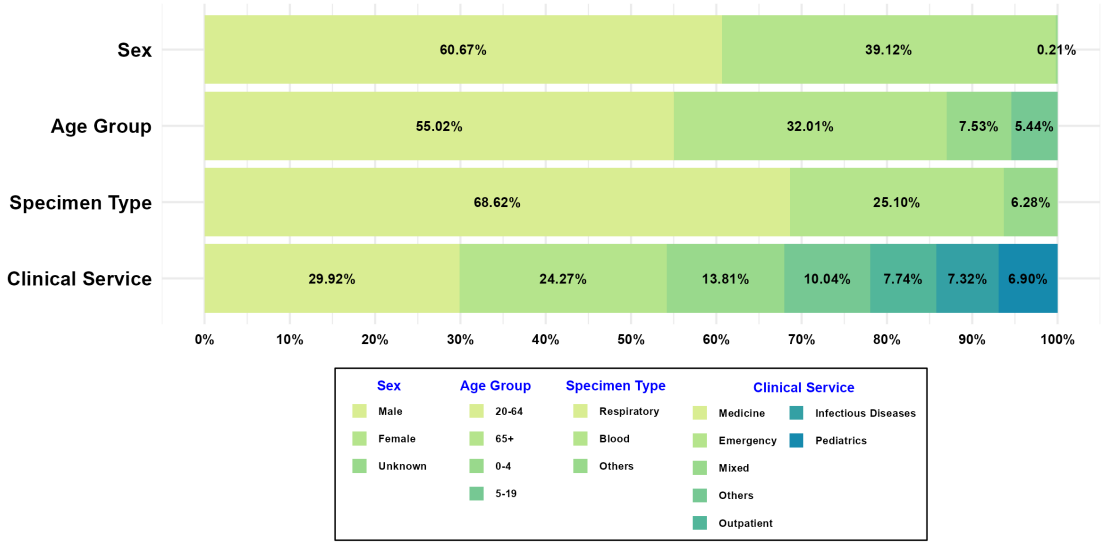


Figure 6. Patient characteristics of *S. pneumoniae* infections, DOH-ARSP, 2024 (n=478)

Figure 7 shows that the percent positive of respiratory infections caused by *S. pneumoniae* in RTM, SLH, PGH, DMC and JLM were 3.57%, 2.97%, 2.43%, 2.10% and 1.60% respectively. The percent positive of respiratory *S. pneumoniae* infections in CRH, EVR, NMC, ZMC, STU, GMH, CMC, CVM, BRH MAR, MMH, NKI and BGH were low, which ranges from 0.06-0.92%.

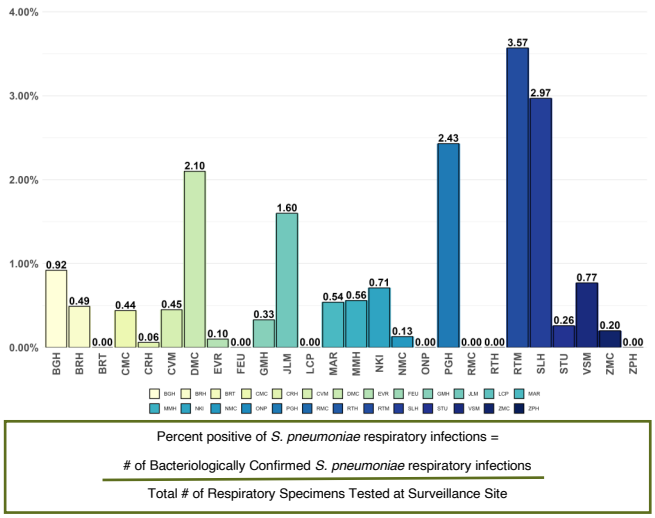


Figure 7. Percent positive of *S. pneumoniae* respiratory infections among all tested respiratory specimens per surveillance site, DOH-ARSP, 2024

All types of Infections

Figure 8 shows the resistance rates among all types of *S. pneumoniae* infections. Penicillin resistance is at 21.35% using meningitis breakpoint, 1.44% using non-menigitis breakpoint and 5.26% using oral breakpoint. Compared with 2023, the overall resistance rates for all *S. pneumoniae* infections are higher in 2024 except for penicillin, co-trimoxazole, and meropenem.

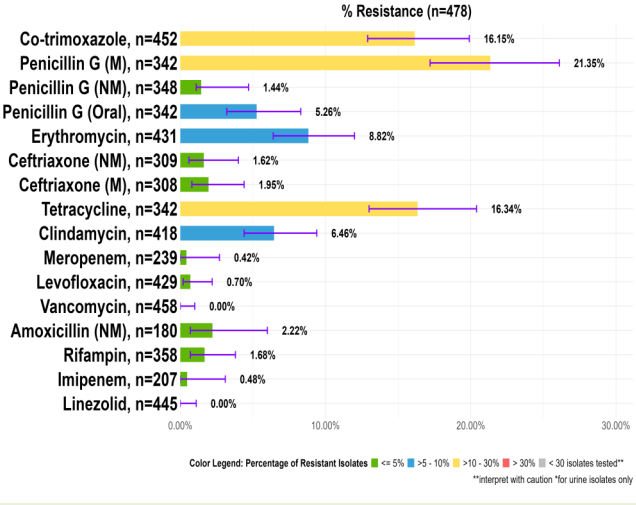
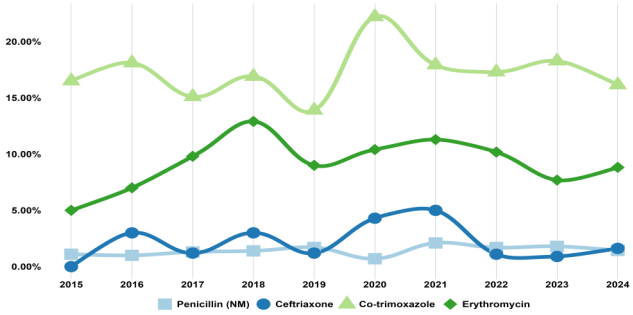


Figure 8. Proportion of all *S. pneumoniae* infections with resistance to tested antibiotics, DOH-ARSP, 2024

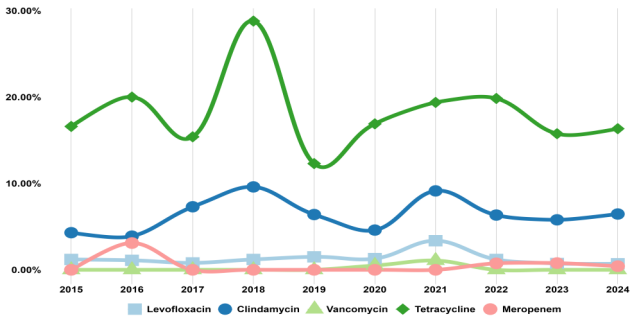
There were two (2) *S. pneumoniae* isolates confirmed to be resistant to ceftriaxone from respiratory infections of two adults in the Visayas. The isolates were noted to be susceptible to erythromycin, meropenem, vancomycin, levofloxacin, and linezolid.

As in 2023, we report the identification of *S. pneumoniae* isolates resistant to meropenem (n=2) and to rifampicin (n=1). The rifampicin resistant isolate was noted to be susceptible to most other antibiotics while the meropenem resistant isolates were susceptible to penicillin, ceftriaxone, erythromycin, levofloxacin, and linezolid. Continued surveillance to watch out for potential emergence and spread of these resistance phenotypes may be warranted in view of these antibiotics being considered as treatment options for penicillin-resistant pneumococcal (penRP) infections.

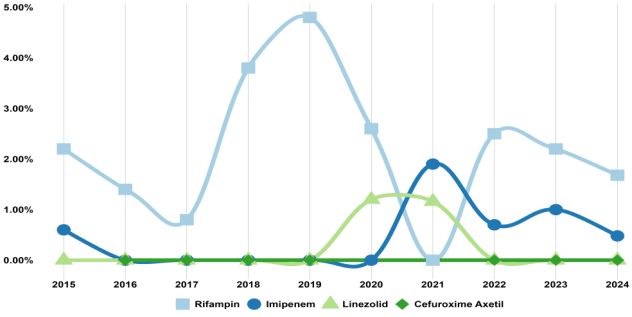
Multiple year analysis shows that the resistance rates over the past decade were statistically significant for penicillin, meropenem and tetracycline.



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Penicillin (NM)	1.1%	1.0%	1.3%	1.4%	1.7%	0.7%	2.1%	1.7%	1.8%	1.4%
Ceftriaxone	0.0%	3.0%	1.2%	3.0%	1.2%	4.3%	5.0%	1.1%	0.9%	1.6%
Co-trimoxazole	16.5%	18.1%	15.1%	16.9%	13.9%	22.2%	17.9%	17.3%	18.3%	16.1%
Erythromycin	5.0%	7.0%	9.8%	12.9%	9.0%	10.4%	11.3%	10.2%	7.7%	8.8%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Levofloxacin	1.2%	1.1%	0.8%	1.2%	1.5%	1.3%	3.4%	1.2%	0.7%	0.7%
Clindamycin	4.3%	3.9%	7.3%	9.6%	6.4%	4.6%	9.1%	6.3%	5.8%	6.5%
Vancomycin	0.0%	0.0%	0.0%	0.0%	0.0%	0.5%	1.1%	0.0%	0.0%	0.0%
Tetracycline	16.6%	20.0%	15.4%	28.8%	12.3%	16.9%	19.4%	19.8%	15.8%	16.3%
Meropenem	0.0%	3.1%	0.0%	0.0%	0.0%	0.0%	0.0%	0.8%	0.8%	0.4%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Rifampin	2.2%	1.4%	0.8%	3.8%	4.8%	2.6%	0.0%	2.5%	2.2%	1.7%
Imipenem	0.6%	0.0%	0.0%	0.0%	0.0%	0.0%	1.9%	0.7%	1.0%	0.5%
Linezolid	0.0%	0.0%	0.0%	0.0%	0.0%	1.2%	1.2%	0.0%	0.0%	0.0%
Cefuroxime Axetil		0.0%	0.0%	0.0%	0.0%			0.0%	0.0%	0.0%

Figure 9. Yearly resistance rates all *S. pneumoniae* infections, DOH-ARSP, 2024

Penicillin resistance rates (NM) in most *S. pneumoniae* infections were less than 1% across all surveillance sites

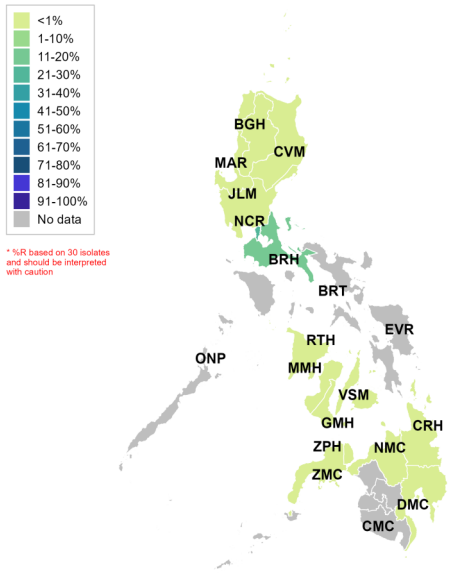


Figure 10. Geographic distribution of penicillin-resistant *S. pneumoniae* infections in the Philippines, DOH-ARSP, 2024

Respiratory Infections

Figures 11 and **12** detail *S. pneumoniae* resistance in respiratory tract infections. For all respiratory infections (**Figure 11**), penicillin resistance remains under 5% (non-meningitic and oral breakpoints). Other key antibiotics, ceftriaxone, clindamycin, and levofloxacin, showed resistance rates below 10%. In contrast, co-trimoxazole and tetracycline had relatively higher resistance, both over 15%.

Figure 12 shows similar resistance rates for lower respiratory tract infections. Overall, resistance rates for both general and lower respiratory tract *S. pneumoniae* infections are relatively higher than for all *S. pneumoniae* infections combined.

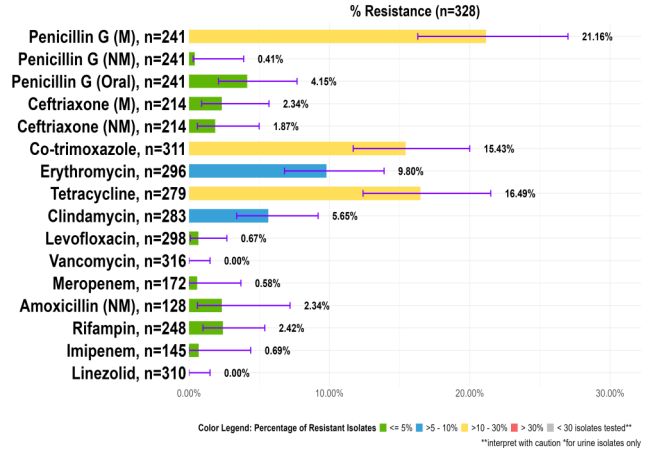


Figure 11. Proportion of *S. pneumoniae* respiratory infections with resistance to tested antibiotics

Lower Respiratory Tract Infections

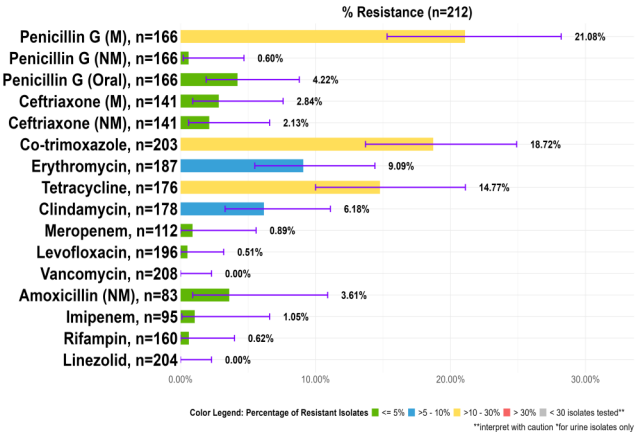
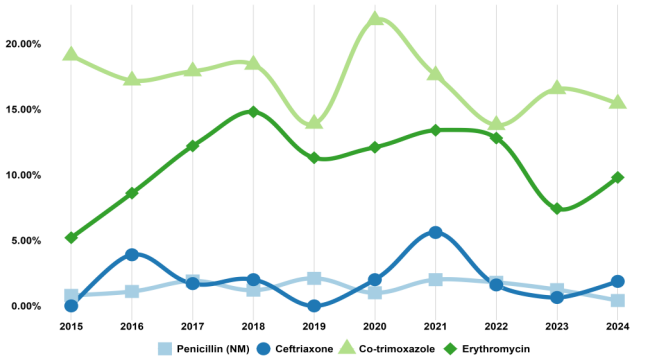
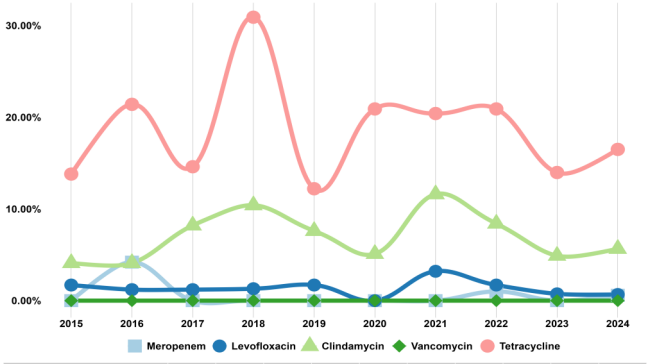


Figure 12. Proportion of *S. pneumoniae* lower respiratory infections with resistance to tested antibiotics, DOH-ARSP, 2024

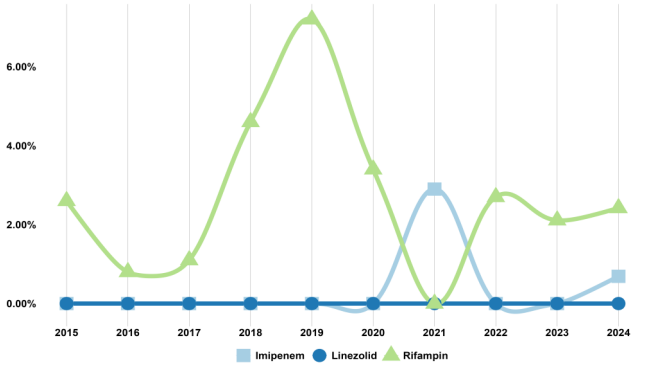
Multiple year analysis shows that the resistance rates over the past decade were statistically significant for penicillin, meropenem and tetracycline.



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Penicillin (NM)	0.8%	1.1%	1.9%	1.2%	2.1%	1.0%	2.0%	1.8%	1.2%	0.4%
Ceftriaxone	0.0%	3.9%	1.7%	2.0%	0.0%	2.0%	5.6%	1.6%	0.6%	1.9%
Co-trimoxazole	19.1%	17.2%	17.9%	18.4%	13.9%	21.8%	17.6%	13.8%	16.5%	15.4%
Erythromycin	5.2%	8.6%	12.2%	14.8%	11.3%	12.1%	13.4%	12.8%	7.4%	9.8%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Meropenem	0.0%	4.2%	0.0%	0.0%	0.0%	0.0%	0.0%	1.0%	0.0%	0.6%
Levofloxacin	1.7%	1.2%	1.2%	1.3%	1.7%	0.0%	3.2%	1.7%	0.7%	0.7%
Clindamycin	4.1%	4.1%	8.2%	10.4%	7.6%	5.1%	11.6%	8.4%	4.9%	5.6%
Vancomycin	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Tetracycline	13.8%	21.4%	14.6%	30.9%	12.2%	20.9%	20.4%	20.9%	14.0%	16.5%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Imipenem	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	2.9%	0.0%	0.0%	0.7%
Linezolid	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Rifampin	2.6%	0.8%	1.1%	4.6%	7.2%	3.4%	0.0%	2.7%	2.1%	2.4%

Figure 13. Yearly resistance rates of *S. pneumoniae* respiratory infections: DOH-ARSP, 2015-2024

Invasive Infections

The resistance rates of invasive *S. pneumoniae* infections (blood and CSF specimens) are shown in **Figure 14**. No resistance was noted to ceftriaxone, vancomycin, rifampin and linezolid. Penicillin (M) resistance was at 21.43%, co-trimoxazole resistance at 19.47% and tetracycline at 12.62%. Resistance to most antibiotics was lower for invasive *S. pneumoniae* infections compared to all *S. pneumoniae* infections except for ceftriaxone.

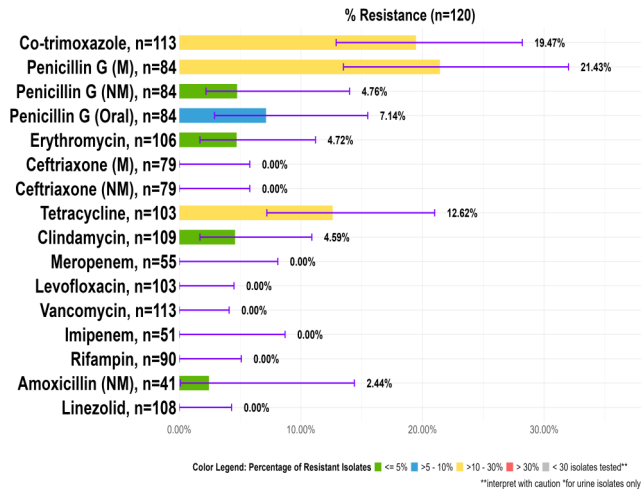
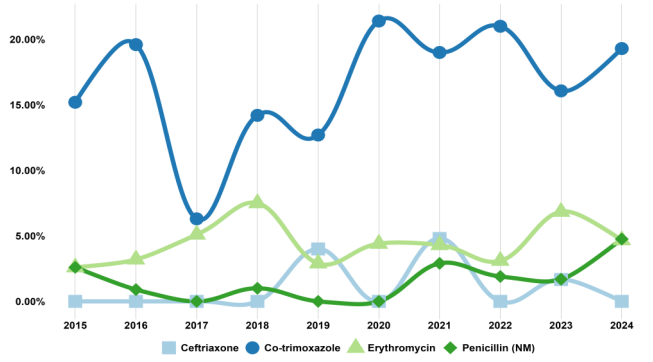
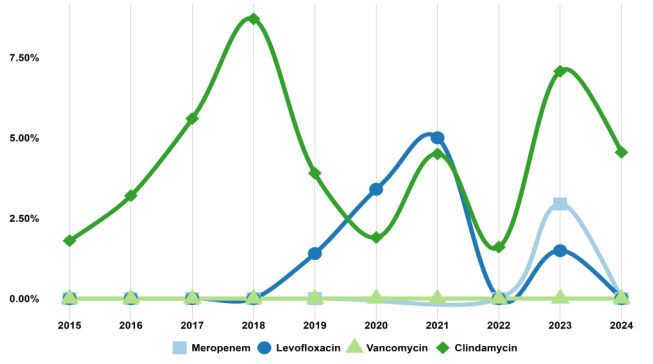


Figure 14. Proportion of invasive *S. pneumoniae* infections with resistance to tested antibiotics, DOH-ARSP, 2024

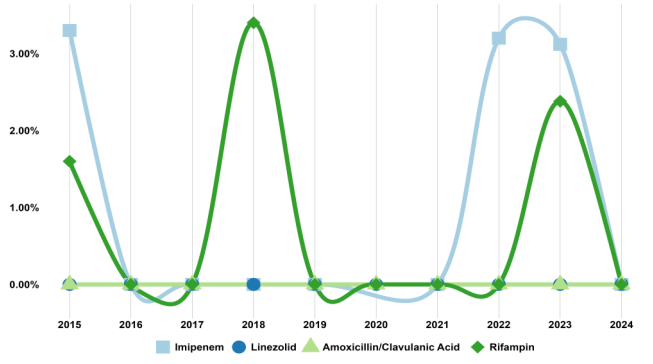
Multi-year analysis shows that the resistance rates over the past decade were statistically significant for invasive *S. pneumoniae* infections.



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Ceftriaxone	0.0%	0.0%	0.0%	0.0%	4.0%	0.0%	4.8%	0.0%	1.7%	0.0%
Co-trimoxazole	15.2%	19.6%	6.3%	14.2%	12.7%	21.4%	19.0%	21.0%	16.1%	19.3%
Erythromycin	2.6%	3.2%	5.1%	7.5%	2.9%	4.4%	4.3%	3.1%	6.8%	4.7%
Penicillin (NM)	2.6%	0.9%	0.0%	1.0%	0.0%	0.0%	2.9%	1.9%	1.7%	4.8%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Meropenem	0.0%	0.0%	0.0%	0.0%	0.0%			0.0%	2.9%	0.0%
Levofloxacin	0.0%	0.0%	0.0%	0.0%	1.4%	3.4%	5.0%	0.0%	1.5%	0.0%
Vancomycin	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Clindamycin	1.8%	3.2%	5.6%	8.7%	3.9%	1.9%	4.5%	1.6%	7.1%	4.5%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Imipenem	3.3%	0.0%	0.0%	0.0%	0.0%		0.0%	3.2%	3.1%	0.0%
Linezolid	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Amoxicillin/Clavulanic Acid	0.0%	0.0%	0.0%		0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Rifampin	1.6%	0.0%	0.0%	3.4%	0.0%	0.0%	0.0%	0.0%	2.4%	0.0%

Figure 15. Yearly resistance rates of invasive *S. pneumoniae* infections, DOH-ARSP, 2015-2024

A total of 111 *S. pneumoniae* isolates were referred to ARSRL in 2024 constituting 39 *S. pneumoniae* serogroup/serotypes. Most (n=36, 31.58%) of the isolates were from the 18-49 age group.

Of the few *S. pneumoniae* isolates from patients 5 years old and below (n=10), the following serogroups/serotypes were noted: 6B (PCV7), 19F (PCV7), 19A (PCV13), 15B (PPSV23), and non-vaccine serotypes 23A, 34 and 35B.

No isolate was from the 6-11 age range. Serotypes 6B, 18C and 15B were observed among the 12-17 age group. Serotype 3 was most observed among 18-49 years old, while among the 50-59 years old, serotypes 34 and 35B were frequently seen. Similarly, among the geriatric population (>60 years old), serotype 3 was the most apparent serotype. Serotype 3 was also the overall most common serotype across all age groups, followed by non-vaccine serotype 34 and PPSV-23 serotype 11A.

Penicillin (NM) resistant *S. pneumoniae* isolates are of serotypes 14 and 18C. One serotype 14 isolate showed resistance to co-trimoxazole. Serotypes of erythromycin resistant isolates include serotypes 11A, 14, 18C, 35A and 35B.

Percent PCV and PPSV coverage (overall and per age group) of *S. pneumoniae* isolates sent to ARSRL for confirmation were determined by dividing the number of isolates with serotypes included in PCV or PPSV over the total number of isolates (overall and per age group). The vaccine coverage of 2024 *S. pneumoniae* isolates are shown in Table 4.

The overall non-vaccine serotype coverage for 2024 was 39.47% while for the population less than 5 years old was 50% and 32.61% for the >60 years old population. The overall non-vaccine coverage for 2023 was at 58.82%, which was lower than the vaccine coverage for 2024 the observed decrease was statistically significant (p=0.0003).

Table 4. Vaccine coverage of 2024 *S. pneumoniae* isolates

Vaccine Coverage			
	All age Group	> 5 years old	≥ 60 years old
PCV 7	14.91%	20.0%	13.02%
PCV 10	14.91%	20.0%	13.02%
PCV 13	35.96%	40.0%	43.45%
PCV 15	39.47%	40.0%	47.795
PCV 20	53.49%	50.0%	63.0%
PPSV 23	57%	50.0%	65.17
Non-vaccine Coverage			
	All age Group	> 5 years old	≥ 60 years old
	39.47%	50.0%	32.61%

Table 5. *S. pneumoniae* serotypes and PCV serotypes per age group, DOH-ARSP, 2024

	SEROTYPE	AGE (years) <1	1-5	6-11	12-17	18-49	50-59	≥60	TOTAL
PCV-7	4								4
	6B								2
	9V								1
	14								1
	18C								2
	19F								4
	23F								3
PCV-10	1								-
	5								-
	7F								-
PCV-13	3								20
	6A								-
	19A								4
PCV-15	22F								4
	33F								-
PCV-20	8								-
	10A								2
	11A								9
	12F								2
	15B								3
PPSV-23	2								-
	9N								-
	20								4
Non- PCV/ PPSV	6C								1
	7C								2
	9L								1
	13								2
	18A								1
	23A								4
	23B								1
	29								2
	34								14
	35A								1
	35B								8
	35F								1
	39								2
	10B								1
	10X								1
	16F								4
TOTAL		4	6	0	3	35	17	46	111

*PCV – Pneumococcal Conjugate Vaccine, PPSV – Pneumococcal Polysaccharide Vaccine

Haemophilus influenzae

A total of **468** *H. influenzae* infections were reported for 2024.

468
infections

The highest contributors were PGH (31.20%), BGH (22.0%) and SLH (11.32%) (Figure 16). Based on island group distribution, Luzon was highest with 78.21% followed by Visayas at 11.11% and Mindanao at 10.68%.

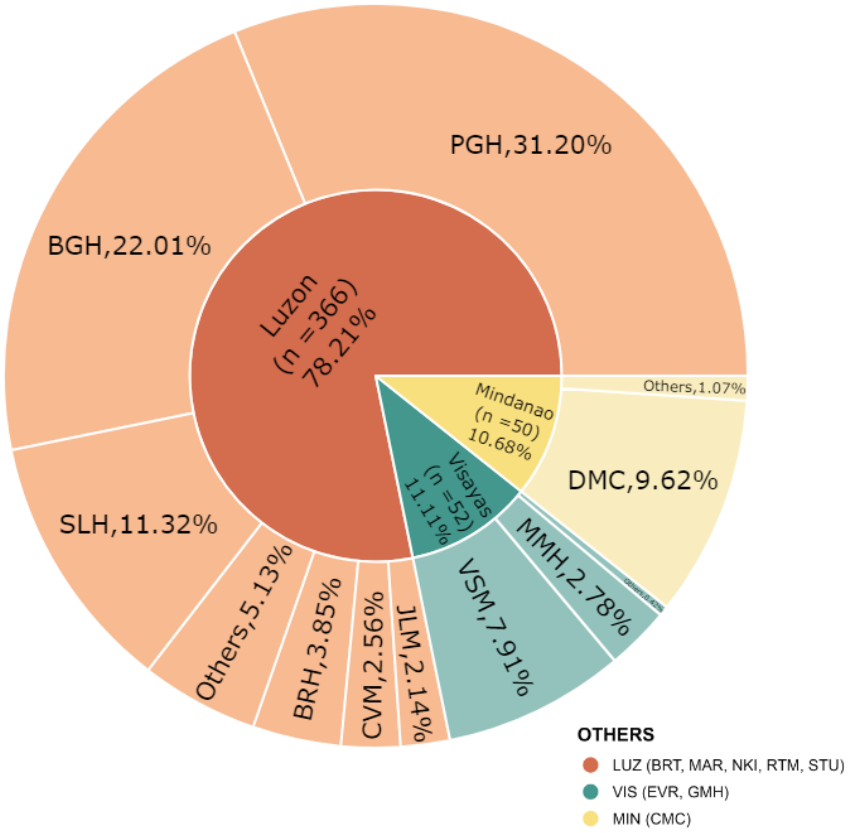


Figure 16. Distribution of *H. influenzae* infections, DOH-ARSP, 2024 (n=468)

More than half of the infections were among male patients at 59.40% and adult patients aged 20-64 years old at 51.28%. Respiratory infection remains the most common type of *H. influenzae* infections, accounting for 90.38%.

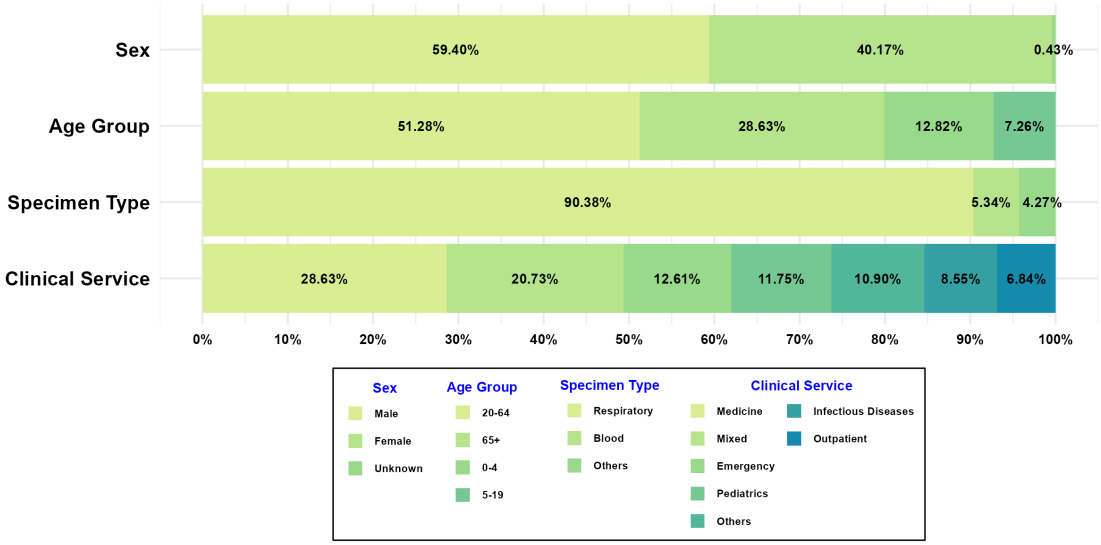
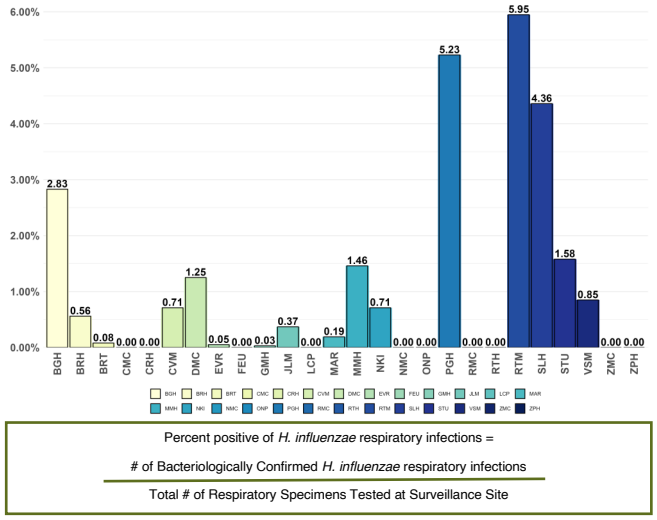


Figure 17. Patient characteristics of *H. influenzae* infections, DOH-ARSP, 2024 (n=468)

The percent positive of respiratory infections due to *H. influenzae* was highest for RTM (5.95%), followed by PGH (5.23%), and SLH (4.36%). While the percent positive of respiratory infections caused by *H. influenzae* ranges for BGH, STU, MMH and DMC were 2.83%, 1.58%, 1.46% and 1.25% respectively. Percent positive of respiratory infections caused by *H. influenzae* in VSM, NKI, BRH, JLM, MAR, BRT, and EVR were less than 1%.



Percent positive of *H. influenzae* respiratory infections =

# of Bacteriologically Confirmed *H. influenzae* respiratory infections

Total # of Respiratory Specimens Tested at Surveillance Site

Figure 18. Percent positive of *H. influenzae* respiratory infections among all tested respiratory specimens per surveillance site, DOH-ARSP, 2024

For 2024, there were 10 *H. influenzae* respiratory infections reported with the isolates confirmed to be resistant to amoxicillin-clavulanic acid. Most of these infections occurred among adult patients and the isolates were susceptible to ceftriaxone ciprofloxacin and meropenem but resistant to trimethoprim-sulfamethoxazole. Compared with data from the past 3 years where the number of amoxicillin-clavulanic acid resistant *H. influenzae* isolates ranged from 1-2, it was noted that for 2024, there was a notable increase in the number of amoxicillin-clavulanic acid resistant *H. influenzae* isolates.

There was likewise one reported *H. influenzae* respiratory infections with the isolate confirmed to be nonsusceptible to cefotaxime. The patient was a 38 year old male diagnosed to have HIV. The isolate was noted to be susceptible to amoxicillin-clavulanic acid, ciprofloxacin and trimethoprim-sulfamethoxazole.

Further, there were three reported *H. influenzae* respiratory infections with the isolates confirmed to be ciprofloxacin nonsusceptible. These infections were among adult patients and the isolates were susceptible to ampicillin, ceftriaxone and to meropenem.

Figure 20 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 and cefuroxime; however, the changes in resistance rates for these two antibiotics were not statistically significant.

All types of Infections

Resistance to ampicillin, cefuroxime and ampicillin-sulbactam was at 13.95%, 13.57% and 8.7% respectively. Resistance to co-trimoxazole and tetracycline resistance was observed to be high across all infections.

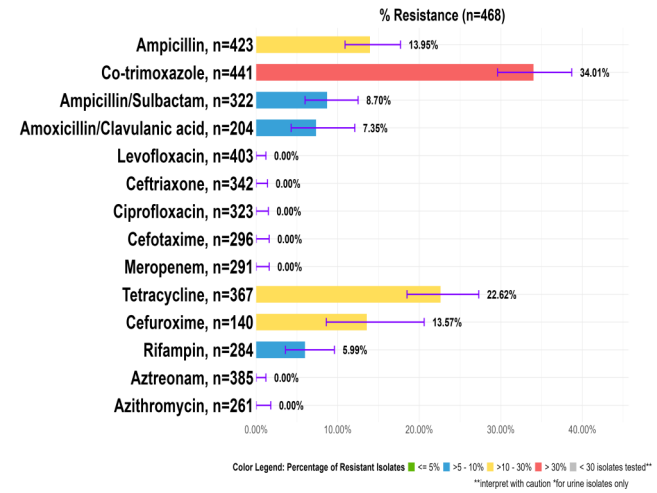
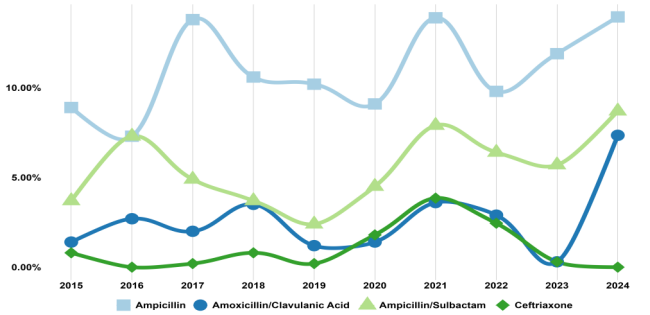
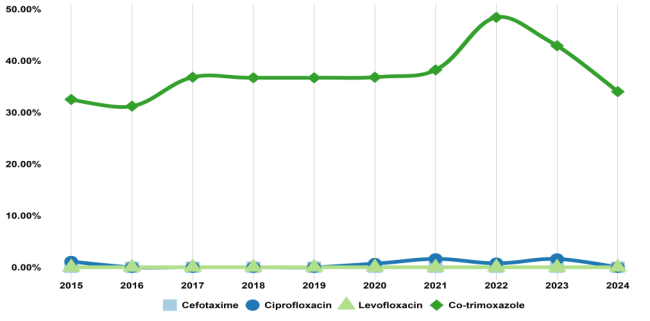


Figure 19. Proportion of all *H. influenzae* infections with resistance to tested antibiotics, DOH-ARSP, 2024

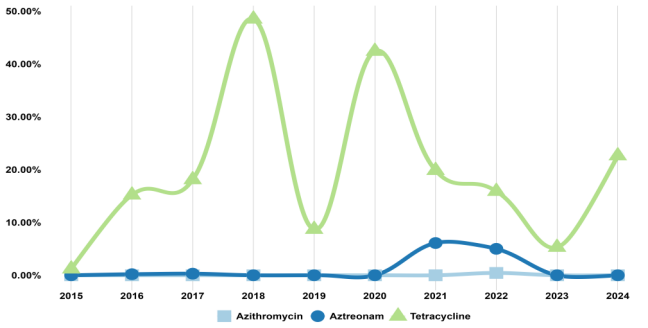


	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Ampicillin	8.9%	7.3%	13.8%	10.6%	10.2%	9.1%	13.9%	9.8%	11.9%	13.9%
Amoxicillin/Clavulanic Acid	1.4%	2.7%	2.0%	3.5%	1.2%	1.4%	3.6%	2.9%	0.3%	7.3%
Ampicillin/Sulbactam	3.7%	7.3%	4.9%	3.7%	2.4%	4.5%	7.9%	6.4%	5.7%	8.7%
Ceftriaxone	0.8%	0.0%	0.2%	0.8%	0.2%	1.8%	3.8%	2.4%	0.3%	0.0%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Cefotaxime	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Ciprofloxacin	1.1%	0.0%	0.0%	0.0%	0.0%	0.7%	1.6%	0.8%	1.6%	0.0%
Levofloxacin	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Co-trimoxazole	32.5%	31.2%	36.8%	36.7%	36.7%	36.8%	38.2%	48.4%	42.9%	34.0%

	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Meropenem	0.0%	0.5%	0.5%	0.0%	0.9%	0.0%	0.0%	1.2%	0.0%	0.0%
Rifampin	5.5%	3.5%	6.5%	16.2%	14.6%	16.7%	25.0%	0.0%	2.2%	6.0%
Cefuroxime	5.8%	3.8%	10.0%	8.1%	0.0%	8.8%	0.0%	0.0%	5.6%	13.6%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Azithromycin	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.4%	0.0%	0.0%
Aztreonam	0.0%	0.2%	0.3%	0.0%	0.0%	0.0%	6.1%	5.0%	0.0%	0.0%
Tetracycline	1.2%	15.2%	18.1%	48.5%	8.7%	42.4%	19.9%	15.9%	5.3%	22.6%

Figure 20. Yearly resistance rates of all *H. influenzae* infections, DOH-ARSP, 2024

Lower Respiratory Tract Infections

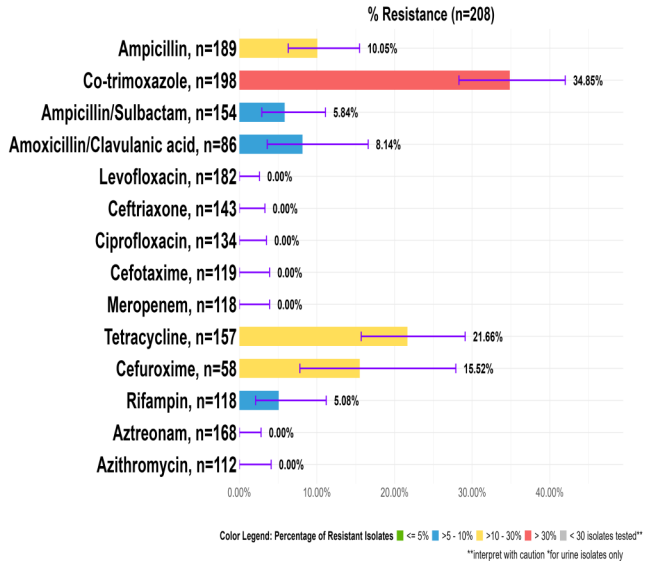


Figure 22. Proportion of *H. influenzae* lower respiratory infections with resistance to tested antibiotics, DOH-ARSP, 2024

Respiratory Infections

Figure 21 shows the proportion of *H. influenzae* respiratory infections with resistance to tested antibiotics. Resistance to amoxicillin-clavulanic acid was less than 10% and cefuroxime at 15%. No resistance was detected to third-generation cephalosporins, fluoroquinolones and azithromycin. **Figure 22**, which specifically details *H. influenzae* lower respiratory tract infections, reflects the same antibiotic resistance trends.

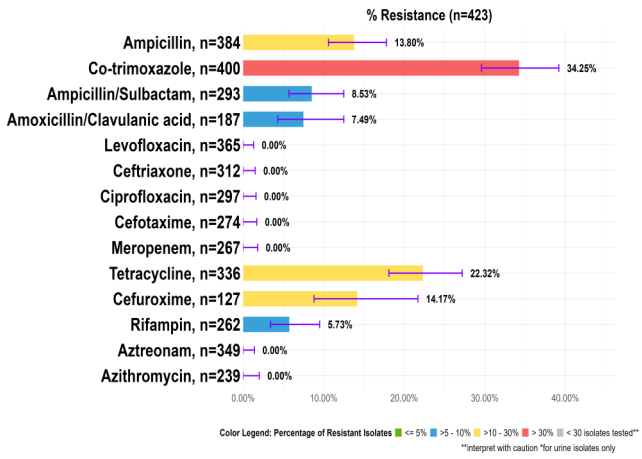


Figure 21. Proportion of *H. influenza* respiratory infections with resistance to tested antibiotics, DOH-ARSP, 2024

Invasive Infections

The resistance rates of invasive *H. influenzae* infections (blood and CSF specimens) from 2022-2024 are shown in **Figure 23**. The data from the past three years were presented in order to obtain reasonable statistical estimates of resistance rates. No resistance to ceftriaxone, and levofloxacin was noted.

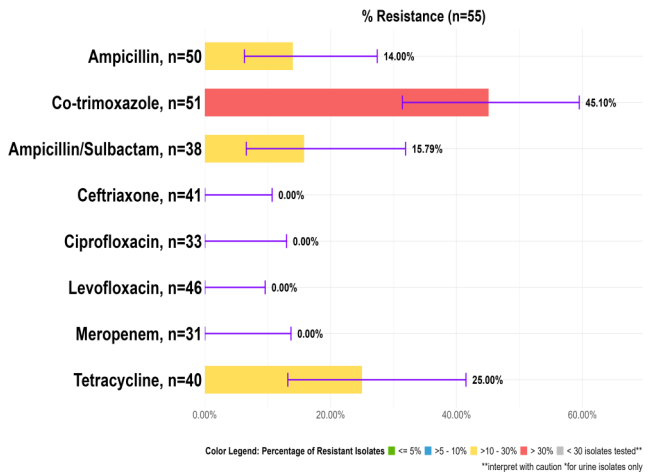


Figure 23. Proportion of invasive *H. influenzae* infections with resistance to tested antibiotics, DOH-ARSP, 2022-2024

Haemophilus influenzae, a significant human pathogen, has demonstrated increasing antimicrobial resistance, posing a substantial public health threat. The epidemiology of resistance in *H. influenzae* is primarily driven by the acquisition of β-lactamase genes leading to ampicillin resistance, and efflux pump mechanisms contributing to macrolide resistance.[1] This resistance complicates treatment for common *H. influenzae* infections such as otitis media, sinusitis, and pneumonia, especially in vulnerable populations like children and the elderly.[2] The impact extends to higher healthcare costs due to the need for more expensive and broad-spectrum antibiotics, increased morbidity, and, in severe cases, treatment failures and mortality. Ongoing surveillance and judicious antibiotic stewardship are crucial to mitigate the rising tide of antimicrobial resistance in *H. influenzae* and preserve the efficacy of current therapeutic options.

Table 6. Patient demographics and clinical characteristics of *H. influenzae* isolates with emerging resistance, 2023-2024, n=6

Accession number	Age	Sex	Specimen Type	Infection Type
23ARS-BGH0113	37	f	sputum	CAI
23ARS-SLH0017	69	m	sputum	HAI
23ARS-VSM0100	71	f	tracheal aspirate	CAI
24ARS-BRT0146	69	m	sputum	CAI
24ARS-CVM0044	70	m	sputum	-
24ARS-VSM0160	34	f	broncho-alveolar lavage	CAI

Table 7. Antimicrobial resistance profile of *H. influenzae* isolates, 2023-2024, n=6

Accession number	AMC	SAM	CRO	CTX	SXT	MEM	CIP	LVX
23ARS-BGH0113	S	S	S	S	S	S	NS	NS
23ARS-SLH0017	S	S	S	S	R	S	NS	NS
23ARS-VSM0100	S	S	S	S	R	S	NS	NS
24ARS-BRT0146	S	S	S	S	S	S	NS	NS
24ARS-CVM0044	R	R	S	S	S	S	S	S
24ARS-VSM0160	S	S	S	S	R	S	NS	NS

We describe in this report, the molecular characterization of three 2024 *H. influenzae* isolates which shows emerging resistance to beta-lactams, folate-pathway inhibitor and quinolone antibiotics. The first isolate was from the sputum sample of a 69-year-old male in BRT. This isolate showed non-susceptibility to quinolone antibiotics, ciprofloxacin and levofloxacin. This resistance profile was explained by the presence of mutations in gyrA (gyrA_D88G, gyrA_S84L) and parC (parC_S84I, parE_D420N) genes. The gyrA gene encodes the A subunit of bacterial DNA gyrase, an essential enzyme involved in DNA replication and supercoiling, while, parC gene encodes the A subunit of topoisomerase IV, a crucial enzyme involved in DNA topology maintenance.[3] Mutations in these two genes are associated with resistance to quinolone-based antibiotics.

One isolate from a 70-year-old male from the sputum specimen in CVM showed resistance to beta-lactam antibiotics: amoxicillin-clavulanic acid and ampicillin-sulbactam. As seen in **Table 7**, no sequence type was assigned yet for this isolate. This isolate harbor four mutations in the ftsI gene (ftsI_G555E, ftsI_M377I, ftsI_R517H, ftsI_Y557H) which encodes penicillin-binding protein 3 (PBP3), a crucial enzyme involved in bacterial cell wall synthesis, particularly during cell division. [4] In *Haemophilus influenzae*, mutations in ftsi are a significant mechanism for beta-lactam resistance.

In VSM, one co-trimoxazole resistant *H. influenzae* was detected from the broncho-alveolar lavage specimen of a 34-year-old female. This isolate was likewise non-susceptible to ciprofloxacin and levofloxacin. Genomic analysis showed that this isolate has mutation in the folP gene, the folA_f15 gene (or specifically, a 15-bp insertion within the folP gene) is linked to cotrimoxazole resistance. This resistance arises because the insertion disrupts the function of the dihydrofolate reductase (DHFR) enzyme, which is a target of trimethoprim, one component of cotrimoxazole.[5] This isolate likewise shows mutations gyrA and parC genes that may explain the characteristic quinolone resistance of this organism.

Table 8. Sequence type and AMR genes detected from *H. influenzae* isolates with emerging resistance, 2023-2024, n=6

Accession number	MLST	Beta-lactam	Cephalosporin	Quinolone	Sulfonamide	Trimethoprim
23ARS-BGH0113	1524	-	-	gyrA_D88G, gyrA_S84L, parC_S84I, parE_D420N	-	folA_F154S
23ARS-SLH0017	1524	-	-	gyrA_D88G, gyrA_S84L, parC_S84I, parE_D420N	folP_G189C, folP_P64PSFLY	folA_F154S
23ARS-VSM0100	1524	-	-	gyrA_D88G, gyrA_S84L, parC_S84I, parE_D420N	folP_G189C	folA_F154S
24ARS-BRT0146	1524	-	-	gyrA_D88G, gyrA_S84L, parC_S84I, parE_D420N	folP_G189C	folA_F154S
24ARS-CVM0044	-	ftsI_G555E, ftsI_M377I, ftsI_R517H, ftsI_Y557H	ftsI_D350N, ftsI_L389F, ftsI_S357N, ftsI_S385T	gyrA_S84L, parC_S84I,	-	-
24ARS-VSM0160	-	-	-	gyrA_D88G, gyrA_S84L, parC_S84I, parE_D420N	-	folA_F154S

In the year 2023, ciprofloxacin resistance was the only emerging resistance observed among *H. influenzae* isolates, however, in this analysis year (2024) resistance to amoxicillin-clavulanic acid, ampicillin-sulbactam and co-trimoxazole were also detected suggesting a continuous AMR evolution for this organism. ST 1524 was the prevailing sequence type since 2023 and ciprofloxacin resistance was mediated by similar mutations in both gyrA and parC genes. Resistance to sulfonamide antibiotics for 2023-2024 was mostly mediated by folP_G189C mutation, while trimethoprim resistance was commonly conferred by folA_F154S. This report highlights that genomic surveillance of emerging resistance is crucial for understanding and controlling AMR as it provides detailed insights into the genetic mechanisms driving resistance, enabling more effective tracking of resistant strains, and informing better public health strategies.

References:

[1] Johannessen, H. A., Zecic, N., Hegstad, K., Ranheim, T. E., and Skaare, D. (2022). Characterization and Fitness Cost of Tn7100, a Novel Integrative and Conjugative Element Conferring Multidrug Resistance in *Haemophilus influenzae*. Front. Microbiol. 13:945411. doi: 10.3389/fmicb.2022.945411

[2] Skaare, D., Anthonisen, I. L., Caugant, D. A., Jenkins, A., Steinbakk, M., Strand, L., et al. (2014a). Multilocus sequence typing and ftsI sequencing: A powerful tool for surveillance of penicillin-binding protein 3-mediated beta-lactam resistance in nontypeable *Haemophilus influenzae*. BMC Microbiol. 14:131. doi: 10.1186/1471-2180-14-131

[3] Nouri R, Ahangarzadeh Rezaee M, Hasani A, Aghazadeh M, Asgharzadeh M. The role of gyrA and parC mutations in fluoroquinolones-resistant *Pseudomonas aeruginosa* isolates from Iran. Braz J Microbiol. 2016 Oct-Dec;47(4):925-930. doi: 10.1016/j.bjm.2016.07.016. Epub 2016 Jul 26. PMID: 27522930; PMCID: PMC5052375.

[4] Skaare D, Allum AG, Anthonisen IL, Jenkins A, Lia A, Strand L, Tveten Y, Kristiansen BE. Mutant ftsI genes in the emergence of penicillin-binding protein-mediated beta-lactam resistance in *Haemophilus influenzae* in Norway. Clin Microbiol Infect. 2010 Aug;16(8):1117-24. doi: 10.1111/j.1469-0691.2009.03052.x. Epub 2009 Sep 8. PMID: 19737286.

[5] Enne VIKing A, Livermore DM, Hall LMC2002.Sulfonamide Resistance in *Haemophilus influenzae* Mediated by Acquisition of sul2 or a Short Insertion in Chromosomal folP. Antimicrob Agents Chemother46:.https://doi.org/10.1128/aac.46.6.1934-1939.2002

Salmonella enterica serovar Typhi

A total of **90** *S.* Typhi infections were reported in 2024.

90
infections

The highest contributors were ZMC (24.44%), CMC (27.78%) and CVM (7.78%). According to island group distribution, 60.0% was from Mindanao, 30.0% from Luzon and 10.0% from Visayas.

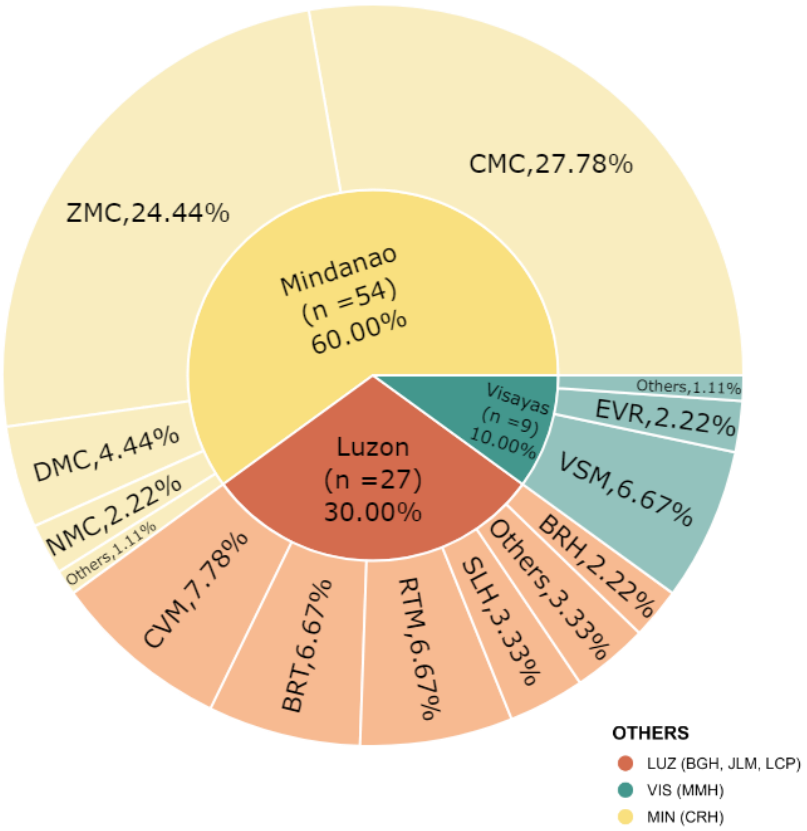


Figure 24. Distribution of *S.* Typhi infections, 2024 (n= 90)

More than half of the infections (56.67%) occurred in male patients, with majority (44.44%) observed in the 5–19 age group. Most infections (88.89%) were detected from blood specimens.

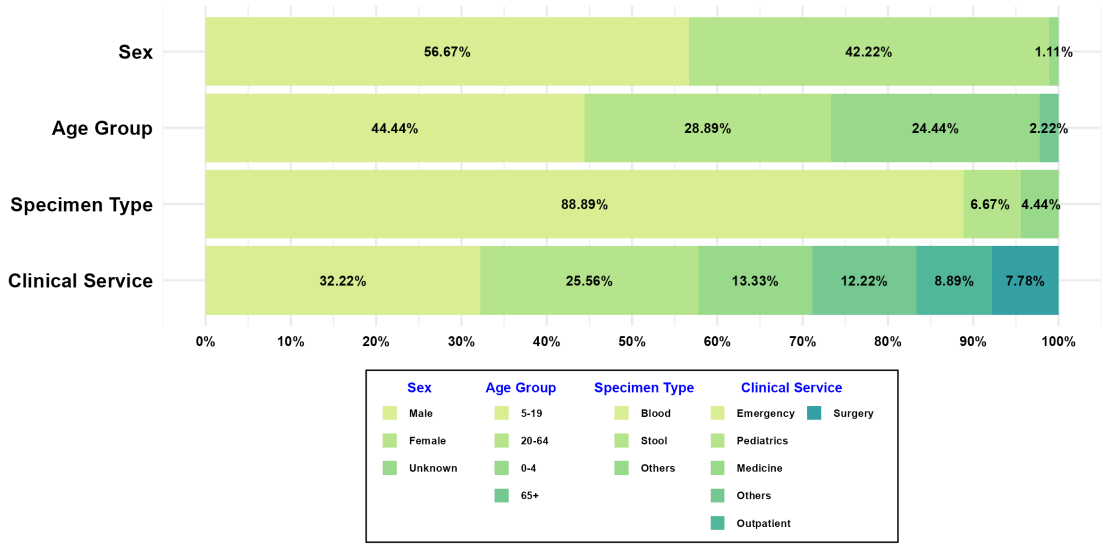


Figure 25. Patient characteristics in *S.* Typhi infections, DOH-ARSP, 2024 (n=90)

Percent positive of blood infection caused by *S. enterica* serovar Typhi for RTM, ZMC and CMC were 3.05%, 0.47% and 0.34% respectively. The observed percent positive for all other surveillance sites were less than 35%

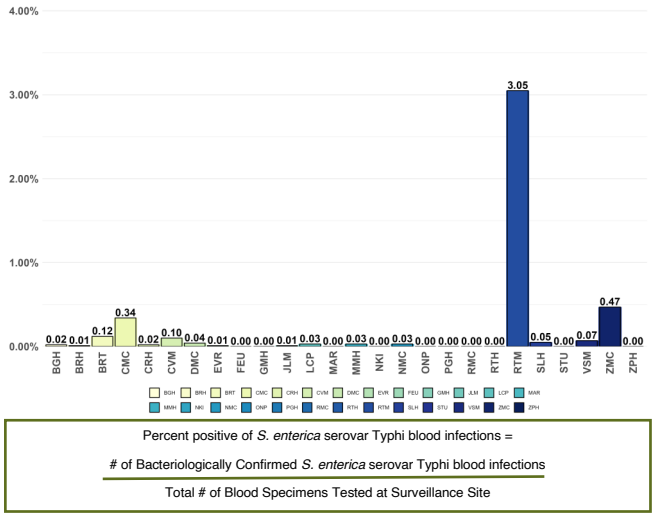


Figure 26. Percent positive of *S. enterica* serovar Typhi bloodstream infections among all tested blood specimens per surveillance site, DOH-ARSP, 2024

All types of Infections

Figure 27 shows the resistance rates of all *S.* Typhi infections for 2024. Resistance to ceftriaxone was at 3.37%, ciprofloxacin at 1.37%, meropenem at 1.52%, and co-trimoxazole at 1.12%. No resistance was observed to chloramphenicol, and azithromycin.

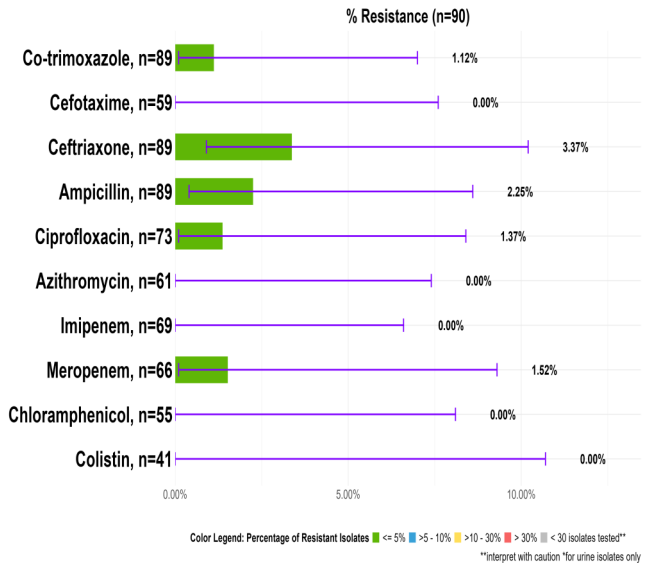
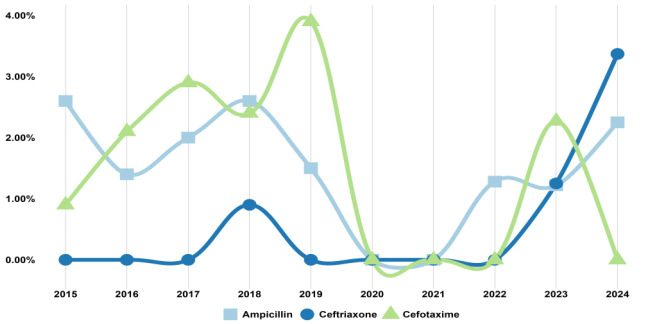
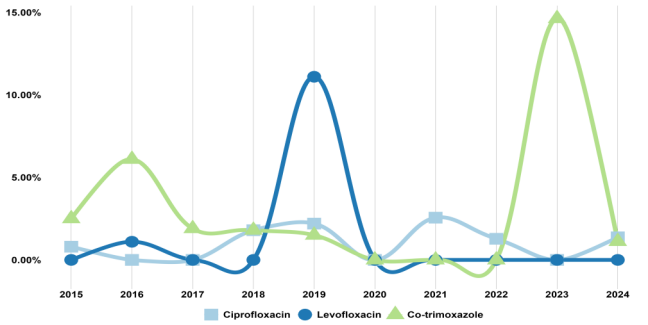


Figure 27. Proportion of all *S.* Typhi infections with resistance to tested antibiotics, DOH-ARSP 2024

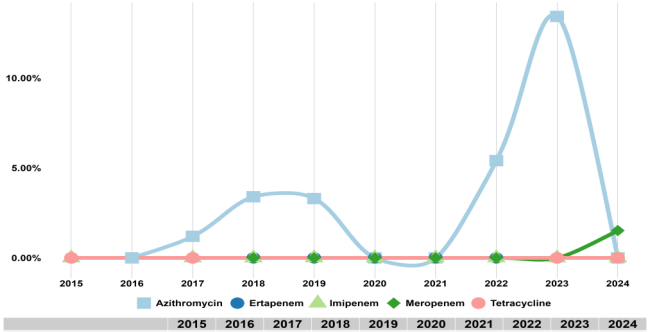
The yearly resistance rates of all *S.* Typhi infections to all antibiotics used for treatment have remained low in the past ten years. However, significant changes were observed for co-trimoxazole (p= 0.0135) and azithromycin (p=0.0121).



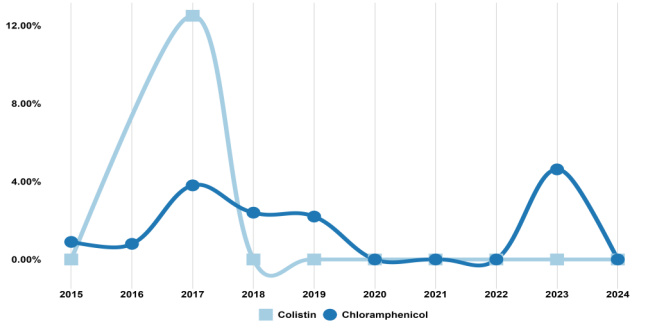
	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Ampicillin	2.6%	1.4%	2.0%	2.6%	1.5%	0.0%	0.0%	1.3%	1.2%	2.2%
Ceftriaxone	0.0%	0.0%	0.0%	0.9%	0.0%	0.0%	0.0%	0.0%	1.2%	3.4%
Cefotaxime	0.9%	2.1%	2.9%	2.4%	3.9%	0.0%	0.0%	0.0%	2.3%	0.0%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Ciprofloxacin	0.8%	0.0%	0.0%	1.8%	2.2%	0.0%	2.6%	1.3%	0.0%	1.4%
Levofloxacin	0.0%	1.1%	0.0%	0.0%	11.1%	0.0%	0.0%	0.0%	0.0%	0.0%
Co-trimoxazole	2.5%	6.1%	1.9%	1.8%	1.5%	0.0%	0.0%	0.0%	14.6%	1.1%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Azithromycin		0.0%	1.2%	3.4%	3.3%	0.0%	0.0%	5.4%	13.4%	0.0%
Ertapenem	0.0%		0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Imipenem	0.0%		0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Meropenem	0.0%		0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	1.5%
Tetracycline	0.0%		0.0%						0.0%	0.0%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Colistin	0.0%		12.5%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Chloramphenicol	0.9%	0.8%	3.8%	2.4%	2.2%	0.0%	0.0%	0.0%	4.6%	0.0%

Figure 28. Yearly resistance rates of all *S.* Typhi infections, DOH-ARSP 2024

Bloodstream Infections

For *S.* Typhi bloodstream infections, antibiotic resistance was generally low, with no resistance detected for most antibiotics. Resistance to ciprofloxacin, meropenem, ceftriaxone, co-trimoxazole and ciprofloxacin was observed but remained below 2%.

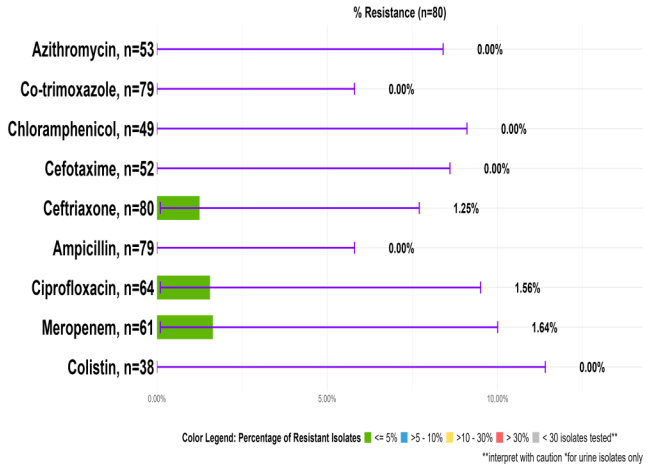


Figure 29. Proportion of *S.* Typhi bloodstream infections with resistance to tested antibiotics, DOH-ARSP, 2024

Diarrheal Disease

The resistance rates of *S.* Typhi diarrheal diseases are seen in **Figure 30.** Resistance to ampicillin and co-trimoxazole was 5.71% and 2.86%, respectively, while no resistance to ciprofloxacin was observed.

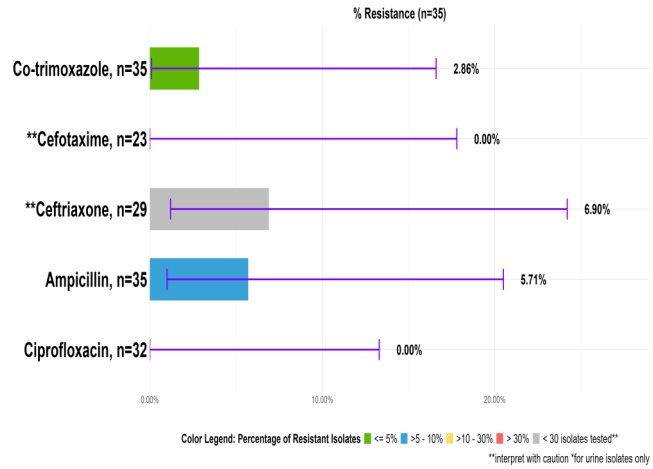


Figure 30. Proportion of *S.* Typhi diarrheal diseases with resistance to tested antibiotics from stool specimens, DOH-ARSP, 2024

Non-typhoidal *Salmonella* species

A total of **351** non-typhoidal *Salmonella* (NTS) infections were reported for 2024.

351
infections

This was a 9.35% increase than the 321 infections noted in 2023. The highest contributing sites were PGH (16.24%) and VSM (13.68%). Infections were mostly reported from Luzon (63.53%) followed by Mindanao (18.52%) and Visayas (17.95%).

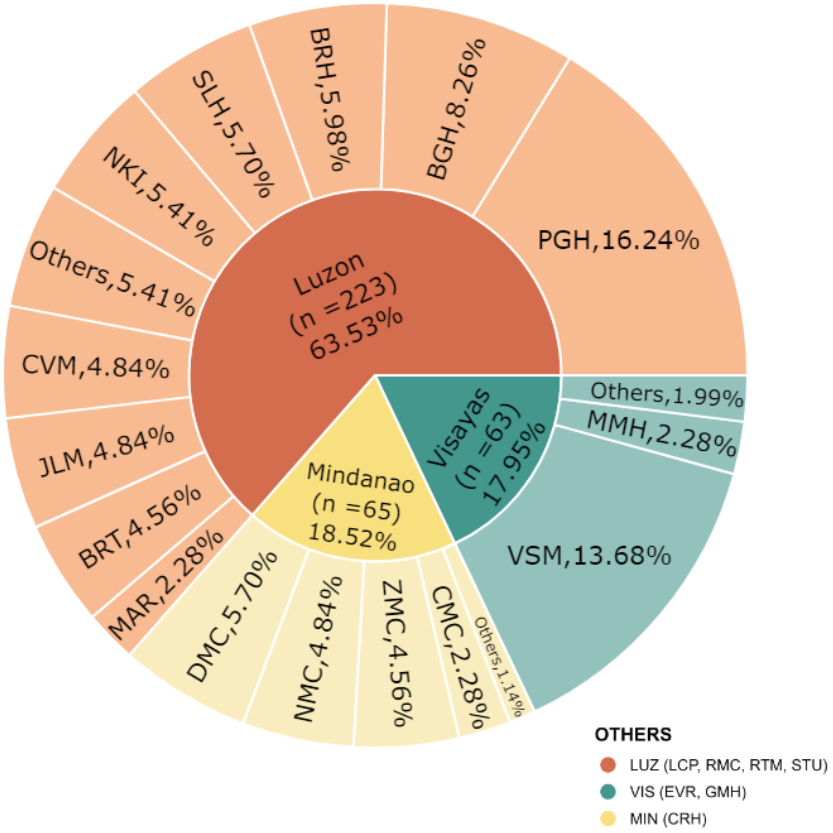


Figure 31. Distribution of NTS infections, DOH-ARSP, 2024 (n=351)

More than half (53.28%) of NTS infections occurred in male patients, and 55.27% were from the 20–64 age group (Figure 32). The majority (40.46%) of the infections were reported from blood specimens.

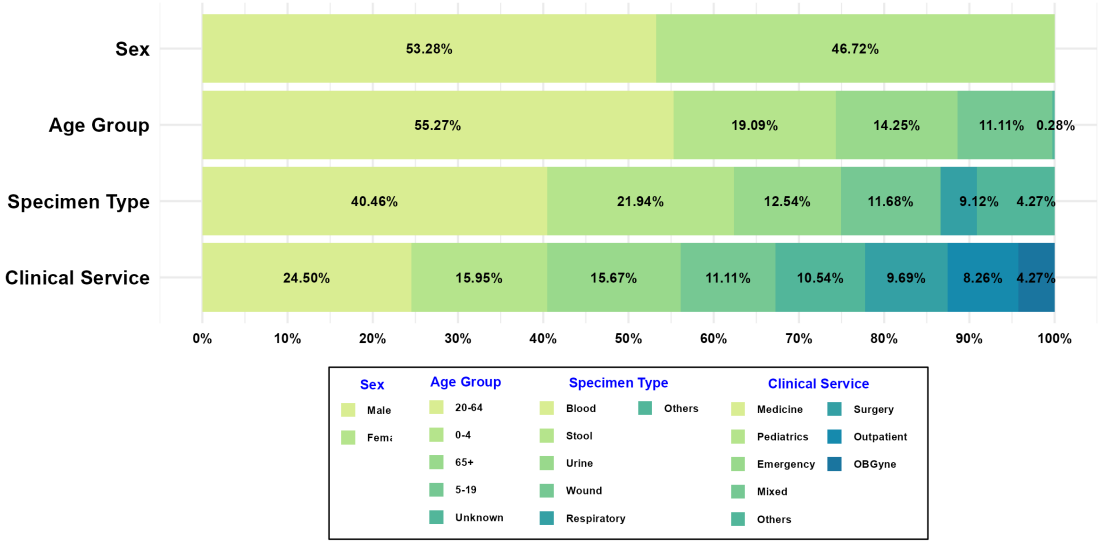


Figure 32. Patient characteristics in NTS infections, DOH-ARSP, 2024 (n=351)

Figure 33 shows that the percent positive of NTS bloodstream infections for all surveillance sites was less than 1.5% with RTM (1.22%) shows the highest percent positive followed by PGH (0.83%) and ZMC (0.34%).

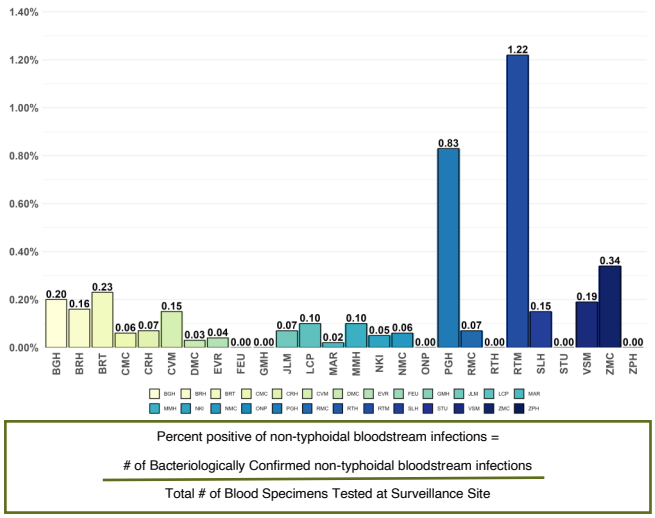


Figure 33. Percent positive of NTS bloodstream infections among all tested blood specimens per surveillance site, DOH-ARSP, 2024

All types of Infections

Figure 34 presents the 2024 resistance rates for all NTS infections. Ciprofloxacin (9.80%) (p=0.0592) and ceftriaxone (19.22%) (p=0.0859) showed decreases compared to 2023, other notable resistance rates included azithromycin (22.22%) (p=0.3673), chloramphenicol (19.90%) (p=0.9595), and co-trimoxazole (14.06%) (p=0.0872). All carbapenems demonstrated resistance rates below 3%.

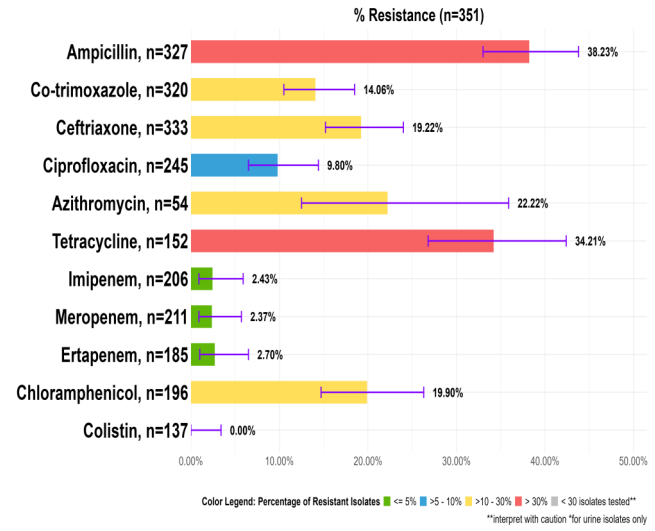
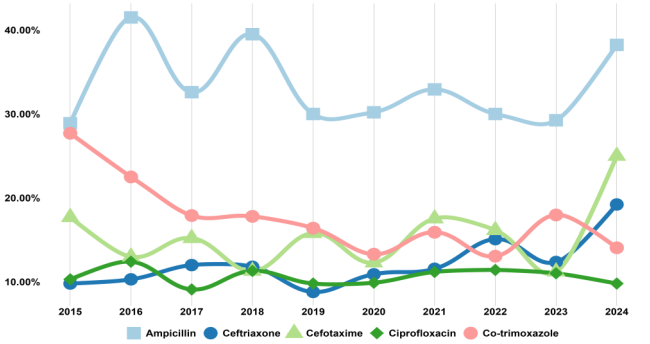


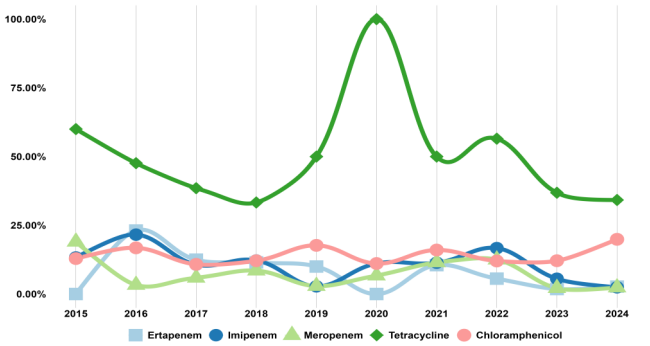
Figure 34. Proportion of all NTS infections with resistance to tested antibiotics, DOH-ARSP, 2024

There were three (3) NTS (1 *Salmonella Infantis*, 2 not serotyped) confirmed to be resistant to cefotaxime and ceftriaxone. These were from blood samples of a 1-year old and two (2) adult patients. The isolates were resistant to ampicillin , chloramphenicol, ceftriaxone, and cefotaxime, intermediate to ciprofloxacin, but susceptible to cotrimoxazole and the carbapenems ertapenem and imipenem.

Figure 35 shows the multi-year analysis of all NTS infections. The decreasing trend in resistance rates for ampicillin and cefotaxime observed from 2021 to 2023 reversed in 2024, with an increase in rates, although this change was not statistically significant. In contrast, resistance rates to ciprofloxacin, carbapenems continued to decrease.



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Ampicillin	28.9%	41.5%	32.6%	39.5%	30.0%	30.2%	32.9%	30.0%	29.2%	38.2%
Ceftriaxone	9.8%	10.3%	12.0%	11.8%	8.8%	10.9%	11.6%	15.1%	12.4%	19.2%
Cefotaxime	17.7%	13.0%	15.2%	11.3%	15.8%	12.3%	17.5%	16.1%	11.2%	25.0%
Ciprofloxacin	10.3%	12.4%	9.1%	11.3%	9.8%	9.9%	11.2%	11.4%	11.0%	9.8%
Co-trimoxazole	27.7%	22.5%	17.9%	17.8%	16.4%	13.3%	15.9%	13.1%	18.0%	14.1%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Ertapenem	0.0%	23.1%	12.5%	11.5%	10.0%	0.0%	10.5%	5.6%	1.8%	2.7%
Imipenem	13.3%	21.6%	10.7%	12.2%	2.8%	11.1%	11.4%	16.7%	5.6%	2.4%
Meropenem	19.0%	3.3%	5.9%	8.5%	2.9%	6.7%	11.5%	12.5%	2.1%	2.4%
Tetracycline	60.0%	47.6%	38.5%	33.3%	50.0%	100.0%	50.0%	56.5%	36.8%	34.2%
Chloramphenicol	12.9%	16.8%	10.8%	12.1%	17.7%	11.1%	16.0%	12.0%	12.1%	19.9%

Figure 35. Yearly resistance rates of all NTS infections, DOH-ARSP 2024

Bloodstream Infections

Figure 36 shows the resistance rates of NTS bloodstream infections. Tetracycline resistance was highest at 23.81% followed by ampicillin at 18.94% and azithromycin at 12.90%. Resistance rates observed in bloodstream infections were lower than the overall cumulative resistance rates for all NTS infections.

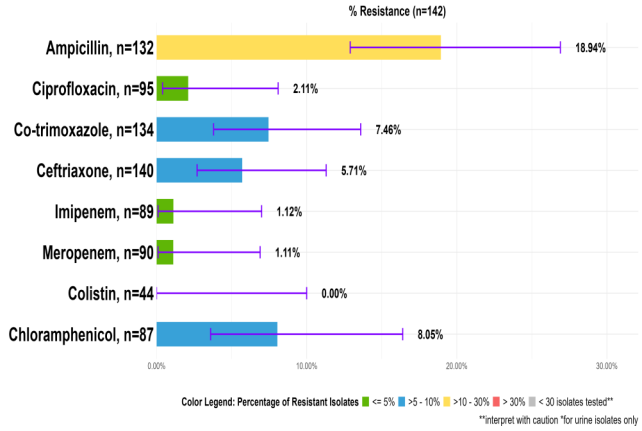
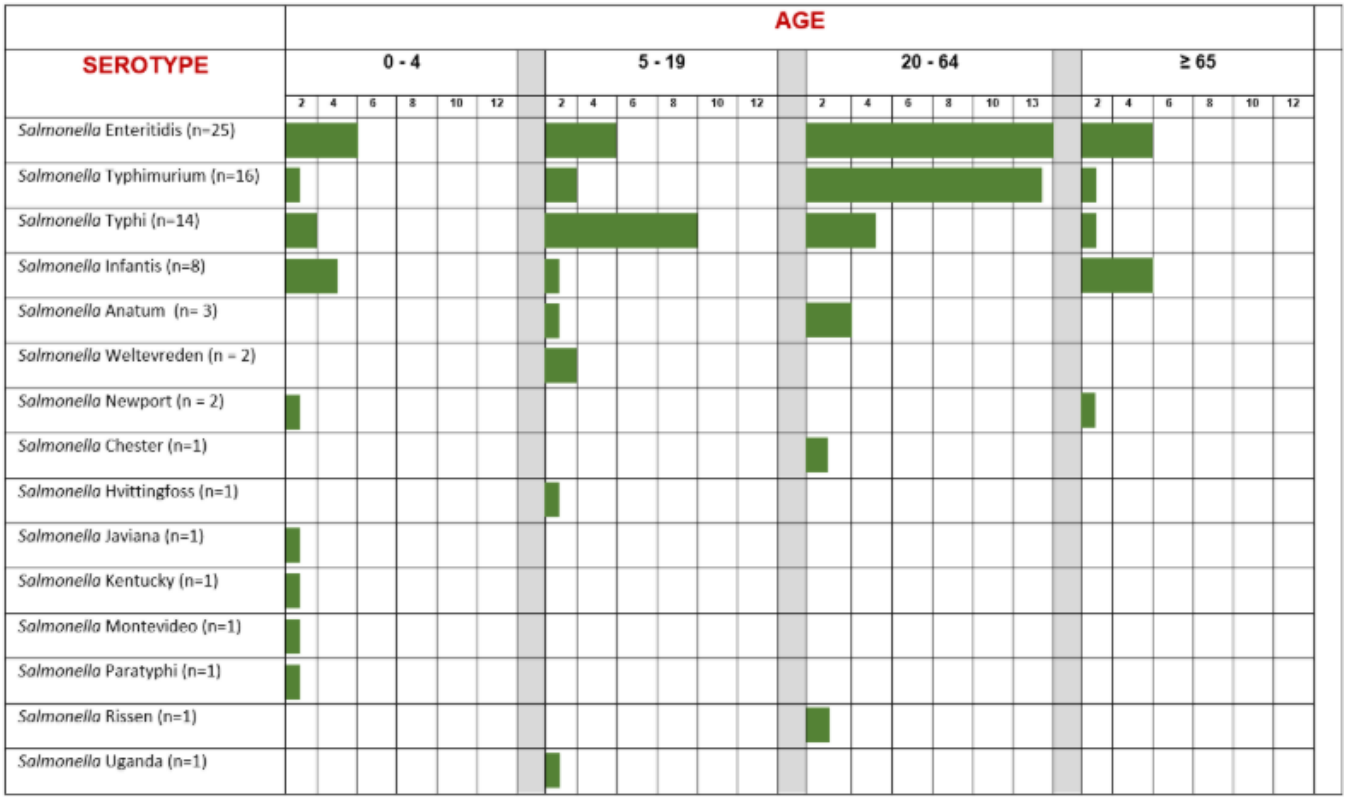


Figure 36. Proportion of NTS bloodstream infections with resistance to tested antibiotics, DOH-ARSP, 2024

There were **78** *Salmonellae* referred to the reference laboratory for serotyping. There were 15 *Salmonella* serotypes identified for 2024. Most of the isolates were *Salmonella enterica* serovar *Enteritidis* (32.05%) followed by *Salmonella enterica* serovar Typhimurium (20.51%) and *Salmonella enterica* serovar Typhi. These three serotypes had been the most common serotypes reported for the past three years. Antimicrobial resistance among NTS may reflect variations in serotypes, its distribution or both.

Table 9. *Salmonella* serotypes per age group, DOH-ARSP, 2024



Shigella species

There were **34** *Shigella* infections reported in 2024.

34
infections

Most infections were from VSM (44.12%), BRH (11.76%) and NKI (8.82%). Based on island group distribution, the majority were from Visayas at 47.06%, Luzon at 35.29% and Mindanao at 17.65%. More than half (55.88%) of the infections were observed among male patients and most (52.94%) are from stool samples.

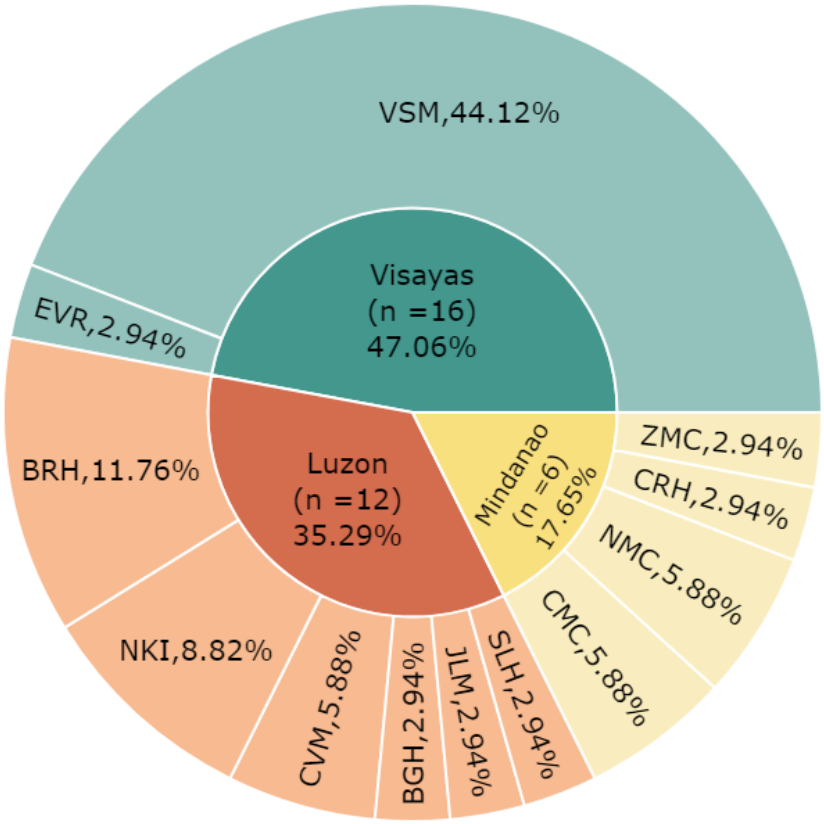


Figure 37. Distribution of *Shigella* infections, DOH-ARSP, 2024 (n=34)

More than half (55.88%) of the infections occurred among female patients and the majority are within the 0-4 (35.29%) and 20-64 (35.29%) age groups. More than half (52.94%) of the infections were observed from stool specimens.

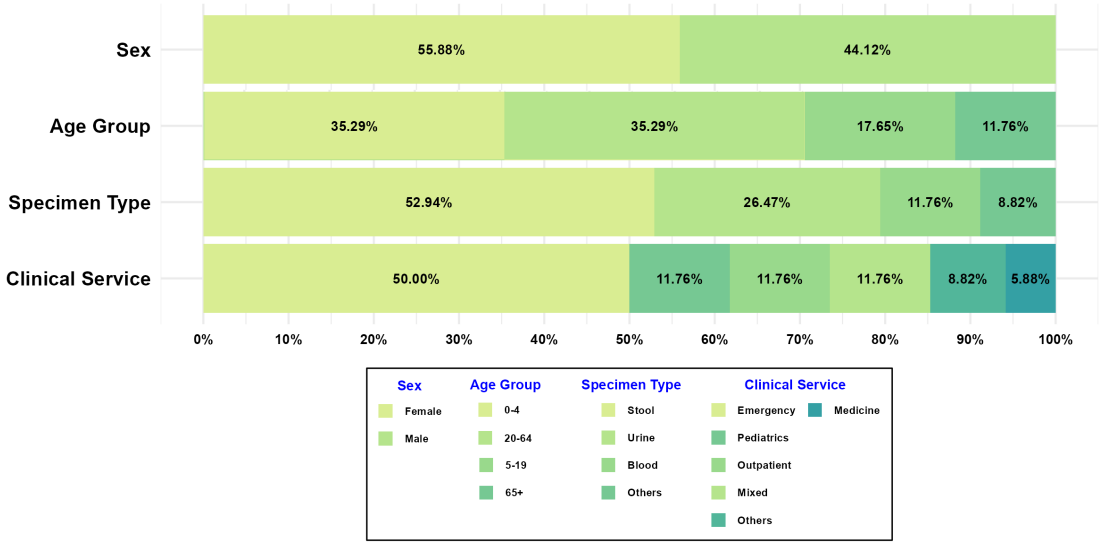


Figure 38. Patient characteristics in *Shigella* infections, DOH-ARSP, 2024 (n=34)

The percent positive of stool infections caused by *Shigella sp.* observed for five surveillance sites including VSM, BRH, CRH, BGH and JLM were 6.07%, 4.76%, 1.43%, 0.84% and 0.72% respectively.

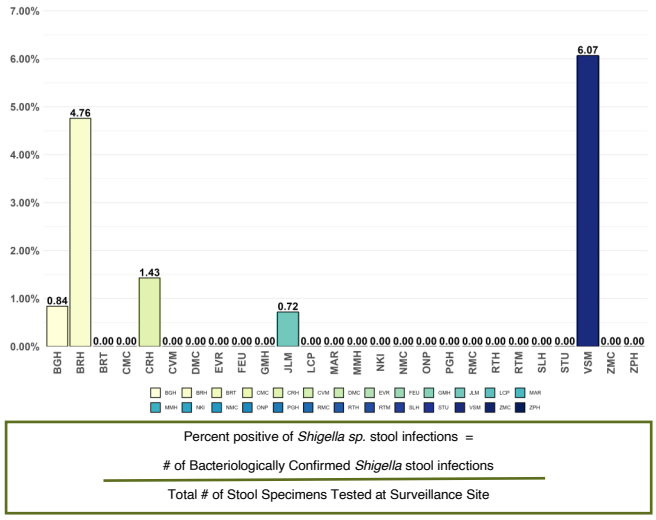


Figure 39. Percent positive of *Shigella sp.* stool infections among all tested stool specimens per surveillance site, DOH-ARSP, 2024

All types of Infections

Figure 40 shows the cumulative resistance rates of all *Shigella* infections. There were few reported *Shigella* infections for 2024. Resistance to ceftriaxone was at 6.67 %, and to ciprofloxacin at 12.50%. A comparison of the cumulative rates obtained from 2006 to 2024 (**Figure 41**) 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 = 0.0005), chloramphenicol (p=0.0009), azithromycin (p=0.0003) and ceftriaxone (p=0.0074).

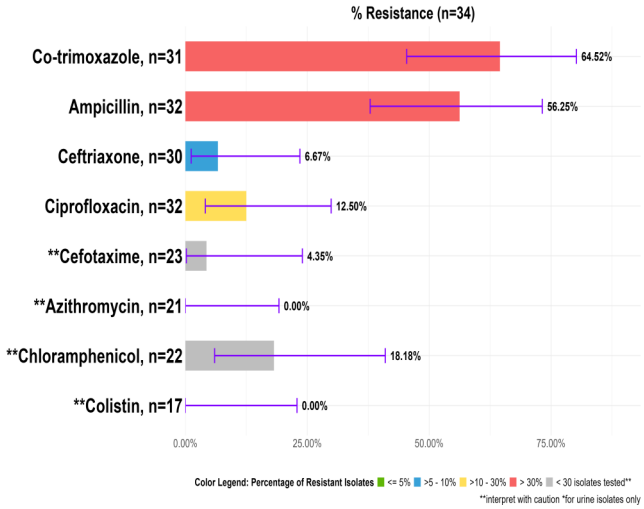
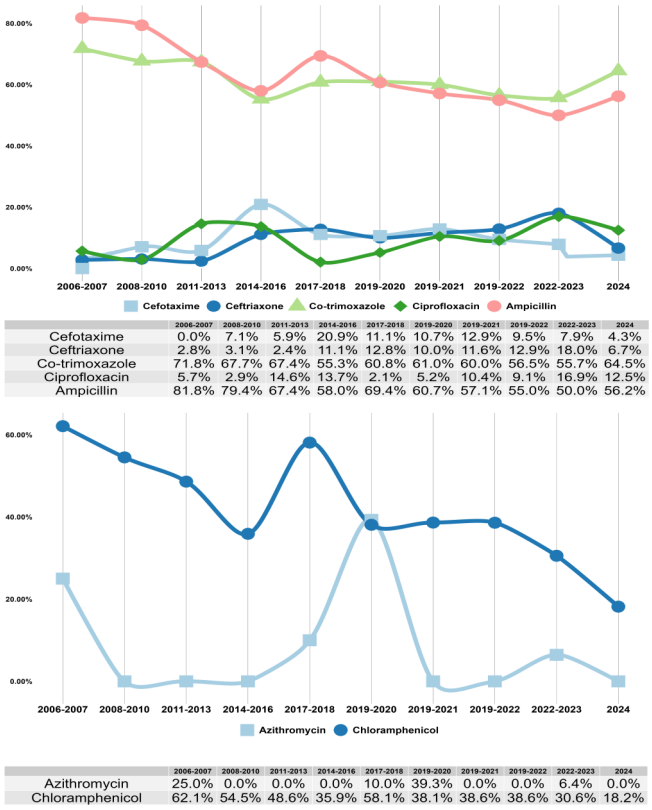


Figure 40. Proportion of all *Shigella* infections with resistance to tested antibiotics, DOH-ARSP, 2024

Multi-year analysis showed that the changes in the resistance rates of *Shigella* infections for ampicillin (p=0.0005), azithromycin (p=0.0003) and ceftriaxone (p=0.0074) were all statistically significant, likewise the decreasing trend seen in chloramphenicol (p=0.0009).



	2006-2007	2008-2010	2011-2013	2014-2016	2017-2018	2019-2020	2019-2021	2019-2022	2022-2023	2024
Cefotaxime	0.0%	7.1%	5.9%	20.9%	11.1%	10.7%	12.9%	9.5%	7.9%	4.3%
Ceftriaxone	2.8%	3.1%	2.4%	11.1%	12.8%	10.0%	11.6%	12.9%	18.0%	6.7%
Co-trimoxazole	71.8%	67.7%	67.4%	55.3%	60.8%	61.0%	60.0%	56.5%	55.7%	64.5%
Ciprofloxacin	5.7%	2.9%	14.6%	13.7%	2.1%	5.2%	10.4%	9.1%	16.9%	12.5%
Ampicillin	81.8%	79.4%	67.4%	58.0%	69.4%	60.7%	57.1%	55.0%	50.0%	56.2%

	2006-2007	2008-2010	2011-2013	2014-2016	2017-2018	2019-2020	2019-2021	2019-2022	2022-2023	2024
Azithromycin	25.0%	0.0%	0.0%	0.0%	10.0%	39.3%	0.0%	0.0%	6.4%	0.0%
Chloramphenicol	62.1%	54.5%	48.6%	35.9%	58.1%	38.1%	38.6%	38.6%	30.6%	18.2%

Figure 41. Yearly resistance rates of all *Shigella* infections, DOH-ARSP, 2024

Diarrheal Disease

The resistance rates for *Shigella* diarrheal diseases from 2023-2024 are shown in **Figure 42**. The high resistance rates were observed for co-trimoxazole (71.43%), ampicillin (53.49%). Ampicillin and co-trimoxazole resistance rates from all specimens were higher in 2024. However, the noted increases were not statistically significant.

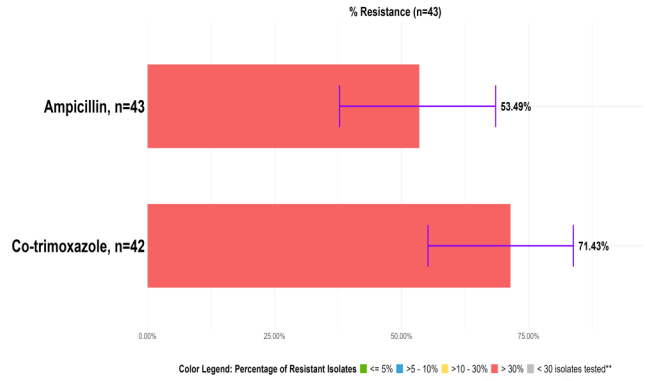


Figure 42. Proportion of *Shigella* diarrheal diseases resistant to tested antibiotics, DOH-ARSP, 2024

Vibrio cholerae

There were **79** *Vibrio cholerae* infections reported for 2024

79
infections

This is 14.50% higher than the reported infections in 2023. The majority of infections were from MMH (37.97%), CMC (21.52%) and ZMC (10.13%). Distribution by island group shows that Visayas have the majority of isolates at 46.84%, Mindanao at 37.97% and Luzon at 15.19%.

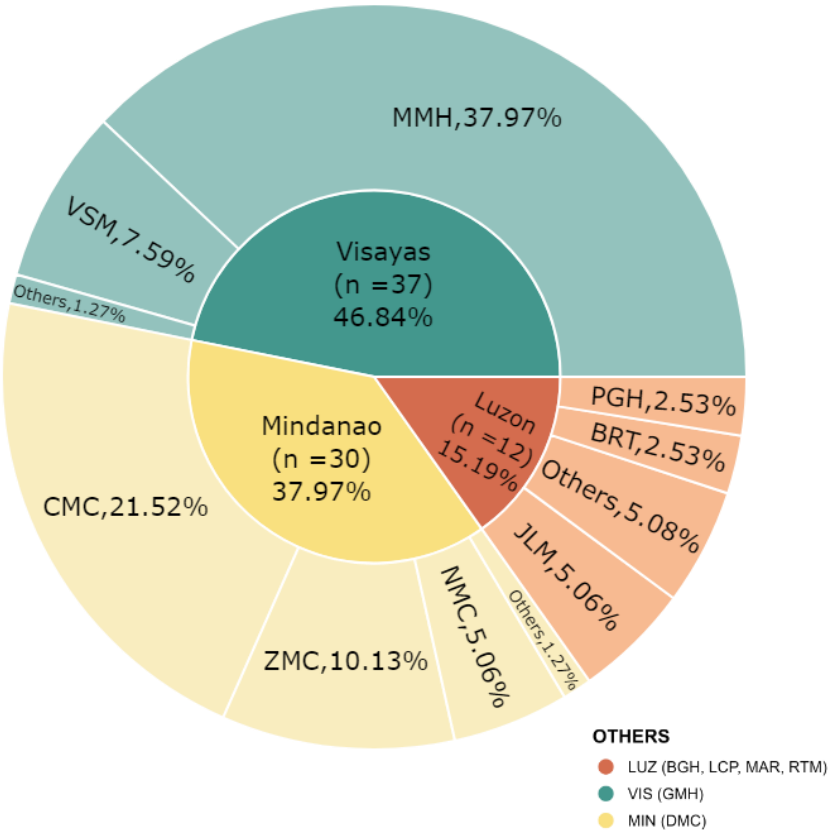


Figure 43. Distribution of *V. cholerae* infections, DOH-ARSP, 2024 (n=79)

More than half (53.16%) of the *V. cholerae* infections were from male patients and were mostly seen among 5-19 (39.24%) and 20-64 (39.24%) age groups. Most (82.28%) of the isolates were detected from stool specimens.

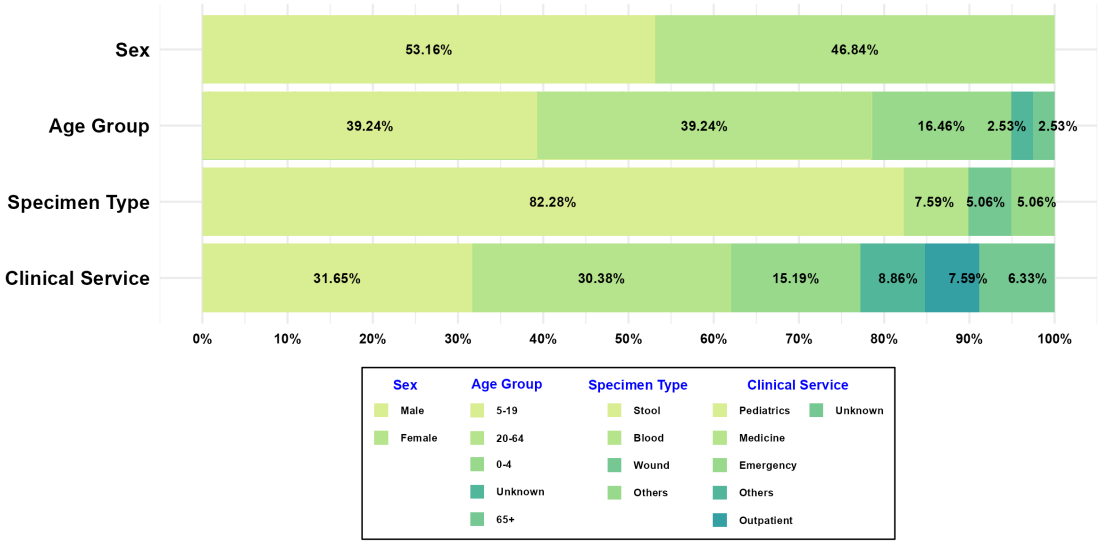


Figure 44. Patient characteristics in *V. cholerae* infections, DOH-ARSP, 2024 (n=79)

As seen in Figure 45, percent positive of stool infections caused by *V. cholerae* were only observed in three surveillance sites including CMC (5.08%), ZMC (4.11%) and BGH (0.84%).

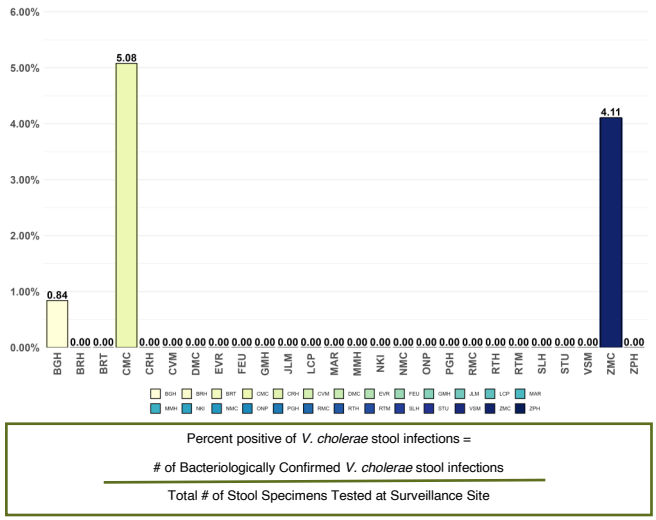


Figure 45. Percent positive of *V. cholerae* stool infections among all tested stool specimens per surveillance site, DOH-ARSP, 2024

All types of Infections

For 2024, all *V. cholerae* infections were susceptible to chloramphenicol and azithromycin. Resistance to ampicillin increased to 28.38% from 16.67% in 2023. Resistance to co-trimoxazole was at 6.85%.

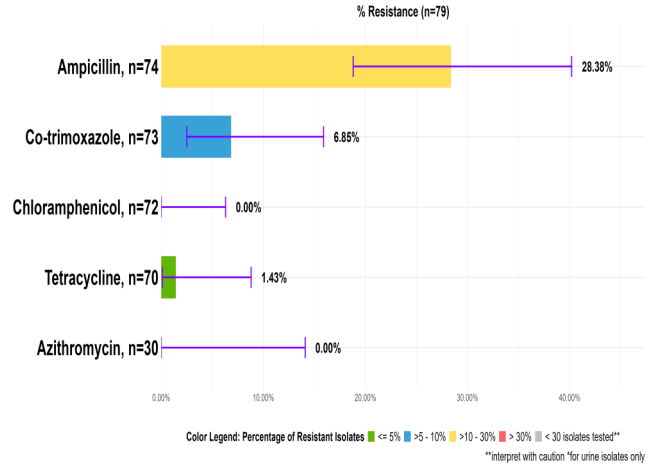
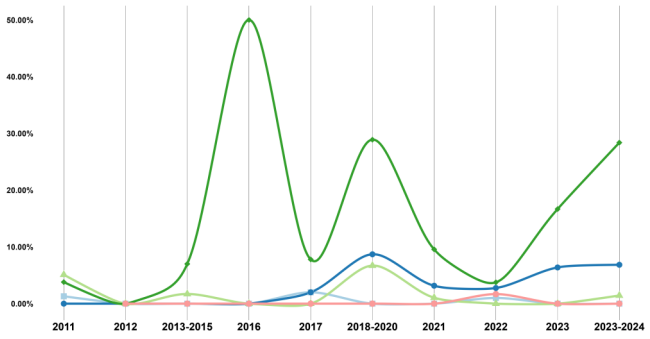


Figure 46. Proportion of all *V. cholerae* infections resistant to tested antibiotics, DOH-ARSP, 2024

Yearly resistance rates of all *V. cholerae* infections are shown in Figure 47. Ampicillin (p=0.699) and co-trimoxazole (p=0.0349) resistance showed increasing trends, with the increase for co-trimoxazole being significant.



	2011	2012	2013-2015	2016	2017	2018-2020	2021	2022	2023	2023-2024
Chloramphenicol	1.3%	0.0%	0.0%	0.0%	2.0%	0.0%	0.0%	1.0%	0.0%	0.0%
Co-trimoxazole	0.0%	0.0%	0.0%	0.0%	2.0%	8.7%	3.2%	2.8%	6.4%	6.8%
Tetracycline	5.1%	0.0%	1.7%	0.0%	0.0%	6.7%	1.0%	0.0%	0.0%	1.4%
Ampicillin	3.8%	0.0%	7.0%	50.0%	7.8%	28.9%	9.6%	3.8%	16.7%	28.4%
Azithromycin (MIC)		0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	1.7%	0.0%	0.0%

Figure 47. Yearly resistance rates of all *V. cholerae* infections, DOH-ARSP, 2024

Neisseria gonorrhoeae

A total of **146** *N. gonorrhoeae* infections were reported for 2024.

146
infections

This is an 80% increase from 81 infections reported in 2023. The highest contributors are CVM (31.51%), VSM (19.86%) and MAR (10.96%) (Figure 48). Based on island group distribution, the highest was Luzon with 67.12% followed by Visayas at 26.71% and Mindanao at 6.16%.

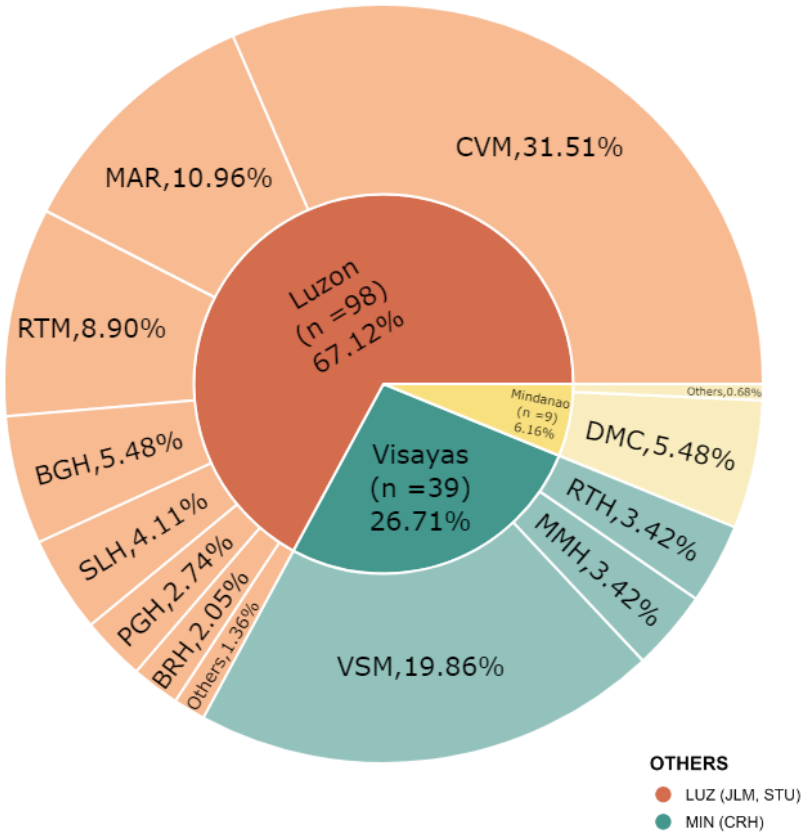


Figure 48. Distribution of *N. gonorrhea* infections, DOH-ARSP, 2024 (n=146)

Majority of infections reported were observed in male patients (86.30%), with the 20-64 age group comprising 73.97%. Genital specimens remain to be the most common source of *N. gonorrhoeae* isolates at 87.67%.

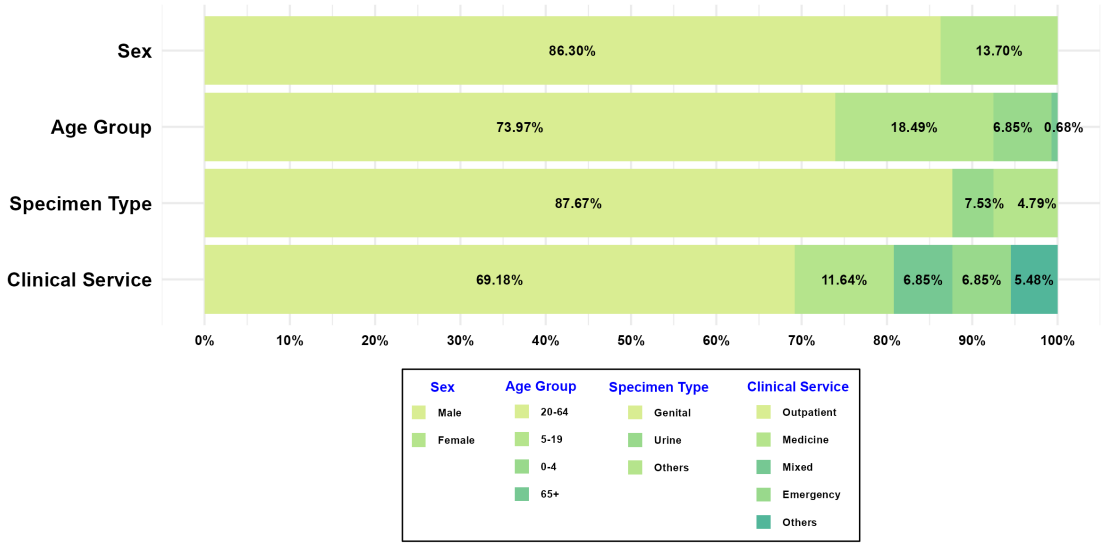


Figure 49. Patient characteristics in *N. gonorrhoeae* infections, DOH-ARSP, 2024 (n=146)

Figure 50 shows that 52.17% and 41.67% of the genital infections in RTM and RTH respectively were caused by *N. gonorrhoeae*. The percent positive of genital infections due to *N. gonorrhoeae* for BGH, CVM, STU and MMH ranges from 8.23-9.23%, while the percent positive for MAR, JLM, PGH, BRH, VSM, DMC and CRH were less than 5.5%.

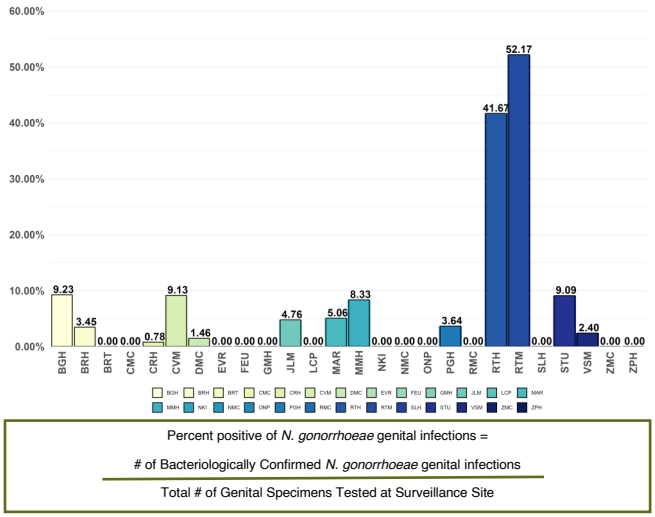


Figure 50. Percent positive of *N. gonorrhoeae* genital infections among all tested genital specimens per surveillance site, DOH-ARSP, 2024

All types of Infections

All isolates from reported infections showed susceptibility to ceftriaxone, cefixime, and azithromycin. The antibiotic resistance rates of gonococcal infections were reported to be 89.76% for ciprofloxacin and 78.40% for tetracycline.

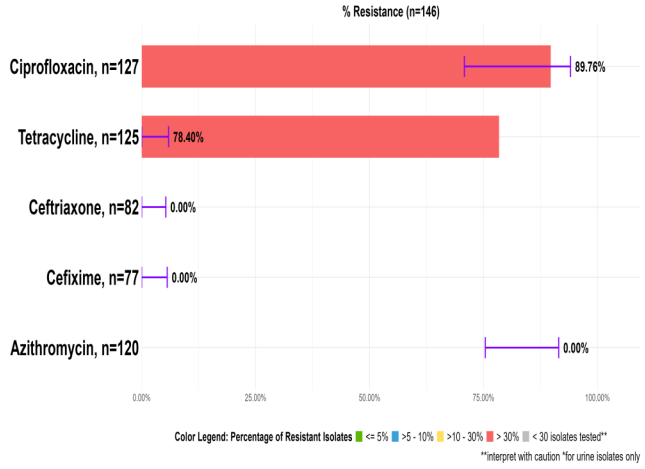
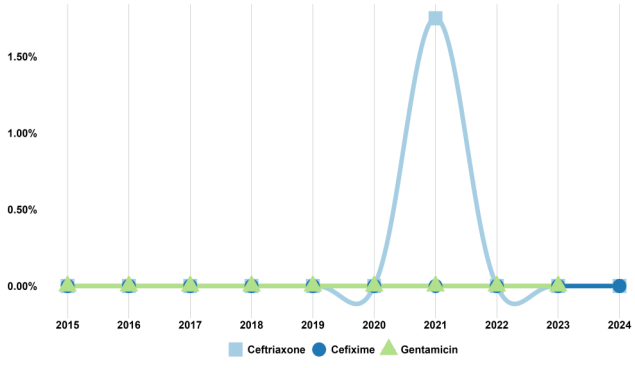
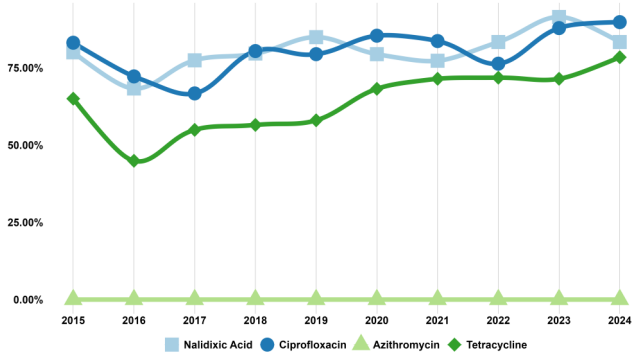


Figure 51. Proportion of all *N. gonorrhea* infections resistant to tested antibiotics, DOH-ARSP, 2024

Figure 52 shows the multi-year analysis of antibiotic resistance rates in gonococcal infections across the different antibiotics. No confirmed resistance to ceftriaxone, cefixime and azithromycin have been reported over the past 10 years. All changes in yearly resistance rates, however, were not statistically significant.



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Ceftriaxone	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	1.8%	0.0%	0.0%	0.0%
Cefixime	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Gentamicin	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Nalidixic Acid	80.0%	68.3%	77.4%	79.6%	84.9%	79.4%	77.3%	83.3%	91.4%	83.3%
Ciprofloxacin	83.1%	72.2%	66.7%	80.4%	79.4%	85.4%	83.6%	76.3%	87.8%	89.8%
Azithromycin	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Tetracycline	65.0%	44.9%	54.9%	56.5%	58.0%	68.2%	71.4%	71.8%	71.4%	78.4%

Figure 52. Yearly resistance rates of all confirmed *N. gonorrhea* infections, DOH-ARSP, 2024

Staphylococcus aureus

There were **8,723** *Staphylococcus aureus* infections reported for 2024

8,723 Infections

An 11.35% increase in *S. aureus* infections was observed from 2023. The majority of *S. aureus* infections came from surveillance sites in Luzon (62.62%) with 29.52% coming from NCR, Mindanao (21.64%), and Visayas (15.74%) (**Figure 53**).

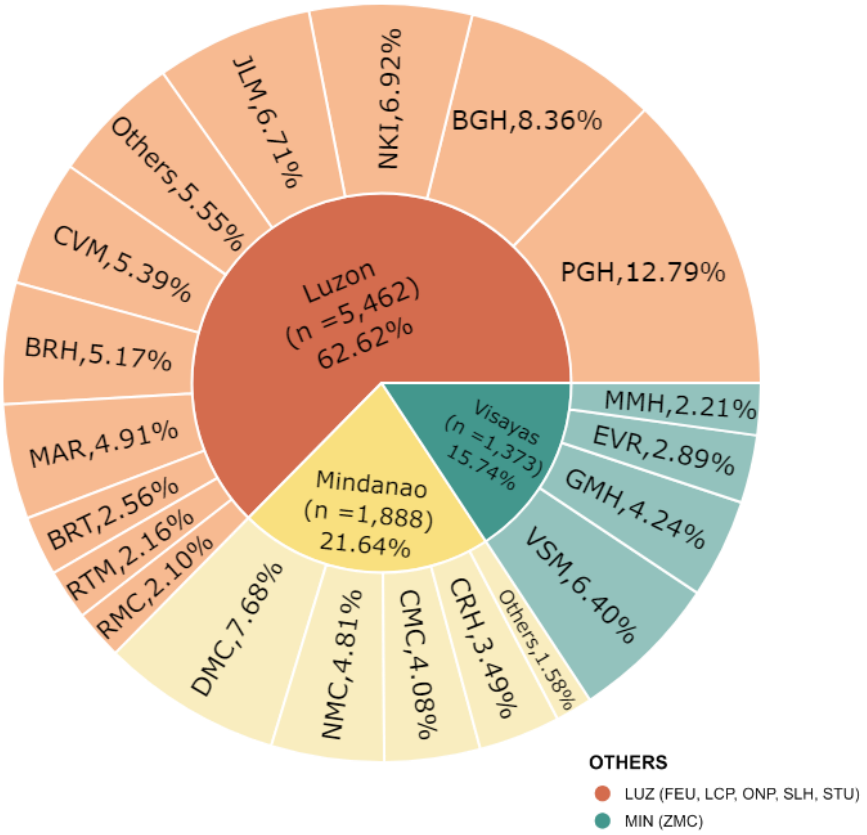


Figure 53. Distribution of *S. aureus* infections , DOH-ARSP, 2024 (n=8,723)

More than half of the infections (54.98%) were observed among male patients and most were from aged 20-64 years old (66.11%). As shown in **Figure 54**, most infections were isolated from wound (39.60%) and blood specimens (22.96%).

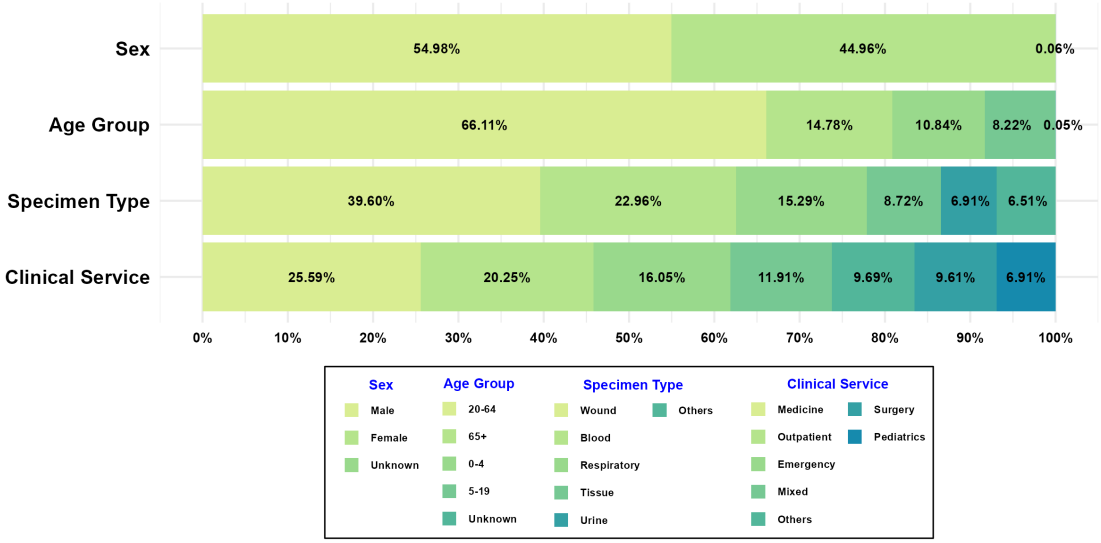


Figure 54. Patient characteristics in *S. aureus* infections, DOH-ARSP, 2024 (n=8,723)

Figure 55 shows that the percent positive of bloodstream infections caused by *S. aureus* in FEU, PGH, NKI, RTM and MMH were 10.14%, 7.14%, 5.59%, 4.88% and 2.70%. The percent positive of BSI due to *S. aureus* for the remaining sites were noted to be from 0.35- 1.96%.

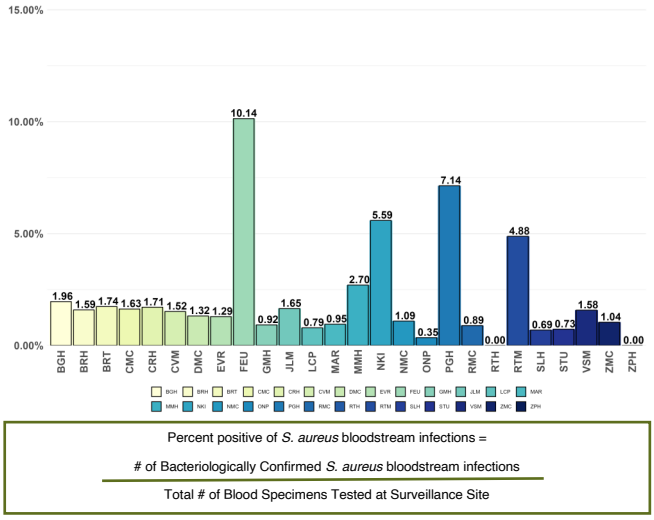


Figure 55. Percent positive of *S. aureus* bloodstream infections among all tested blood specimens per surveillance site, DOH-ARSP, 2024

Among the wound infections from RTM and FEU, 60% and 44.44% of which were caused by *S. aureus* (Figure 56). The percent positive of wound infections due to *S. aureus* for BGH, CRH, CVM, GMH, MAR and PGH were more than 20%. While percent positive of *S. aureus* wound infections for VSM, ZMC, ONP, NKI, BRH, NMC, CMC, DMC, JLM, SLH and STU ranges from 11.42-19.91%. The four sites with less than 10% percent positive of *S. aureus* infections include BRT, EVR, LCP and RMC.

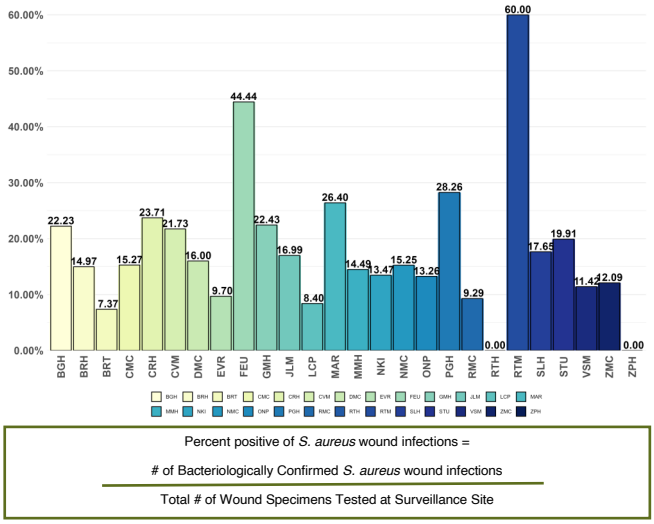


Figure 56. Percent positive of *S. aureus* wound infections among all tested wound specimens per surveillance site, DOH-ARSP, 2024

All types of Infections

Figure 57 shows the resistance rates of isolates from all *S. aureus* infections. Oxacillin resistance was at 38.44%, clindamycin at 9.86% and cotrimoxazole at almost 40%. Resistance to vancomycin and linezolid were less than 2%. Although there were reported vancomycin resistance in 2024, this was not confirmed in the reference laboratory. All isolates from *S. aureus* infections tested against daptomycin were susceptible.

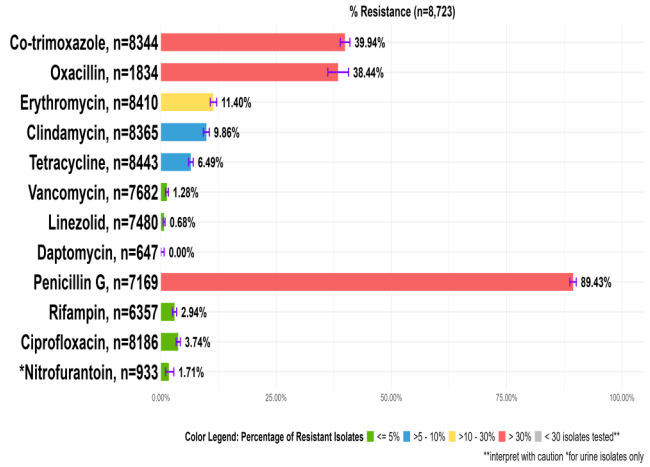
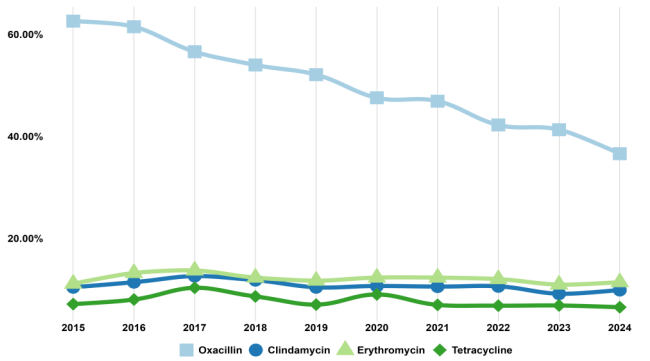
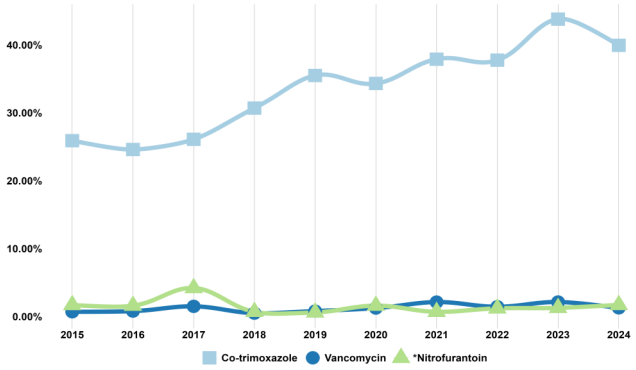


Figure 57. Proportion of all *S. aureus* infections resistant to tested antibiotics, DOH-ARSP, 2024 (n=8,723)

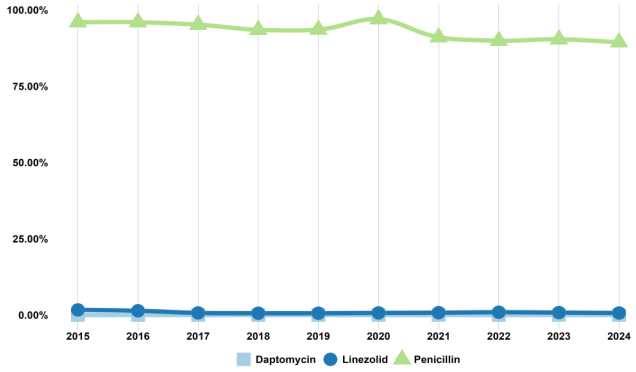
The resistance rates of all *S. aureus* infections are shown in Figure 58. Over the past decade, statistically significant changes in resistance rates were observed for oxacillin (p=0.0000), vancomycin (p=0.0001), and co-trimoxazole (p=0.0000), with oxacillin resistance showing a steadily decreasing trend.



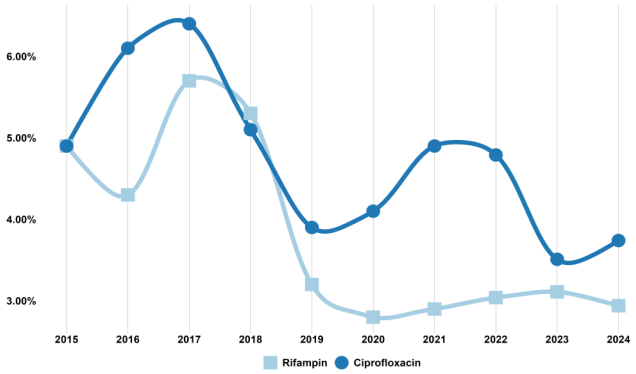
	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Oxacillin	62.6%	61.5%	56.6%	54.0%	52.1%	47.6%	46.9%	42.2%	41.3%	36.6%
Clindamycin	10.4%	11.4%	12.6%	11.8%	10.4%	10.6%	10.5%	10.6%	9.1%	9.9%
Erythromycin	11.1%	13.2%	13.7%	12.3%	11.7%	12.3%	12.3%	12.0%	10.9%	11.4%
Tetracycline	7.1%	8.0%	10.3%	8.6%	7.0%	9.0%	7.0%	6.8%	6.8%	6.5%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Co-trimoxazole	25.9%	24.6%	26.1%	30.7%	35.5%	34.3%	37.9%	37.8%	43.8%	39.9%
Vancomycin	0.7%	0.8%	1.5%	0.5%	0.8%	1.2%	2.1%	1.4%	2.1%	1.3%
*Nitrofurantoin	1.7%	1.6%	4.2%	0.7%	0.6%	1.6%	0.7%	1.2%	1.3%	1.7%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Daptomycin	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Linezolid	1.7%	1.4%	0.7%	0.6%	0.6%	0.7%	0.8%	0.9%	0.8%	0.7%
Penicillin	96.0%	96.0%	95.2%	93.5%	93.6%	97.0%	91.1%	89.9%	90.4%	89.4%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Rifampin	4.9%	4.3%	5.7%	5.3%	3.2%	2.8%	2.9%	3.0%	3.1%	2.9%
Ciprofloxacin	4.9%	6.1%	6.4%	5.1%	3.9%	4.1%	4.9%	4.8%	3.5%	3.7%

Figure 58. Yearly resistance rates of all *S. aureus* infections

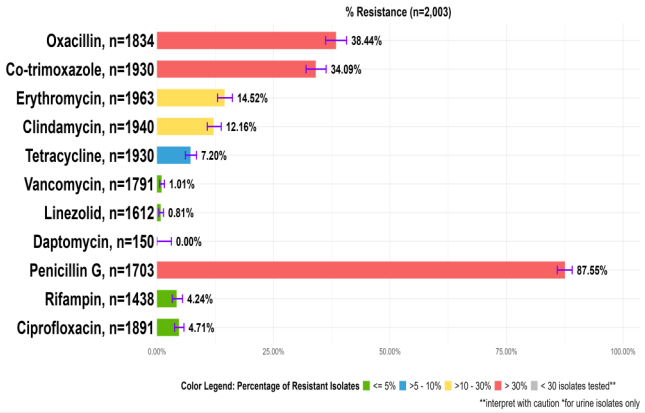
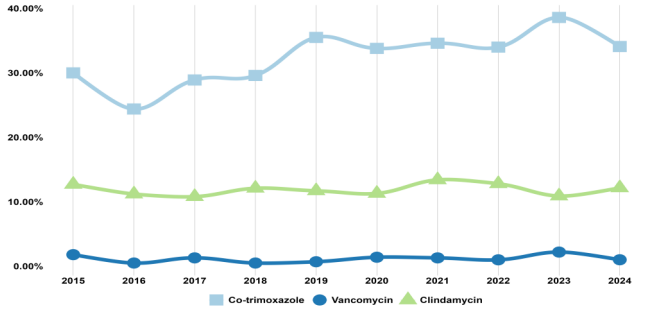
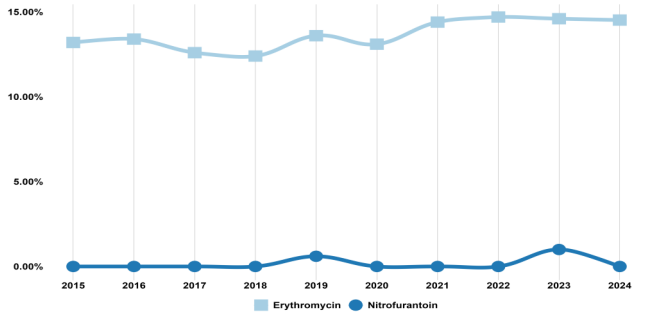


Figure 59. Proportion of *S. aureus* bloodstream infections resistant to tested antibiotics, DOH-ARSP, 2024 (n=2,003)

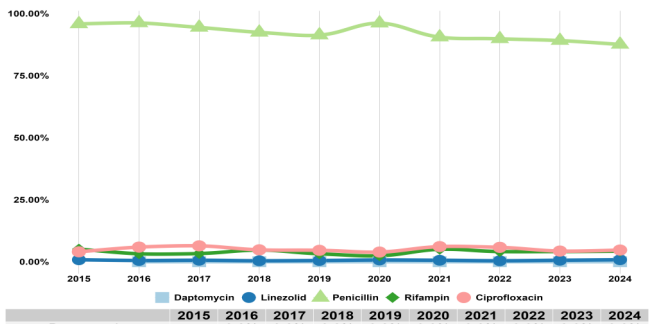
Figure 60 illustrates the resistance rates of *S. aureus* bloodstream infections. The resistance rates in bloodstream infections were generally similar to those observed across all infection types, although there was slightly higher resistance to erythromycin (14.52%) and rifampin (4.24%).



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Co-trimoxazole	30.0%	24.4%	28.9%	29.6%	35.5%	33.8%	34.6%	34.0%	38.6%	34.1%
Vancomycin	1.8%	0.5%	1.3%	0.5%	0.7%	1.4%	1.3%	1.0%	2.2%	1.0%
Clindamycin	12.7%	11.2%	10.8%	12.1%	11.7%	11.3%	13.4%	12.8%	10.9%	12.2%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Erythromycin	13.2%	13.4%	12.6%	12.4%	13.6%	13.1%	14.4%	14.7%	14.6%	14.5%
Nitrofurantoin	0.0%	0.0%	0.0%	0.0%	0.6%	0.0%	0.0%	0.0%	1.0%	0.0%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Daptomycin		0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Linezolid	0.8%	0.5%	0.6%	0.4%	0.5%	0.7%	0.6%	0.4%	0.6%	0.8%
Penicillin	95.8%	96.2%	94.4%	92.4%	91.3%	96.1%	90.5%	89.8%	89.1%	87.5%
Rifampin	5.1%	3.2%	3.3%	4.7%	3.2%	2.4%	5.0%	4.1%	4.1%	4.2%
Ciprofloxacin	4.0%	5.9%	6.4%	4.8%	4.6%	3.9%	6.1%	5.8%	4.3%	4.7%

Figure 60. Yearly resistance rates of *S. aureus* bloodstream infections

Bloodstream Infections

Figure 59 shows the resistance rates of *S. aureus* bloodstream infections. While generally similar to rates from all specimens, resistance to clindamycin (12.16%) and erythromycin (14.52%) was higher in bloodstream infections.

Skin and Soft Tissue Infections

Resistance to oxacillin was at 33.62%, clindamycin at 8.01% and linezolid at less than 1%. Although there were reported vancomycin resistance, this was not confirmed in the reference laboratory. Compared to the resistance rates observed in all infections, *S. aureus* skin and soft tissue infections showed slightly higher resistance to clindamycin, erythromycin, rifampin, and slightly lower resistance to co-trimoxazole **(Figure 61)**.

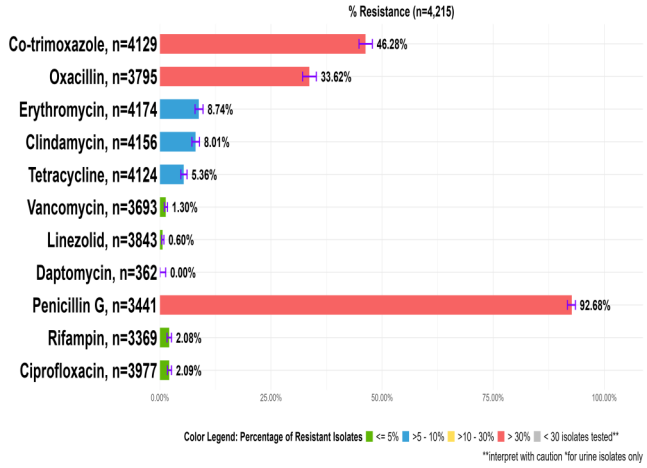
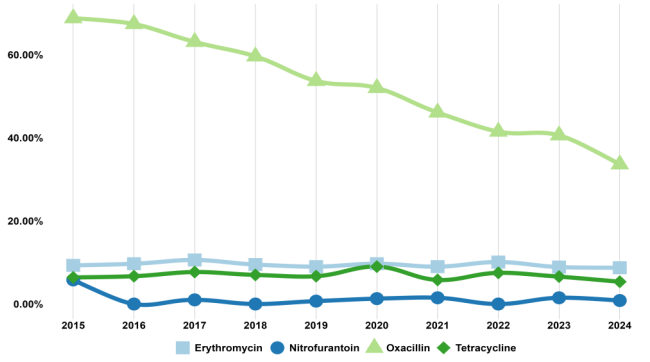
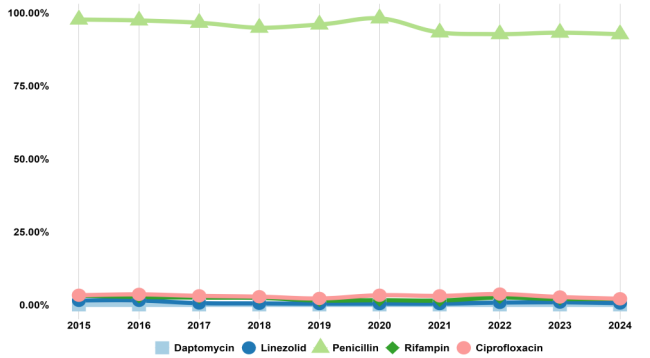


Figure 61. Proportion of *S. aureus* skin and soft tissue infections resistant to tested antibiotics, DOH-ARSP, 2024 (n=4,215)



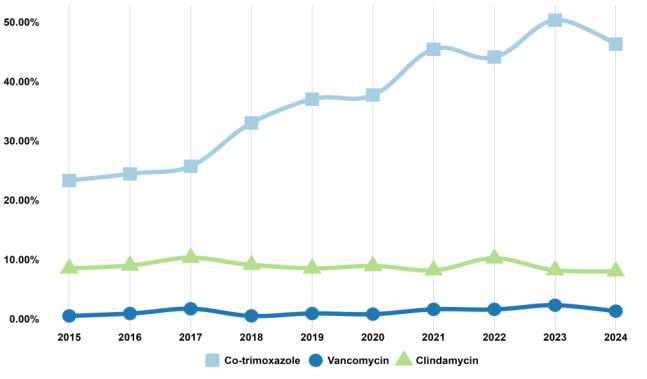
	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Erythromycin	9.3%	9.7%	10.6%	9.5%	9.0%	9.7%	9.0%	10.1%	8.9%	8.7%
Nitrofurantoin	5.8%	0.0%	1.0%	0.0%	0.7%	1.3%	1.5%	0.0%	1.5%	0.8%
Oxacillin	68.8%	67.4%	63.1%	59.6%	53.7%	52.0%	46.1%	41.5%	40.6%	33.6%
Tetracycline	6.4%	6.7%	7.7%	7.0%	6.7%	9.0%	5.8%	7.5%	6.6%	5.4%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Daptomycin	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Linezolid	1.5%	1.5%	0.6%	0.5%	0.4%	0.4%	0.4%	0.8%	0.9%	0.6%
Penicillin	97.7%	97.4%	96.6%	94.9%	96.0%	98.1%	93.3%	92.7%	93.2%	92.7%
Rifampin	3.2%	2.7%	2.5%	2.4%	1.5%	1.6%	1.5%	2.5%	2.0%	2.1%
Ciprofloxacin	3.3%	3.6%	3.1%	2.8%	2.2%	3.3%	3.1%	3.7%	2.7%	2.1%

Figure 62. Yearly resistance rates of *S. aureus* skin and soft tissue infections, DOH-ARSP, 2024

Figure 62 shows the multi-year analysis of *S. aureus* skin and soft tissue infections. Co-trimoxazole (p=0.000) resistance showed an increasing trend over the last ten years, while the resistance rate to oxacillin (p=0.0000) was noted to be decreasing. Multi-year analysis showed that the differences noted across years were significant for all antibiotics except for daptomycin (p=0.9324) and linezolid (p=0.6532)



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Co-trimoxazole	23.3%	24.4%	25.7%	33.0%	37.0%	37.7%	45.4%	44.1%	50.3%	46.3%
Vancomycin	0.5%	0.9%	1.7%	0.5%	0.9%	0.8%	1.6%	1.6%	2.3%	1.3%
Clindamycin	8.5%	9.0%	10.3%	9.1%	8.5%	8.9%	8.2%	10.2%	8.2%	8.0%

In 2024, one (1) linezolid-resistant methicillin-resistant *Staphylococcus aureus* (MRSA) isolate was identified and confirmed by the reference laboratory. This isolate was recovered from a presumptive healthcare-associated bloodstream infection in a 70-year-old patient at EVR. It exhibited resistance to clindamycin and erythromycin, yet remained susceptible to daptomycin and vancomycin. While linezolid resistance has been reported over the past ten years, with rates ranging from 0.4% to 1.5%, this 2024 isolate represents the first confirmed case by the reference laboratory within that period.

Despite vancomycin's role as a last-resort treatment for MRSA infections, recent evidence highlights increasing treatment failures and a rising global prevalence of vancomycin-resistant *Staphylococcus aureus* (VRSA). Locally, though no VRSA isolates have been confirmed by the national reference laboratory, its reported presence across the country, primarily from wound infections, is noted. The reference laboratory continuously monitors this organism of concern across all surveillance sites, emphasizing ongoing surveillance and good laboratory practices.

The oxacillin resistance rates of *S. aureus* infections across different regions in the country was shown in **Figure 63-A**. Surveillance sites in Visayas and Mindanao have MRSA rates from 40-60%, while surveillance sites in Luzon have MRSA rates from 11-30%. **Figure 63 - B** shows the geographic distribution of vancomycin-resistance among *S. aureus* infections across different regions. Resistance rates to most surveillance sites were less than 10%.

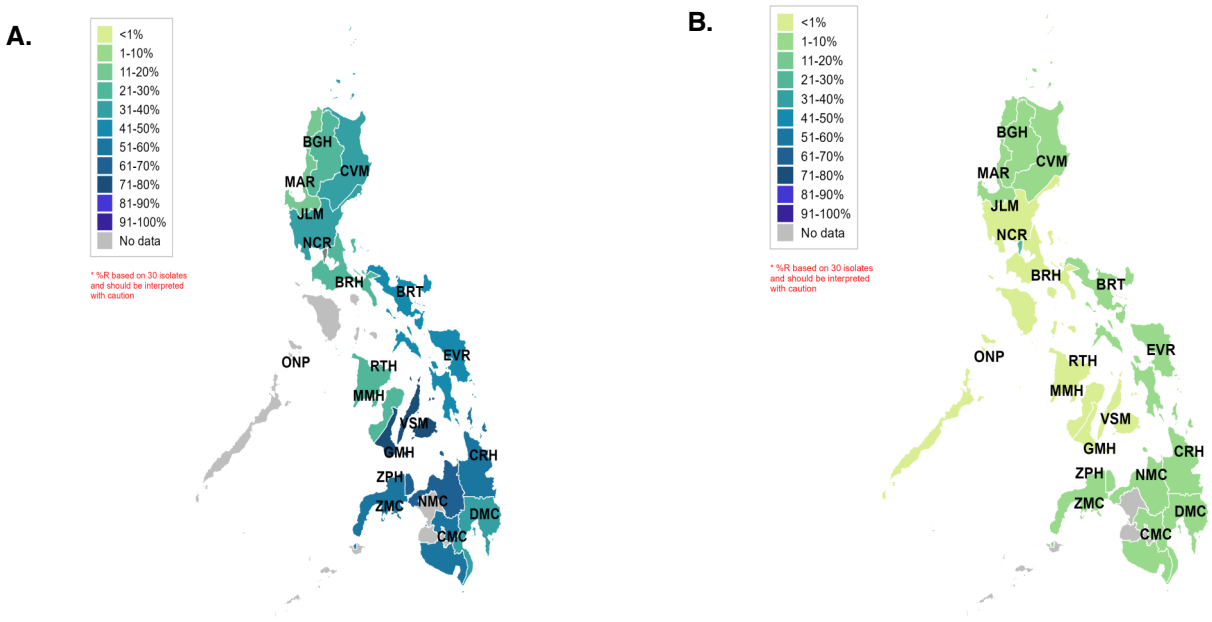
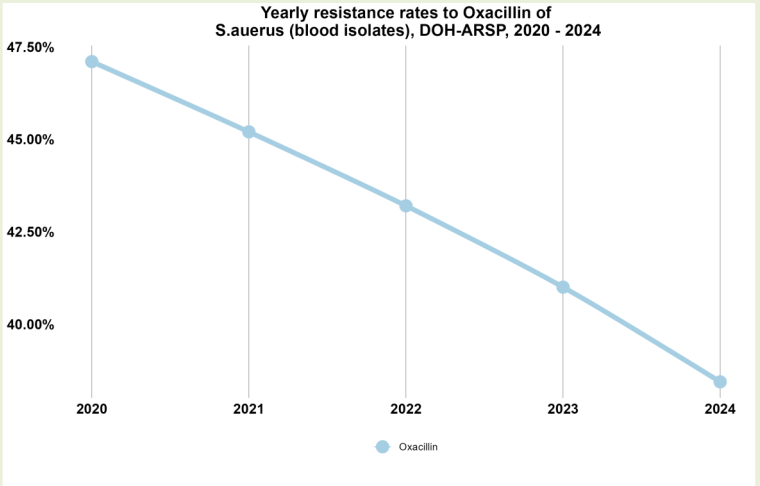


Figure 63. Geographic distribution of A) oxacillin-resistant and B) vancomycin- resistant *S. aureus* infections in the Philippines, DOH, ARSP, 2024.

Antimicrobial resistance (AMR) indicator 3.d of the Sustainable Development Goal (SDG) focuses on the proportion of patients with MRSA bloodstream infections (BSI). MRSA remains a significant global burden, recognized as a leading cause of both health-care-associated and community-acquired infections. Its persistent prevalence and potential to cause severe infections contribute to substantial morbidity, mortality, and health-care costs, making it a major ongoing concern.

Figure 64 shows a decreasing trend in MRSA resistance rates in blood isolates over the past decade, from 47.1% in 2020 to 38.4% in 2023. Using blood isolates as a proxy for MRSA BSI, ARSP data suggests the Philippines is making progress towards achieving this SDG target.

Proportion of Blood stream infections (BSIs) due to ***Staphylococcus aureus***



	2020	2021	2022	2023	2024
Oxacillin	47.1%	45.2%	43.2%	41.0%	38.4%

Figure 64 Yearly resistance rates of *S. aureus* bloodstream infections, DOH-ARSP, 2024

Methicillin-resistant *Staphylococcus aureus*

There were **3,006** methicillin-resistant *S. aureus* (MRSA) infections reported for 2024.

3,006 infections

This is a 22.83% increase from what was reported in 2023. The top contributing surveillance sites are PGH (12.97%), VSM (8.45%) and DMC (7.39%) (Figure 65). Luzon contributed the majority of infections (55.12%) followed by Visayas (17.03%) and Mindanao (27.84%).

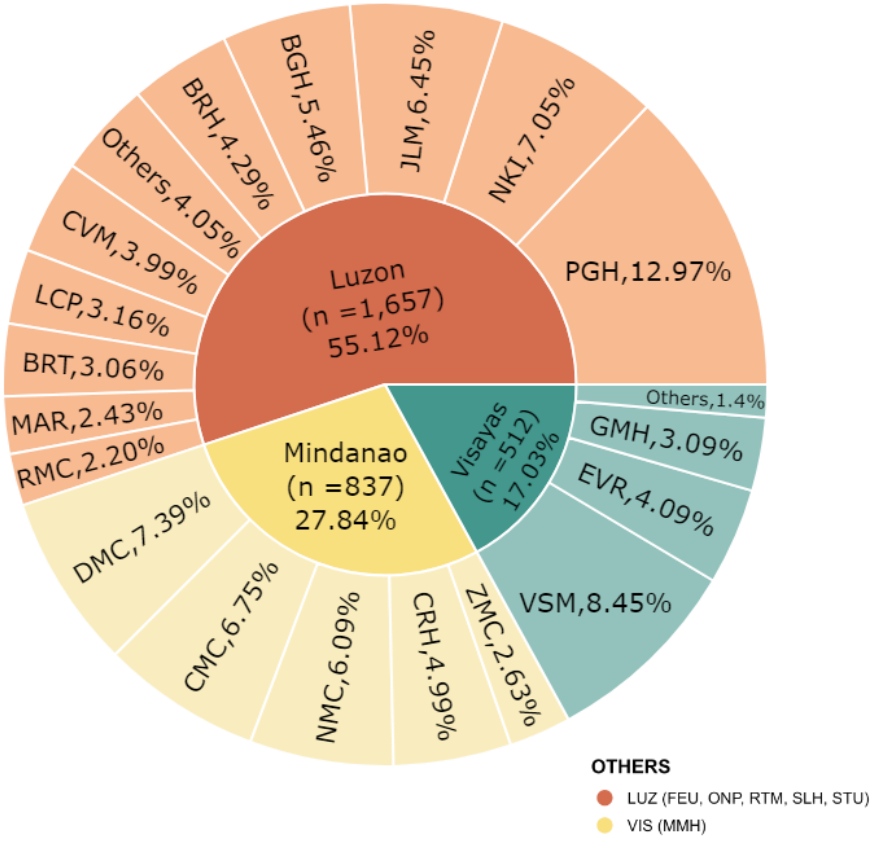


Figure 65. Distribution of MRSA infections, DOH-ARSP, 2024 (n=3,006)

Slightly over half of the infections were observed among male patients (54.56%), and most (64.04%) occurring in the 20-64 age group. Most are wound infections (36.06%).

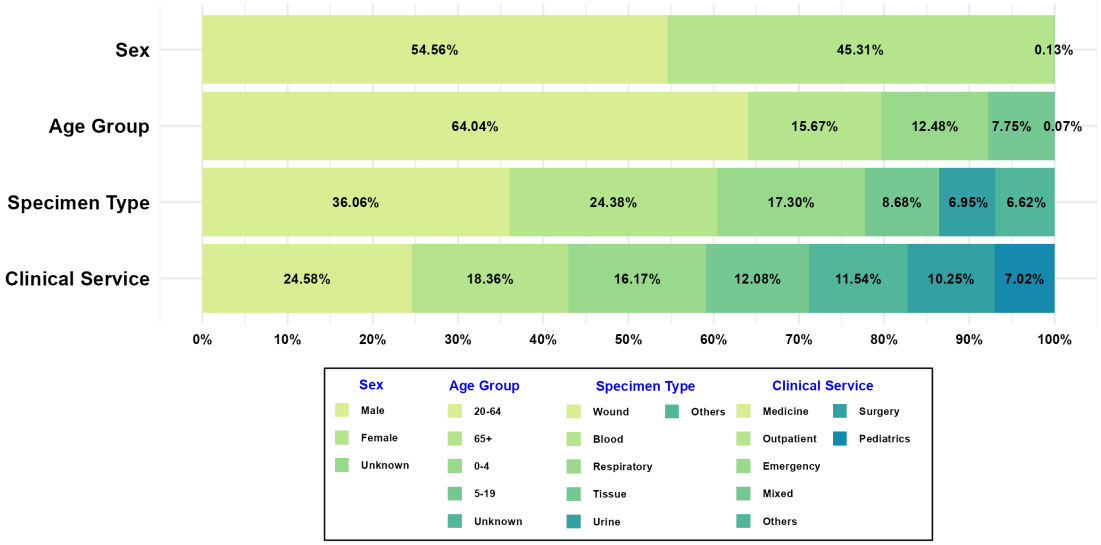


Figure 66. Patient characteristics in MRSA infections, DOH-ARSP, 2024 (n=3,006)

The percent positive of BSI caused by MRSA were less than 1% for most (n=19) surveillance sites. While the percent positive of bloodstream infections caused by MRSA for FEU, PGH, NKI and RTM were 4.35%, 2.36%, 1.96% and 1.22%, respectively (Figure 67).

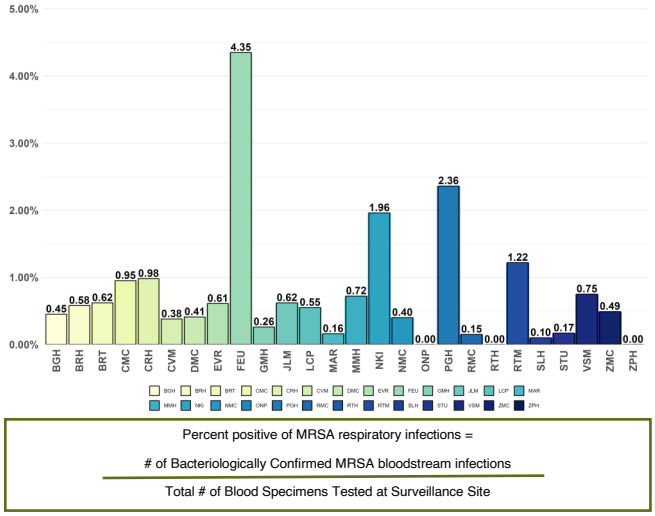


Figure 67. Percent positive of MRSA bloodstream infections among all tested blood specimens per surveillance site, DOH-ARSP, 2024

For most (n=14) surveillance sites, MRSA percent positivity in wound infections was less than 5%. (Figure 68). While the percent positive of bloodstream infections caused by MRSA for RTM, CRH, PGH, CMC, ZMC, FEU, NMC, CVM, STU and MAR were 12.69%, 9.42%, 9.19%, 8.74%, 7.84%, 7.41%, 6.33%, 5.80%, 5.69% and 5.23%, respectively.

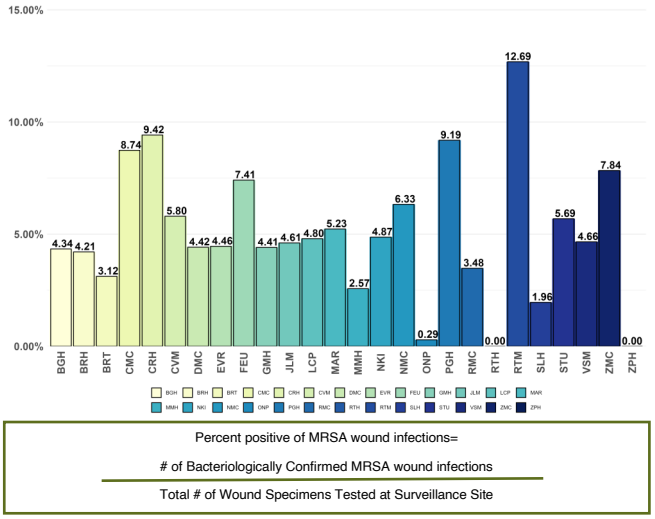


Figure 68. Percent positive of MRSA wound infections among all tested wound specimens per surveillance site, DOH-ARSP, 2024

All types of Infections

Figure 69 shows the resistance rates of all MRSA infections. Resistance to clindamycin was at 6.96% and erythromycin at 19.95%, while resistance to vancomycin was at 2.82% and linezolid at 1.30%. Resistance rates to tetracycline, rifampicin, ciprofloxacin and nitrofurantoin were all less than 10%. Resistance to co-trimoxazole was still notably high at 45.23%. It was noted that from 2023, the decrease in resistance rates for vancomycin and cotrimoxazole were found to be statistically significant (p=0.0051 and p=0.0000, respectively).

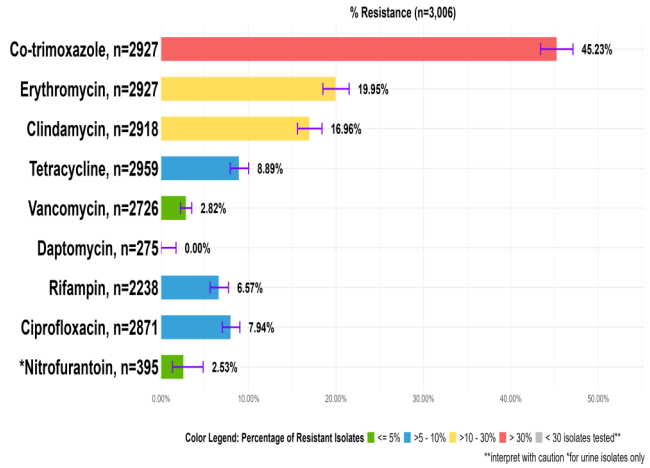
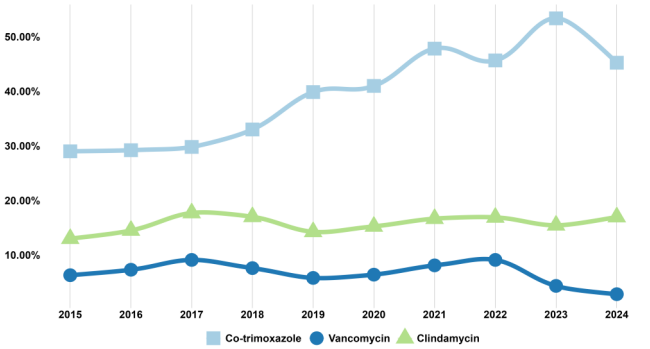
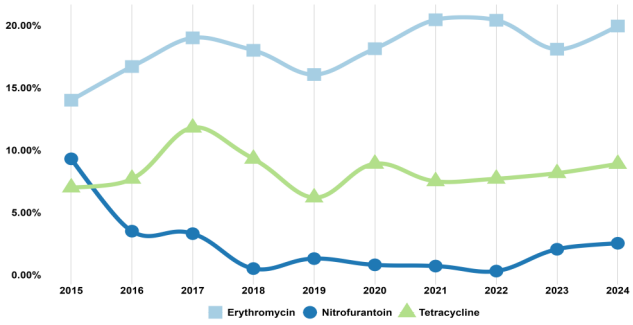


Figure 69. Proportion of all MRSA infections resistant to tested antibiotics, DOH-ARSP, 2024 (n=3,006)

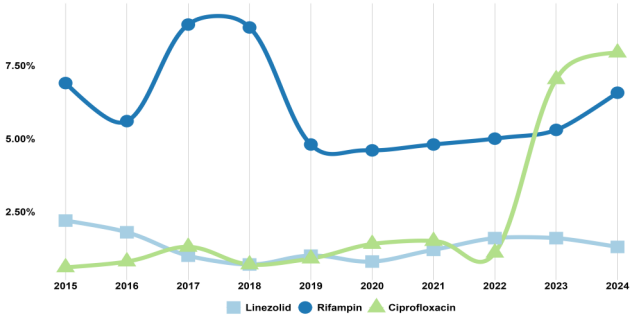
Figure 70 illustrates the multiple year analysis and shows the changes in antibiotic resistance rates in MRSA infections over the past decade. A decrease in the resistance rates of vancomycin and nitrofurantoin was observed. The reported vancomycin resistance was not confirmed in the reference laboratory. The resistance rate for ciprofloxacin drastically increased in 2023, a trend that continued into 2024. 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), rifampin (p=0.0002), and ciprofloxacin (p=0.0000).



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Co-trimoxazole	29.0%	29.2%	29.8%	33.0%	39.9%	41.0%	47.8%	45.6%	53.4%	45.2%
Vancomycin	6.3%	7.3%	9.1%	7.6%	5.8%	6.4%	8.1%	9.1%	4.3%	2.8%
Clindamycin	13.0%	14.5%	17.7%	17.0%	14.2%	15.2%	16.7%	16.9%	15.4%	17.0%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Erythromycin	14.0%	16.7%	19.0%	18.0%	16.1%	18.1%	20.4%	20.4%	18.1%	19.9%
Nitrofurantoin	9.3%	3.5%	3.3%	0.5%	1.3%	0.8%	0.7%	0.3%	2.0%	2.5%
Tetracycline	7.0%	7.7%	11.8%	9.3%	6.2%	8.9%	7.5%	7.7%	8.2%	8.9%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Linezolid	2.2%	1.8%	1.0%	0.7%	1.0%	0.8%	1.2%	1.6%	1.6%	1.3%
Rifampin	6.9%	5.6%	8.9%	8.8%	4.8%	4.6%	4.8%	5.0%	5.3%	6.6%
Ciprofloxacin	0.6%	0.8%	1.3%	0.7%	0.9%	1.4%	1.5%	1.1%	7.0%	7.9%

Figure 70. Yearly resistance rates of MRSA infection

Bloodstream Infections

Figure 71 illustrates that MRSA bloodstream infections exhibited slightly higher resistance rates to clindamycin, erythromycin, vancomycin, tetracycline, linezolid, and ciprofloxacin compared to the resistance rates observed in all specimens.

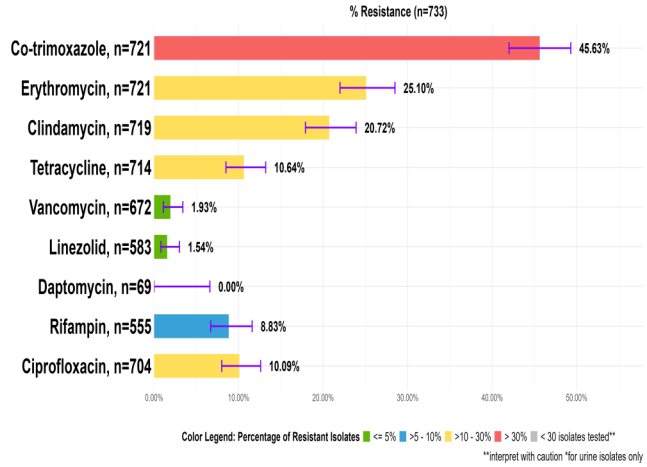


Figure 71. Proportion of MRSA bloodstream infections resistant to tested antibiotics, DOH-ARSP, 2024 (n=733)

Skin and Soft Tissue Infections

For MRSA skin and soft tissue infections (**Figure 72**), resistance to erythromycin was at 14.97%, clindamycin at 13.42% and tetracycline at 6.44%. Resistance rates to rifampin and ciprofloxacin were 4.58% and 4.52% respectively while resistance rates to vancomycin and linezolid were at 2.90% and 1.26% respectively. No daptomycin resistant MRSA skin and soft tissue isolate was detected in 2024. Compared with the resistance rates of all MRSA infections, resistance rates of MRSA skin and soft tissues infections are slightly lower for clindamycin, erythromycin, tetracycline, rifampin and ciprofloxacin.

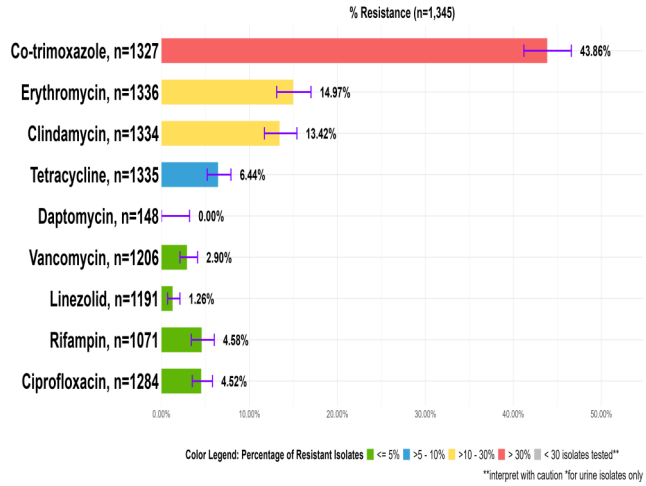


Figure 72. Proportion of MRSA skin and soft tissue infections with resistance to tested antibiotics, DOH-ARSP, 2024 (n=1,345)

Methicillin-Susceptible *Staphylococcus aureus*

There were **5,393** methicillin-susceptible *S. aureus* (MSSA) infections reported in 2024.

5,393 infections

This is a 22.83% increase from what was reported in 2023. The highest contributors were PGH (13.35%), BGH (10.42%) and DMC (7.9%) (Figure 73). Based on island group distribution, the highest was Luzon with 66.42%, followed by Mindanao at 17.75%, and Visayas at 15.84%.

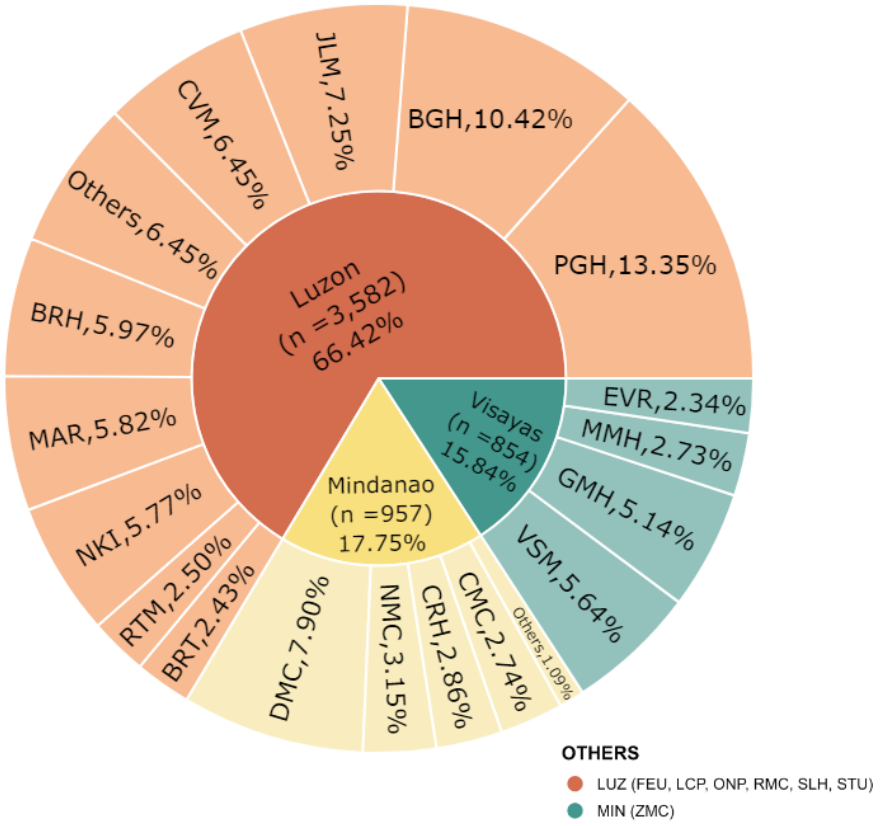


Figure 73. Distribution of MSSA infections, DOH-ARSP, 2024 (n=5,393)

A little more than half (55.42%) of infections were from male patients and the majority (67.03%) were from the 20-64 age group. The majority of the infections were from wound specimens (41.85%).

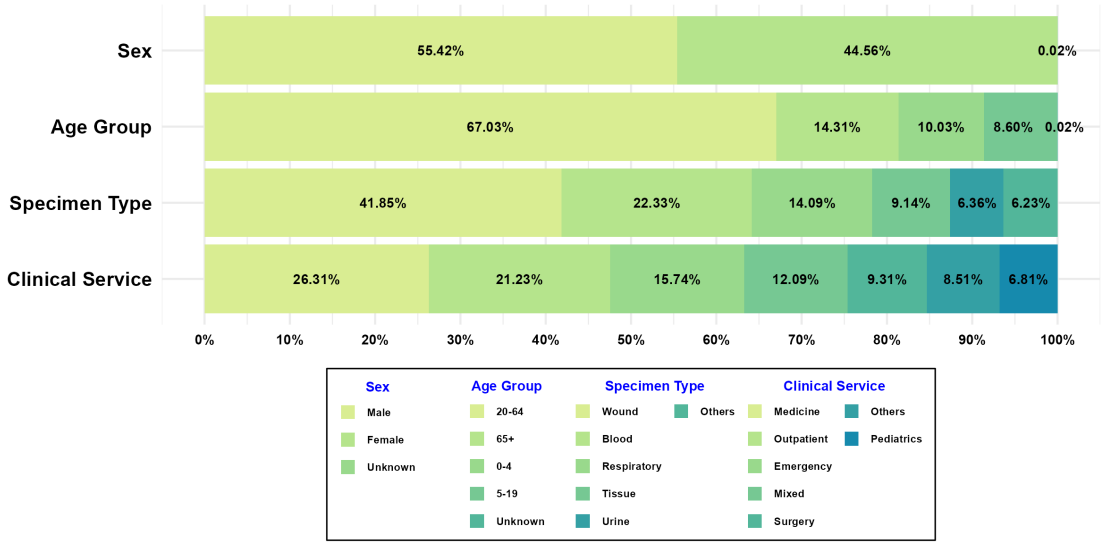


Figure 74. Patient characteristics in in MSSA infections, DOH-ARSP, 2024 (n=5,393)

Figure 75 shows that the percent positive of BSI caused by MSSA for FEU, PGH, RTM, NKI and MMH were 5.80%, 4.75%, 3.05%, 2.99% and 1.86% respectively. While the percent positive of BSI due to MSSA for most (n=18) of the surveillance sites were less than 1.5%.

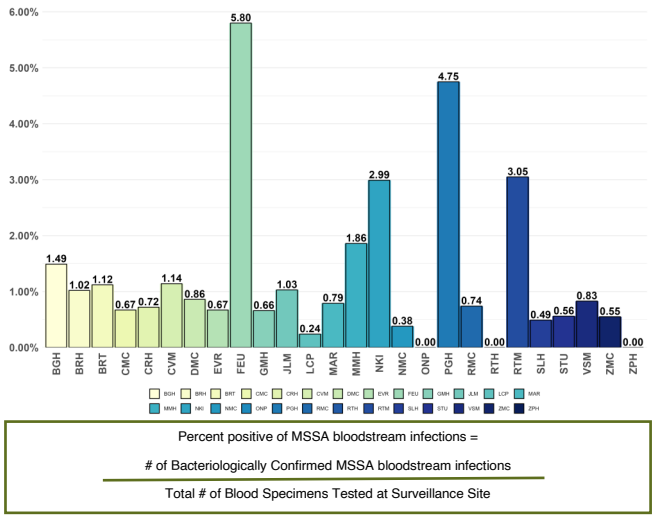


Figure 75. Percent positive of MSSA bloodstream infections among all tested blood specimens per surveillance site, DOH-ARSP, 2024

Figure 76 shows that the percent positive of wound infections caused by MSSA for RTM, FEU and MAR were 44.23%, 37.04% and 21.05%, respectively. While the percent positive of wound infections due to MSSA for BRH, DMC, MMH, JLM, STU, CRH, SLH, CVM, BGH, GMH, and PGH were within 10-20%. While the remaining 9 surveillance sites showed percent positive from 3.60-7.20%.

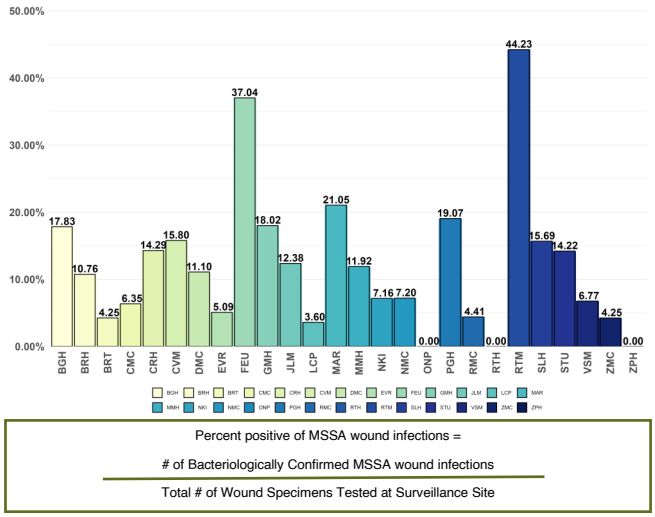


Figure 76. Percent positive of MSSA wound infections among all tested wound specimens per surveillance site, DOH-ARSP, 2024

All types of Infections

Resistance to tested antibiotics among all types of MSSA infections are shown in Figure 77. Likewise, resistance to MSSA SSIs and BSIs are shown in Figures 78 and 79, respectively. Resistance to first line antibiotics other than oxacillin such as erythromycin, clindamycin and tetracycline ranged from 5-6%.

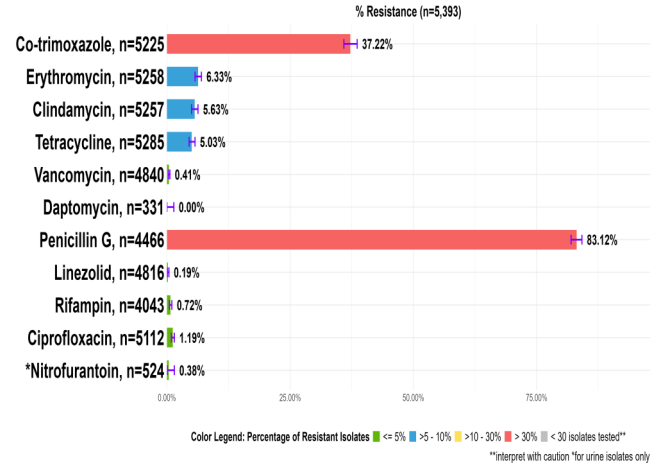


Figure 77. Proportion of all MSSA infections resistant to tested antibiotics, DOH-ARSP, 2024 (n=5,393)

Bloodstream Infections

Figure 78 shows the proportion of MSSA bloodstream infections. Resistance to penicillin, co-trimoxazole, erythromycin and clindamycin were 79.53%, 27.28%, 7.81% and 6.95%, respectively.

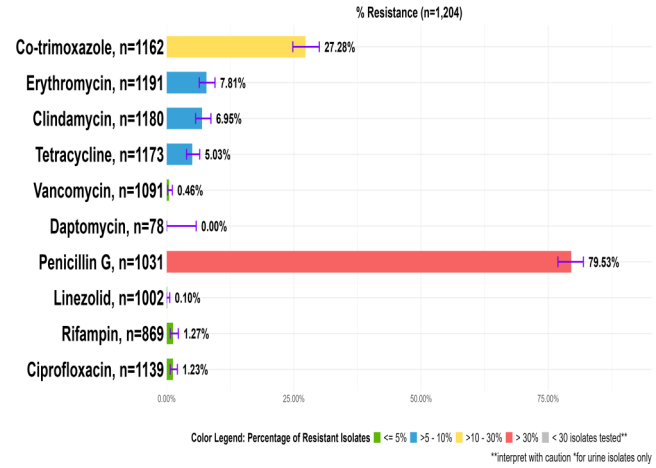


Figure 78. Proportion of MSSA bloodstream infections with resistance to tested antibiotics, DOH-ARSP, 2024 (n=1,204)

Skin and Soft Tissue Infections

Figure 79 shows the proportion of MSSA skin and soft tissue infections. Resistance to penicillin, co-trimoxazole, erythromycin and clindamycin were 88.73%, 47.97%, 5.46% and 4.94%, respectively.

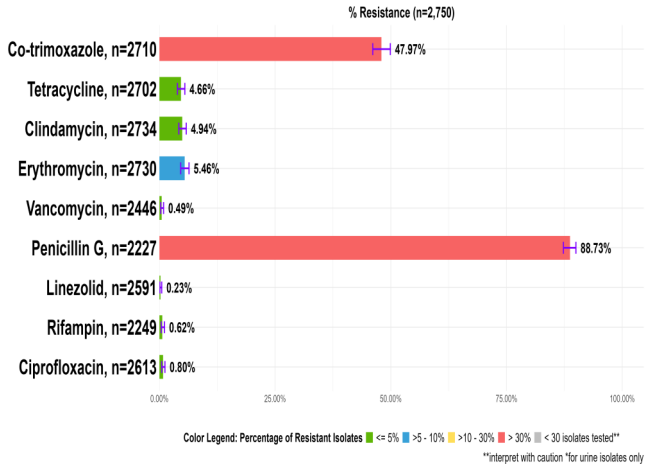


Figure 79. Proportion of MSSA skin and soft tissue infections with resistance to tested antibiotics, DOH-ARSP, 2024 (n=2,750)

Introduction:

The *Staphylococcus aureus* remains a leading cause of both healthcare-associated and community-acquired infections worldwide. In the Philippines, oxacillin resistance rates have steadily declined over the past five years, decreasing from 52% in 2019 to 40% in 2023 [1]. This genomic survey report provides critical insights into the evolution, epidemiology, and dissemination of methicillin-resistant *Staphylococcus aureus* (MRSA) clones, highlighting their significant public health implications.

Results and Discussion

Demographics and characteristics of *Staphylococcus aureus* isolates

Only 41 *S. aureus* isolates sent to Antimicrobial Resistance Surveillance Reference Laboratory (ARSRL) were included in the analysis. Of the 41 the *S. aureus* submitted for whole genome sequencing (WGS), 35 isolates were confirmed as MRSA *in silico*, while six isolates were methicillin-susceptible *S. aureus*.

The age range of the patients was < 1 to 79 years; 9.76% (n = 4) of the isolates were from patients aged < 1 year. Of the 41 isolates, 63.41% were recovered from male patients (n = 26) and 36.59% from females (n = 15). The most common specimen sources were blood and wound, each representing 12 isolates (29.27%), followed by tracheal aspirate (12.20%, n = 5). Most of the infections (78.05%, n =32) were classified as community-associated *S. aureus* (CA-MRSA) infection.

Resistance Profile

Isolates were tested for susceptibility to 11 antibiotics from nine classes. All the isolates were susceptible to vancomycin, linezolid, and daptomycin. All MRSA resistant to penicillin, oxacillin and cefoxitin, consistent with the presence of *blaZ* and *mecA* (Table 10). Ten isolates were resistant to sulfamethoxazole/trimethoprim and carried the *dfrG* gene. Comparisons between phenotypic and genotypic data are presented for seven key antibiotics from six classes (Table 10).

Table 10. Oxacillin resistance genes of MRSA isolates by surveillance sites, ARSP 2024

Oxacillin and other resistance genes			
Surveillance Site	blaZ (n, %)	*mecA (n, %)*	other (n, %)
BGH	4, 57%	7, 100%	0, 0%
BRH	3, 50%	4, 100%	0, 0%
BRT	3, 60%	5, 100%	0, 0%
CMC	0, 0%	0, 0%	0, 0%
CRH	0, 0%	0, 0%	0, 0%
CVM	3, 100%	3, 100%	0, 0%
DMC	3, 75%	4, 100%	0, 0%
EVR	0, 0%	2, 100%	0, 0%
FEU	0, 0%	0, 0%	0, 0%
GMH	0, 0%	0, 0%	0, 0%
JLM	0, 0%	0, 0%	0, 0%
LCP	0, 0%	0, 0%	0, 0%

Table 11. Number of resistant isolates to select antibiotics tested

Surveillance Site	Vancomycin %R	Linezolid %R	Daptomycin %R
BGH	0, 0%	0, 0%	0, 0%
BRH	0, 0%	0, 0%	0, 0%
BRT	0, 0%	0, 0%	0, 0%
CMC	0, 0%	0, 0%	0, 0%
CRH	0, 0%	0, 0%	0, 0%
CVM	0, 0%	0, 0%	0, 0%
DMC	0, 0%	0, 0%	0, 0%
EVR	0, 0%	0, 0%	0, 0%
FEU	0, 0%	0, 0%	0, 0%
GMH	0, 0%	0, 0%	0, 0%
JLM	0, 0%	0, 0%	0, 0%
LCP	0, 0%	0, 0%	0, 0%
MAR	0, 0%	0, 0%	0, 0%

Oxacillin and other resistance Genes			
Surveillance Site	blaZ (n, %)	*mecA (n, %)*	other (n, %)
MAR	4, 67%	5, 100%	0, 0%
MMH	0, 0%	1, 100%	0, 0%
NKI	0, 0%	0, 0%	0, 0%
NMC	0, 0%	0, 0%	0, 0%
ONP	0, 0%	0, 0%	0, 0%
PGH	0, 0%	0, 0%	0, 0%
RMC	0, 0%	0, 0%	0, 0%
RTH	0, 0%	0, 0%	0, 0%
RTM	0, 0%	0, 0%	0, 0%
SLH	1, 50%	2, 100%	0, 0%
STU	2, 100%	2, 100%	0, 0%
VSM	0, 0%	0, 0%	0, 0%
ZMC	0, 0%	0, 0%	0, 0%
ZPH	0, 0%	0, 0%	0, 0%

Surveillance Site	Vancomycin %R	Linezolid %R	Daptomycin %R
MAR	0, 0%	0, 0%	0, 0%
MMH	0, 0%	0, 0%	0, 0%
NKI	0, 0%	0, 0%	0, 0%
NMC	0, 0%	0, 0%	0, 0%
ONP	0, 0%	0, 0%	0, 0%
PGH	0, 0%	0, 0%	0, 0%
RMC	0, 0%	0, 0%	0, 0%
RTH	0, 0%	0, 0%	0, 0%
RTM	0, 0%	0, 0%	0, 0%
SLH	0, 0%	0, 0%	0, 0%
STU	0, 0%	0, 0%	0, 0%
VSM	0, 0%	0, 0%	0, 0%
ZMC	0, 0%	0, 0%	0, 0%
ZPH	0, 0%	0, 0%	0, 0%

Genotypic Findings

The MLST, X region of the protein A (spa) gene type, Staphylococcal Cassette Chromosome mec (*SCCmec*) type, and virulence genes were predicted *in silico* from the WGS data for the 41 *S. aureus* isolates. A total of 20 different multilocus sequence types (STs) were identified. The most prevalent were ST30 and ST97, each accounting for 7 isolates (17.1%), followed by ST6, which was detected in 5 isolates (12.2%). Most of the isolates belonged to clonal complexes CC97, CC30, and CC5, each comprising 7 isolates (17.07%). The most prevalent spa types were t019 and t239, each identified in 5 isolates (12.2%). All of the MRSA isolates were typed as SCC*mec*IV.

The MRSA population were dominated by CC97-ST97-SCC*mec*-typeIV (n=7, 17.07%) detected among CA-MRSA isolates recovered in the provinces of Ilocos, Batangas, Bicol and Eastern Visayas. All of these isolates possessed virulence genes: hld, *hlg*ABC, *ica*C, *luk*DE, *sak-scn-sel*X-sph and *spl*ABE. The clonal complex 97 (CC97) has been previously linked to livestock-associated MRSA (LA-MRSA) infection[3]. This finding underscores the zoonotic potential of these strains and their ability to affect both human and animal populations.

The CC30-*spa*-t019-SCC*mec*-typeIV-PVL+ (Southwest Pacific MRSA clone, n=5, 12.20%) was isolated across the country. Furthermore, two isolates only exhibited resistance to sulfamethoxazole/trimethoprim, and carried the *dfrG* resistance genes. Previous study in the Philippines linked CC30 genomes to Southwest Pacific clones with evidence of global dissemination [2].

Interestingly, the ST8-*spa*-t008-SCC*mec*-IV-PVL+ clone (n = 2), detected in Ilocos and Davao, were related to the pandemic USA300 lineage, a major cause of skin and soft tissue infections (SSTIs) and invasive diseases globally [4]. Additionally, previous study in the Philippines identified the multidrug-resistant ST239-*spa*-t030-SCC*mec*-typeIII strain as a prevalent healthcare-associated MRSA (HA-MRSA) clone. Our findings revealed the emergence of an ST239-SCC*mec*-typeIV isolate (n = 1), which exhibited resistance only to penicillin and oxacillin, harbored the blaZ and mecA resistance genes. The detection of SCC*mec* type IV in ST239 indicates a genetic shift from the classical type III, commonly linked to HA-MRSA. Unlike the larger SCC*mec* type III, the type IV is smaller and more mobile, facilitating horizontal gene transfer and potentially enabling the clone to spread more efficiently across both healthcare and community settings.

Table 12. Comparison of genomic predictions of antibiotic resistance with phenotypic antimicrobial susceptibility test.

Antibiotic Class	Antibiotic	Resistant Isolates	False Negative	False Positive	Concordance %	Resistance genes
Penicillin	Penicillin	37	0	0	100	blaZ, mecA
Penicillin	Oxacillin	35	0	0	100	mecA
Folate pathway antagonist	Sulfamethoxazole/ Trimethoprim	10	0	1	90%	dfrG
Macrolide	Erythromycin	5	1	0	80%	*erm*A , *erm*B, *erm*C, *erm*T, *msr*A, *mph*C
Lincosamide	Clindamycin	5	2	0	60%	ermA , ermB, ermC, ermT, lnuA, lnuB, lsaE
Tetracycline	Tetracycline	1	0	0	100%	*tet*L ,*tet*K , *tet*M
Fluoroquinolone	Ciprofloxaxin	1	0	0	100%	*Gyr*A_S84L, *Gyr*A_G106D *parC*_S80F *parC*_E84F

Conclusion

This highlights the evolving nature of MRSA, with the potential for these strains to spread beyond healthcare settings into the community. Expanding WGS-based surveillance allows us determine the national population structure of MRSA and identify emergence of high-risk clones for development of stringent infection prevention and control measures to prevent cross-species transmission.

References:

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Enterococcus faecalis

A total of **3,714** *E. faecalis* infections were reported for 2024

3,714 infections

The highest reported cases are in PGH (16.99%), DMC (10.53%), and NKI (10.23%) (**Figure 80**). Based on island group distribution, the highest was Luzon with 61.07%. followed by Mindanao at 23.13%, and Visayas at 15.81%.

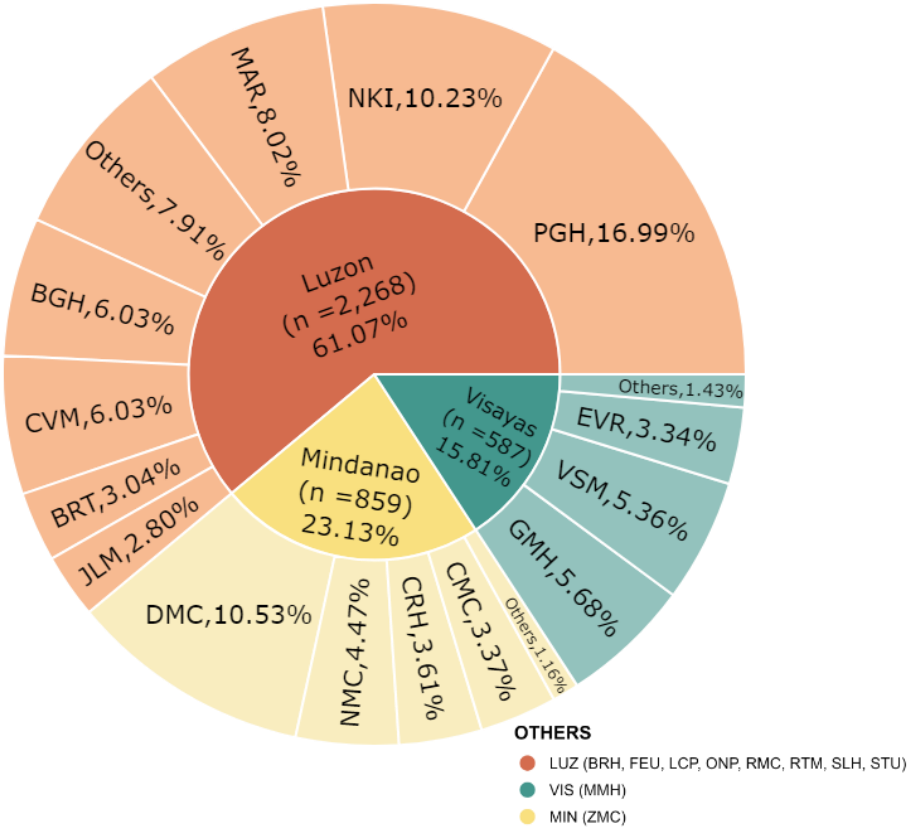


Figure 80. Distribution of *E. faecalis* infections, DOH-ARSP, 2024 (n=3,714)

More than half (55.09%) of the infections were from male patients and from the 20-64 age group (65.62%). Urinary infections are the most common type of *E. faecalis* infections, accounting for 50.24%.

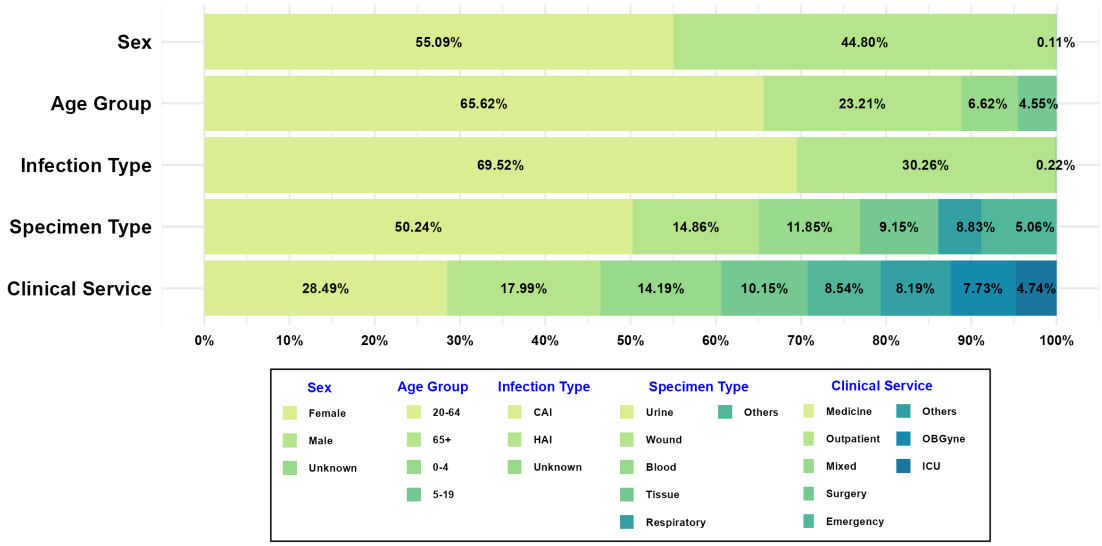
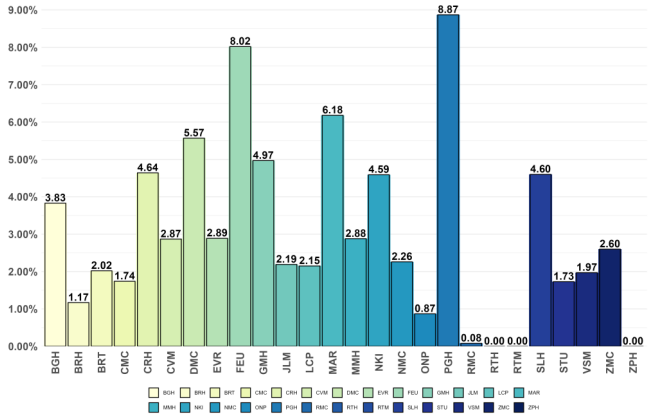


Figure 81. Patient characteristics in in *E. faecalis* infections, DOH-ARSP, 2024 (n=3,714)

The percent positive of urinary tract infections (UTIs) caused by *E. faecalis* in RTM, FEU, MAR, DMC, GMH, CRH, SLH, and NKI were above 4% (Figure 82). While the percent positive of urinary tract infections for most of the surveillance sites ranges from 0.08-3.83%.



Percent positive of *E. faecalis* urinary tract infections =

# of Bacteriologically Confirmed *E. faecalis* urinary tract infections

Total # of Urine Specimens Tested at Surveillance Site

Figure 82. Percent positive of *E. faecalis* urinary tract infections among all tested urine specimens per surveillance site, DOH-ARSP, 2024

All types of Infections

The resistance rates of all *E faecalis* infections to penicillin was at 13.08% and 8.40% to ampicillin. Resistance to vancomycin and linezolid were at 2.76% and 2.65%. All infections were susceptible to daptomycin. Resistance against high-level gentamicin and high-level streptomycin were at 17.17% and 16.58%, respectively. From 2023, there was a decrease in resistance rates of penicillin and vancomycin, while an increase was observed in ampicillin. These changes were found to be statistically significant.

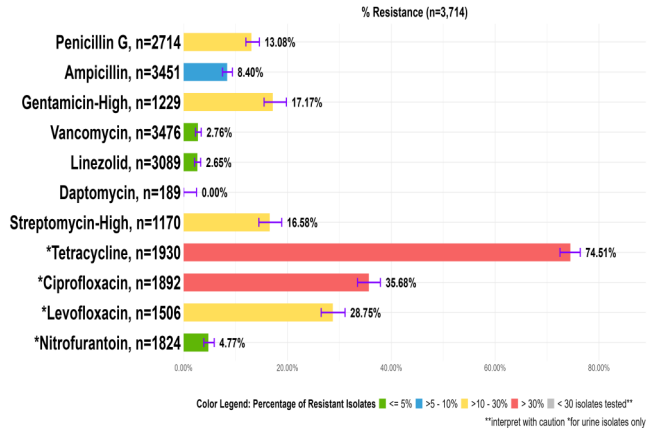
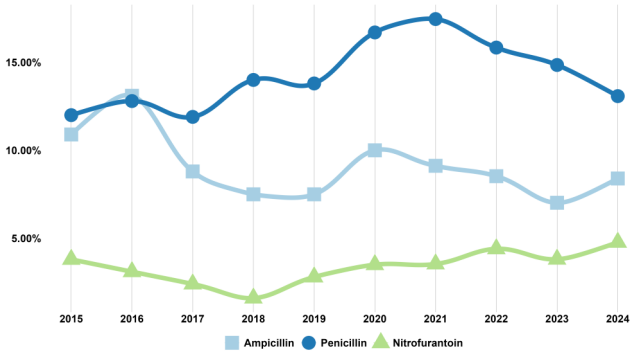
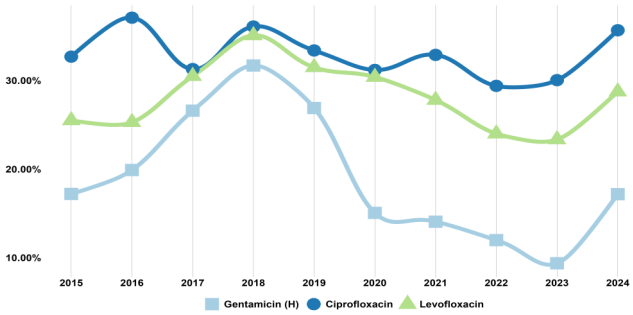


Figure 83. Proportion of all *E. faecalis* infections resistant to tested antibiotics, DOH-ARSP, 2024 (n=3,714)

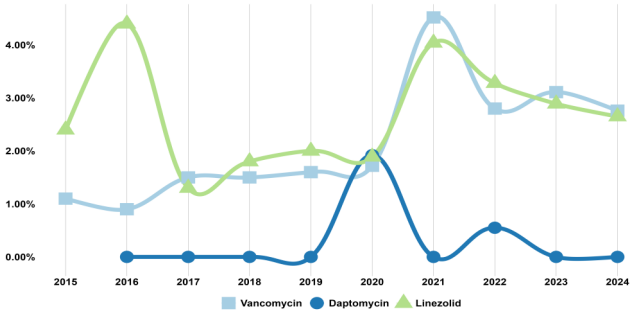
Figure 84 shows the trend in resistance rates of all *E. faecalis* infections in the last ten years. No drastic fluctuations in resistance rates were observed, except for high level gentamicin and high level streptomycin, which are statistically significant. The changes in resistance rates over the past 10 years were statistically significant for ampicillin (p=0.0273), penicillin (p=0.0237), high-level gentamicin (p=0.0000), vancomycin, and high-level streptomycin.



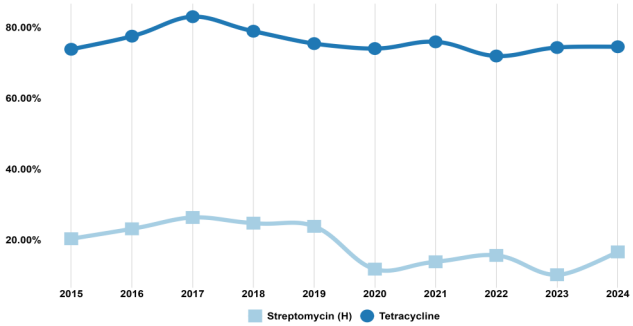
	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Ampicillin	10.9%	13.1%	8.8%	7.5%	7.5%	10.0%	9.1%	8.5%	7.0%	8.4%
Penicillin	12.0%	12.8%	11.9%	14.0%	13.8%	16.7%	17.5%	15.8%	14.8%	13.1%
Nitrofurantoin	3.8%	3.1%	2.4%	1.6%	2.8%	3.5%	3.5%	4.4%	3.8%	4.8%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Gentamicin (H)	17.2%	19.9%	26.6%	31.7%	26.9%	15.1%	14.1%	12.0%	9.4%	17.2%
Ciprofloxacin	32.7%	37.1%	31.3%	36.1%	33.4%	31.2%	32.9%	29.4%	30.1%	35.7%
Levofloxacin	25.5%	25.3%	30.5%	35.1%	31.5%	30.4%	27.8%	24.0%	23.4%	28.8%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Vancomycin	1.1%	0.9%	1.5%	1.5%	1.6%	1.7%	4.5%	2.8%	3.1%	2.8%
Daptomycin		0.0%	0.0%	0.0%	0.0%	1.9%	0.0%	0.6%	0.0%	0.0%
Linezolid	2.4%	4.4%	1.3%	1.8%	2.0%	1.9%	4.0%	3.3%	2.9%	2.6%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Streptomycin (H)	20.3%	23.1%	26.3%	24.7%	23.8%	11.7%	13.8%	15.6%	10.1%	16.6%
Tetracycline	73.8%	77.5%	83.0%	78.9%	75.4%	74.0%	75.9%	71.9%	74.3%	74.5%

Figure 84. Yearly resistance rates of all *E. faecalis* infections

Bloodstream Infections

Figure 85 shows the resistance rates of *E. faecalis* bloodstream infections. Resistance to penicillin was at 16.15%, ampicillin at 10.73%, vancomycin at 4.09% and linezolid at 2.15%. Resistance against high-level gentamicin was at 26.24% and high-level streptomycin at 22.07%. Compared with resistance rates of *E. faecalis* from all samples, resistance of *E. faecalis* from blood isolates were generally higher.

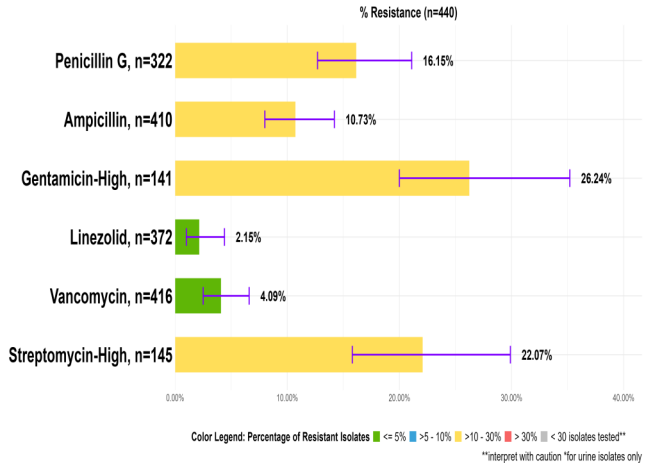
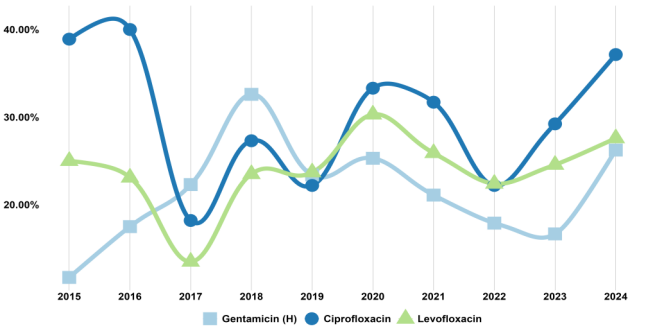
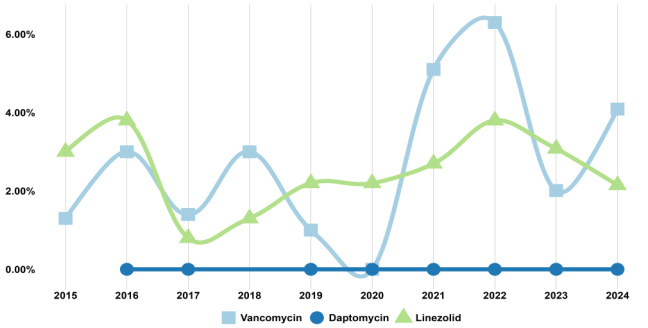


Figure 85. Proportion of *E. faecalis* bloodstream infections with resistance to tested antibiotics, DOH-ARSP, 2024 (n=440)

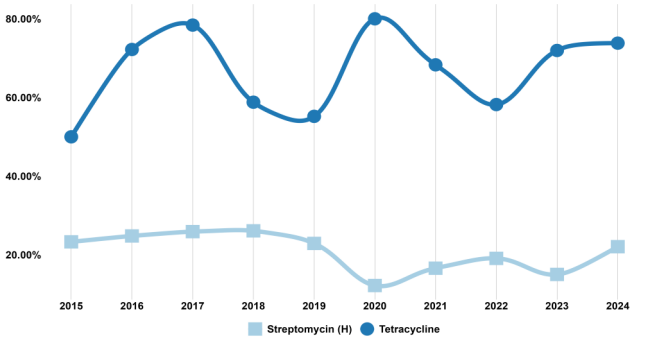
	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Ampicillin	13.9%	15.8%	5.1%	7.7%	8.5%	8.3%	11.0%	7.3%	7.6%	10.7%
Penicillin G	18.9%	15.2%	6.9%	10.1%	12.7%	16.4%	20.4%	14.9%	17.9%	16.1%
Nitrofurantoin		0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	8.3%	6.1%	4.8%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Gentamicin (H)	11.7%	17.5%	22.3%	32.6%	23.4%	25.3%	21.1%	17.9%	16.7%	26.2%
Ciprofloxacin	38.9%	40.0%	18.2%	27.3%	22.2%	33.3%	31.7%	22.2%	29.2%	37.1%
Levofloxacin	25.0%	23.1%	13.5%	23.5%	23.7%	30.3%	25.9%	22.4%	24.6%	27.6%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Vancomycin	1.3%	3.0%	1.4%	3.0%	1.0%	0.0%	5.1%	6.3%	2.0%	4.1%
Daptomycin		0.0%	0.0%		0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Linezolid	3.0%	3.8%	0.8%	1.3%	2.2%	2.2%	2.7%	3.8%	3.1%	2.1%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Streptomycin (H)	23.3%	24.8%	25.9%	26.1%	22.9%	12.2%	16.6%	19.1%	15.0%	22.1%
Tetracycline	50.0%	72.2%	78.4%	58.8%	55.2%	80.0%	68.3%	58.2%	72.0%	73.8%

Figure 86. Yearly resistance rates of *E. faecalis* bloodstream infections, ARSP-DOH, 2024

Urinary Tract Infections

Figure 87 shows that *E. faecalis* urinary tract infections showed resistance to penicillin G at 14.62% and ampicillin at 8.03%. Resistance rates for the following treatment options remained under 5%: nitrofurantoin, vancomycin, daptomycin, and linezolid.

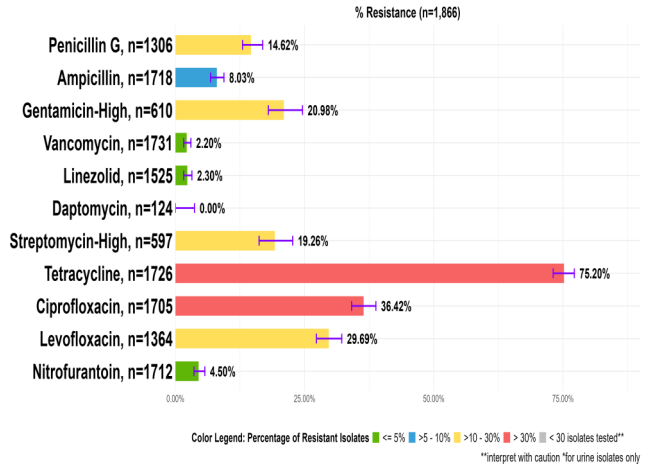
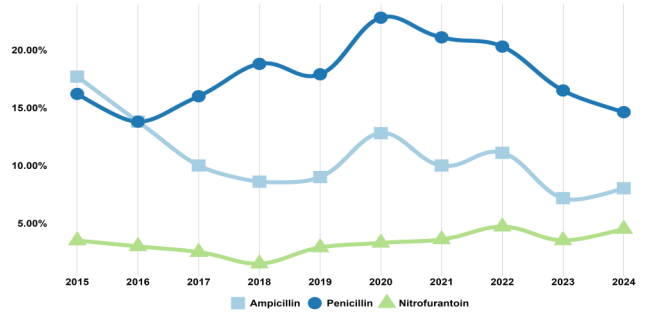
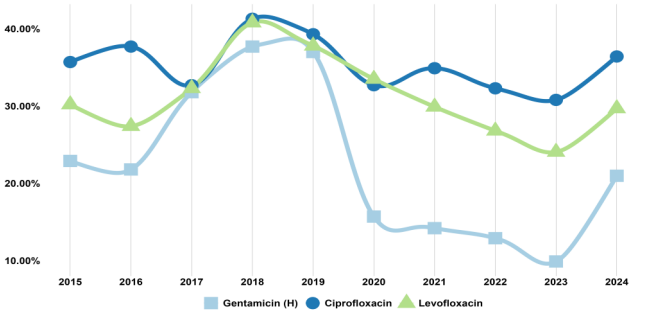


Figure 87. Proportion of *E. faecalis* urinary tract infections with resistance to tested antibiotics, DOH-ARSP, 2024 (n=1,866)

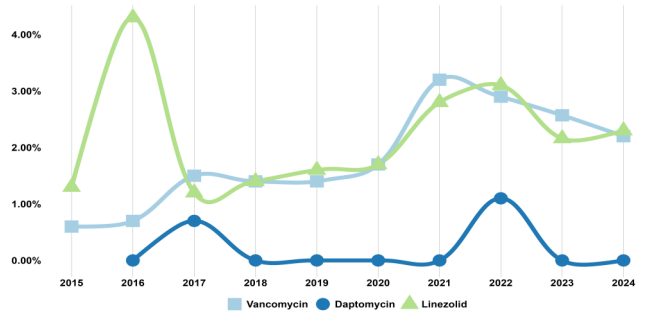
Yearly resistance rates of *E. faecalis* urinary tract infections were relatively steady in the past 10 years. Similar trend changes were observed for muti-year trends for bloodstream infections. Daptomycin resistance remained less than less than 2% in the past 10 years.



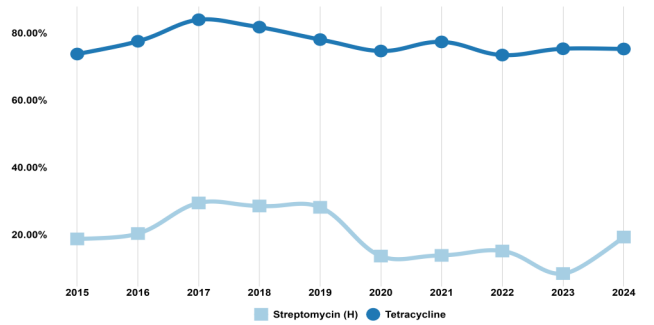
	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Ampicillin	17.7%	13.8%	10.0%	8.6%	9.0%	12.8%	10.0%	11.1%	7.2%	8.0%
Penicillin	16.2%	13.8%	16.0%	18.8%	17.9%	22.8%	21.1%	20.3%	16.5%	14.6%
Nitrofurantoin	3.5%	3.0%	2.5%	1.5%	2.9%	3.3%	3.6%	4.7%	3.5%	4.5%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Gentamicin (H)	22.9%	21.8%	31.8%	37.7%	37.0%	15.7%	14.2%	12.9%	9.9%	21.0%
Ciprofloxacin	35.7%	37.7%	32.7%	41.3%	39.3%	32.7%	34.9%	32.3%	30.8%	36.4%
Levofloxacin	30.2%	27.4%	32.3%	40.8%	37.8%	33.5%	29.9%	26.8%	24.1%	29.7%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Vancomycin	0.6%	0.7%	1.5%	1.4%	1.4%	1.7%	3.2%	2.9%	2.6%	2.2%
Daptomycin		0.0%	0.7%	0.0%	0.0%	0.0%	0.0%	1.1%	0.0%	0.0%
Linezolid	1.3%	4.3%	1.2%	1.4%	1.6%	1.7%	2.8%	3.1%	2.2%	2.3%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Streptomycin (H)	18.7%	20.3%	29.4%	28.5%	28.1%	13.6%	13.8%	15.1%	8.4%	19.3%
Tetracycline	73.7%	77.5%	83.9%	81.7%	78.0%	74.6%	77.3%	73.4%	75.3%	75.2%

Figure 88. Yearly resistance rates of *E. faecalis* urinary tract infection, DOH-ARSP, 2024

Five (5) confirmed cases of vancomycin-resistant *Enterococcus faecalis* were identified from clinical specimens. These included two (2) blood isolates from a 35-year-old and a 24-year-old patient, two (2) wound isolates from a 6-month-old patient, and one (1) urine isolate from a 62-year-old female. Two specimens originated from Luzon (JLM and MAR), while the remaining three were from the Visayas region. Four out of five infections were presumptively healthcare-associated.

There were confirmed linezolid-resistant isolates reported in 2024. The molecular characterization of these emerging resistant isolates are described in succeeding sections.

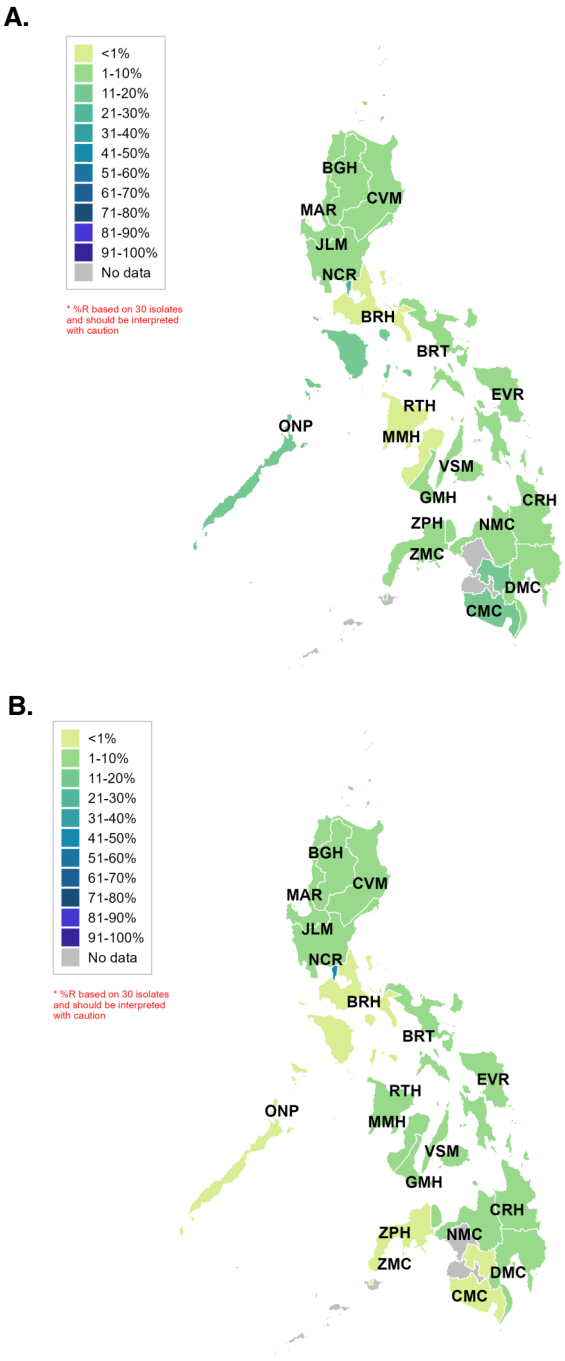


Figure 89. Geographic distribution of A) vancomycin-resistant and B) linezolid-resistant *E. faecalis* infections in the Philippines, DOH, ARSP, 2024.

Introduction:

Enterococcus faecalis, a multidrug-resistant opportunistic pathogen, is a major cause of hospital-acquired infections. Linezolid-resistant *E. faecalis* is an emerging global health threat, with resistance rates in the Philippines rising from 1.9% in 2020 to 4% in 2021, then decreasing to 2.89% in 2023[1]. This report provides the genomic features of linezolid-resistant *E. faecalis* isolates in the Philippines in 2024.

Results and Discussion

Demographics and characteristics of *Enterococcus faecalis* isolates

A total of 17 linezolid-resistant *Enterococcus faecalis* (LRE) isolates collected through surveillance sites of ARSP in 2024 were identified from patients with varying infection types and specimen sources. Isolates were derived from patients with healthcare-associated infections (HAI), 41.18% (n=7), and community-acquired infections (CAI) (58.82%, n=10). Urine was the most common specimen source (41.18%, n=7), followed by wound (23.53%, n=4), tissue (17.65%, n=3), blood (11.76%, n=2), and aspirate (5.88%, n=1) samples. Patient ages ranged from 6 months to 78 years, and were distributed between males (52.9%, n=9) and females (47.06%, n=8).

Molecular characterization of Linezolid-resistant *Enterococcus faecalis* (2024)

All seventeen (17) *E. faecalis* isolates were susceptible to vancomycin but resistant to linezolid, with minimum inhibitory concentrations (MICs) ranging from 8–64 μg/mL. Whole-genome sequencing identified sequence type (ST)16 as the most prevalent (n=9, 52.9%), previously reported in both humans and livestock. Other Sequence Types (STs) detected included ST403, ST368, ST36, ST179, ST506, and ST780.

The detection of *optr*A in ST16 and *cfr(D)* in ST780 from confirmed linezolid-resistant *E. faecalis* isolates in 2024 highlights the emergence of distinct genetic mechanisms of linezolid resistance in different clones. ST16, a known high-risk, globally distributed clone, suggests clonal dissemination of *optrA*-mediated resistance[2,3], while ST780 carrying cfr(D) points to horizontal gene transfer in less common lineages[4,5]. Both genes are located on mobile genetic elements, raising concerns over the spread of resistance across species and regions. The results highlight the critical importance of improving genomic surveillance and infection control measures to halt the spread of linezolid-resistant *E. faecalis.*

Table 13. Genomic characterization of linezolid resistant *E. faecalis* isolates, DOH-ARSP, 2024

Accession No.	Specimen Source	Infection Type	MLST	Linezolid MIC (ug/mL)	Linezolid MIC	AMR Resistance Genes
24ARS-CRH0140	Blood	HAI	16	32	R	optrA, aac(6')-Ie/aph(2'')-Ia, aph(3')-IIIa, spw, fexA, lnu(B), lsa(A), lsa(E), sat4, tet(M), dfrG
24ARS-DMC0129	Urine	HAI	16	64	R	optrA, aac(6')-Ie/aph(2'')-Ia, spw, fexA, lnu(B), lsa(A), lsa(E), ant(6)-Ia, sat4, tet(M), dfrG
24ARS-DMC0392	Urine	HAI	16	32	R	optrA, aac(6')-Ie/aph(2'')-Ia, aph(3')-IIIa, spw, fexA, erm(B), lnu(B), lsa(A), lsa(E), sat4, tet(M), dfrG
24ARS-VSM0252	Wound	HAI	16	16	R	optrA, aac(6')-Ie/aph(2'')-Ia, aph(3')-IIIa, spw, fexA, lnu(B), lsa(A), lsa(E), sat4, tet(M), dfrG
24ARS-VSM0389	Wound	HAI	16	64	R	optrA, aac(6')-Ie/aph(2'')-Ia, aph(3')-IIIa, fexA, lsa(A), sat4, tet(M), dfrG
24ARS-BGH0059	Tissue	HAI	403	16	R	optrA, aac(6')-Ie/aph(2'')-Ia, spw, fexA, erm(B), lnu(B), lsa(A), lsa(E), ant(9)-Ia, tet(L), tet(M)
24ARS-BGH0193	Tissue	HAI	-	≥8	R	optrA, spw, fexA, erm(B), lnu(B), lsa (A), lsa(E), tet(M), dfrG
24ARS-NKI0031	Blood	CAI	16	≥8	R	optrA, aac(6')-Ie/aph(2'')-Ia, aph(3')-IIIa, spw, fexA, erm(B), lnu(B), lsa(A), lsa(E), ant(9)-Ia, sat4, tet(M)

Accession No.	Specimen Source	Infection Type	MLST	Linezolid MIC (ug/mL)	Linezolid MIC	AMR Resistance Genes
24ARS-DMC0256	Urine	CAI	16	8	R	optrA, aac(6')-Ie/aph(2'')-Ia, spw, fexA, lnu(B), lsa(A), lsa(E), ant(6)-Ia, sat4, tet(M), dfrG
24ARS-BGH0089	Wound	CAI	16	8	R	optrA, fexA, erm(B), lsa(A), tet(M)
24ARS-STU0124	Wound	CAI	16	≥8	R	optrA, aac(6')-Ie/aph(2'')-Ia, aph(3')-IIIa, spw, fexA, erm(B), lnu(B), lsa(A), lsa(E), sat4, tet(M), dfrG
24ARS-DMC0393	Urine	CAI	36	16	R	optrA, fexA, lsa(A), str, tet(M), tet(O/W/32/O)
24ARS-BGH0062	Urine	CAI	179	8	R	erm(B), lsa(A), str, tet(M), dfrG
24ARS-BGH0058	Tissue	CAI	368	≥8	R	aph(3')-IIIa, erm(B), lsa(A), ant(9)-Ia, ant(6)-Ia, sat4, tet(L), tet(M)
24ARS-BGH0061	Urine	CAI	403	8	R	optrA, fexA, lsa(A), ant(9)-Ia, ant(6)-Ia, tet(L), tet(M)
24ARS-MAR0217	Aspirate	CAI	506	8	R	optrA, aph(3')-IIIa, fexA, erm(B), lsa(A), ant(9)-Ia, tet(L), tet(M), dfrG
24ARS-DMC0306	Urine	CAI	780	32	R	optrA, aph(3')-IIIa, cfr(D), fexA, erm(B), lsa(A), ant(9)-Ia, tet(L), tet(M), dfrG

The table presented below summarizes resistance data for Linezolid, High-level Gentamicin, High-level Streptomycin, and Tetracycline across various isolates. Linezolid showed high resistance (n=17) with 88.24% concordance due to two false positives; associated genes included *optrA, cfr(D)*, and *fex*A. Gentamicin had 100% concordance with no false results and the presence of *aac(6')-Ie/aph(2")-Ia*. Streptomycin showed inconsistent results, with one group having 60% false negatives and only 20% concordance, while others showed full agreement; resistance genes included *aph(3')-IIIa, ant(6)-Ia*, and *str*. Tetracycline had perfect concordance across all urine specimens, with resistance linked to tet genes. Overall, genotypic and phenotypic resistance were generally consistent, except for variability in Streptomycin results.

Table 14. Comparison of genomic predictions of antibiotic resistance with phenotypic antimicrobial Susceptibility test, 2024

Antibiotic Class	Antibiotic	No. of isolates	Resistant Isolates	False Negative	FN%	False Positive	FP%	Concordance %	Resistance genes
Oxazolidinones	Linezolid	17	16	0	0%	2	11.76%	88.24%	optrA
			1	0	0%	0	0%	100%	optrA, cfr(D)
			17	0	0%	0	0%	100%	fexA
Aminoglycosides	High-level Gentamicin	17	9	0	0%	0	0%	100%	aac(6')-Ie/aph(2'')-Ia
	High-level Streptomycin	17	5	3	60.00 %	1	20.00%	20.00%	aph(3')-IIIa
			1	0	0%		0%	100%	aph(3')-IIIa, ant(6)-Ia
			3	0	0%		0%	100%	ant(6)-Ia
			1	1	100%		0%	0%	str
Tetracyclines	Tetracycline	7	2	0	0%	0	0%	100%	tet(L), tet(M)
			4	0	0%	0	0%	100%	tet(M)
			1	0	0%	0	0%	100%	tet(M), tet(O/W/32/O)

The Sequence Type 16 (ST16) showed a multidrug-resistant profile, being resistant to linezolid, high-level gentamicin, and high-level streptomycin, while remaining susceptible to vancomycin, ampicillin, and penicillin. Linezolid resistance suggests the presence of genes like *optrA* or *cfr*, while aminoglycoside resistance may involve *aac(6′)-Ie-aph(2″)-Ia, ant(6)-Ia*, or *aph(3′)-IIIa*. The resistance profile of ST16 indicates its potential as a high-risk clone, emphasizing the need for genomic surveillance and strict infection control.

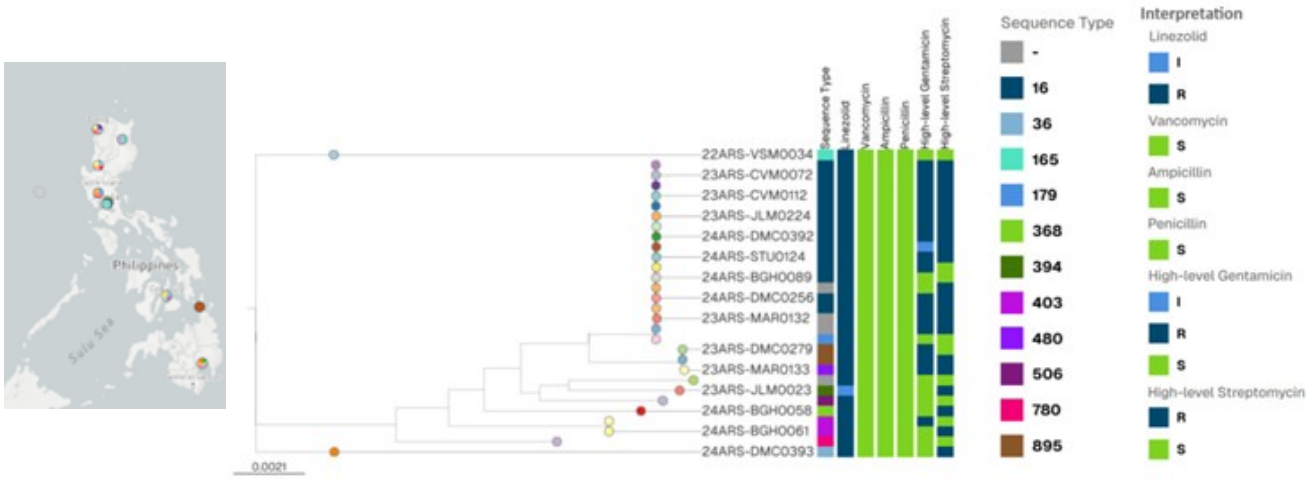


Figure 90. Phylogenetic Tree of Linezolid-Resistant *Enterococcus faecalis* (2022-2024). Metablocks: Geographical location of ST type. A. ST16, B. ST403, C. ST368, D. ST36, E. ST179, F. ST506, and G. ST780, linezolid, vancomycin, ampicillin, penicillin, high-level gentamycin AST

Conclusion

The emergence and spread of linezolid-resistant *E. faecalis* in both community and hospital settings is a pressing concern. The widespread presence of resistance genes like *optrA* calls for heightened surveillance, strict infection control measures, and ongoing antimicrobial stewardship efforts.

References:

[1] ARSP 2023 annual report data summary. Muntinlupa: Antimicrobial Resistance Surveillance Reference Laboratory, Department of Health; 2023.

[2] Wang Y, et al. 2015. J. Antimicrob. Chemother. 70(8):2182-90 A novel gene, optrA, that confers transferable resistance to oxazolidinones and phenicols and its presence in *Enterococcus faecalis* and *Enterococcus faecium* of human and animal origin. (PMID 25977397)

[3] Zhou W, et al. 2019. Distribution of the optrA gene in *Enterococcus* isolates at a tertiary care hospital in China. https://doi.org/10.1016/j.jgar.2019.01.001

[4] Nasir, S.A.R., et. al. 2024. Linezolid-resistant *Enterococcus faecium* clinical isolates from Pakistan: a genomic analysis. BMC Microbiol 24, 347. https://doi.org/10.1186/s12866-024-03491-2

[5] Guerin F, et al. 2020. J. Antimicrob. Chemother. : Molecular and functional analysis of the novel cfr(D) linezolid resistance gene identified in *Enterococcus faecium*. (PMID 32277823)

Enterococcus faecium

A total of **1,931** *E. faecium* infections were reported for 2024.

1,931 infections

The highest reported cases are in DMC (19.99%), PGH (18.02%) and GMH (8.54%) (Figure 91). Based on island group distribution, Luzon contributed the highest percentage of *E faecium* infections with 47.18% followed by Mindanao at 31.80% and Visayas at 21.03%.

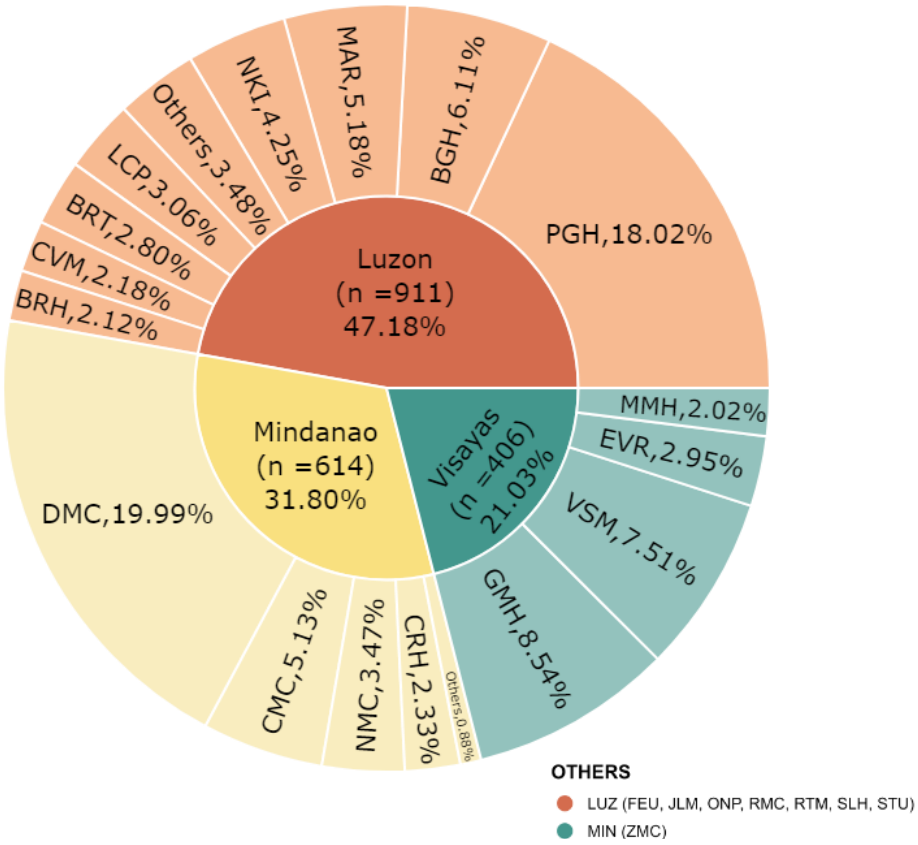


Figure 91 . Distribution of *E. faecium* infections, DOH-ARSP, 2024 (n=1,931)

More than half (58.93%) of the infections were from male patients and the 20-64 age group (53.50%). Urine specimens are the most common source of *E. faecium* infections at 62.51%.

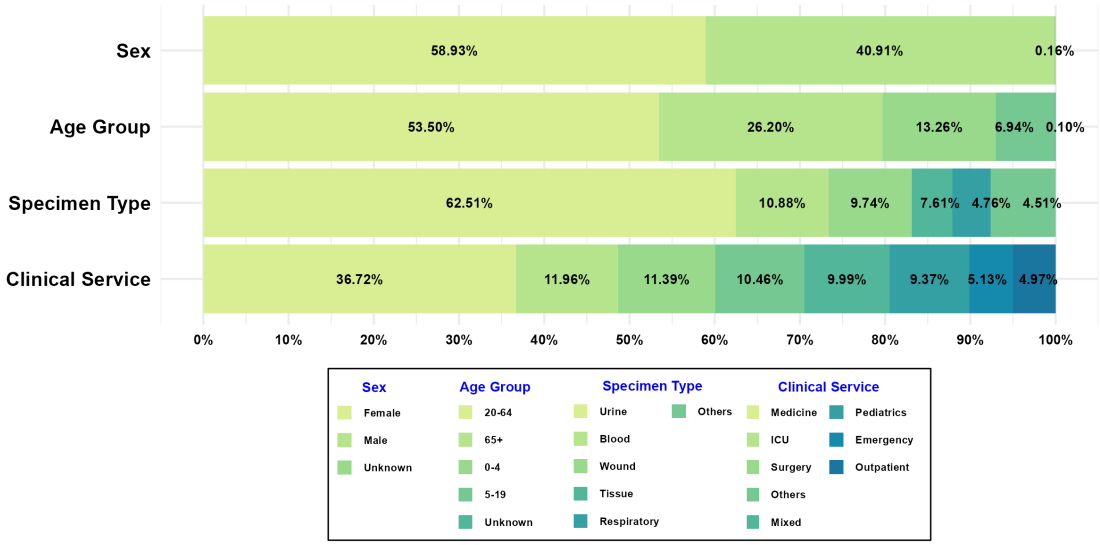


Figure 92. Patient characteristics in *E. faecium* infections, DOH-ARSP, 2024 (n=1,931)

The percent positive of *E. faecium* urinary tract infections for DMC, PGH, GMH, LCP were 7.0%, 6.08%, 4.01% and 3.97%. While the proportions of urinary tract infections due to *E. faecium* for MMH, CRH, BGH, RTM, MAR ranges from 2.13-2.88%. The percent positive of surveillance sites for 15 other surveillance sites were less than 2%.

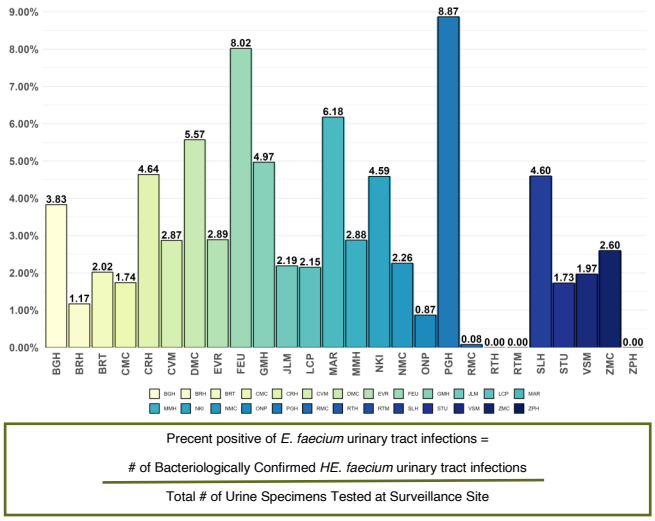


Figure 93. Percent positive of *E. faecium* urinary tract infections among all tested urine specimens per surveillance site, DOH-ARSP, 2024

All types of Infections

Figure 94 shows the resistance rates for all *E. faecium* infections. High resistance rates were observed across all types of infections for penicillin (92.55%) and ampicillin (89.83%). Relatively high resistance rates were noted for vancomycin (22.54%) and nitrofurantoin (50.43%). Resistance to other treatment options remained low, with linezolid at 1.11% and no daptomycin resistance detected in 2024.

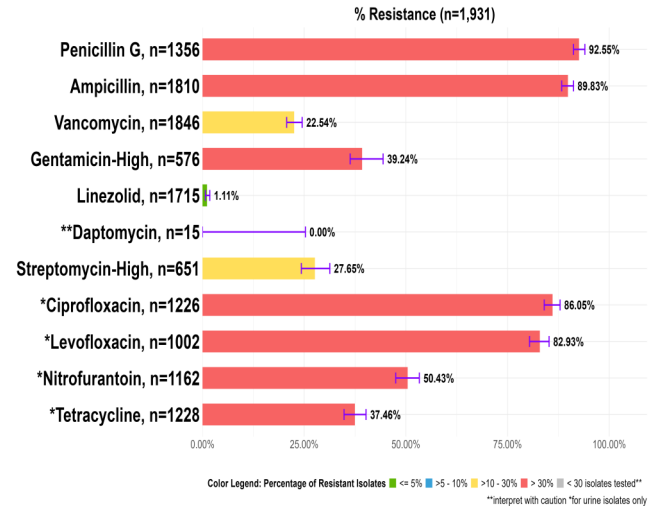
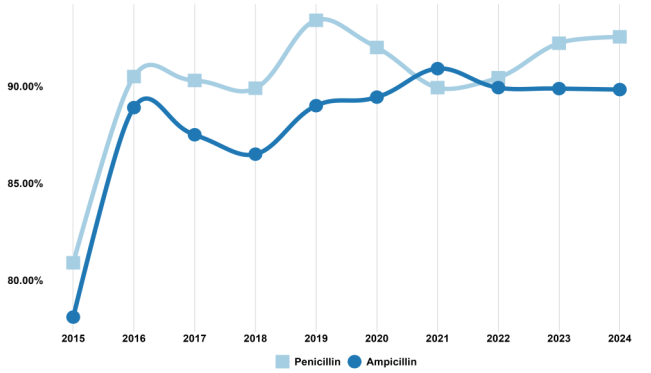
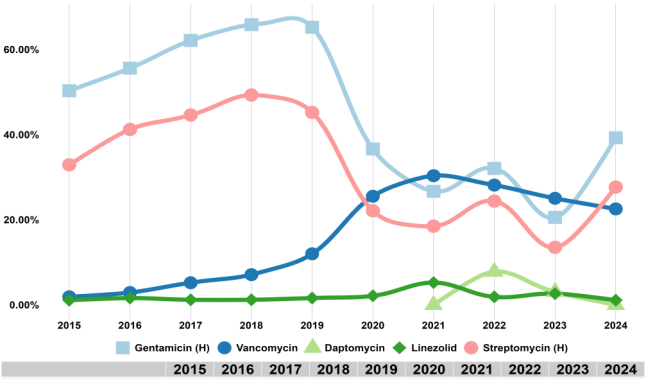


Figure 94. Proportion of all *E. faecium* infections with resistance to tested antibiotics, DOH-ARSP, 2024 (n=1,931)

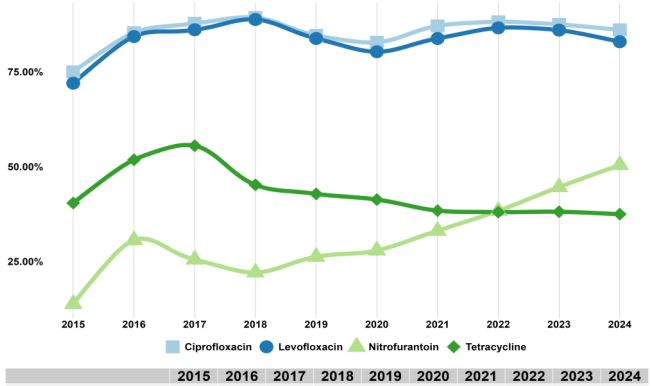
Resistance to penicillin (p=0.000) and ampicillin (p=0.000) has remained consistently high over the past ten years (**Figure 95**). Vancomycin (p=0.000) resistance has also been notably elevated, ranging from 22-33% in the last five years. Conversely, an increasing trend for nitrofurantoin (p=0.000) resistance has been observed over the past decade, while linezolid (p=0.000) and daptomycin (p=0.000) resistance rates have shown a decreasing trend for the last five years.



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Penicillin	80.9%	90.5%	90.3%	89.9%	93.4%	92.0%	89.9%	90.4%	92.2%	92.5%
Ampicillin	78.1%	88.9%	87.5%	86.5%	89.0%	89.4%	90.9%	89.9%	89.9%	89.8%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Gentamicin (H)	50.3%	55.6%	62.1%	65.8%	65.2%	36.6%	26.7%	32.0%	20.6%	39.2%
Vancomycin	1.9%	2.9%	5.2%	7.1%	12.0%	25.5%	30.3%	28.1%	25.0%	22.5%
Daptomycin							0.0%	7.8%	3.1%	0.0%
Linezolid	1.1%	1.6%	1.2%	1.2%	1.6%	2.1%	5.2%	1.9%	2.6%	1.1%
Streptomycin (H)	32.9%	41.2%	44.6%	49.3%	45.2%	22.1%	18.5%	24.4%	13.5%	27.6%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Ciprofloxacin	75.0%	85.3%	87.8%	89.3%	84.6%	82.8%	87.1%	88.2%	87.5%	86.0%
Levofloxacin	72.0%	84.3%	86.1%	88.8%	83.8%	80.3%	83.8%	86.6%	86.0%	82.9%
Nitrofurantoin	13.8%	30.6%	25.5%	22.1%	26.2%	27.9%	33.1%	38.4%	44.6%	50.4%
Tetracycline	40.4%	51.8%	55.5%	45.2%	42.8%	41.3%	38.4%	38.0%	38.1%	37.5%

Figure 95. Yearly resistance rates of all *E. faecium* infections

Bloodstream Infections

Figure 96 illustrates the resistance rates of *E. faecium* bloodstream infections. Vancomycin resistance stands at 21.36%, and linezolid at 1.55%. High-level gentamicin resistance was observed at 52.46%, while high-level streptomycin resistance was 23.33%. These resistance rates for *E. faecium* bloodstream infections are similar to those observed across all *E. faecium* infections.

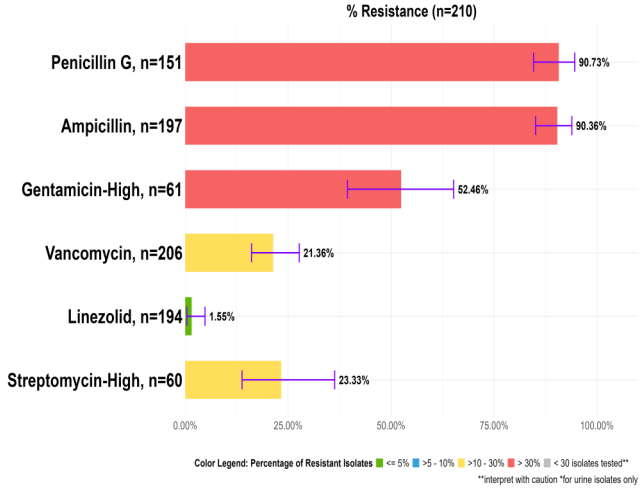
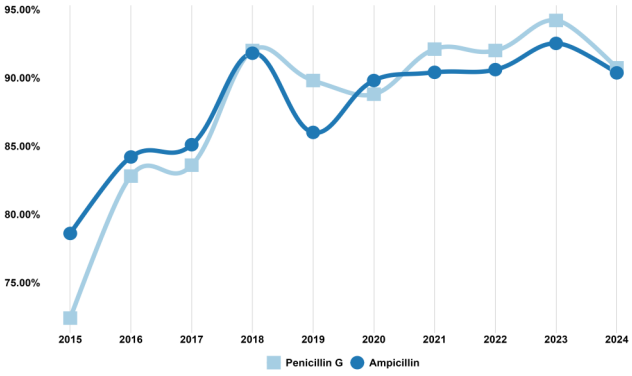
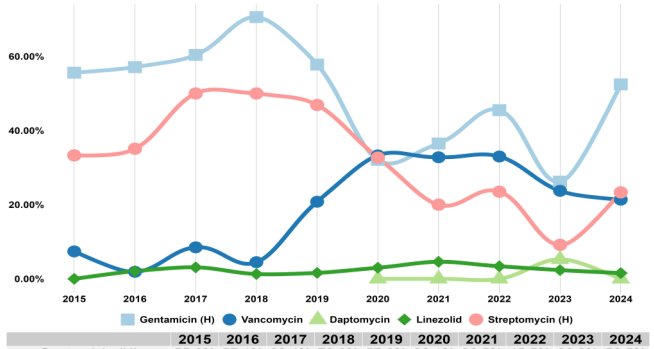


Figure 96. Proportion of *E. faecium* bloodstream infections with resistance to tested antibiotics, DOH-ARSP, 2024 (n=210)

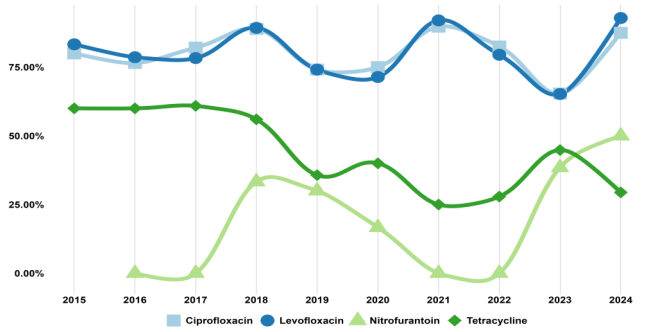
Figure 97 shows the multi-year analysis of the resistance rates of *E. faecium* bloodstream infections: ampicillin (p=0.000), penicillin (p=0.000), vancomycin (p=0.000), linezolid (p=0.000), daptomycin (p=0.000), high-level gentamycin (p=0.000) and streptomycin (p=0.000).



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Penicillin G	72.4%	82.8%	83.6%	92.0%	89.8%	88.8%	92.1%	92.0%	94.2%	90.7%
Ampicillin	78.6%	84.2%	85.1%	91.8%	86.0%	89.8%	90.4%	90.6%	92.5%	90.4%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Gentamicin (H)	55.6%	57.1%	60.4%	70.6%	57.8%	32.1%	36.5%	45.5%	26.2%	52.5%
Vancomycin	7.4%	1.9%	8.5%	4.5%	20.8%	33.3%	32.8%	33.0%	23.7%	21.4%
Daptomycin						0.0%	0.0%	0.0%	5.3%	0.0%
Linezolid	0.0%	2.1%	3.1%	1.3%	1.6%	3.0%	4.6%	3.4%	2.4%	1.6%
Streptomycin (H)	33.3%	35.1%	50.0%	50.0%	46.9%	32.7%	20.0%	23.5%	9.2%	23.3%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Ciprofloxacin	80.0%	76.5%	82.1%	88.9%	74.1%	75.0%	89.7%	82.5%	65.4%	87.5%
Levofloxacin	83.3%	78.6%	78.3%	89.3%	74.1%	71.4%	92.0%	79.5%	65.2%	92.9%
Nitrofurantoin	0.0%	0.0%	33.3%	30.0%	16.7%	0.0%	0.0%	38.5%	50.0%	
Tetracycline	60.0%	60.0%	60.9%	56.0%	35.7%	40.0%	25.0%	27.9%	44.8%	29.4%

Figure 97. Yearly resistance rates of *E. faecium* bloodstream infections, DOH-ARSP, 2024

Urinary Tract Infections

Figure 98 illustrates the resistance rates of *E. faecium* urinary tract infections. High resistance rates were noted for nitrofurantoin (50.58%) and vancomycin (22.26%). While linezolid resistance remained low at 1.20% and no daptomycin resistance was detected (0%), 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.

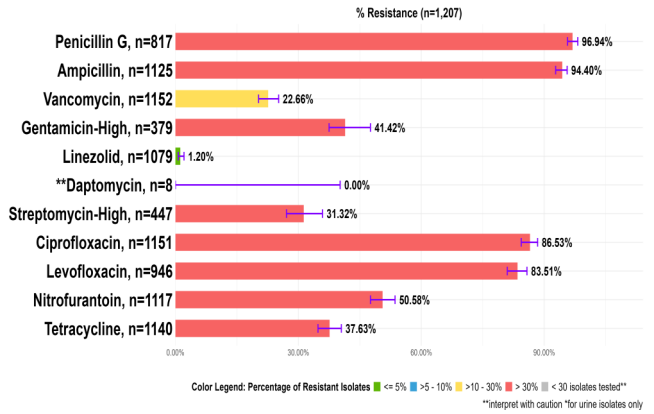
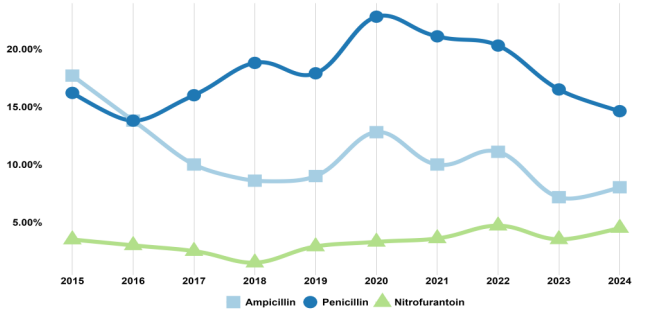
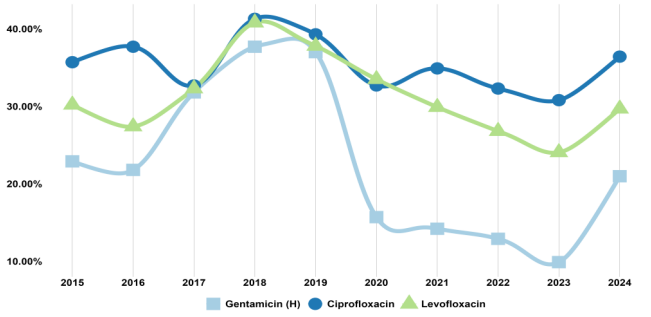


Figure 98. Proportion of *E. faecium* urinary tract infections with resistance to tested antibiotics, DOH-ARSP, 2024 (n=1,207)

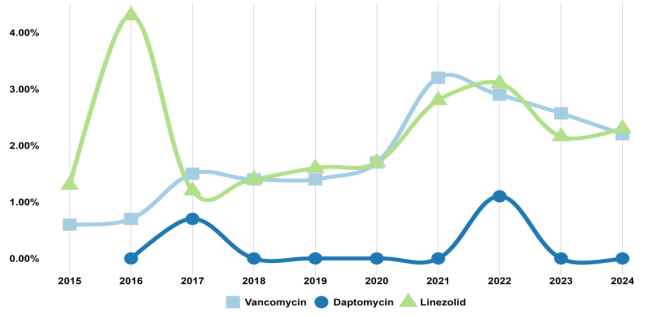
A steady increase in nitrofurantoin resistance has been observed in *E. faecium* urine tract infections over the past 10 years (**Figure 99**). Similar muti-year trends were also observed for *E. faecium* bloodstream infections.



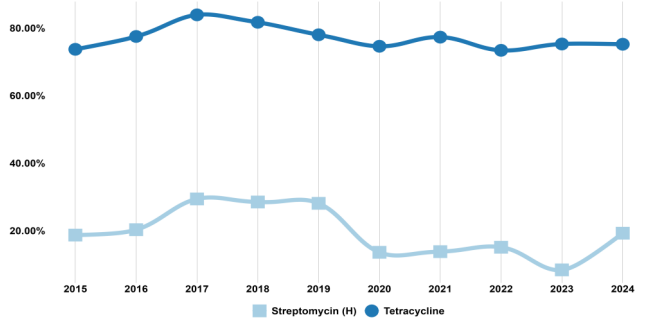
	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Ampicillin	17.7%	13.8%	10.0%	8.6%	9.0%	12.8%	10.0%	11.1%	7.2%	8.0%
Penicillin	16.2%	13.8%	16.0%	18.8%	17.9%	22.8%	21.1%	20.3%	16.5%	14.6%
Nitrofurantoin	3.5%	3.0%	2.5%	1.5%	2.9%	3.3%	3.6%	4.7%	3.5%	4.5%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Gentamicin (H)	22.9%	21.8%	31.8%	37.7%	37.0%	15.7%	14.2%	12.9%	9.9%	21.0%
Ciprofloxacin	35.7%	37.7%	32.7%	41.3%	39.3%	32.7%	34.9%	32.3%	30.8%	36.4%
Levofloxacin	30.2%	27.4%	32.3%	40.8%	37.8%	33.5%	29.9%	26.8%	24.1%	29.7%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Vancomycin	0.6%	0.7%	1.5%	1.4%	1.4%	1.7%	3.2%	2.9%	2.6%	2.2%
Daptomycin		0.0%	0.7%	0.0%	0.0%	0.0%	0.0%	1.1%	0.0%	0.0%
Linezolid	1.3%	4.3%	1.2%	1.4%	1.6%	1.7%	2.8%	3.1%	2.2%	2.3%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Streptomycin (H)	18.7%	20.3%	29.4%	28.5%	28.1%	13.6%	13.8%	15.1%	8.4%	19.3%
Tetracycline	73.7%	77.5%	83.9%	81.7%	78.0%	74.6%	77.3%	73.4%	75.3%	75.2%

Figure 99. Yearly resistance rates of *E. faecium* urinary tract infections, DOH-ARSP 2024

Figure 100-A shows the geographical distribution of vancomycin-resistant *E. faecium* isolates across the country. Rates of surveillance sites from Luzon, Visayas and Mindanao vary which range from 1-50%. **Figure 100 -B** shows the geographical distribution of linezolid-resistant *E. faecium* isolates across the country. Resistance rates across the country are relatively low from 1-205.

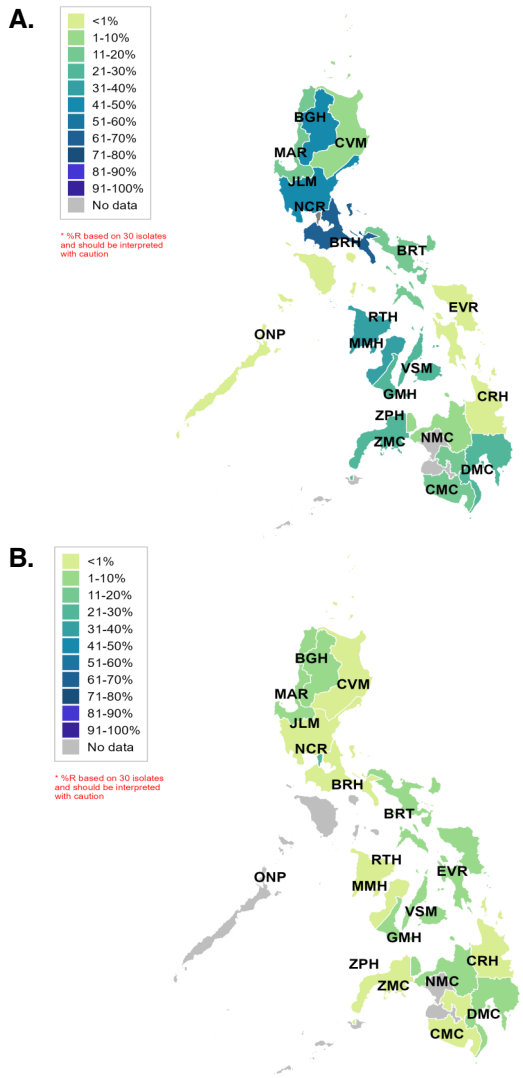


Figure 100. Geographic distribution of A) vancomycin-resistant and B) linezolid-resistant *E. faecium* infections in the Philippines, DOH, ARSP, 2024.

Escherichia coli

A total of **14,894** *E. coli* Infections were reported and analyzed in 2024

14,894 infections

PGH (15.74%) contributed most to the number of isolates followed by DMC (8.31%) and BGH (7.7%). Based on island group distribution, 59.75% were from Luzon.

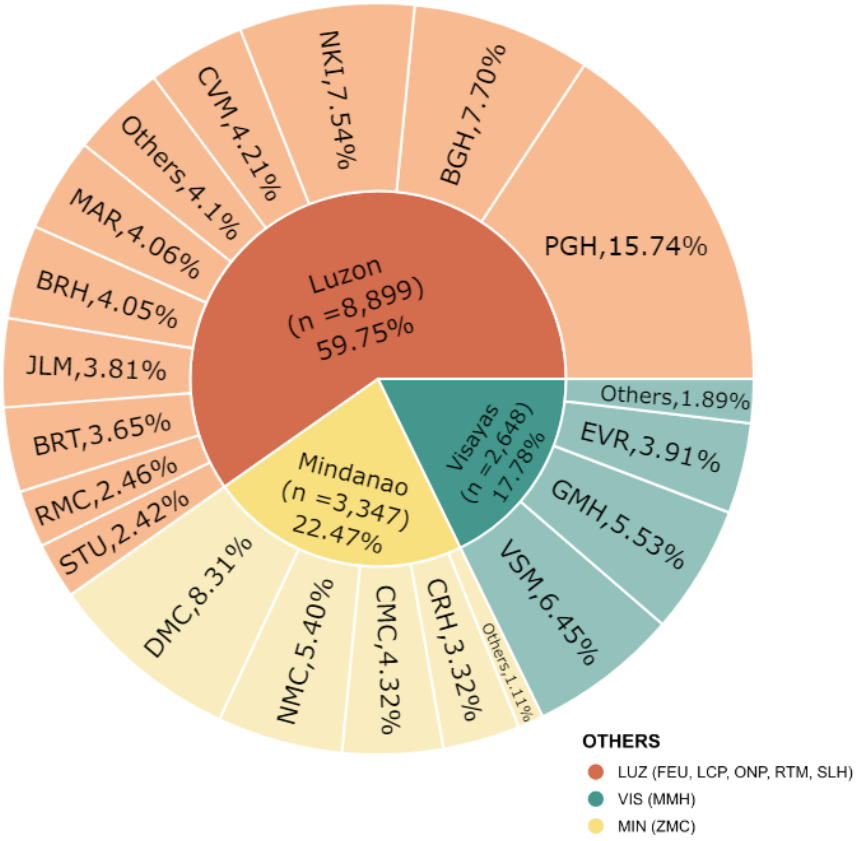


Figure 101. Distribution of *E. coli* infections, DOH-ARSP, 2024 (n=14, 894)

More than half (62.31%) of the infections were from female patients and most (63.04%) were aged 20-64 years old. More than half (79.27%) were presumptive community acquired infections.

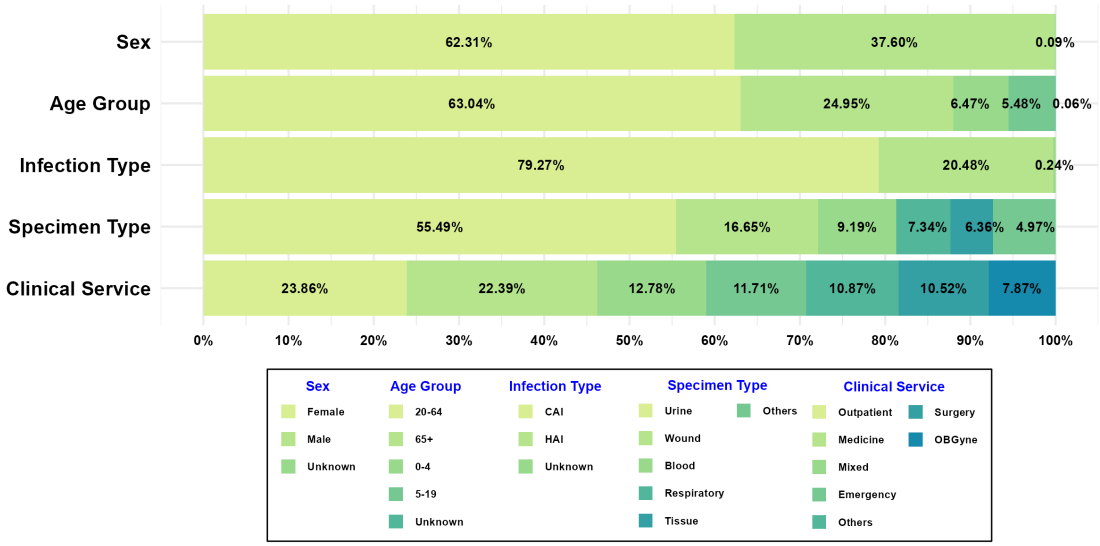
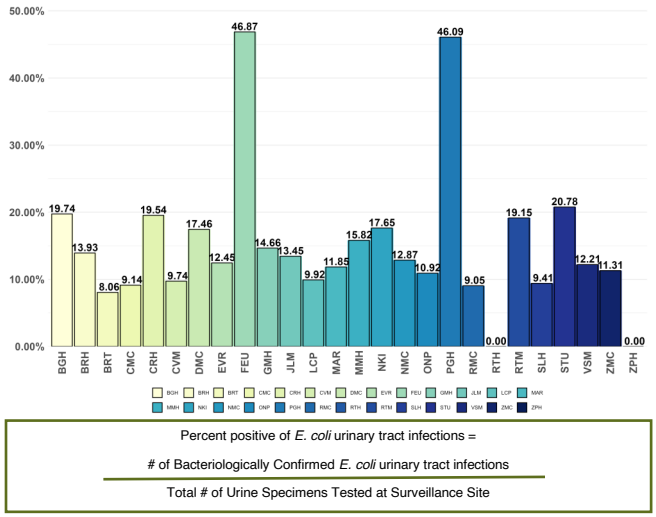


Figure 102. Patient characteristics in *E.coli* infections, DOH-ARSP, 2024 (n=14,894)

Figure 103 shows the percent positive of urinary tract infections caused by *E. coli*. Highest percent positive were observed for FEU (46.87%) and PGH (46.09%). While the percent positive of urinary tract infections due to *E. coli* for STU, BGH, CRH, RTM, NKI, DMC, and MMH were more than 15%. Other surveillance sites have percent positive from 8.06-14.66%.



Percent positive of *E. coli* urinary tract infections =

# of Bacteriologically Confirmed *E. coli* urinary tract infections

Total # of Urine Specimens Tested at Surveillance Site

Figure 103. Percent positive of *E. coli* urinary tract infections among all tested urine specimens per surveillance site, DOH-ARSP, 2024

All types of Infections

Cumulative resistance rates of all *E. coli* infections are shown in Figure 104. Resistance rates of *E. coli* to almost all antibiotics were above 5% high resistance rates for beta-lactams and quinolones. Resistance to nitrofurantoin was at 5.25%, and to co-trimoxazole at 52.14%. Resistance to the combination antibiotics amoxicillin-clavulanic acid was at 22.82% and piperacillin-tazobactam at 17.52%. Resistance to carbapenems are from 7-9% and 2.28% to colistin. All antibiotics tested showed significant differences when the 2023 and 2024 resistance rates were compared except for amoxicillin-clavulanic acid, cefoxitin, gentamicin, nitrofurantoin, meropenem, ertapenem and colistin.

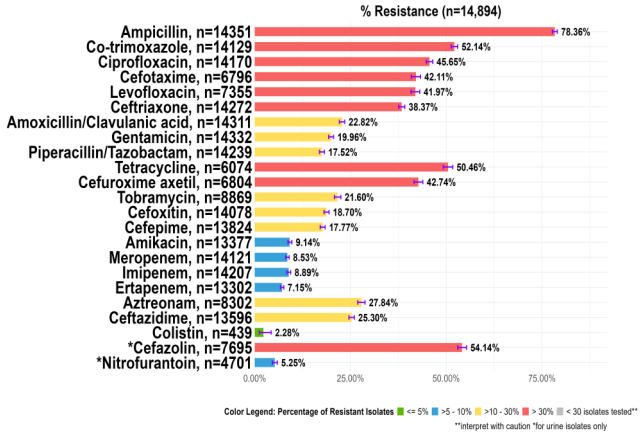
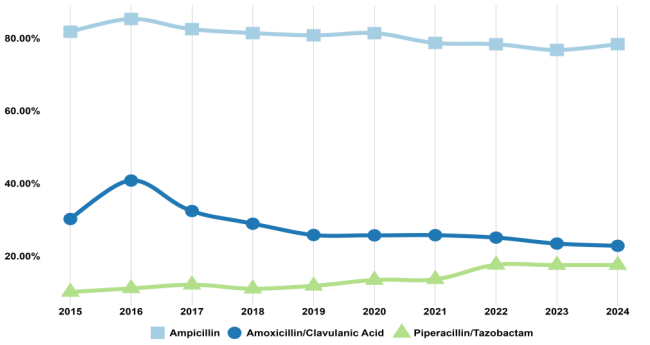
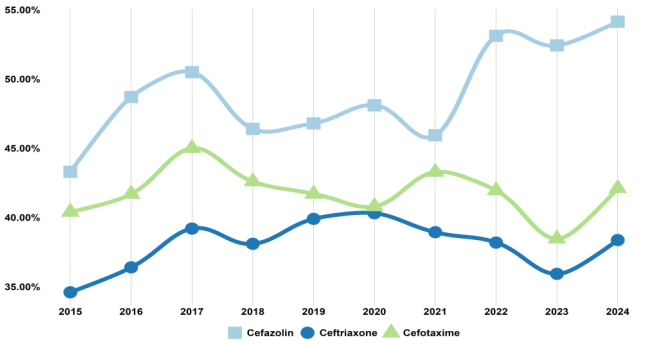


Figure 104. Proportion of all *E. coli* infections with resistance tested to antibiotics, DOH-ARSP, 2024

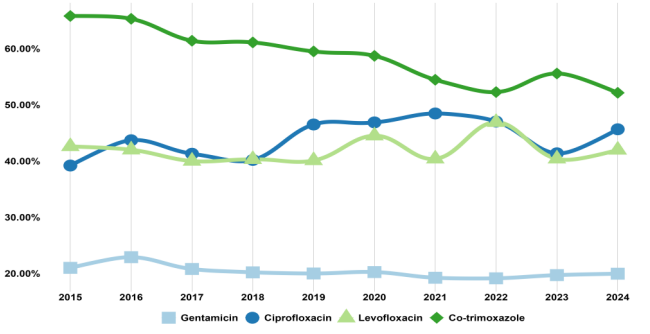
The yearly resistance rates of all *E. coli* infections are shown in Figure 105. There has been a decrease in the resistance rates of amoxicillin-clavulanic acid (p=0.0000). While the observed increasing rates for cefazolin (p=0.0000), amikacin (p=0.0000) and cefuroxime (p=0.0000).



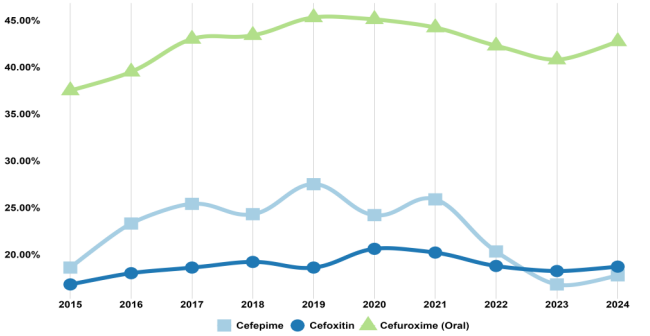
	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Ampicillin	81.8%	85.3%	82.5%	81.4%	80.8%	81.4%	78.7%	78.3%	76.8%	78.4%
Amoxicillin/Clavulanic Acid	30.2%	40.8%	32.4%	28.9%	25.8%	25.7%	25.8%	25.1%	23.4%	22.8%
Piperacillin/Tazobactam	10.1%	11.1%	12.1%	11.0%	11.8%	13.4%	13.6%	17.6%	17.5%	17.5%



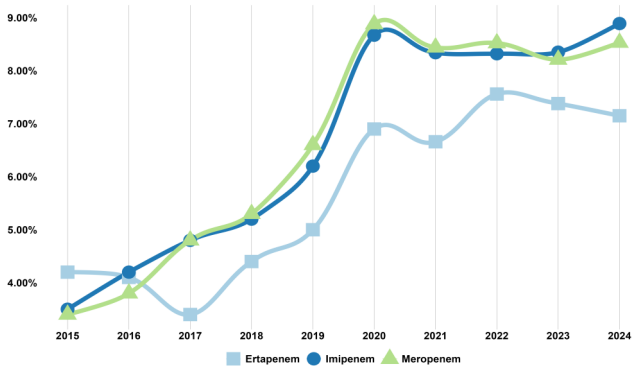
	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Cefazolin	43.3%	48.7%	50.5%	46.4%	46.8%	48.1%	45.9%	53.1%	52.4%	54.1%
Ceftriaxone	34.6%	36.4%	39.2%	38.1%	39.9%	40.3%	38.9%	38.2%	35.9%	38.4%
Cefotaxime	40.4%	41.7%	45.0%	42.6%	41.7%	40.8%	43.3%	42.0%	38.5%	42.1%



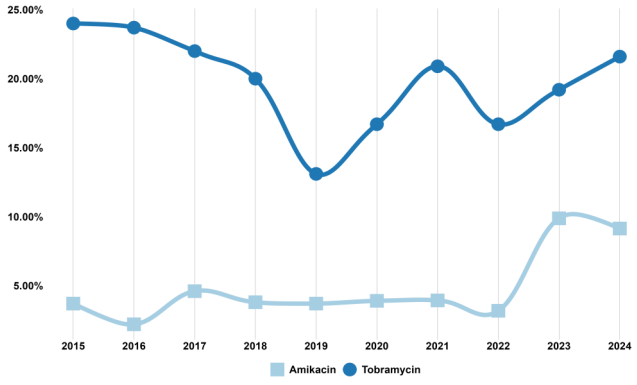
	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Gentamicin	21.0%	22.9%	20.8%	20.2%	20.0%	20.2%	19.2%	19.1%	19.7%	20.0%
Ciprofloxacin	39.2%	43.7%	41.3%	40.2%	46.5%	46.9%	48.5%	47.0%	41.4%	45.6%
Levofloxacin	42.6%	42.0%	40.0%	40.3%	40.1%	44.5%	40.4%	46.9%	40.4%	42.0%
Co-trimoxazole	65.8%	65.3%	61.4%	61.1%	59.5%	58.7%	54.5%	52.3%	55.6%	52.1%



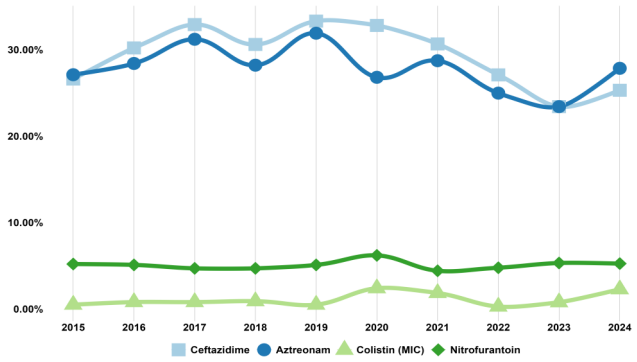
	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Cefepime	18.6%	23.3%	25.4%	24.3%	27.5%	24.2%	25.9%	20.3%	16.8%	17.8%
Cefoxitin	16.8%	18.0%	18.6%	19.2%	18.6%	20.6%	20.2%	18.8%	18.2%	18.7%
Cefuroxime (Oral)	37.5%	39.5%	43.0%	43.4%	45.3%	45.1%	44.2%	42.3%	40.8%	42.7%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Ertapenem	4.2%	4.1%	3.4%	4.4%	5.0%	6.9%	6.7%	7.6%	7.4%	7.2%
Imipenem	3.5%	4.2%	4.8%	5.2%	6.2%	8.7%	8.3%	8.3%	8.3%	8.9%
Meropenem	3.4%	3.8%	4.8%	5.3%	6.6%	8.9%	8.4%	8.5%	8.2%	8.5%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Amikacin	3.7%	2.2%	4.6%	3.8%	3.7%	3.9%	3.9%	3.2%	9.9%	9.1%
Tobramycin	24.0%	23.7%	22.0%	20.0%	13.1%	16.7%	20.9%	16.7%	19.2%	21.6%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Ceftazidime	26.6%	30.2%	32.9%	30.6%	33.3%	32.8%	30.7%	27.1%	23.4%	25.3%
Aztreonam	27.1%	28.4%	31.2%	28.2%	31.9%	26.8%	28.7%	25.0%	23.4%	27.8%
Colistin (MIC)	0.5%	0.8%	0.8%	0.9%	0.5%	2.4%	1.8%	0.3%	0.8%	2.3%
Nitrofurantoin	5.2%	5.1%	4.7%	4.7%	5.1%	6.2%	4.4%	4.8%	5.3%	5.2%

Figure 105. Yearly resistance rates of all *E. coli* infections, DOH-ARSP, 2024

Figure 106 shows the resistance rates of all *E. coli* infections to carbapenems across regions represented by surveillance sites. Resistance to imipenem and meropenem across regions were mostly from 1-20%. Ertapenem resistance was diverse and noted to have 41-50% resistance in BRT.

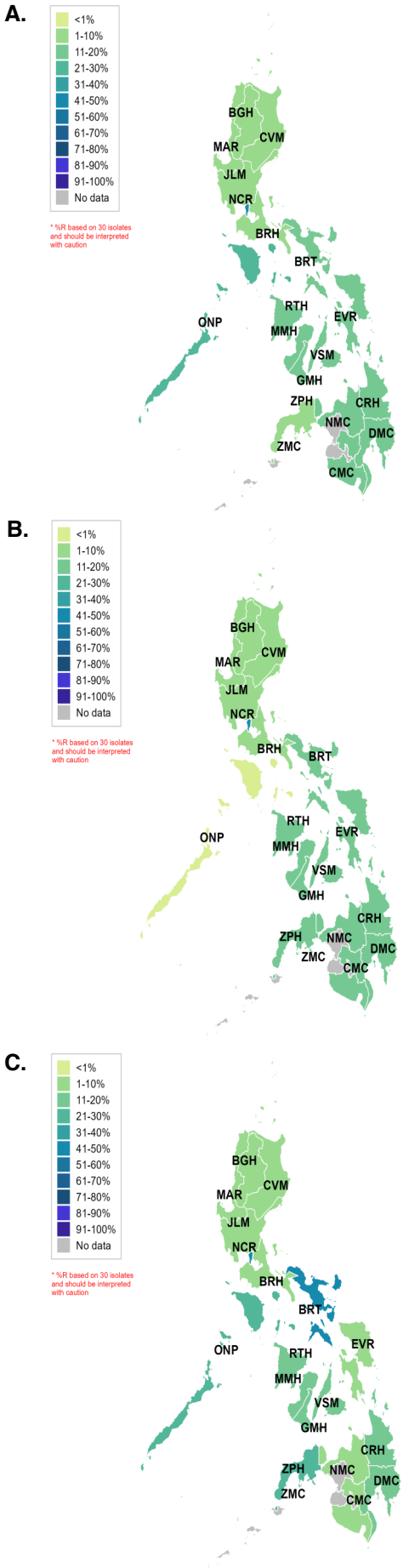


Figure 106. Resistance maps of *E. coli* infections for (A) Imipenem, (B) Ertapenem and (C) Meropenem

Bloodstream Infections

Figure 107 shows the resistance rates of *E.coli* bloodstream infections. High resistance for almost all antibiotics including the cephalosporins, quinolones, and aminoglycosides were observed. Resistance to carbapenems was relatively lower and ranged from 6-8%. Relatively low resistance were likewise noted for the combination antibiotics imipenem/relebactam, ceftolozane/tazobactam and colistin. Compared to the resistance rates for all *E. coli* infections, relatively lower resistance rates for most antibiotics were noted in *E. coli* bloodstream infections.

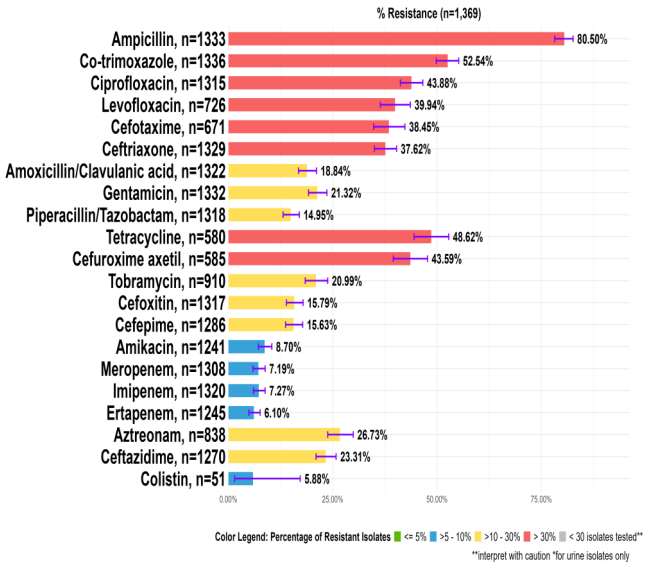
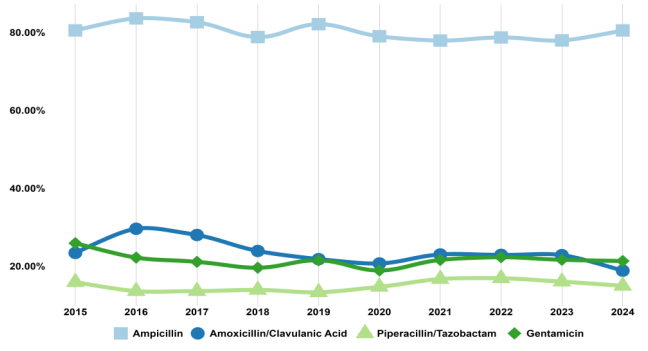
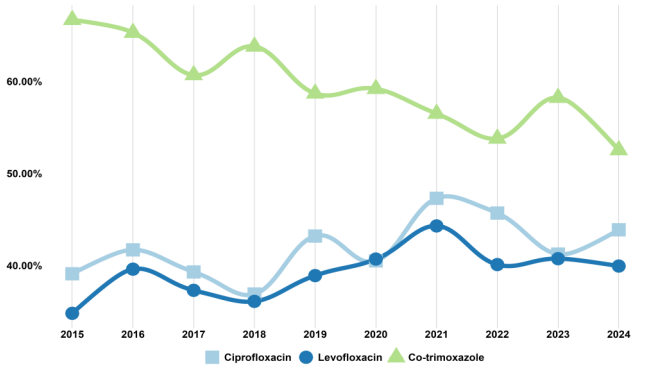


Figure 107. Proportion of *E.coli* bloodstream infections with resistance to tested antibiotics, DOH-ARSP, 2024

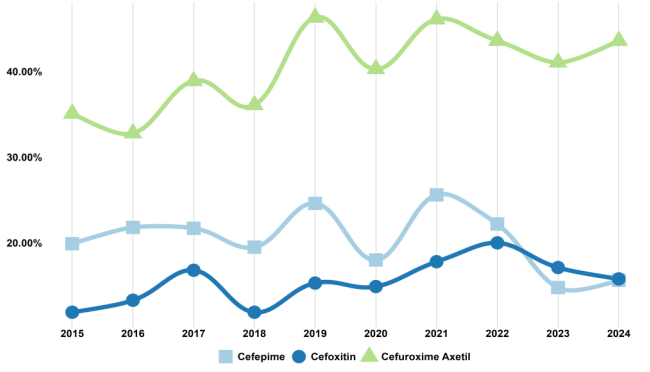
The yearly resistance rates of *E. coli* bloodstream infections is shown in Figure 108. There had been a noted decrease in the resistance rates for cefepime (p=0.0000), amoxicillin-clavulanic acid (p= 0.0000) and tetracycline (p=0.0000).



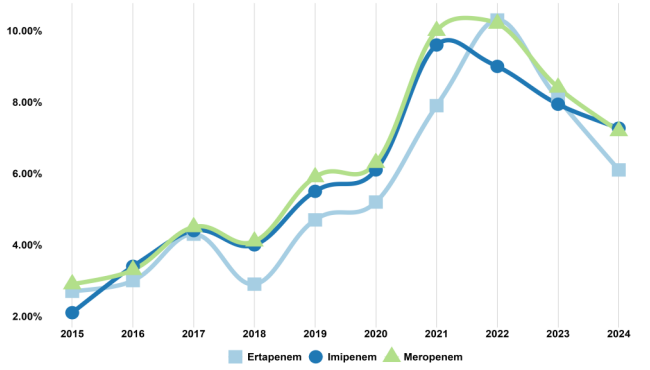
	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Ampicillin	80.5%	83.6%	82.6%	78.8%	82.1%	79.0%	77.9%	78.7%	77.9%	80.5%
Amoxicillin/Clavulanic Acid	23.4%	29.6%	28.0%	23.9%	21.8%	20.7%	23.0%	22.9%	22.8%	18.8%
Piperacillin/Tazobactam	15.9%	13.6%	13.6%	13.9%	13.3%	14.7%	16.7%	16.9%	16.0%	14.9%
Gentamicin	25.9%	22.2%	21.1%	19.6%	21.5%	18.9%	21.6%	22.3%	21.6%	21.3%



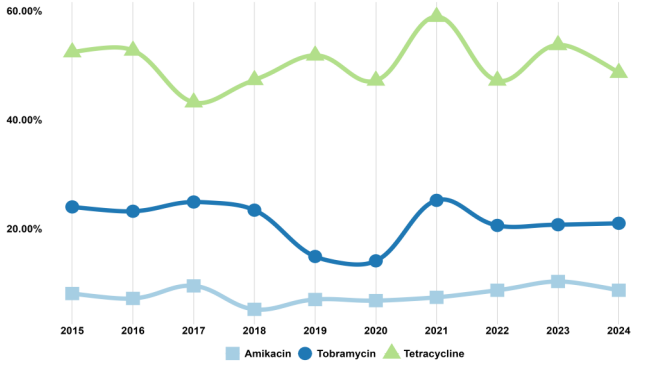
	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Ciprofloxacin	39.1%	41.7%	39.3%	36.9%	43.2%	40.5%	47.3%	45.7%	41.2%	43.9%
Levofloxacin	34.8%	39.6%	37.3%	36.1%	38.9%	40.7%	44.3%	40.1%	40.7%	39.9%
Co-trimoxazole	66.7%	65.3%	60.7%	63.8%	58.7%	59.2%	56.5%	53.8%	58.2%	52.5%



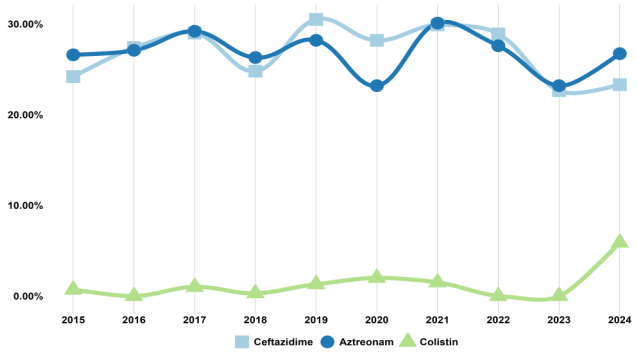
	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Cefepime	19.9%	21.8%	21.7%	19.5%	24.6%	18.0%	25.6%	22.2%	14.8%	15.6%
Cefoxitin	11.9%	13.3%	16.8%	11.9%	15.3%	14.9%	17.8%	20.0%	17.1%	15.8%
Cefuroxime Axetil	35.1%	32.8%	38.9%	36.1%	46.3%	40.3%	46.1%	43.6%	41.1%	43.6%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Ertapenem	2.7%	3.0%	4.3%	2.9%	4.7%	5.2%	7.9%	10.3%	8.1%	6.1%
Imipenem	2.1%	3.4%	4.4%	4.0%	5.5%	6.1%	9.6%	9.0%	7.9%	7.3%
Meropenem	2.9%	3.3%	4.5%	4.1%	5.9%	6.3%	10.0%	10.2%	8.4%	7.2%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Amikacin	8.1%	7.2%	9.5%	5.2%	7.0%	6.8%	7.4%	8.7%	10.3%	8.7%
Tobramycin	24.0%	23.2%	24.9%	23.4%	14.9%	14.1%	25.2%	20.6%	20.7%	21.0%
Tetracycline	52.4%	52.7%	43.2%	47.3%	51.8%	47.2%	58.9%	47.2%	53.7%	48.6%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Ceftazidime	24.2%	27.4%	29.0%	24.8%	30.5%	28.2%	29.9%	28.9%	22.7%	23.3%
Aztreonam	26.6%	27.1%	29.2%	26.3%	28.2%	23.2%	30.1%	27.6%	23.2%	26.7%
Colistin	0.7%	0.0%	1.0%	0.3%	1.3%	2.0%	1.5%	0.0%	0.0%	5.9%

Figure 108. Yearly resistance rates of *E.coli* bloodstream infections

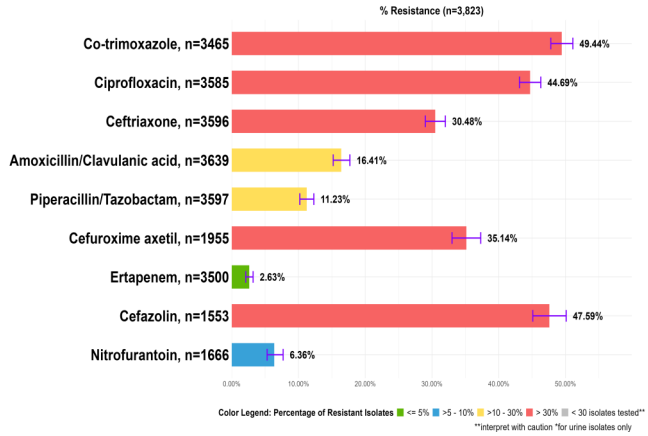


Figure 110. Proportion of *E.coli* urinary tract infections with resistance to tested antibiotics among out-patients, DOH-ARSP, 2024

Urinary Tract Infections

Resistance rates of in-patient *E. coli* urinary tract infections were shown in **Figure 109**. Among urinary *E. coli* infections in in-patients, high resistance to co-trimoxazole, cefazolin and ciprofloxacin were 52.83%, 54.02% and 50.32% respectively, while resistance to nitrofurantoin was at 4.67%

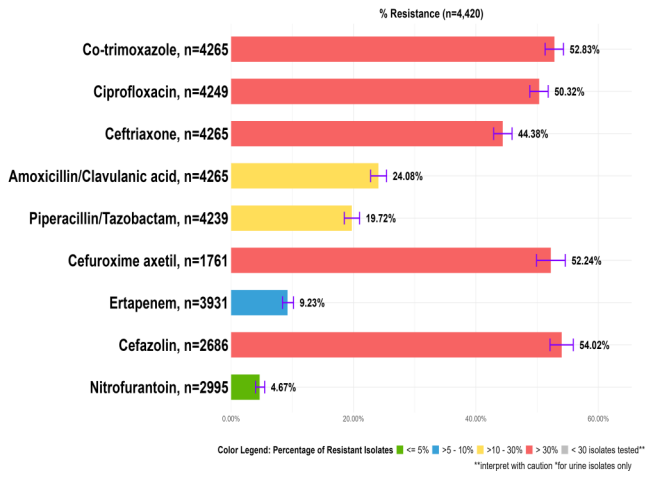
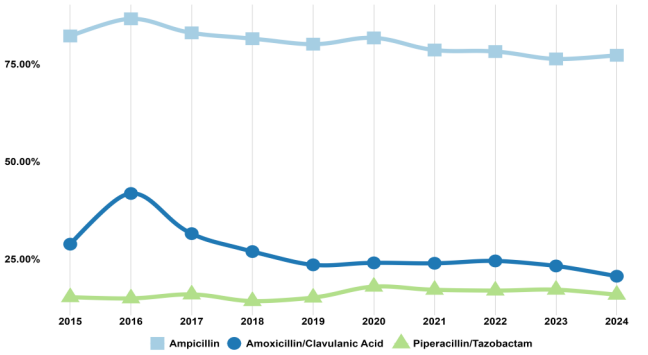


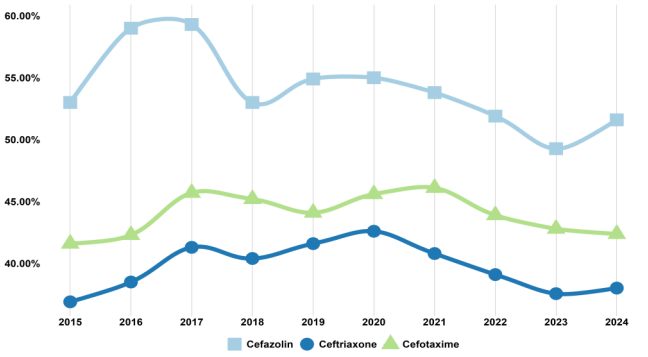
Figure 109. Proportion of *E.coli* urinary tract infections with resistance to tested antibiotics among in-patients, DOH-ARSP, 2024

Resistance rates of out-patient *E. coli* urinary tract infections were shown in **Figure 110**. Among urinary *E. coli* infections in out-patients, high resistance to co-trimoxazole, cefazolin and ciprofloxacin were 49.44%, 47.59% and 44.69%% respectively, while resistance to nitrofurantoin was at 6.36%.

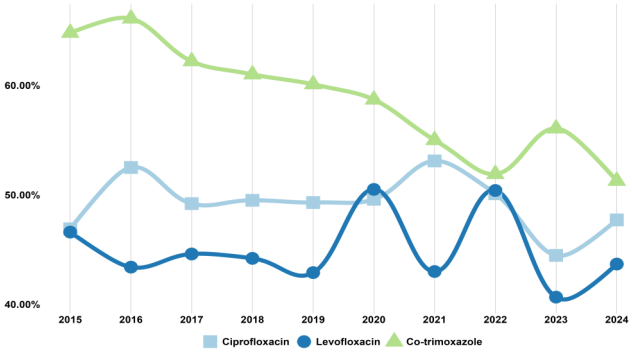
The yearly resistance rates of *E. coli* urinary tract infections are shown in **Figure 111**. Over the past years, there had been a decrease in the resistance rates for the following antibiotics: amoxicillin-clavulanic acid (p=0.0000), meropenem (p=0.0265), ceftazidime (p=0.0176), tetracycline (p=0.0076), and cefoxitin (p=0.0013).



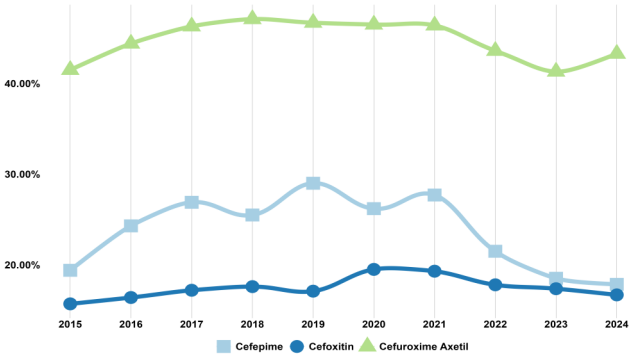
	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Ampicillin	82.2%	86.6%	83.0%	81.5%	80.1%	81.7%	78.6%	78.2%	76.3%	77.2%
Amoxicillin/Clavulanic Acid	28.8%	41.8%	31.5%	26.9%	23.5%	24.0%	23.9%	24.5%	23.2%	20.6%
Piperacillin/Tazobactam	15.2%	14.9%	15.9%	14.2%	15.1%	17.9%	17.1%	16.9%	17.1%	15.8%



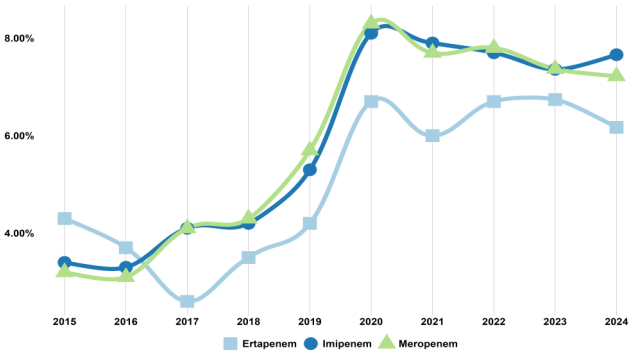
	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Cefazolin	53.0%	59.0%	59.3%	53.0%	54.9%	55.0%	53.8%	51.9%	49.3%	51.6%
Ceftriaxone	36.9%	38.5%	41.3%	40.4%	41.6%	42.6%	40.8%	39.1%	37.6%	38.0%
Cefotaxime	41.6%	42.3%	45.7%	45.2%	44.1%	45.6%	46.1%	43.9%	42.8%	42.4%



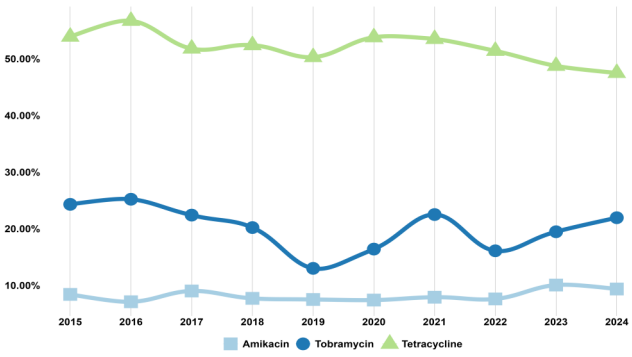
	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Ciprofloxacin	46.9%	52.5%	49.2%	49.5%	49.3%	49.6%	53.1%	50.1%	44.5%	47.7%
Levofloxacin	46.6%	43.4%	44.6%	44.2%	42.9%	50.5%	43.0%	50.4%	40.7%	43.7%
Co-trimoxazole	64.8%	66.1%	62.2%	61.0%	60.1%	58.7%	55.0%	51.9%	56.1%	51.3%



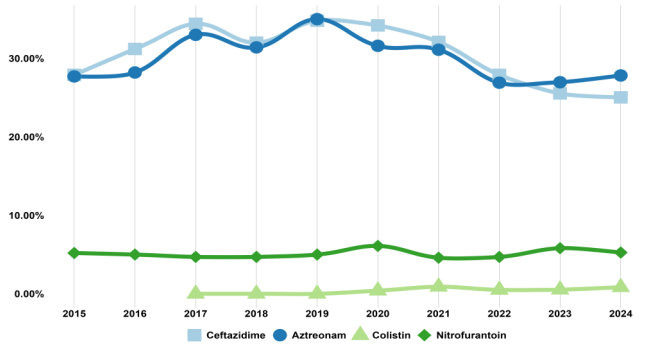
	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Cefepime	19.4%	24.3%	26.9%	25.5%	29.0%	26.2%	27.7%	21.5%	18.5%	17.9%
Cefoxitin	15.7%	16.4%	17.2%	17.6%	17.1%	19.5%	19.3%	17.8%	17.4%	16.7%
Cefuroxime Axetil	41.5%	44.4%	46.3%	47.1%	46.7%	46.5%	46.4%	43.6%	41.3%	43.3%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Ertapenem	4.3%	3.7%	2.6%	3.5%	4.2%	6.7%	6.0%	6.7%	6.7%	6.2%
Imipenem	3.4%	3.3%	4.1%	4.2%	5.3%	8.1%	7.9%	7.7%	7.4%	7.7%
Meropenem	3.2%	3.1%	4.1%	4.3%	5.7%	8.3%	7.7%	7.8%	7.4%	7.2%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Amikacin	8.4%	7.1%	9.0%	7.7%	7.5%	7.4%	7.9%	7.6%	10.1%	9.3%
Tobramycin	24.3%	25.2%	22.4%	20.2%	13.0%	16.4%	22.5%	16.1%	19.5%	21.9%
Tetracycline	53.9%	56.7%	51.8%	52.4%	50.3%	53.8%	53.5%	51.4%	48.8%	47.5%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Ceftazidime	27.9%	31.2%	34.4%	32.0%	34.8%	34.2%	32.1%	27.9%	25.5%	25.0%
Aztreonam	27.7%	28.2%	33.0%	31.4%	35.0%	31.6%	31.1%	26.9%	27.0%	27.8%
Colistin			0.0%	0.0%	0.0%	0.4%	0.9%	0.5%	0.5%	0.8%
Nitrofurantoin	5.2%	5.0%	4.7%	4.7%	5.0%	6.1%	4.6%	4.7%	5.8%	5.3%

Figure 111. Yearly resistance rates of *E. coli* urinary tract infection, DOH-ARSP, 2024

Carbapenem - resistant

Figure 112 shows the resistance rates of carbapenem-resistant *E. coli* infections for 2024. Among these infections, resistance to most antibiotics ranged from 60-90%. The resistance rate for amikacin was at 35.66% and 3.07% for colistin.

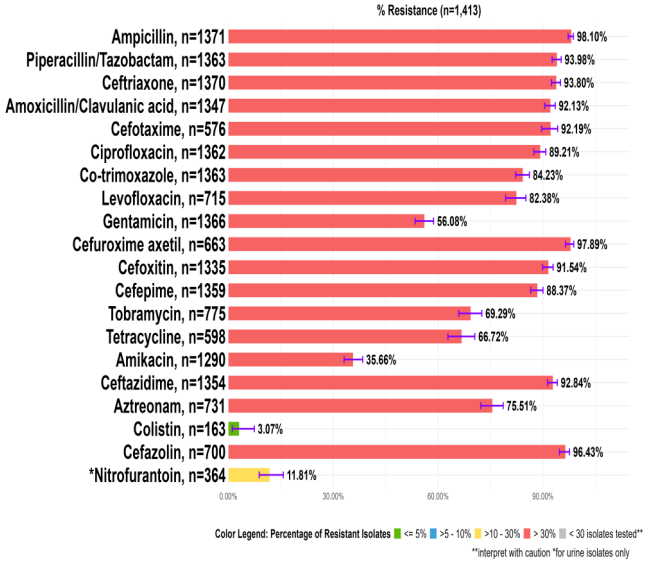


Figure 112. Proportion of all carbapenem-resistant *E. coli* infections, DOH-ARSP, 2024

From the subset of 2024 *E. coli* infections screened phenotypically for ESBL production (n= 11,215), the positivity rate was at 26.19%. The yearly resistance rates of ESBL-positive *E. coli* infections were observed to be fluctuating with a notable increase in rates in 2024. Multi-year analysis showed that the overall changes in ESBL rates for ten years was not statistically significant.

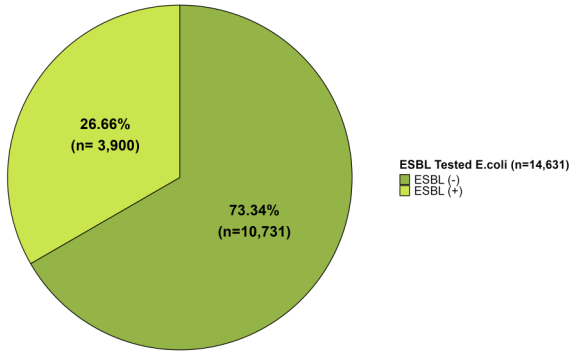
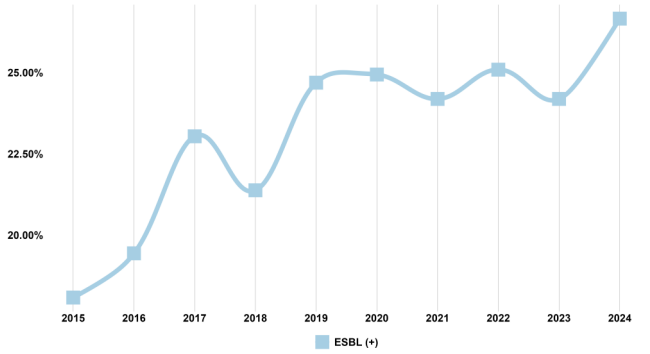


Figure 113. Percentage of ESBL positive and negative *E. coli* infections.



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
ESBL (+)	18.1%	19.4%	23.0%	21.4%	24.7%	24.9%	24.2%	25.1%	24.2%	26.7%

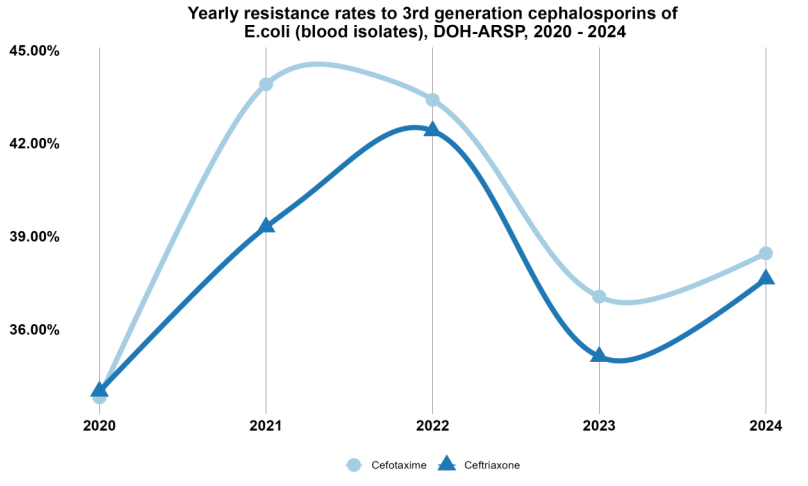
Figure 114. Yearly resistance rates of ESBL-positive *E. coli* infections, DOH-ARSP, 2015-2024

Figure 114 shows the yearly resistance rates of ESBL-positive *E. coli* infections from 2015 to 2024. The resistance rate increased to 26.66% in the previous year, and this difference was statistically significant (p=0.0034). Moreover, the overall changes in the resistance rates over the years are statistically significant (p=0.0000).

One of the AMR indicators in the SDG monitoring framework—is the proportion of patients with bloodstream infections caused by *E. coli* resistant to third-generation cephalosporins. This indicator is particularly concerning in the pediatric population, where the increasing prevalence of third-generation cephalosporin-resistant organisms in neonatal sepsis is especially alarming, as it is associated with higher morbidity and mortality—particularly in low- and middle-income countries (LMICs).

As shown in **Figure 115**, resistance to ceftriaxone and cefotaxime among *E. coli* isolates from bloodstream infections decreased in 2023 but showed a slight increase in 2024. Using *E. coli* blood isolates as a proxy for patients with resistant bloodstream infections, ARSP data indicate that resistance rates remain variable, and a sustained downward trend has yet to be achieved. These findings suggest that the Philippines may need to further strengthen and implement targeted interventions to meet this SDG indicator.

Proportion of blood stream infections (BSIs) due to ***Escherichia coli*** resistant to 3rd generation cephalosporins



	2020	2021	2022	2023	2024
Cefotaxime	33.8%	43.9%	43.4%	37.0%	38.5%
Ceftriaxone	34.0%	39.3%	42.4%	35.1%	37.6%

Figure 115. Yearly resistance rates of *E.coli* bloodstream infections, DOH-ARSP, 2024

Introduction

Colistin remains one of the few effective treatments for multidrug-resistant Gram-negative infections and is classified by the World Health Organization as a "highest-priority critically important antimicrobial" [1]. *Escherichia coli* can develop colistin resistance through chromosomal mutations in the *pmrA/pmrB* genes or via plasmid-mediated *mcr* genes, initially identified as *mcr*-1 in 2015 and now encompassing *mcr*-1 through *mcr*-10 [2]. The Philippines reported its first *mcr*-1-positive *E. coli* infections in 2018, indicating local dissemination and raising concerns about strains resistant to both carbapenems and colistin [3]. This report summarizes surveillance data from ARSP surveillance hospitals and details the genetic characteristics of colistin-resistant *E. coli.*

Results and Discussion

Characteristics and AMR Phenotypic Profile

In 2024, five colistin-resistant *E. coli* isolates underwent whole-genome sequencing (WGS) and antimicrobial resistance (AMR) profiling. These isolates were obtained from patients ranging in age from 1 day to 80 years. Three isolates were recovered from blood samples and two from urine. Most patients presented with multidrug-resistant infections. Notably, one urine isolate from an 80-year-old patient exhibited resistance to nearly all tested antibiotic classes, while an isolate from a neonate remained susceptible to all other antibiotics tested. Four of the five isolates were susceptible to carbapenems, whereas four exhibited resistance to cephalosporins (Table 15).

Table 15. Antimicrobial resistance data

Sample	Age	Specimen	Colistin MIC	Polymyxin	Carbapenem	Cephalosporin	Aminoglycoside	Fluoroquinolone	Beta-lactam	Folate pathway antagonist	Tetracyclines
22ARS-JLM0012	64	Urine	4	Colistin	None	Cefotaxime, Ceftazidime, Ceftriaxone, Cefepime	Gentamicin, Tobramycin	Ciprofloxacin	None	None	None
23ARS-BRH0037	74	Urine	8	Colistin	None	None	Gentamicin	Ciprofloxacin	None	None	None
23ARS-JLM0081	75	Urine	8	Colistin	None	Cefotaxime, Ceftriaxone	None	Ciprofloxacin	None	Trimethoprim/ Sulfamethoxazole	Tetracycline
23ARS-NMC0005	67	Tissue	8	Colistin	None	None	None	None	None	None	None
24ARS-CRH0017	80	Urine	4	Colistin	Imipenem, Meropenem, Ertapenem	Cefotaxime, Ceftazidime, Ceftriaxone, Cefepime	Gentamicin, Tobramycin	Ciprofloxacin, Levofloxacin	Amoxicillin/ Clavulanic acid, Piperacillin/ Tazobactam, Ceftazidime/ Avibactam, Ceftolozane/ Tazobactam	Trimethoprim/ Sulfamethoxazole	Tetracycline
24ARS-CRH0084	27	Blood	4	Colistin	None	Cefotaxime, Ceftriaxone	None	None	None	None	None
24ARS-EVR0035	1d	Blood	4	Colistin	None	None	None	None	None	None	None
24ARS-NKI0048	23	Blood	>32	Colistin	None	Cefotaxime, Ceftazidime, Ceftriaxone, Cefepime	Amikacin, Tobramycin	Ciprofloxacin, Levofloxacin	Amoxicillin/ Clavulanic acid, Piperacillin/ Tazobactam	Trimethoprim/ Sulfamethoxazole	Tetracycline
24ARS-VSM0550	57	Urine	8	Colistin	None	Cefotaxime, Ceftriaxone	Amikacin	Ciprofloxacin, Levofloxacin	None	Trimethoprim/ Sulfamethoxazole	Tetracycline

Typing

WGS-based typing revealed significant genetic diversity among the colistin-resistant *E. coli* isolates, identifying four distinct sequence types (STs), five serotypes, and three phylogenetic groups (Table 16). Two isolates belonged to the epidemic ST131 lineage, underscoring the potential for colistin resistance to arise across diverse *E. coli* lineages.

AMR Gene Profile

WGS detected plasmid-borne *mcr*-1.1 in one of the five colistin-resistant isolates (ST2083, serotype -:H6, phylogroup B1), which also harbored *blaNDM-7* and multiple genes conferring resistance to aminoglycosides, quinolones, beta-lactams, co-trimoxazole, and tetracyclines. Four isolates carried cephalosporin resistance genes. Both ST131 isolates produced extended-spectrum β-lactamases—one with

Table 16. Strain types of *Escherichia coli* isolates

Sample	MLST	Serotype	Phylotype
22ARS-JLM0012	90	O8:H9	C
23ARS-BRH0037	93	O7:H4	A
23ARS-JLM0081	224	O8:H23	B1
23ARS-NMC0005	223	O159:H21	B1
24ARS-CRH0017	2083	-:H6	B1
24ARS-CRH0084	127	O6:H31	B2
24ARS-EVR0035	10	O107/O117:H27	A
24ARS-NKI0048	131	O25:H4	B2
24ARS-VSM0550	131	O16:H5	B2

blaCTX-M-15 and *blaOXA-1*, the other with *blaCTX-M-27*—and possessed *gyrA* and *parC* mutations conferring fluoroquinolone resistance (Table 17). Overall, phenotypic and genotypic findings were largely concordant, with WGS effectively identifying the genes responsible for most observed antibiotic resistances.

Table 17. Antimicrobial resistance genes of *Escherichia coli* isolates

Sample	Colistin	Carbapenem	Cephalosporin	Aminoglycoside	QUINOLONE	BETA-LACTAM	Co-trimoxazole	Tetracycline	Plasmids
24ARS-CRH0017	mcr-1.1	blaNDM-7	blaCMY-42	aac(6')-Ib-cr5, aac(3)-IId, aph(3')-Ia, aph(6)-Id, aph (3")-Ib	gyrA_D87N, gyrA_S83L, parC_S80I, parE_S458A	blaTEM-1	sul2	tet(B)	IncA/C2, IncFIA, IncFIB(AP001918), IncFII(pAMA1167-NDM-5), IncI, IncI1, IncQ1, IncX3, IncX4
24ARS-CRH0084	None	None	blaCTX-M-15	None	None	None	None	None	IncFIB(AP001918), IncFII
24ARS-EVR0035	None	None	None	None	None	None	None	None	IncFII(pCoo)
24ARS-NKI0048	None	None	blaCTX-M-15, blaOXA-1	aac(6')-Ib-cr5, aadA5	gyrA_D87N, gyrA_S83L, parE_I529L, parC_E84V, parC_S80I	None	sul1, dfrA17	tet(A)	Col(BS512), Col(MG828), Col156, IncFIA, IncFII
24ARS-VSM0550	None	None	blaCTX-M-27	aadA5, aph(3")-Ib, aph (6)-Id	parE_E460D, parC_S80I, gyrA_D87N, gyrA_S83L	None	sul1, sul2, dfrA17	tet(A)	Col156, IncFIA, IncFIB(AP001918), IncFII(pRSB107), IncI1

Trends from 2022–2024

Surveillance data from 2022 to 2024 indicate an upward trend in both the number of colistin-resistant *E. coli* cases and the diversity of their resistance profiles. Only one case was reported in 2022, increasing to three in 2023 and five in 2024. These isolates were identified across various surveillance hospitals in different regions of the Philippines, suggesting widespread dissemination rather than localized emergence. WGS revealed plasmid-borne *mcr-1.1* genes in five of the nine colistin-resistant isolates from 2022–2024; however, four isolates from 2024 did not harbor detectable mcr variants. All *mcr*-positive isolates—including the single case in 2022 and three cases in 2023—consistently carried the *mcr-1.1* gene, typically on common plasmid backbones (e.g., *IncX*4 plasmid in one 2023 isolate, and *IncI*2/*IncF* types in others) (Figure 116).

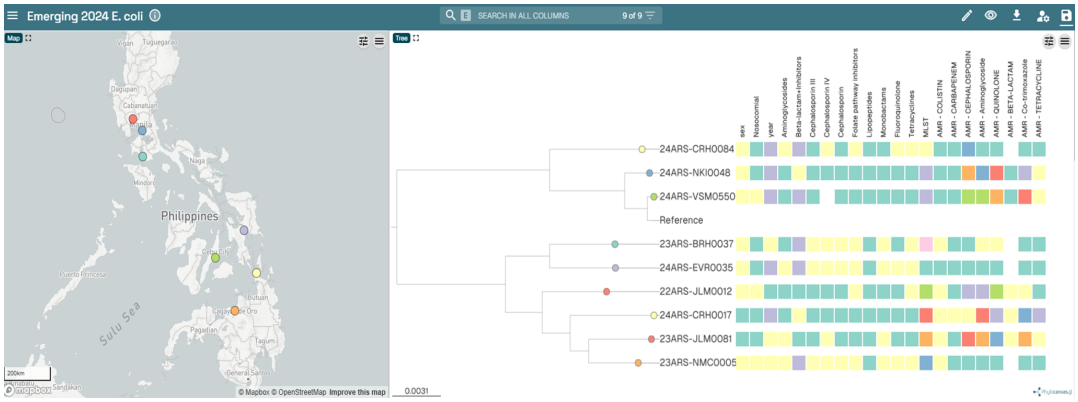


Figure 116. Phylogenetic tree and metadata of colistin-resistant *E.coli* isolates (2022–2024) visualized using Microreact

Discussion

Among the five colistin-resistant *E. coli* isolates analyzed in 2024, only one carried the *mcr-1.1* gene, suggesting that the remaining strains may rely on cryptic mechanisms—such as transcriptional silencing, phase-variable resistance loci, or low-level heteroresistance—undetectable by standard genomic pipelines. Targeted investigations, including transcriptomics, population-analysis profiling, and lipid A structural assays, are warranted to uncover non-canonical or epigenetic pathways of resistance [4]. Phenotype–genotype concordance remained high overall: ESBL genes (notably *blaCTX-M* variants) explained third-generation cephalosporin resistance, *blaNDM-7* matched the single carbapenem-resistant profile, and fluoroquinolone/aminoglycoside resistances correlated with canonical *gyrA/parC* mutations or plasmid genes. The isolates spanned diverse phylogroups A, B1, B2, and C, indicating sporadic rather than clonal spread; nonetheless, two multidrug-resistant ST131 strains and one nearly pan-resistant *mcr-1.1* + *blaNDM-7* isolate demand vigilant monitoring [5]. Integrating whole-genome sequencing with routine AST—and supplementing it with functional assays—will be crucial for timely detection and containment of both plasmid-mediated and mutational colistin resistance in the Philippines.

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Structured Genomic Survey

Background

Antimicrobial-resistant *Escherichia coli* is a critical public health threat, recognized globally as a priority for surveillance[1]. In particular, *E. coli* that produce extended-spectrum β-lactamases (ESBLs) or carbapenemases can cause difficult-to-treat infections, eroding the efficacy of our last-line antibiotics[2]. The World Health Organization's AMR strategy highlights ESBL-producing *E. coli* as a key One Health indicator organism, and lists third-generation cephalosporin-resistant and carbapenem-resistant Enterobacterales among the highest priority pathogens requiring urgent containment.

As part of the Antimicrobial Resistance Surveillance Program (ARSP), we conducted a genomic survey of *Escherichia coli* isolates to identify and characterize their antimicrobial-resistance mechanisms—including ESBLs and carbapenemases.

Results and Discussion

Isolate Characteristics and Resistance Profiles

Fifty-four (54) *Escherichia coli* isolates that were sequenced from 13 ARSP surveillance sites was included in the analysis. Phenotypic testing showed 25 isolates (46 %) resistant to both third-generation cephalosporins and carbapenems, 20 (37 %) resistant only to cephalosporins, and nine fully susceptible—yielding overall ESBL/*AmpC*-mediated cephalosporin resistance in 83 % and carbapenem resistance in 46 %. Patients ranged from newborns to 88 years (56 % aged 25–64, 20 % ≥65, 13 % <15) with a female majority (61 %), and cases were evenly divided between community-onset (54 %) and hospital-acquired (46 %). Urine was the predominant specimen (52 %), followed by blood (19 %), with smaller numbers from respiratory, wound, catheter, stool, and abscess samples.

Table 18. Distribution summary of clinical specimens

Laboratory distribution		Specimen type distribution		Age group distribution		Sex distribution		Nosocomial (Yes/No)	
Laboratory	**Count**	**Specimen Type**	**Count**	**Age group**	**Count**	**Sex**	**Count**	**Nosocomial**	**Count**
BGH	2	Abscess	1	15–24 years	6	Female	33	No	29
BRT	7	Blood	10	1–4 years	1	Male	21	Yes	25
CRH	3	Catheter	2	25–64 years	30				
CVM	8	Other	1	5–14 years	2				
EVR	1	Stool	1	65+ years	11				
GMH	1	Tissue	3	<1 year	4				
JLM	2	Tracheal Aspirate	5						
MAR	10	Urine	28						
NKI	1	Wound	3						
SLH	3								
STU	3								
VSM	9								
ZMC	4								

Genomic Resistance Determinants

WGS showed that carbapenem resistance was almost entirely NDM-driven: *blaNDM-5* in 16 isolates, *blaNDM-7* in six, *blaNDM-1* in two, and two *blaNDM-5* carriers that also harboured *blaKPC-2*; no *OXA-48*-like genes were found. Cephalosporin resistance centred on *blaCTX-M-15* (21 isolates) with few *blaCTX-M-27,* -55 and -14 alleles. *AmpC* genes—mainly *blaCMY* variants and one *blaDHA-1*—plus narrow-spectrum *blaOXA*-1/-10 frequently co-occurred. This β-lactamase stacking overlapped with genes or mutations for aminoglycoside, fluoroquinolone, sulfonamide/trimethoprim and tetracycline resistance, rendering all Ceph+Carb isolates multidrug- or extensively drug-resistant. The nine most susceptible isolates harboured only intrinsic or narrow-spectrum β-lactamase genes (*blaEC ± blaSHV-1*) and do not have any ESBL, *AmpC* or carbapenemase determinants, explaining their full cephalosporin/carbapenem susceptibility despite carrying sporadic non-β-lactam resistance markers such as *gyrA/parC* mutations or *dfrA* alleles.

Table 19. Carbapenemase genes of carba-R or ESBL + *E. coli* isolates by surveillance sites, ARSP 2024

	CARBAPEBEMASE GENES						
Surveillance Site	KPC-2 (n, %)	NDM-1 (n, %)	NDM-5 (n, %)	NDM-7 (n, %)	Oxa-48-like (n, %)	VIM (n, %)	other (n, %)
BGH	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
BRH	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
BRT	0, 0%	1, 14%	4, 57%	0, 0%	0, 0%	0, 0%	1, 14%
CMC	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
CRH	0, 0%	0, 0%	0, 0%	1, 33%	0, 0%	0, 0%	0, 0%
CVM	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
DMC	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
EVR	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
FEU	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
GMH	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
JLM	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
LCP	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
MAR	2, 20%	0, 0%	9, 90%	1, 10%	0, 0%	0, 0%	0, 0%
MMH	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
NKI	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
NMC	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
ONP	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
PGH	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
RMC	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
RTH	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
RTM	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
SLH	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
STU	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
VSM	0, 0%	1, 11%	2, 22%	3, 33%	0, 0%	0, 0%	1, 11%
ZMC	0, 0%	0, 0%	1, 25%	1, 25%	0, 0%	0, 0%	1, 25%
ZPH	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%

	BETA-LACTAMASE GENES																
Surveillance Site	blaLEc	blaLAP-2	blaOXA	blaSHV-1	blaTEM-1	blaC-MY-2	blaC-MY-42	blaC-MY-145	blaCTX-M-14	blaCTX-M-15	blaCTX-M-27	blaCTX-M-55	blaCTX-M-65	blaCTX-M-123	blaEC-5	blaOXA-1	blaOXA-10
BGH	1, 50%	0, 0%	0, 0%	0, 0%	1, 50%	0, 0%	0, 0%	0, 0%	0, 0%	1, 50%	0, 0%	1, 50%	0, 0%	0, 0%	1, 50%	1, 50%	0, 0%
BRH	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
BRT	5, 71%	0, 0%	0, 0%	0, 0%	5, 71%	1, 0%	0, 0%	0, 0%	0, 0%	5, 71%	0, 0%	0, 0%	0, 0%	1, 14%	0, 0%	1, 14%	1, 14%
CMC	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
CRH	3, 100%	0, 0%	0, 0%	0, 0%	1, 33%	0, 0%	1, 33%	0, 0%	0, 0%	2, 67%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
CVM	7, 88%	0, 0%	0, 0%	0, 0%	2, 25%	0, 0%	0, 0%	0, 0%	0, 0%	6, 75%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	1, 13%	0, 0%
DMC	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
EVR	1, 100%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	1, 100%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
FEU	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
GMH	1, 100%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
JLM	2, 100%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
LCP	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
MAR	10, 100%	0, 0%	7, 70%	0, 0%	6, 60%	0, 0%	0, 0%	7, 70%	0, 0%	2, 20%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
MMH	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
NKI	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	1, 100%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	1, 100%	0, 0%
NMC	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
ONP	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
PGH	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
RMC	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
RTH	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
RTM	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
SLH	2, 67%	0, 0%	0, 0%	1, 33%	1, 33%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	1, 33%	0, 0%	1, 33%	1, 33%	0, 0%	0, 0%
STU	3, 100%	1, 33%	0, 0%	0, 0%	3, 100%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	1, 33%	1, 33%	1, 33%	0, 0%	0, 0%	0, 0%
VSM	8, 89%	0, 0%	2, 22%	0, 0%	7, 78%	2, 0%	1, 11%	0, 0%	0, 0%	2, 22%	1, 11%	0, 0%	0, 0%	0, 0%	0, 0%	3, 33%	2, 22%
ZMC	3, 75%	0, 0%	0, 0%	0, 0%	2, 50%	1, 25%	0, 0%	0, 0%	0, 0%	2, 50%	0, 0%	0, 0%	1, 25%	0, 0%	0, 0%	2, 0%	2, 0%
ZPH	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%

Table 20. Number of resistant isolates to select antibiotics tested

Surveillance Site	Colistin %R
BGH	2, 100%
BRH	0, 0%
BRT	7, 100%
CMC	0, 0%
CRH	3, 100%
CVM	8, 100%
DMC	0, 0%
EVR	1, 100%
FEU	0, 0%
GMH	1, 100%
JLM	2, 100%
LCP	0, 0%
MAR	10, 100%
MMH	0, 0%
NKI	1, 100%
NMC	0, 0%
ONP	0, 0%
PGH	0, 0%
RMC	0, 0%
RTH	0, 0%
RTM	0, 0%
SLH	3, 100%
STU	3, 100%
VSM	9, 100%
ZMC	4, 100%
ZPH	0, 0%

Molecular Typing Overview

MLST assigned the 54 isolates to 25 sequence types spread across phylogroups A (14 isolates), B2 (11), C (11), B1 (6), F (5), D (5) and clade I (2). Majorirty were ST361 O9:H30 (8 isolates), ST410 H9/O8:H9 (7), ST131 O25:H4 and O16:H5 (6), and ST2851 O100:H18 (4), with the remaining STs occurring once or twice each (e.g., ST38 O7:H18, ST648 O153:H42, ST770 O25:H51/O102:H51), showing the survey's broad genetic and serotype diversity. See **Table 21.**

Table 21. Molecular typing summary of *E. coli*

Surveillance sites	ST	Serotype(s)	Phylogroup(s)	Isolates	% of total
CRH, MAR	361	O9:H30	A	8	14.80%
BGH, BRT, VSM, ZMC	410	–:H9, O8:H9	C	7	13.00%
BRT, CVM, NKI, VSM, ZMC	131	O25:H4, O16:H5	B2	6	11.10%
BRT, MAR	2851	O100:H18	C	4	7.40%
CRH, ZMC	127	O6:H31	B2	2	3.70%
CVM, STU	770	O25:H51, O102:H51	clade I	2	3.70%
SLH, STU	648	O153:H42	F	2	3.70%
VSM	448	O23:H19	B1	2	3.70%
MAR, VSM	10	O89:H10, O89:H17	A	2	3.70%
CVM	38	O7:H18	D	2	3.70%
CVM	62	O7:H45	F	2	3.70%
Singletons: 15 additional STs occurred once each, together accounting for 27.8 % (15/54) of the collection.					

Discussion

The survey confirms that NDM metallo-β-lactamases—mainly *blaNDM*-5, with lesser *blaNDM*-7, *blaNDM*-1 and occasional *blaKPC*-2—now prevalent on carbapenem resistance in local cases of *E. coli.* This pattern mirrors the global rise of NDM in *E. coli*[4] and parallels some recent Asian reports of NDM-5–positive ST361[5]. Most NDM producers also carried *blaCMY AmpC* and *blaCTX-M*-15 ESBLs, creating extensively drug-resistant profiles that severely constrain β-lactam therapy. MLST revealed broad diversity yet a clear skew toward high-risk international lineages—ST361 O9:H30 (8 isolates) and ST410 H9/O8:H9 (6) alongside ST131 O25:H4/O16:H5 (6) and other minor STs[3][6]. These clones occurred at surveillance sites across country, indicating nationwide dissemination rather than isolated hospital events. The few phenotypically susceptible isolates lacked any acquired ESBL, *AmpC*, or carbapenemase genes, confirming genotype–phenotype concordance despite limited sampling of susceptible strains.

Conclusion

This genomic survey clearly shows what AMR genes drives the nationwide spread of carbapenemase- and ESBL-producing *E. coli*, providing evidence to tighten infection control and prioritize focused surveillance using high-resolution approach such as WGS.

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Klebsiella pneumoniae

A total of **18,378** *K. pneumoniae* infections were reported in 2024

18,378
infections

PGH (13.53%) contributed most to the number of infections followed by VSM (9.36%) and GMH (7.45%). Based on island group distribution, more than half (56.74%) of the infections were from Luzon, 23.91% from Visayas and 19.35% from Mindanao.

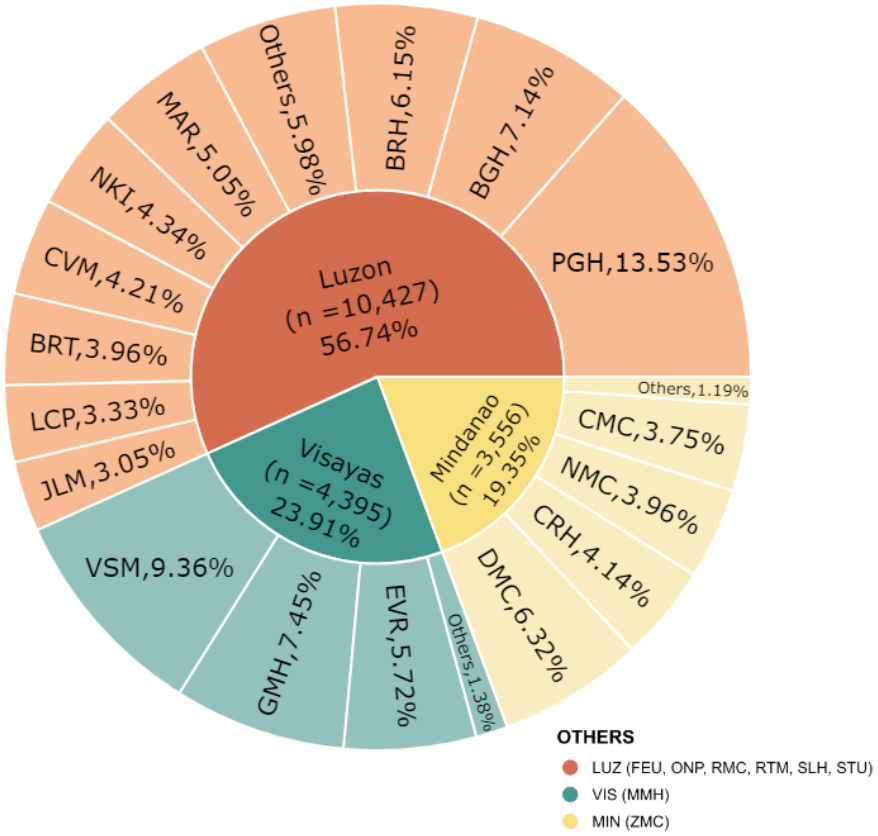


Figure 117. Distribution of *Klebsiella pneumoniae* infections, DOH-ARSP, 2024 (n=18,378)

More than half (61.01%) of the infections were from the 20-64 age group and most (54.70%) were from male patients. Many were respiratory infections (49.54%), with some urinary infections (19.22%) and wound infections (13.09%). Most (62.64%) of the infections were community acquired (Figure 118).

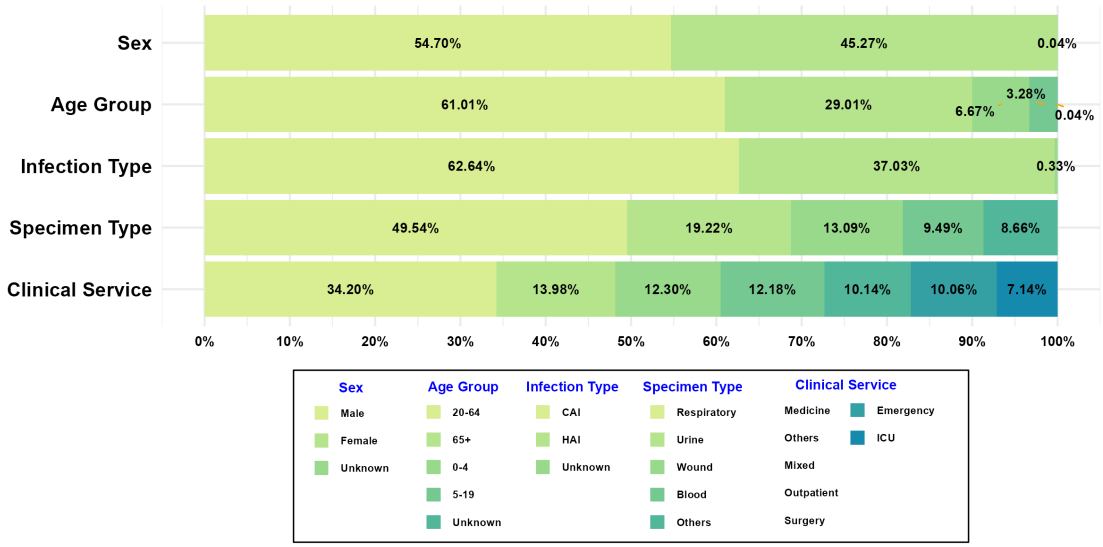
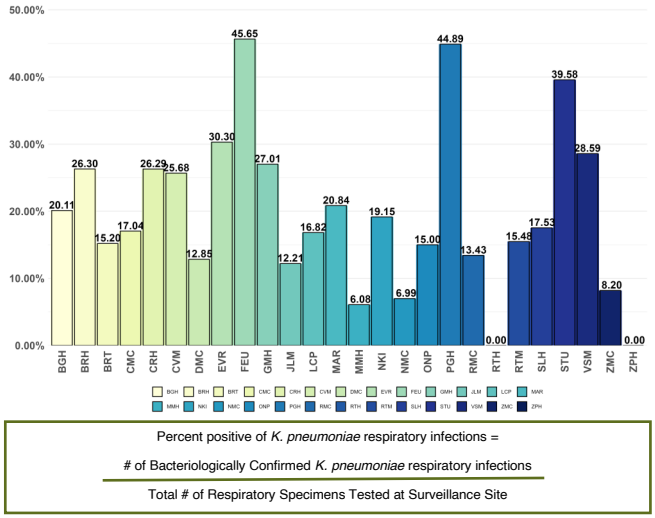


Figure 118. Patient characteristics in *K. pneumoniae* infections, DOH-ARSP, 2024 (n=18,378)

Figure 119 shows the percent positive of respiratory infections caused by *K. pneumoniae* for FEU, PGH, STU, EVER and VSM were 45.65%, 44.89%, 39.58%, 30.30% and 28.59% respectively. The range of percent positive of respiratory infections due to *K. pneumoniae* for GMH, BRH, CRH, CVM, MAR, and BGH were more than 20%. While the percent positive of the remaining surveillance sites ranged from 6.08-19.15%, and no *K. pneumoniae* causing respiratory infection was observed for RTH.



Percent positive of *K. pneumoniae* respiratory infections =

# of Bacteriologically Confirmed *K. pneumoniae* respiratory infections

Total # of Respiratory Specimens Tested at Surveillance Site

Figure 119. Percent positive of *K. pneumoniae* respiratory infections among all tested respiratory specimens per surveillance site, DOH-ARSP, 2024

All types of Infections

Figure 120 shows the resistance rates of all *K. pneumoniae* infections. As known to be commonly resistant to multiple antibiotics, resistance rates of *K. pneumoniae* infections to most antibiotics were high with many being above 30%. Resistance to reserve antibiotics meropenem, imipenem and ertapenem were at 16.48%, 16.38% and 15.72% respectively and to colistin at 3.06%

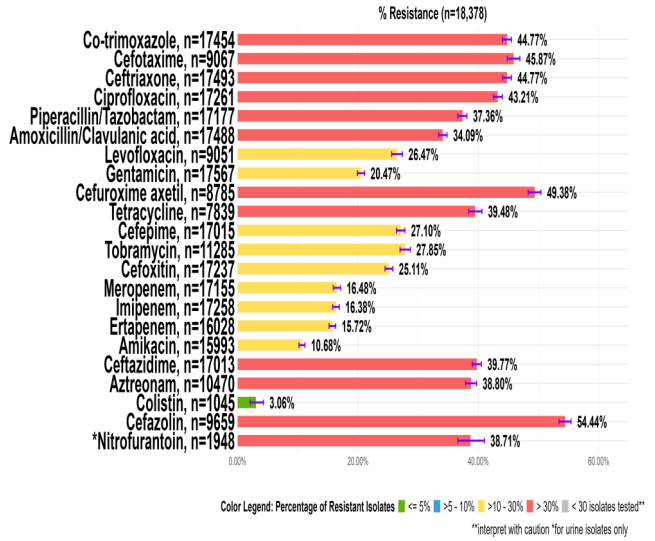
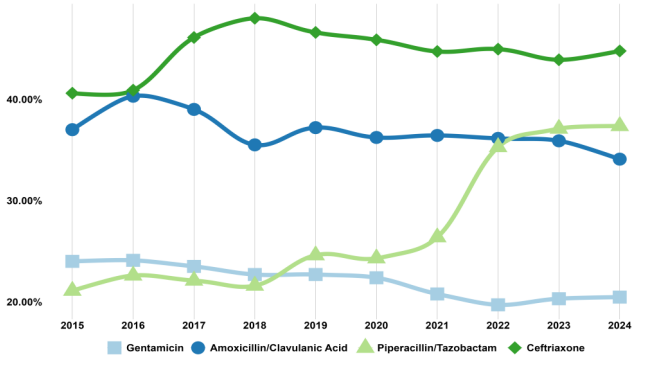
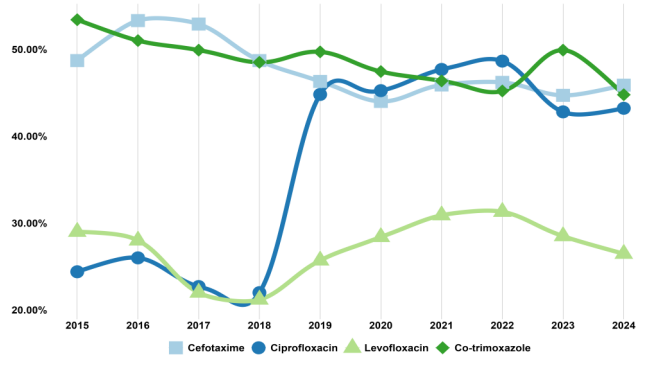


Figure 120. Proportion of all *K. pneumoniae* infections resistant to tested antibiotics, DOH-ARSP, 2024

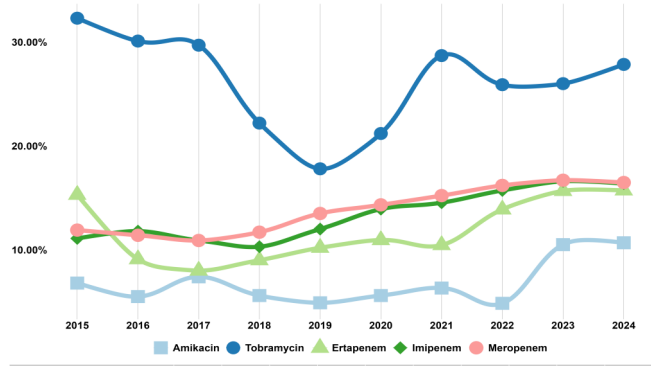
There was a significant increase in the resistance rates of the following antibiotics from 2023 to 2024: aztreonam (p=0.0145) and cefotaxime (p=0.0008). On the other hand, there was a significant decrease in the resistance rates for the following antibiotics: amoxicillin-clavulanic acid (p= 0.0003), co-trimoxazole (p=0.0000) and cefazolin (p=0.0089).



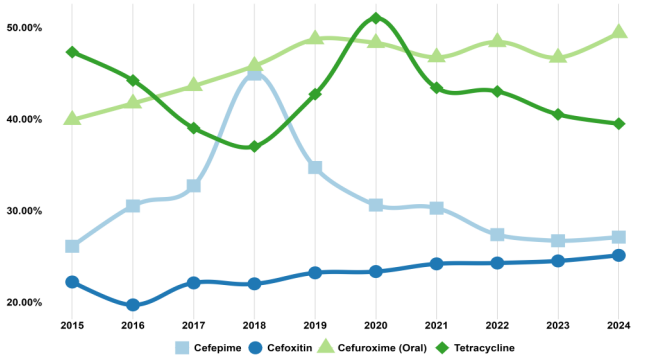
	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Gentamicin	24.0%	24.1%	23.5%	22.7%	22.7%	22.4%	20.8%	19.7%	20.3%	20.5%
Amoxicillin/Clavulanic Acid	37.0%	40.3%	39.0%	35.5%	37.2%	36.2%	36.4%	36.1%	35.9%	34.1%
Piperacillin/Tazobactam	21.1%	22.6%	22.1%	21.6%	24.6%	24.3%	26.4%	35.3%	37.1%	37.4%
Ceftriaxone	40.6%	40.9%	46.1%	48.0%	46.6%	45.9%	44.7%	45.0%	43.9%	44.8%



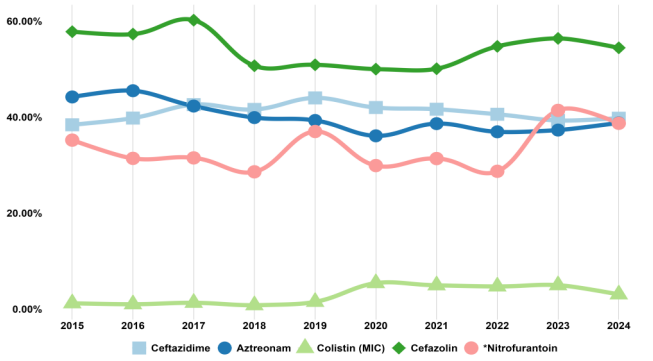
	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Cefotaxime	48.7%	53.3%	52.9%	48.7%	46.3%	44.0%	45.9%	46.2%	44.7%	45.9%
Ciprofloxacin	24.4%	26.0%	22.7%	22.0%	44.8%	45.2%	47.7%	48.6%	42.8%	43.2%
Levofloxacin	29.0%	28.0%	22.0%	21.2%	25.7%	28.4%	30.9%	31.3%	28.5%	26.5%
Co-trimoxazole	53.4%	51.0%	49.9%	48.5%	49.7%	47.4%	46.4%	45.2%	49.9%	44.8%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Amikacin	6.8%	5.5%	7.4%	5.6%	4.9%	5.6%	6.3%	4.8%	10.5%	10.7%
Tobramycin	32.3%	30.1%	29.7%	22.2%	17.8%	21.2%	28.7%	25.9%	26.0%	27.9%
Ertapenem	15.3%	9.1%	8.0%	9.0%	10.2%	10.9%	10.5%	13.9%	15.7%	15.7%
Imipenem	11.1%	11.8%	10.9%	10.3%	12.0%	13.9%	14.5%	15.7%	16.6%	16.4%
Meropenem	11.9%	11.4%	10.9%	11.7%	13.5%	14.3%	15.2%	16.2%	16.7%	16.5%



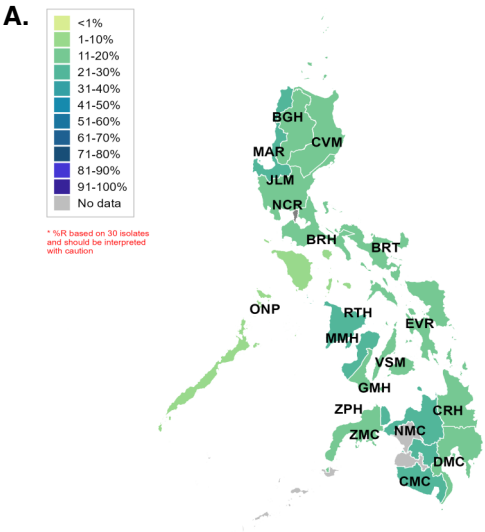
	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Cefepime	26.1%	30.5%	32.7%	44.9%	34.7%	30.6%	30.3%	27.4%	26.7%	27.1%
Cefoxitin	22.2%	19.7%	22.1%	22.0%	23.2%	23.3%	24.2%	24.3%	24.5%	25.1%
Cefuroxime (Oral)	39.9%	41.7%	43.6%	45.8%	48.7%	48.3%	46.7%	48.4%	46.7%	49.4%
Tetracycline	47.3%	44.2%	39.0%	37.0%	42.7%	51.0%	43.4%	43.0%	40.5%	39.5%

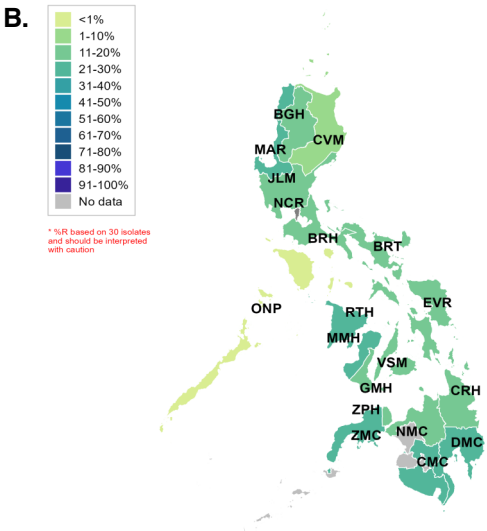


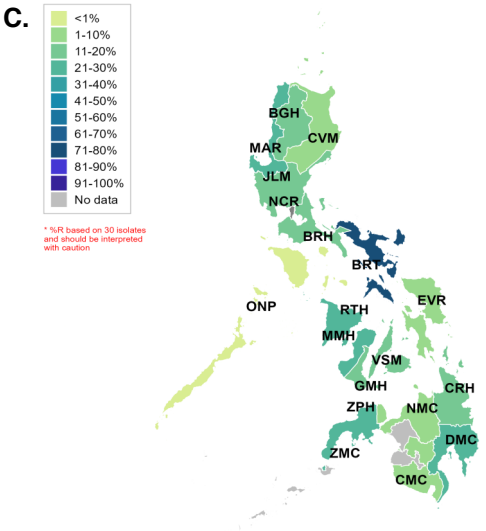
	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Ceftazidime	38.4%	39.8%	42.6%	41.6%	44.0%	42.0%	41.6%	40.6%	39.3%	39.8%
Aztreonam	44.2%	45.5%	42.3%	39.9%	39.3%	36.1%	38.6%	36.9%	37.3%	38.8%
Colistin (MIC)	1.2%	1.0%	1.3%	0.8%	1.5%	5.4%	5.0%	4.7%	5.0%	3.1%
Cefazolin	57.8%	57.3%	60.2%	50.7%	50.9%	50.0%	50.1%	54.7%	56.4%	54.4%
*Nitrofurantoin	35.2%	31.4%	31.5%	28.6%	37.0%	29.9%	31.4%	28.7%	41.4%	38.7%

Figure 121. Yearly resistance rates of all *K. pneumoniae* infections, DOH-ARSP, 2024

Figure 122 shows the geographical distribution of carbapenem and colistin resistance of all *K. pneumoniae* infections across regions of the country. Carbapenem resistance among *K. pneumoniae* infections ranged from 1–30% in surveillance sites across Luzon and the Visayas, and from 21–40% in Mindanao sites. Confirmed colistin-resistant infections were reported in nearly all surveillance sites, except ONP.







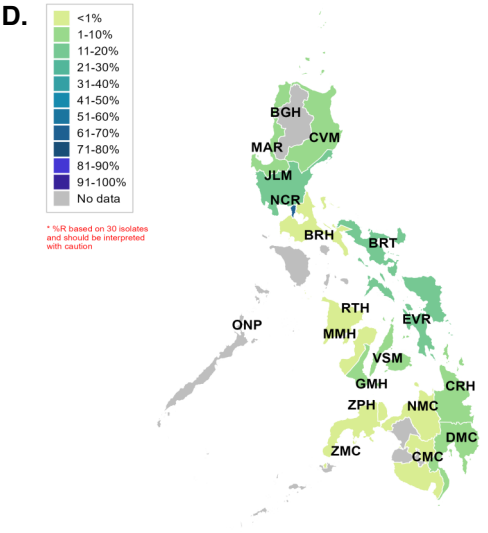


Figure 122. Resistance maps of *K. pneumoniae* infections for a) Imipenem b) Meropenem c) Ertapenem and d) Colistin

Bloodstream Infections

Resistance rates for *K. pneumoniae* blood stream infections, lower respiratory infections, and urine infections are shown on Figures 123, 125 and 127, respectively. Colistin resistance among *K. pneumoniae* bloodstream infections was 5.23%. *K. pneumoniae* bloodstream and urinary tract infections showed relatively higher resistance rates while respiratory infections exhibited relatively lower resistance rates to most antibiotics.

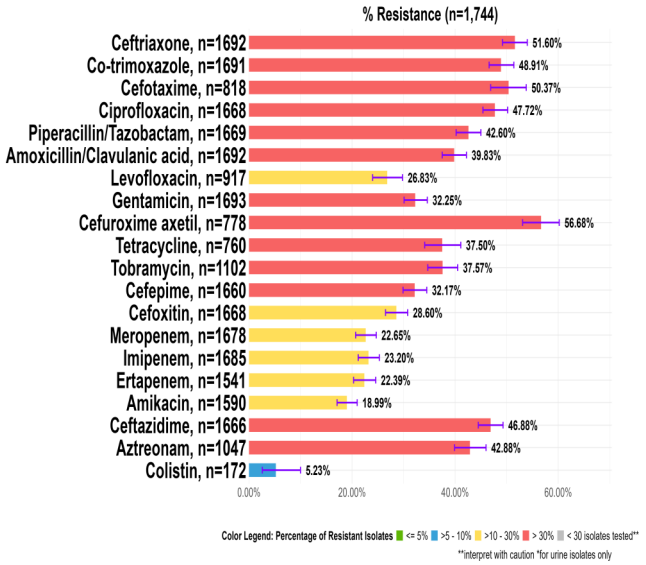
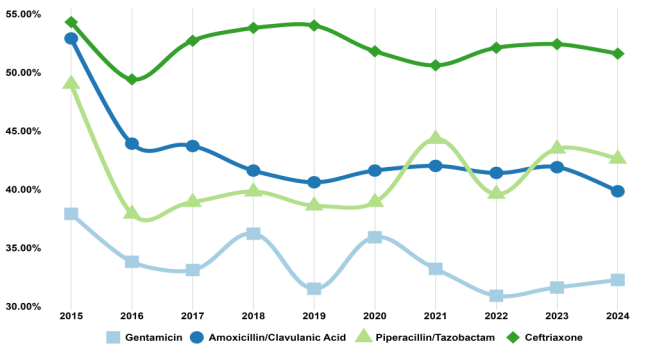
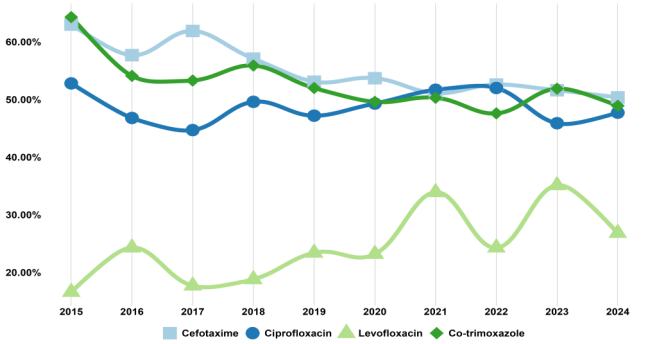


Figure 123. Proportion of all *K. pneumoniae* bloodstream infections with resistance to tested antibiotics, DOH-ARSP, 2024

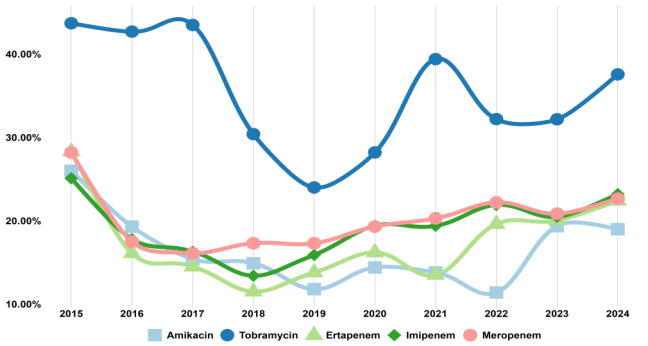
The yearly resistance rates of *K. pneumoniae* bloodstream infections are seen on Figure 124. Multi-year analysis showed significant differences for all tested antibiotics except for cefazolin (p=0.0745) and ceftazidime (p=0.6654).



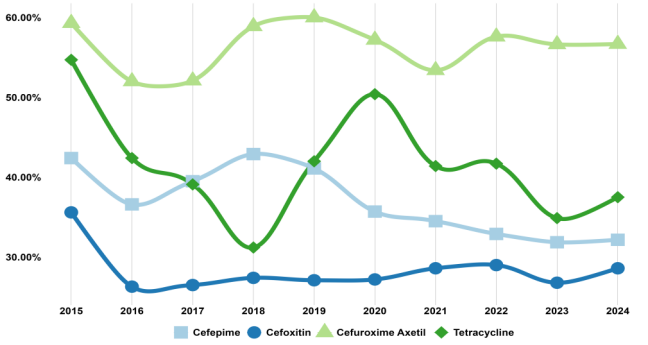
	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Gentamicin	37.9%	33.8%	33.1%	36.2%	31.5%	35.9%	33.2%	30.9%	31.6%	32.2%
Amoxicillin/Clavulanic Acid	52.9%	43.9%	43.7%	41.6%	40.6%	41.6%	42.0%	41.4%	41.9%	39.8%
Piperacillin/Tazobactam	49.0%	37.9%	38.9%	39.8%	38.6%	38.9%	44.3%	39.6%	43.5%	42.6%
Ceftriaxone	54.3%	49.4%	52.7%	53.8%	54.0%	51.8%	50.6%	52.1%	52.4%	51.6%



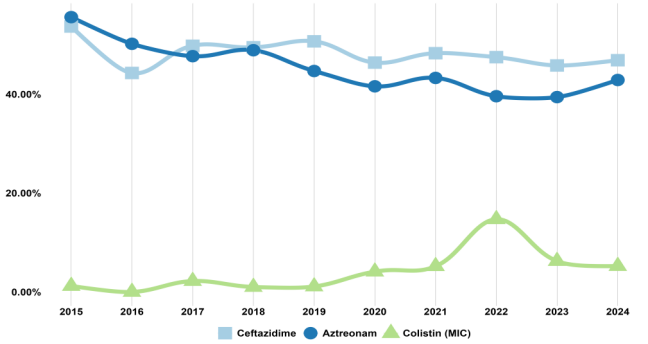
	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Cefotaxime	63.0%	57.7%	61.9%	57.1%	53.1%	53.7%	51.1%	52.6%	51.6%	50.4%
Ciprofloxacin	52.8%	46.8%	44.7%	49.6%	47.2%	49.3%	51.7%	52.0%	45.9%	47.7%
Levofloxacin	16.6%	24.3%	17.7%	18.8%	23.4%	23.2%	33.9%	24.3%	35.1%	26.8%
Co-trimoxazole	64.3%	54.1%	53.3%	55.9%	52.0%	49.6%	50.3%	47.6%	51.9%	48.9%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Amikacin	26.0%	19.3%	15.4%	14.9%	11.8%	14.4%	13.8%	11.4%	19.4%	19.0%
Tobramycin	43.7%	42.7%	43.5%	30.4%	24.0%	28.2%	39.4%	32.2%	32.2%	37.6%
Ertapenem	28.3%	16.1%	14.5%	11.5%	13.8%	16.2%	13.5%	19.6%	20.1%	22.4%
Imipenem	25.1%	17.8%	16.3%	13.4%	15.9%	19.4%	19.4%	21.9%	20.5%	23.2%
Meropenem	28.2%	17.5%	16.1%	17.3%	17.3%	19.3%	20.3%	22.2%	20.9%	22.6%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Cefepime	42.4%	36.6%	39.5%	42.9%	41.1%	35.7%	34.5%	32.9%	31.9%	32.2%
Cefoxitin	35.6%	26.3%	26.5%	27.4%	27.1%	27.2%	28.6%	29.0%	26.8%	28.6%
Cefuroxime Axetil	59.3%	52.0%	52.1%	58.9%	60.0%	57.2%	53.4%	57.6%	56.6%	56.7%
Tetracycline	54.7%	42.4%	39.1%	31.2%	42.0%	50.4%	41.4%	41.7%	34.9%	37.5%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Ceftazidime	53.7%	44.3%	49.8%	49.5%	50.7%	46.4%	48.3%	47.5%	45.8%	46.9%
Aztreonam	55.6%	50.2%	47.7%	48.9%	44.7%	41.6%	43.3%	39.6%	39.4%	42.9%
Colistin (MIC)	1.2%	0.0%	2.2%	1.0%	1.1%	4.1%	5.2%	14.7%	6.2%	5.2%

Figure 124. Yearly resistance rates of *K. pneumoniae* bloodstream infections, DOH-ARSP, 2024

Lower Respiratory Tract Infections

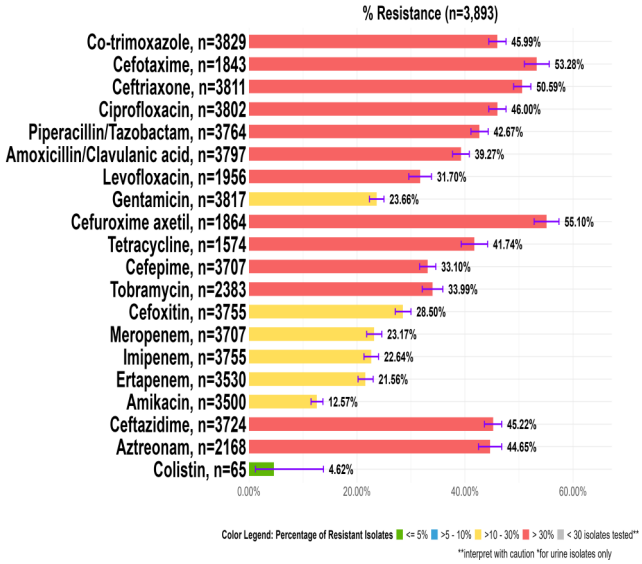
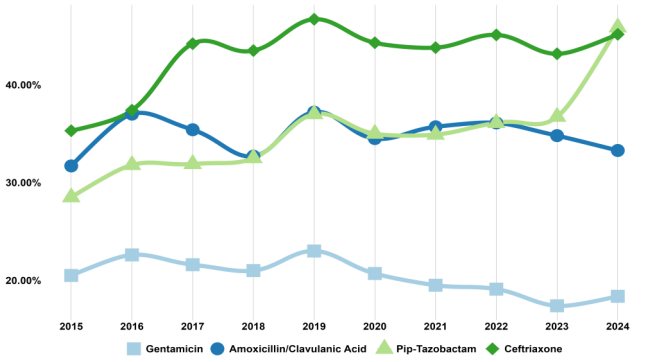
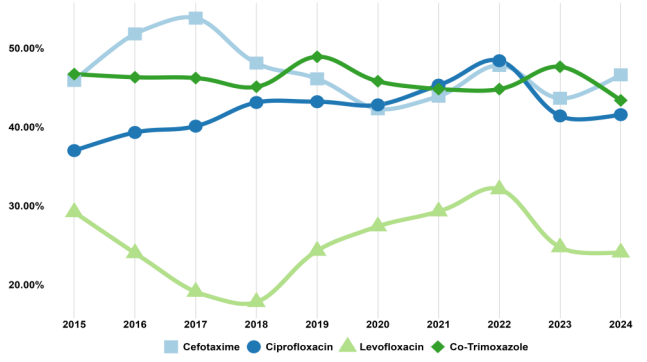


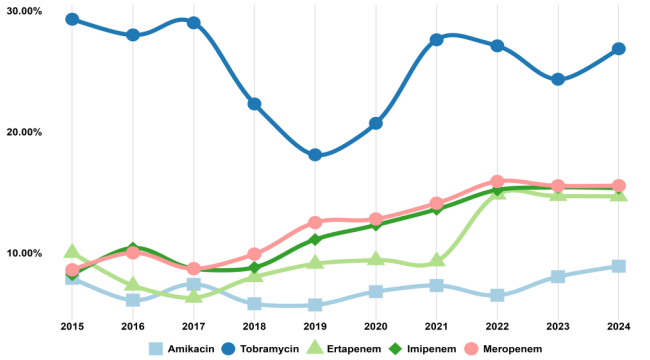
Figure 125. Proportion of *K. pneumoniae* lower respiratory tract infections with resistance to tested antibiotics DOH-ARSP, 2024



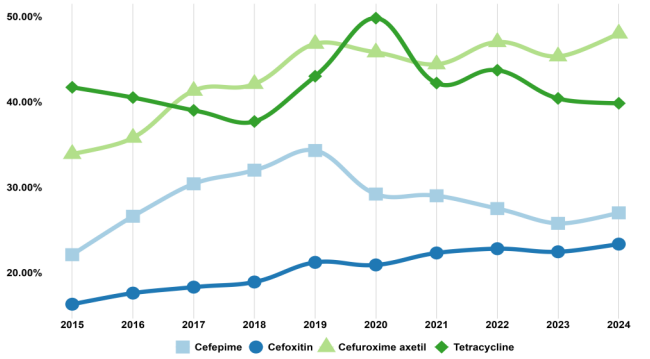
	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Gentamicin	20.5%	22.6%	21.6%	21.0%	23.0%	20.7%	19.5%	19.1%	17.4%	18.4%
Amoxicillin/Clavulanic Acid	31.7%	37.0%	35.4%	32.7%	37.2%	34.5%	35.7%	36.1%	34.8%	33.3%
Pip-Tazobactam	28.5%	31.8%	31.9%	32.5%	37.0%	35.0%	34.9%	36.1%	36.7%	45.9%
Ceftriaxone	35.3%	37.4%	44.2%	43.5%	46.7%	44.3%	43.8%	45.1%	43.2%	45.2%



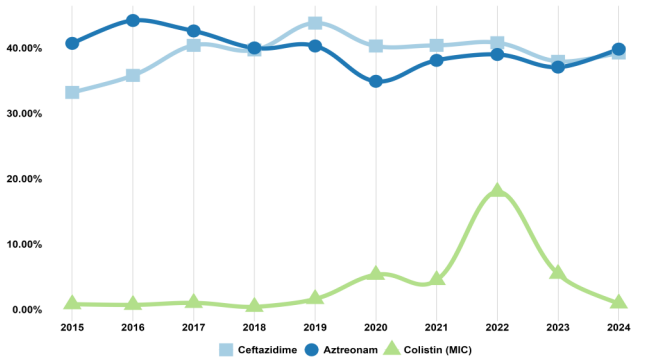
	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Cefotaxime	45.9%	51.8%	53.8%	48.1%	46.1%	42.3%	43.9%	47.8%	43.6%	46.6%
Ciprofloxacin	37.0%	39.3%	40.1%	43.1%	43.2%	42.8%	45.3%	48.4%	41.4%	41.6%
Levofloxacin	29.2%	24.0%	19.1%	17.8%	24.3%	27.4%	29.3%	32.1%	24.8%	24.1%
Co-Trimoxazole	46.7%	46.3%	46.2%	45.1%	48.9%	45.8%	44.8%	44.8%	47.6%	43.4%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Amikacin	7.9%	6.1%	7.4%	5.8%	5.7%	6.8%	7.3%	6.5%	8.0%	8.9%
Tobramycin	29.3%	28.0%	29.0%	22.3%	18.1%	20.7%	27.6%	27.1%	24.3%	26.9%
Ertapenem	10.0%	7.3%	6.3%	8.0%	9.1%	9.4%	9.3%	14.8%	14.7%	14.7%
Imipenem	8.2%	10.4%	8.7%	8.8%	11.1%	12.3%	13.6%	15.2%	15.4%	15.3%
Meropenem	8.6%	10.0%	8.7%	9.9%	12.5%	12.8%	14.1%	15.9%	15.5%	15.6%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Cefepime	22.1%	26.6%	30.4%	32.0%	34.3%	29.2%	29.0%	27.5%	25.8%	27.0%
Cefoxitin	16.3%	17.6%	18.3%	18.9%	21.2%	20.9%	22.3%	22.8%	22.4%	23.3%
Cefuroxime axetil	33.9%	35.8%	41.3%	42.1%	46.8%	45.8%	44.4%	47.0%	45.3%	48.0%
Tetracycline	41.7%	40.5%	39.0%	37.7%	43.0%	49.8%	42.2%	43.7%	40.4%	39.8%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Ceftazidime	33.2%	35.8%	40.4%	39.7%	43.8%	40.3%	40.4%	40.8%	38.0%	39.2%
Aztreonam	40.7%	44.2%	42.6%	40.0%	40.3%	34.9%	38.1%	39.0%	37.1%	39.8%
Colistin (MIC)	0.8%	0.7%	1.0%	0.4%	1.6%	5.3%	4.5%	18.0%	5.4%	0.9%

Figure 126. Yearly resistance rates of *K pneumoniae* respiratory infections, DOH-ARSP, 2024

Urinary Tract Infections

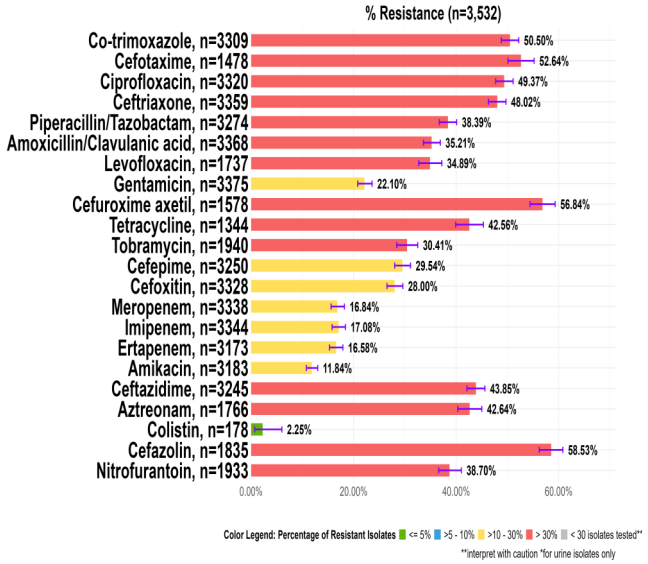
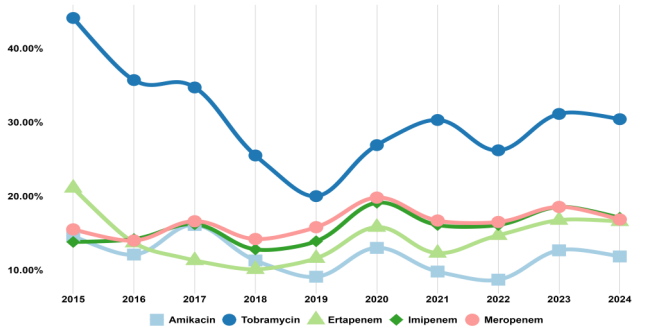
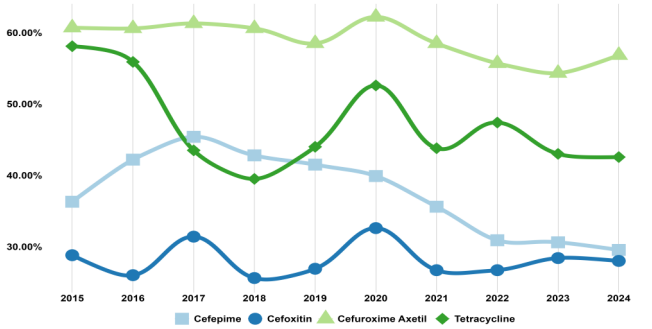


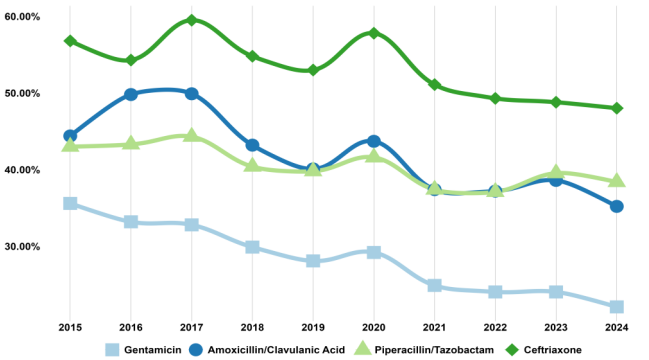
Figure 127. Proportion of *K. pneumoniae* urinary tract infections with resistance to tested antibiotics, DOH-ARSP, 2024



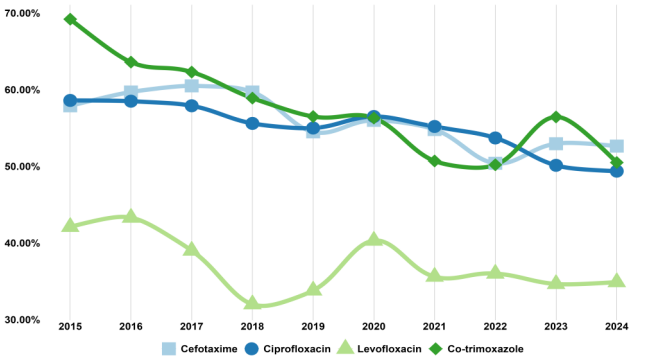
	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Amikacin	14.6%	12.1%	16.1%	11.3%	9.1%	13.0%	9.8%	8.7%	12.7%	11.8%
Tobramycin	44.1%	35.7%	34.7%	25.5%	20.0%	26.9%	30.3%	26.2%	31.1%	30.4%
Ertapenem	21.1%	13.7%	11.3%	10.1%	11.6%	15.8%	12.3%	14.7%	16.8%	16.6%
Imipenem	13.8%	14.2%	16.2%	12.8%	13.9%	19.1%	16.1%	16.1%	18.5%	17.1%
Meropenem	15.5%	14.0%	16.6%	14.2%	15.8%	19.8%	16.7%	16.5%	18.5%	16.8%



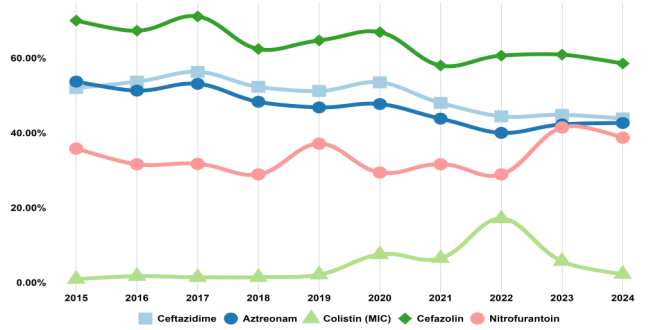
	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Cefepime	36.3%	42.2%	45.4%	42.8%	41.5%	39.9%	35.6%	30.9%	30.6%	29.5%
Cefoxitin	28.8%	26.0%	31.4%	25.6%	26.9%	32.6%	26.7%	26.7%	28.4%	28.0%
Cefuroxime Axetil	60.7%	60.6%	61.3%	60.6%	58.5%	62.2%	58.5%	55.7%	54.3%	56.8%
Tetracycline	58.1%	55.9%	43.5%	39.5%	44.0%	52.6%	43.8%	47.4%	43.0%	42.6%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Gentamicin	35.6%	33.2%	32.8%	29.9%	28.1%	29.2%	24.9%	24.1%	24.1%	22.1%
Amoxicillin/Clavulanic Acid	44.4%	49.8%	49.9%	43.2%	40.1%	43.7%	37.4%	37.2%	38.6%	35.2%
Piperacillin/Tazobactam	43.0%	43.3%	44.3%	40.4%	39.8%	41.6%	37.4%	37.1%	39.5%	38.4%
Ceftriaxone	56.8%	54.3%	59.5%	54.8%	53.0%	57.8%	51.1%	49.3%	48.8%	48.0%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Cefotaxime	57.9%	59.7%	60.5%	59.7%	54.5%	56.0%	54.8%	50.4%	53.0%	52.6%
Ciprofloxacin	58.6%	58.5%	57.9%	55.6%	55.0%	56.5%	55.2%	53.7%	50.1%	49.4%
Levofloxacin	42.1%	43.3%	39.0%	32.0%	33.8%	40.3%	35.6%	36.0%	34.6%	34.9%
Co-trimoxazole	69.2%	63.6%	62.3%	58.9%	56.5%	56.3%	50.7%	50.2%	56.4%	50.5%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Ceftazidime	52.0%	53.7%	56.3%	52.3%	51.2%	53.5%	48.0%	44.4%	44.8%	43.9%
Aztreonam	53.7%	51.3%	53.1%	48.3%	46.8%	47.7%	43.8%	40.0%	42.2%	42.6%
Colistin (MIC)	0.9%	1.7%	1.4%	1.4%	2.1%	7.5%	6.5%	17.1%	5.7%	2.2%
Cefazolin	70.0%	67.3%	71.1%	62.4%	64.7%	66.9%	58.0%	60.6%	60.9%	58.5%
Nitrofurantoin	35.8%	31.6%	31.7%	28.9%	37.1%	29.4%	31.6%	28.9%	41.4%	38.7%

Figure 128. Yearly resistance rates of *K. pneumoniae* urinary tract infections

Healthcare-associated Infections

Figure 129 shows the resistance rates of presumptive healthcare-acquired *K. pneumoniae* infections. Resistance rates to most antibiotics were more than 30%. Cefazolin resistance was highest at 68.06% followed by cefuroxime-axetil at 64.08% and cefotaxime (60.95%). Lowest resistance rates were observed for colistin at 2.92%.

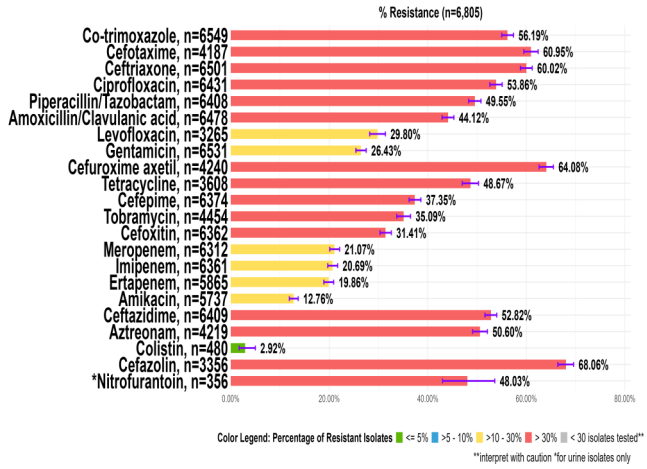


Figure 129. Proportion of presumptive healthcare-acquired *K. pneumoniae* infections with resistance to tested antibiotics, DOH-ARSP, 2024

Colistin - resistant

Figure 130 presents the resistance rates of colistin-resistant *K. pneumoniae* infections. Among these isolates, resistance to most antibiotics was high, ranging from 30% to 74%. Resistance rates were highest for cefazolin (74.15%), ceftriaxone (71.87%), tobramycin (55.18%), and gentamicin (32.93%). The lowest resistance rate was observed for amikacin at 18.90%.

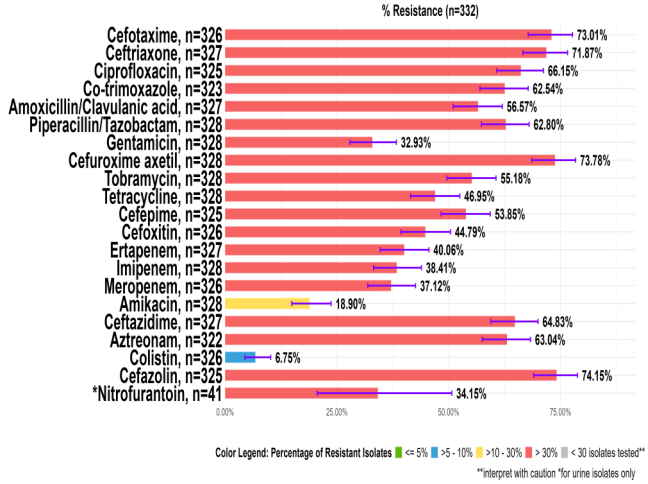


Figure 130. Proportion of all colistin-resistant *K. pneumoniae* infections, DOH-ARSP, 2024

Carbapenem - resistant

Figure 131 shows the resistance rates of carbapenem-resistant *K. pneumoniae* infections. Among these infections, resistance rates to most antibiotics were high ranging from 40-90%. Cefazolin resistance was at 97.82%, tobramycin at 78.37%, gentamicin 55.78% and ceftriaxone at 96.15%. Resistance rates to amikacin, tetracycline and colistin were 41.34%, 52.09% and 4.51% respectively.

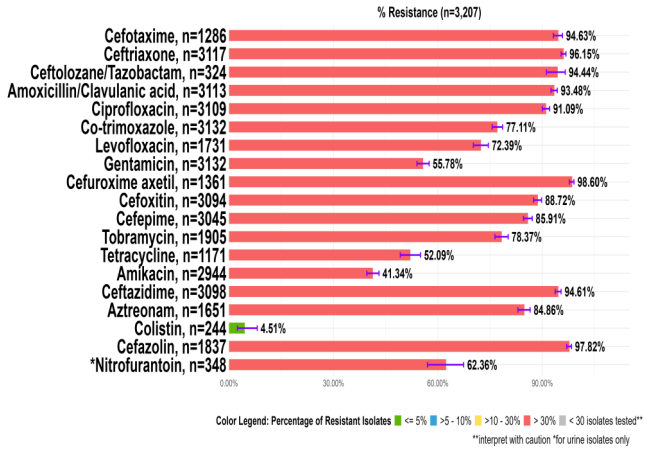


Figure 131. Proportion of all carbapenem-resistant *K. pneumoniae* infections, DOH-ARSP, 2024

From the subset of 2024 *K. pneumoniae* infections screened phenotypically for ESBL production (n= 5,528), positivity rate was at 30.99%. The yearly resistance rates of ESBL positive *K. pneumoniae* infections were observed to be fluctuating with a notable increase in rates in 2024. Multi-year analysis showed that the overall changes in ESBL rates for ten years was not statistically significant.

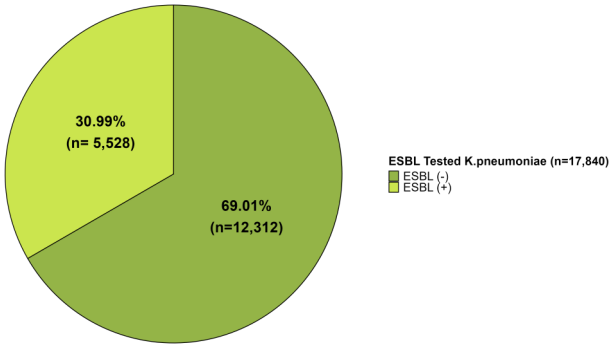
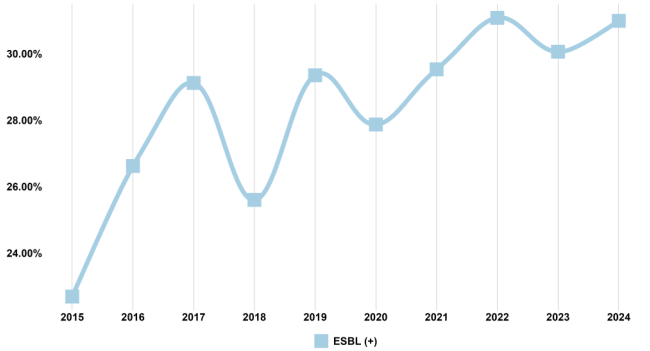


Figure 132. Percentage of ESBL positive and negative infections among tested *K. pneumoniae* infections.



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
ESBL (+)	22.7%	26.6%	29.1%	25.6%	29.4%	27.9%	29.5%	31.1%	31.8%	31.0%

Figure 133. Yearly resistance rates of ESBL positive *K. pneumoniae* infections, DOH-ARSP, 2015-2024

Introduction:

Colistin-resistant *Klebsiella pneumoniae* (Kpn) represents a serious clinical and public health threat due to its resistance to one of the few remaining antibiotics effective against multidrug-resistant Gram-negative bacteria. Colistin, often considered a last-resort treatment for infections caused by carbapenem-resistant organisms, is increasingly compromised by resistance mechanisms in Kpn. These are primarily driven by chromosomal mutations in genes such as *pmrAB, phoPQ,* and *mgrB*, which alter the bacterial outer membrane and reduce colistin binding [1]. Even more concerning is the emergence of plasmid-mediated *mcr-1* genes [2] which can spread horizontally between bacterial species, facilitating rapid dissemination across human, animal, and environmental reservoirs.

Clinically, colistin-resistant Kpn is frequently associated with severe and life-threatening infections, including bloodstream infections, ventilator-associated pneumonia (VAP) and catheter-associated urinary tract infections, particularly in hospitalized or immunocompromised patients [3,4]. These infections are associated with significantly higher morbidity and mortality due to limited treatment options.

This report highlights the surveillance of colistin-resistant Kpn in the Philippines and the genomic characterization of these emerging resistant isolates to identify resistance mechanisms and sequence types, and to monitor the dissemination of high-risk clones.

Results and Discussion

In 2024, a total of 16 colistin-resistant Kpn isolates were identified from various clinical specimens, including blood, sputum, tracheal aspirate, and urine. These resistant strains were associated with a wide spectrum of infections, such as community-acquired and hospital-acquired pneumonia (CAP/HAP), COVID-19-associated pneumonia, urinary tract infections, and cellulitis. Patients' age ranged from 2 months to 75 years, representing both the pediatric and adult populations. Most infections (56.3%) were hospital-acquired, while 43.8% were community-acquired.

Table 22. Distribution summary of clinical specimens

Accession No.	Age	Sex	Specimen Type	Infection Type	Ward
24ARS-BRT0006	64	f	sp	CAI	mwd
24ARS-BRT0009	45	m	wd	CAI	swd
24ARS-BRT0011	69	m	bl	CAI	iso
24ARS-CRH0030	17	m	sp	HAI	ereid 1
24ARS-DMC0098	2m	f	ur	HAI	pcu2
24ARS-DMC0464	75	m	bl	HAI	pw1
24ARS-EVR0037	27	m	sp	HAI	swd
24ARS-EVR0161	1	f	bl	HAI	pwd
24ARS-GMH0029	73	f	ta	CAI	mcu
24ARS-JLM0018	44	m	ab	HAI	scu
24ARS-JLM0020	69	m	sp	HAI	mwd
24ARS-NKI0050	71	f	as	CAI	opd
24ARS-NKI0051	58	f	ur	CAI	opd
24ARS-SLH0060	13	f	ta	CAI	pav 3
24ARS-STU0024	74	m	ur	CAI	op
24ARS-STU0104	59	f	bl	CAI	mwd

AMR Phenotypic Profile

The colistin resistant Kpn isolates showed high resistance across multiple antibiotic classes, (Table 23) with resistance rates exceeding 80% for most cephalosporins, folate pathway inhibitors, and monobactams. Interestingly, 43.8% of these colistin-resistant isolates remained susceptible to carbapenems, indicating that colistin resistance does not necessarily coincide with carbapenem resistance. There is relatively lower resistance to tetracyclines (31.3%) and aminoglycosides (43.8%) which highlights the limited therapeutic options available for treating infections caused by these multidrug-resistant strains.

Table 23. Resistance rates of colistin-resistant *Klebsiella pneumoniae*

Antibiotic Class	% Resistant
Aminoglycosides	43.8%
Beta-lactam + Inhibitor	62.5%
Cephalosporin I	81.3%
Cephalosporin III	81.3%
Cephalosporin IV	62.5%
Cephamycin	62.5%
Carbapenems	56.3%
Folate Pathway Inhibitors	81.3%
Monobactams	87.5%
Fluoroquinolones	75.0%
Tetracyclines	31.3%

Molecular characterization of Colistin-Resistant Kpn

In silico species identification revealed that most isolates were *Klebsiella pneumoniae* (9, 56.3%), followed by *K. quasipneumoniae* subsp. *similipneumoniae* (5,31.3%), and *K. variicola* subsp. *variicola* (2, 12.5%). The isolates were genetically diverse, with no dominant clone identified. Sequence types (STs) included ST147, ST15, ST273, ST6403, and others. This genetic heterogeneity suggests that colistin resistance likely emerged independently across multiple lineages rather than from the clonal expansion of a single outbreak strain.

Among the 16 colistin-resistant Kpn isolates identified in 2024, only three (3) harbored mutations in the *pmrB* gene, a key determinant in outer membrane modification reducing colistin susceptibility. Notably, none of the 2024 isolates carried the plasmid-mediated *mcr-1* gene, a major driver of transmissible colistin resistance. Carbapenem resistance among colistin resistant Kpn was primarily driven by the presence of *blaNDM-1*, *blaNDM-5* and *blaNDM-7* genes while cephalosporin resistance was associated with extended-spectrum β-lactamases (ESBLs), particularly *blaCTX-M-15*. The *aac(6')-Ib-cr5* gene, which mediates resistance to both aminoglycosides and fluoroquinolones, was also detected among the isolates. A summary of the isolates including their sequence types (MLST) and associated resistance genes, is presented in Table 24.

Table 24. MLST and Resistance Gene Profiles of 2024 Colistin-Resistant *Klebsiella* Isolates

Accession No.	ST	B-lactam	Ceph	Carba	Colistin	Quinolone	Agly	Agly Quinolone	Sulfona-mide	Trim	Tet
24ARS_BRT0006	ST273	blaSHV-11 blaTEM-1	blaCTX-M-15 blaOXA-1 blaOXA-10	blaNDM-1	pmrB_R256G	gyrA_S83I parC_S80I qnrB1, qnrS1 qnrVC1	aac(6')-Ib3 aac(3)-Iie		sul1	dfrA1 dfrA14 dfrA15	tet(A)
24ARS-BRT0009	ST6403	blaOKP-B	blaCTX-M-15	blaNDM-7		qnrS1			sul1, sul2	dfrA12	tet(A)
24ARS-BRT0011	ST147	blaSHV-11	blaCTX-M-15 blaOXA-10	blaNDM-1	pmrB_R256G	gyrA_D87A gyrA_S83Y parC_S80I qnrS1			sul1 sul2	dfrA12 dfrA14	tet(A)
24ARS_CRH0030	ST1887-2LV	blaOKP-B	blaCTX-M-15			qnrS1			sul1,sul2	dfrA12	tet(A)
24ARS_DMC0098	ST17	blaSHV-11 blaTEM-1				oqxA oqxB25		aac(6')-Ib-cr5	sul1	dfrA27	tet(A)
24ARS-DMC0464	ST285-2LV	blaLEN-11									
24ARS-EVR0037	ST1224	blaOKP-B-21	blaDHA-1			oqxA oqxB qnrB4			sul1		
24ARS-EVR0161	ST1887-2LV	blaOKP-B									
24ARS-GMH0029	ST147	blaSHV-11	blaCTX-M-15 blaOXA-1	blaNDM-7	pmrB_R256G	gyrA_S83I parC_S80I qnrB1	aac(3)-IIe	aac(6')-Ib-cr5	sul1	dfrA14 dfrA27	tet(A)
24ARS-JLM0018	ST39	blaSHV-11	blaCTX-M-15 blaOXA-1	blaNDM-5		qnrB6 qnrS1	aac(3)-IId	aac(6')-Ib-cr5	sul1 sul2	dfrA12 dfrA27 dfrA7	tet(A)
24ARS-JLM0020	ST6403	blaOKP-B	blaCTX-M-15			qnrS1			sul1,sul2	dfrA12	tet(A)
24ARS-NKI0050	ST2050	blaSHV-1									
24ARS-NKI0051	ST15	blaSHV-28	blaCTX-M-15 blaOXA-1	blaNDM-5		gyrA_D87A gyrA_S83F parC_S80I qnrB1, qnrS1	aac(3)-IIe	aac(6')-Ib-cr5	sul1 sul2	dfrA12 dfrA51	
24ARS-SLH0060	ST327	blaSHV-1	blaOXA-10	blaNDM-1		qnrS1			sul1, sul2	dfrA14	
24ARS-STU0024	ST39	blaSHV-11	blaCTX-M-15			qnrS1			sul1, sul2	dfrA7	
24ARS-STU0104	ST318-2LV	blaLEN-17									

Colistin resistance 2022-2024

From 2022 to 2024, a total of 59 colistin-resistant Kpn isolates were sequenced across 3 surveillance years. One of the most prominent clones identified was ST147, a well-known high-risk clone associated with multidrug resistance and hospital outbreaks. ST147 accounted for 6 isolates across all three years, making it the most consistently detected high-risk clone over the surveillance period. The phylogenetic relationships and distribution of STs are further illustrated in **Figure 134**. In the past years, colistin resistance was primarily mediated by mutations in chromosomal regulators (*pmrB, mgrB*) detected across multiple surveillance sites indicating widespread distribution rather than regional clustering. Only one case of *mcr-1* has been detected to date, suggesting limited plasmid-mediated resistance locally.

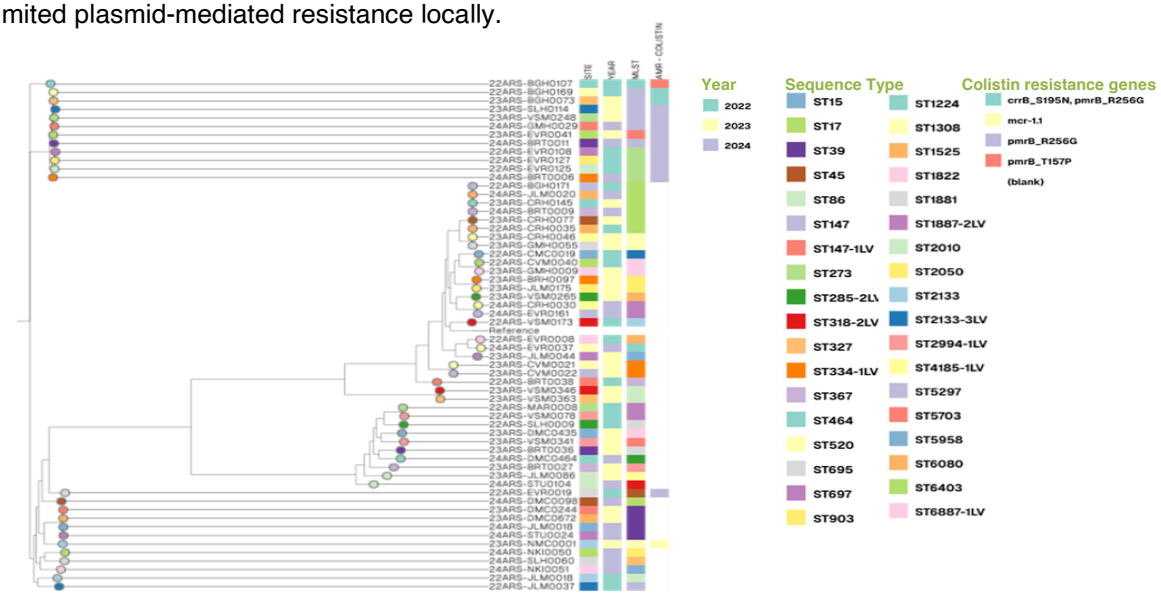


Figure 134. Phylogenetic tree and metadata of colistin-resistant Kpn isolates (2022–2024) visualized using Microreact

Discussion

This surveillance highlights the growing threat of colistin-resistant Kpn species in the Philippines. The isolates were genetically diverse, with no dominant clone, suggesting that resistance likely emerged independently rather than from a single outbreak. Despite colistin resistance, 43.8% of isolates remained susceptible to carbapenems, indicating that colistin resistance does not necessarily coincide with carbapenem resistance. High resistance rates were observed for cephalosporins, monobactams, and folate pathway inhibitors, limiting treatment options. The detection of *pmrB* mutations among 2024 Kpn isolates aligns with earlier findings that chromosomal mechanisms remain the dominant drivers of colistin resistance. Although plasmid-mediated resistance mechanisms like *mcr-1* remain rare, their potential for horizontal gene transfer warrants sustained surveillance efforts. Similarly, the repeated identification of ST147 across multiple sites and years signals its capacity for persistence and possible spread, emphasizing its importance as a high-risk clone. While no clear geographic or temporal clustering was observed overall, the broad distribution of resistance mechanisms reinforces the need for continued genomic surveillance.

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Introduction:

Klebsiella pneumoniae (Kpn) is a clinically significant opportunistic pathogen frequently implicated in severe healthcare-associated infections. Among these, Kpn bloodstream infections are particularly concerning due to their high morbidity and mortality rates [1]. These infections can lead to severe sepsis and septic shock, especially in vulnerable populations such as the elderly, immunocompromised individuals, and patients with comorbidities like diabetes or chronic liver disease [2]. The emergence of multidrug-resistant (MDR) and extensively drug-resistant (XDR) strains, including carbapenem-resistant Kpn (CRKP), has further complicated treatment options and outcomes [3].

Over the past three years, ARSP data showed an upward trend in carbapenem resistance among Kpn bloodstream infections. In 2021, carbapenem resistance rates were reported at 20.3% for meropenem and 19.4% for imipenem. The following year, 2022, saw an increase in resistance rates to 22% for meropenem and 21.9% for imipenem. By 2023, resistance rates slightly decreased to 20.85% for meropenem and 20.48% for imipenem [4]. Despite the decline, the resistance levels remain alarmingly high, underscoring the ongoing threat posed by CRKP strains.

Given these trends, genomic surveillance is crucial to monitor the evolving threat of Kpn and its increasing resistance to critical antibiotics like carbapenems. Whole genome sequencing (WGS) offers valuable insights into the genetic mechanisms behind resistance, allowing for the detection of key resistance genes [5].

Patient Demographics and Clinical Characteristics

A total of 56 Kpn bloodstream infections were reported in 2024. Males accounted for a greater proportion of cases (58.9%) compared to females (41.1%). Patient ages ranged from 2 days to 82 years, with the highest incidence among adults aged 20–60 years. More than half were community-acquired (51.7%) while hospital-acquired infections accounted for 48.2%.

Phenotypic Resistance Patterns

Among the 56 Kpn bloodstream infections, 48.2% were carbapenem resistant while 51.7% remained susceptible. All CRKP isolates demonstrated resistance to third-generation cephalosporins (cefotaxime, ceftazidime, ceftriaxone), as well as to β-lactam/β-lactamase inhibitor combinations (amoxicillin-clavulanic acid and piperacillin-tazobactam). Additionally, high resistance rates were observed for ciprofloxacin (96.3%) and aminoglycosides, specifically gentamicin (88.9%) and tobramycin (88.9%). Lower resistance rates were observed for amikacin (40.7%) and trimethoprim-sulfamethoxazole (44.4%). These data are summarized in Table 25.

Table 25. Resistance rates among carbapenem-resistant Kpn bloodstream isolates, 2024.

Antibiotic Class	% Resistant
Carbapenems	
Meropenem	100
Imipenem	100
Cephalosporins	
Cefotaxime	100
Ceftazidime	100
Ceftriaxone	100
β-lactam/β-lactamase inhibitors	
Amoxicillin-clavulanic acid	100
Piperacillin-tazobactam	100
Fluoroquinolone	
Ciprofloxacin	96.3
Aminoglycosides	
Gentamicin	88.9
Tobramycin	88.9
Amikacin	40.7
Trimethoprim-sulfamethoxazole	44.4

Molecular characterization of Kpn from bloodstream infections

In silico species identification showed that most isolates were *Klebsiella pneumoniae* subsp. *pneumoniae* (87.5%). A smaller proportion were identified as *Klebsiella variicola* subsp. *variicola* (5.4%), *Klebsiella quasipneumoniae* subsp. *quasipneumoniae* (3.6%), and *Klebsiella quasipneumoniae* subsp. *similipneumoniae* (3.6%).

Multilocus sequence typing (MLST) revealed significant genetic diversity, with 37 unique sequence types (STs) identified. Among the carbapenem-resistant *K. pneumoniae* (CRKP) bloodstream isolates, high-risk international clones such as ST147 and ST39 were predominant. ST147 was found in six isolates across four surveillance sites (BRH, GMH, SLH, STU), while ST39 was detected in seven isolates from MAR.

Of the 56 confirmed *K. pneumoniae* bloodstream infections, 27 isolates (48.2%) were found to harbor carbapenemase genes. Among these, *blaNDM-5* and *blaNDM-1* were the most frequently detected, each present in 10 of the 27 isolates (37.0%). *blaNDM-7* was found in six isolates (22.2%), while *blaKPC-2* was least prevalent, identified in a single isolate (3.7%).

Serotyping

In silico capsular typing revealed diversity among the 27 CRKP isolates, identifying 17 unique K-locus (KL) types. The most frequently detected KL type was KL23, found in 5 isolates (19.2%), followed by KL1, observed in 4 isolates (15.4%). KL31 was present in 2 isolates, while the remaining KL types were each detected in a single isolate. Analysis of the O-locus types showed a predominance of the O1/O2 antigen complex, which was detected in 19 of 27 isolates (73.1%).

Phylogenetic analysis identified a tight cluster of 8 CRKP isolates from MAR, all belonging to ST39 and harboring the *blaNDM-5* (**Figure 135**). Five of these were recovered from neonates (aged 3–12 days) in the SNCU and ICUC, all classified as nosocomial infections and collected within a three-week period, indicating a likely clonal transmission.

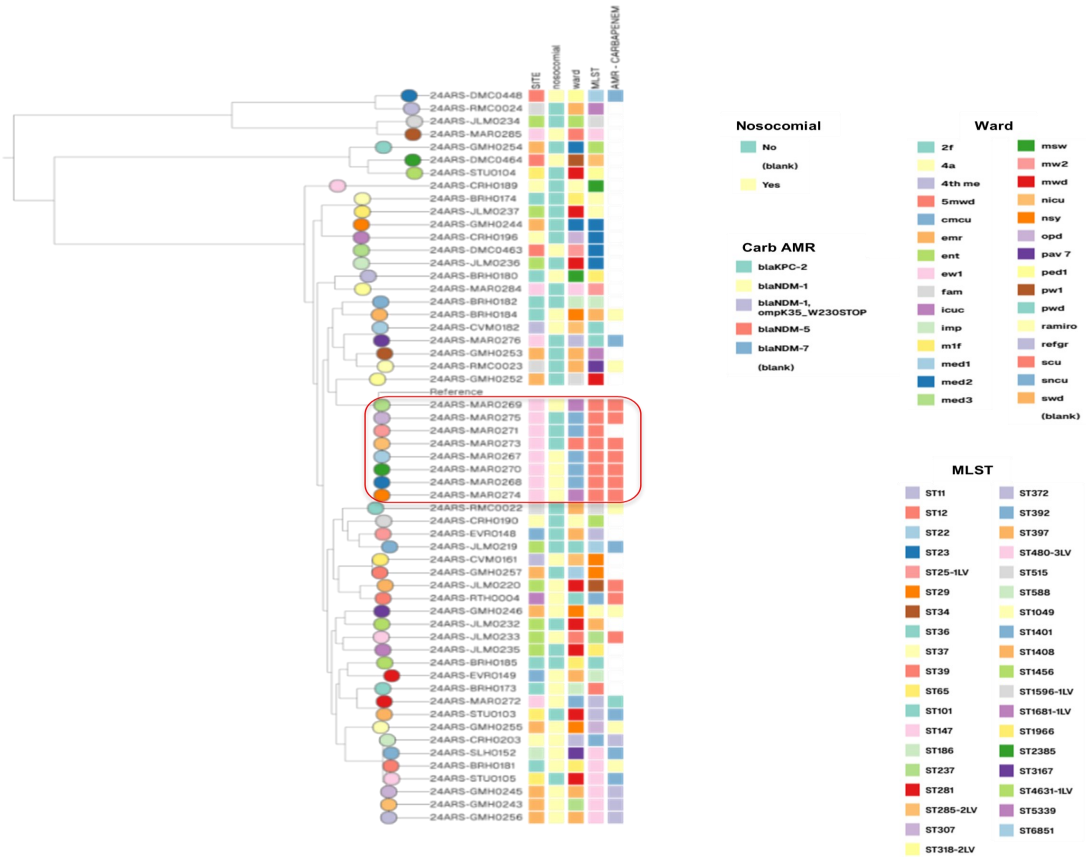

Figure 135. Phylogenetic tree and metadata of 2024 CRKP isolates from bloodstream infections

Discussion

The findings from the 2024 structured survey highlight the complex and evolving landscape of Kpn bloodstream infections in the Philippines, with a significant clinical burden posed by CRKP. The phenotypic resistance data confirm that all CRKP isolates exhibit multidrug resistance (MDR), making treatment increasingly challenging.

The molecular epidemiology of CRKP isolates reflects a genetically diverse population, marked by the predominance of high-risk international clones, ST147, and ST39, which are lineages previously linked to healthcare-associated outbreaks and resistance. These clones are notorious for harboring carbapenemase genes and spreading efficiently within and between healthcare facilities. The presence of several other STs suggests simultaneous introduction and evolution of multiple lineages, further complicating infection control efforts.

The detection of multiple carbapenemase genes, particularly *blaNDM-1, blaNDM-5*, and *blaNDM-7*, which together comprise the vast majority (over 90%) of CRKP isolates, has critical treatment implications. NDM variants are metallo-β-lactamases that confer resistance to almost all β-lactam antibiotics, and they are not inhibited by current β-lactamase inhibitors such as avibactam or vaborbactam. In contrast, *KPC-2*, though rare in this cohort (3.7%), remains clinically significant due to its susceptibility to newer inhibitor combinations like ceftazidime-avibactam, meropenem-vaborbactam, and imipenem-relebactam.

In silico capsular typing further supports the genetic heterogeneity of CRKP in the country. This wide distribution of capsular types suggests parallel evolution or multiple introductions of diverse CRKP strains across different healthcare facilities. The capsular diversity has important implications for pathogenesis, immune system evasion, and potential vaccine design.

Overall, this study highlights that CRKP bloodstream infections in the Philippines are driven by a diverse set of high-risk clones, carrying transmissible resistance genes, particularly NDM types, and a wide array of capsular types. The observed diversity in both sequence types and capsular types (KL-types) suggests multiple introductions or the emergence of various high-risk clones within the healthcare setting. Moreover, insights into sequence and KL-type distribution are critical for informing future therapeutic strategies and the design of effective vaccines against circulating high-risk Kpn clones.

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Pseudomonas aeruginosa

A total of **10,735** *Pseudomonas aeruginosa* infections were reported for 2024.

10,735 infections

Large contributors of *P. aeruginosa* data were PGH (12.29%), VSM (10.22%) and DMC (7.85%). Based on island group distribution, surveillance sites from Luzon contributed more than half (59.77%) of the infections, 22.22% from Visayas, and 18.02% from Mindanao.

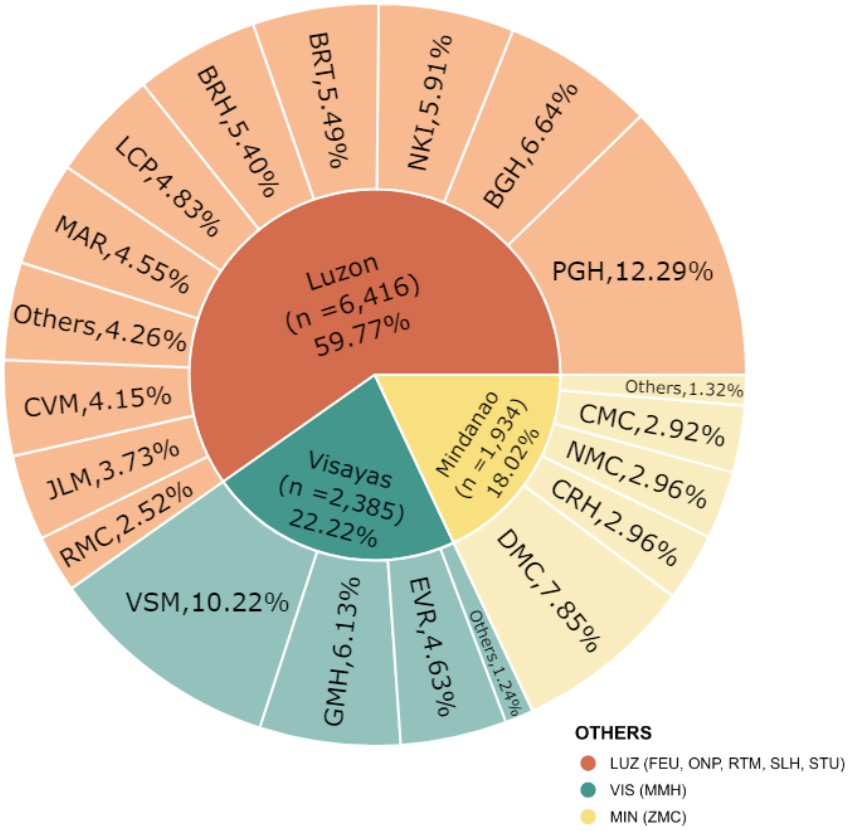


Figure 136. Distribution of *Pseudomonas aeruginosa* infections, DOH-ARSP, 2024 (n= 10,735)

Most (62.79%) *P. aeruginosa* infections were obtained from patients aged 20-64 years old and from male patients (56.65%). A large proportion of the infections were from respiratory (46.57%) and wound (18.96%) specimens. Additionally, more than half (61.0%) were community-acquired.

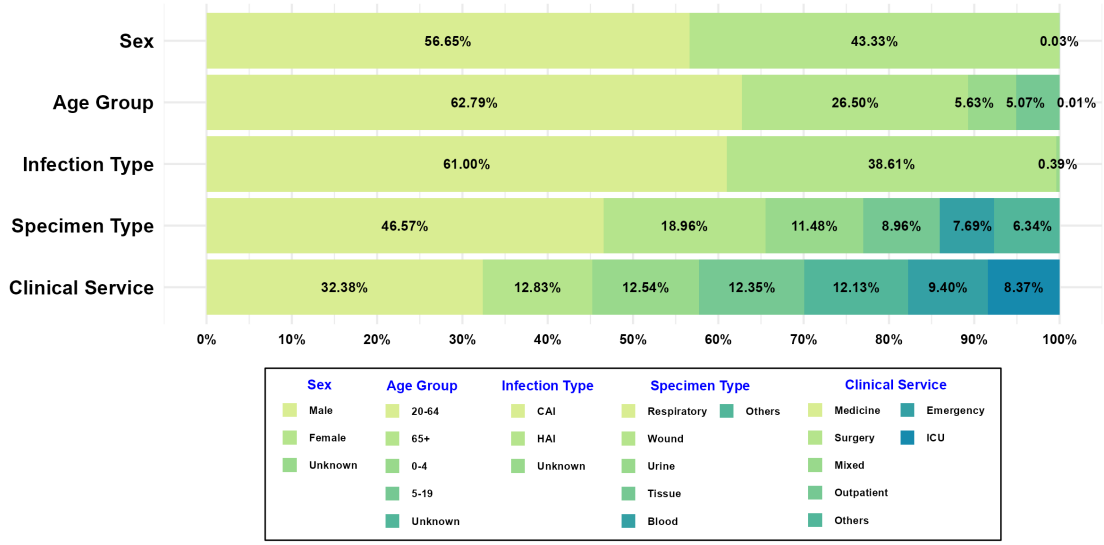


Figure 137. Patient characteristics in *P. aeruginosa* infections, DOH-ARSP, 2024 (n=10,735)

The percent positive of respiratory infections caused by *P. aeruginosa* for FEU, PGH, STU and VSM were 26.09%, 22.75%, 19.53% and 15.86% respectively. The percent positive of respiratory infections due to *P. aeruginosa* for ONP, BGH, RMC, MAR, CRH, EVR, BRT, GMH, LCP, and CVM were ≥ 10%. While the percent positive of the remaining surveillance sites ranged from 1.46-9.98%, and no *P. aeruginosa* causing respiratory infection was observed for RTH and ZPH.

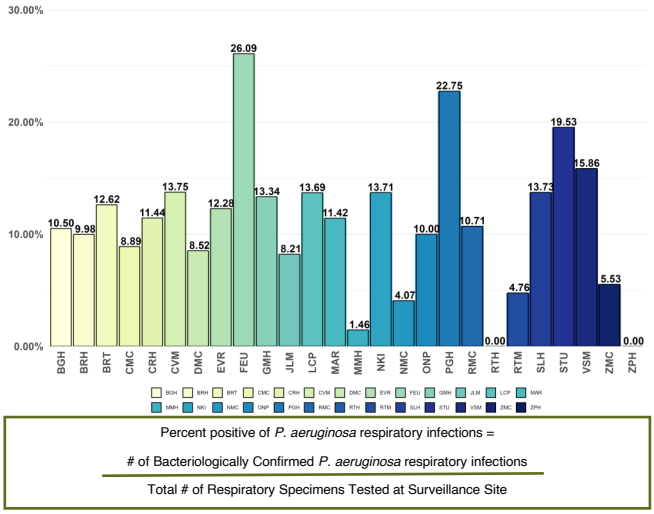


Figure 138. Percent positive of *P. aeruginosa* respiratory infections among all tested respiratory specimens per surveillance site, DOH-ARSP, 2024

All types of Infections

Figure 139 shows the cumulative resistance rates of all *P. aeruginosa* infections in 2024. The resistance to beta-lactams are as follows: ceftazidime was at 15.37%, cefepime at 12.36%, aztreonam at 13.96% and piperacillin-tazobactam at 18.78%. For fluoroquinolone resistance, levofloxacin and ciprofloxacin resistance were at 10.33% and 14.08% respectively. There were significant increases in the resistance rates of the following antibiotics from 2023 to 2024: ceftazidime (p=0.0000), ciprofloxacin (p=0.0002), levofloxacin (p=0.0012), imipenem (p=0.0153) and meropenem (p=0.0113), while significant decreases were noted for piperacillin-tazobactam (p=0.0293) and tobramycin (p=0.0000).

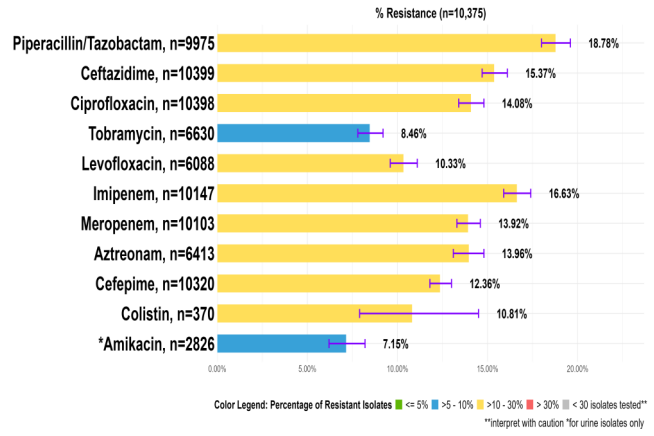
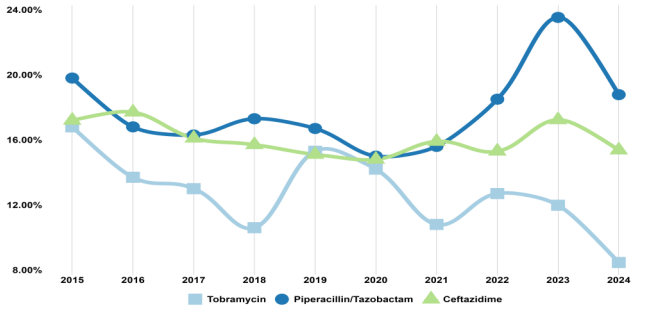
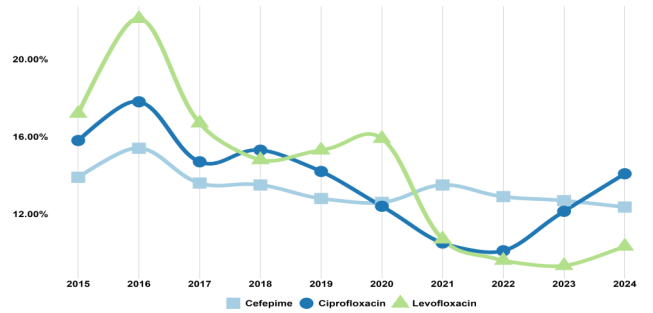


Figure 139. Proportion of all *P. aeruginosa* infections with resistance to tested antibiotics, DOH-ARSP, 2024

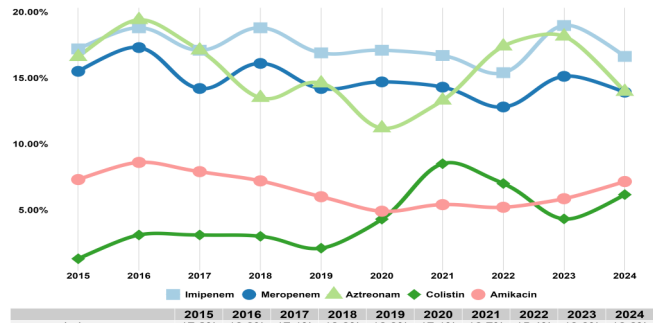
Figure 140 shows the yearly resistance rates of all *P. aeruginosa* infections. The multi-year analysis showed an increasing trend for ciprofloxacin (p=0.0000) and amikacin (p=0.0000) while a decreasing trend was observed for cefepime (p=0.000) and tobramycin (p=0.000).



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Tobramycin	16.8%	13.7%	13.0%	10.6%	15.3%	14.2%	10.8%	12.7%	12.0%	8.5%
Piperacillin/Tazobactam	19.8%	16.8%	16.3%	17.3%	16.7%	15.0%	15.6%	18.5%	23.5%	18.8%
Ceftazidime	17.2%	17.7%	16.1%	15.7%	15.1%	14.8%	15.9%	15.3%	17.2%	15.4%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Cefepime	13.9%	15.4%	13.6%	13.5%	12.8%	12.6%	13.5%	12.9%	12.7%	12.4%
Ciprofloxacin	15.8%	17.8%	14.7%	15.3%	14.2%	12.4%	10.5%	10.1%	12.1%	14.1%
Levofloxacin	17.2%	22.1%	16.7%	14.8%	15.3%	15.9%	10.7%	9.6%	9.3%	10.3%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Imipenem	17.2%	18.8%	17.1%	18.8%	16.9%	17.1%	16.7%	15.4%	19.0%	16.6%
Meropenem	15.5%	17.3%	14.2%	16.1%	14.2%	14.7%	14.3%	12.8%	15.1%	13.9%
Aztreonam	16.6%	19.4%	17.1%	13.5%	14.6%	11.2%	13.3%	17.4%	18.1%	14.0%
Colistin	1.3%	3.1%	3.1%	3.0%	2.1%	4.3%	8.5%	7.0%	4.3%	6.2%
Amikacin	7.3%	8.6%	7.9%	7.2%	6.0%	4.9%	5.4%	5.2%	5.8%	7.2%

Figure 140. Yearly resistance rates of all *P. aeruginosa* infections, DOH-ARSP, 2024

Bloodstream Infections

Figure 141 shows the resistance rates of *P. aeruginosa* bloodstream infections. Resistance to ceftazidime was at 11.54%, cefepime at 10.18%, aztreonam at 13.24% and piperacillin-tazobactam at 13.76%. Resistance to fluoroquinolone were as follows: ciprofloxacin at 10.08% and levofloxacin at 5.02%. Resistance to carbapenems are as follows: meropenem at 10.92% and imipenem at 13.87%. Compared to the cumulative resistance rates across all infection types, bloodstream infections caused by *P. aeruginosa* have relatively lower resistance to all tested antibiotics.

Resistance rates for *P. aeruginosa* lower respiratory infections and presumptive health care associated infections are shown on **Figures 144,** and **145**, respectively.

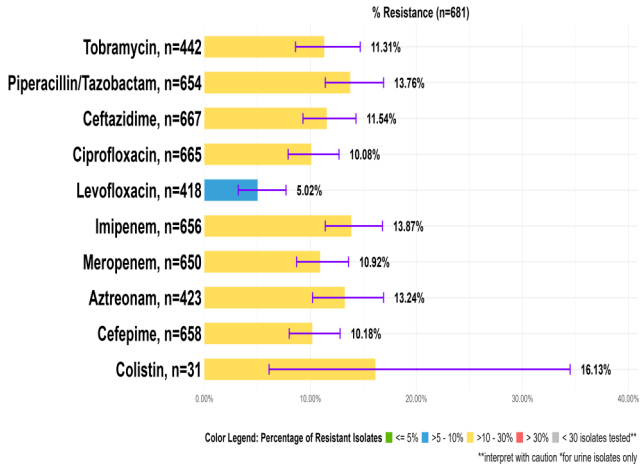
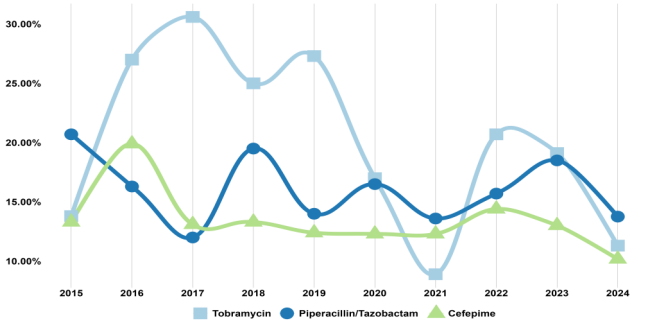
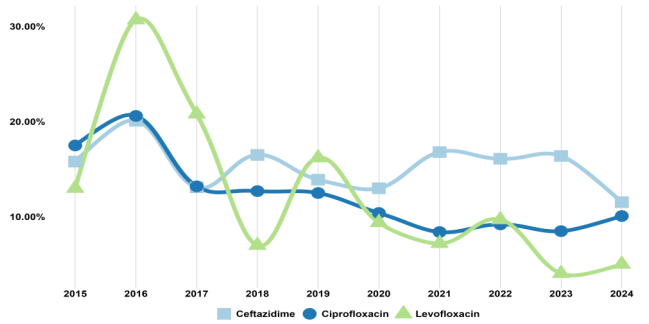


Figure 141. Proportion of *P. aeruginosa* bloodstream infections with resistance to tested antibiotics, DOH-ARSP, 2024

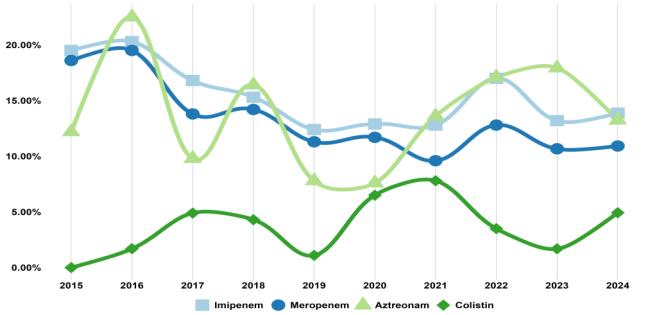
Figure 142 shows the yearly resistance rates of *P. aeruginosa* bloodstream infections. The multi-year analyses for these subset likewise showed statistically significant increasing trends for ciprofloxacin (p=0.0000) and amikacin (p=0.0000) while a decreasing trend was observed for cefepime (p=0.000) and tobramycin (p=0.000).



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Tobramycin	13.8%	27.0%	30.6%	25.0%	27.3%	17.0%	8.9%	20.7%	19.1%	11.3%
Piperacillin/Tazobactam	20.7%	16.3%	12.0%	19.5%	14.0%	16.5%	13.6%	15.7%	18.5%	13.8%
Cefepime	13.3%	19.9%	13.1%	13.3%	12.4%	12.3%	12.3%	14.4%	13.0%	10.2%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Ceftazidime	15.8%	20.1%	13.1%	16.5%	13.9%	13.0%	16.8%	16.1%	16.4%	11.5%
Ciprofloxacin	17.5%	20.6%	13.2%	12.7%	12.5%	10.4%	8.4%	9.2%	8.5%	10.1%
Levofloxacin	13.0%	30.7%	20.8%	7.0%	16.2%	9.4%	7.2%	9.7%	4.0%	5.0%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Imipenem	19.5%	20.3%	16.8%	15.3%	12.4%	12.9%	12.8%	17.0%	13.2%	13.9%
Meropenem	18.6%	19.5%	13.8%	14.2%	11.3%	11.7%	9.6%	12.8%	10.7%	10.9%
Aztreonam	12.2%	22.5%	9.8%	16.4%	7.8%	7.6%	13.6%	17.1%	17.9%	13.2%
Colistin	0.0%	1.7%	4.9%	4.3%	1.1%	6.5%	7.8%	3.5%	1.7%	4.9%

Figure 142. Yearly resistance rates of *P. aeruginosa* bloodstream infections, DOH-ARSP, 2024

Lower Respiratory Tract Infections

Figure 143 shows the resistance rates of *P. aeruginosa* lower respiratory infections. Most of the resistance rates were below 30%. Resistance to ceftazidime and cefepime were 23.67% and 20.32% respectively. Resistance to ciprofloxacin and levofloxacin were 21.59% and 17.06% respectively. Colistin resistance was at 5.98%.

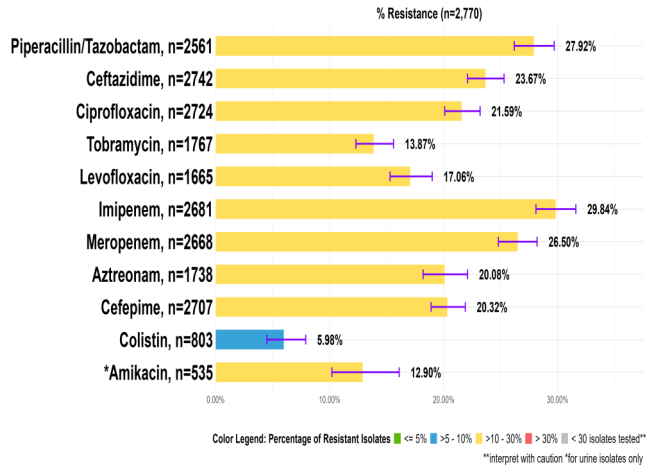


Figure 143. Proportion of *P. aeruginosa* lower respiratory tract infections with resistance to tested antibiotics, DOH-ARSP, 2024

Healthcare-associated Infections

The resistance rates of presumptive healthcare-associated *P. aeruginosa* infections are shown in **Figure 144**. Resistance to ceftazidime was at 22.83%, cefepime at 17.87%, aztreonam at 19.07%. Resistance to fluoroquinolones were as follows: ciprofloxacin (18.23%) and levofloxacin (13.47%). Carbapenem resistance were as follows: meropenem (20.15%) and imipenem (21.89%). Low resistance rates among HAI were seen for imipenem/ relebactam (6.77%) and ceftolozane/ tazobactam (9.63%)

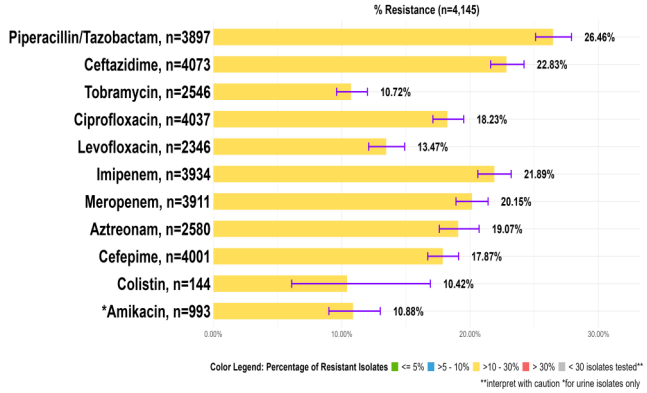


Figure 144. Proportion of *P. aeruginosa* isolates from presumptive healthcare-associated infections with resistance to tested antibiotics, DOH-ARSP, 2024

Emerging Resistance

Pseudomonas aeruginosa is a significant opportunistic pathogen, particularly in healthcare settings, and the emergence of colistin-resistant strains poses a critical public health challenge. While the global prevalence of colistin-resistant P. aeruginosa is generally below 10%, it has been steadily increasing, notably from 2% in 2006 -2010 to 5% in 2020-2023, with higher rates observed in immunocompromised patients like those with cystic fibrosis.[1] This resistance, often acquired through chromosomal mutations or occasionally plasmid-mediated mcr genes, severely limits treatment options for multi-drug resistant (MDR) and extensively drug-resistant (XDR) infections. Consequently, colistin-resistant *P. aeruginosa* infections are associated with higher rates of treatment failure, prolonged hospital stays, and increased mortality, underscoring the urgent need for stringent infection control and antimicrobial stewardship.

In 2024, a total of 38 colistin-resistant *P. aeruginosa* isolates from 14 surveillance sites were molecularly characterized. Most (68.42%) of the isolates were from patients aged 20-64 years old followed by the ≥65 age group (n=9), 5-19 (n=2) and 0-4 (n=1) years old. More than half (n=22, 57.89%) of the isolates were from male patients and were mostly from respiratory samples: tracheal aspirate (n= 17, 44.74%) and sputum (n=5, 13.16%). More isolates were from presumptive nosocomial infections (n=16, 42.10%).

Table 26. Patient demographics and clinical characteristics of colistin-resistant *P. aeruginosa* isolates

Antibiotic	No. of Resistant Isolates	Percentage
Tobramycin	9	23.68
Piperacillin-tazobactam	18	47.36
Ceftazidime	16	42.1
Cefepime	14	36.84
Aztreonam	15	39.47
Imipenem	19	50
Meropenem	19	50
Ciprofloxacin	14	36.84
Levofloxacin	17	44.74

The colistin-resistant *P. aeruginosa* isolates showed resistance to nine other antibiotics from seven antibiotic classes. As seen in **Table 26,** half of these isolates were likewise resistant to carbapenem antibiotics, while resistance to piperacillin-tazobactam, levofloxacin, ceftazidime and aztreonam were 47.36%, 44.74%, 42.1% and 39.47% respectively. Resistance to cefepime and ciprofloxacin were both at 36.84%, while resistance to tobramycin was relatively lower at 23.68%. It can be observed that many of the colistin-resistant *P. aeruginosa* were still susceptible to multiple antibiotics. Notably, there were n=14 colistin-resistant isolates that showed susceptibility to all other tested antibiotics.

A total of 27 sequence types (ST) were noted among the colistin-resistant isolates, and ST155 (n=3,7.89%) and ST277 (n=3, 7.89%) were the dominating sequence types followed by ST155 (n=2, 5.26%). The top three serotypes among these isolates were serotypes O11 (n=12, 31.58%), O5 (n=8, 21.05%) and O6 (n=7, 18.42%). Colistin resistance among *P. aeruginosa* isolates was primarily mediated by the specific mutation in *pmrB* gene including *pmrB_v15I* (n=10, 26.31%), *pmrB_H340R* (n=1, 2.63%) and *pmrB_T343A* (n=1, 2.63%). The most common carbapenem resistance mechanism observed was the mutation in the *oprD* gene [2] (*oprD*_v359L) which

was detected in 8 isolates, which are found to be susceptible to both imipenem and meropenem. All isolates harbor beta-lactam resistance genes which includes 16 types of *blaOXA* genes, with *blaOXA-50* (n=7, 18.42%) being the most frequent. Concordance between genomic and phenotypic resistance to piperacillin-tazobactam was at 47.36%. Similarly, *blaPDC* which confers resistance to cephalosporin antibiotics were present on all isolates, and blaPDC5 (n=12,31.58%) was the most common gene found. Chloramphenicol resistance gene *catB7* was likewise observed across all isolates, and fluoroquinolone resistance was primarily mediated by *crpP* gene (n=22, 57.89%).

There were 53 colistin-resistant *P. aeruginosa* isolates sequenced and analyzed for three years (2022-2024), from 14 surveillance sites. Evidently, the analysis year 2024 has the greatest number of colistin-resistant isolates collected followed by 2022(n=12, 31.58%) and only few for 2023 (n=3, 7.89%). It can be observed that the most common sequence types, ST155 and ST277 were only seen in 2024, while ST 357 was apparent for three years. In addition, serotype O11 was the dominating serotype throughout the analysis years. The resistance mechanism that confers resistance to colistin and other antibiotics among 2022 and 2023 *P. aeruginosa* isolates were similar with that of 2024 isolates. However, the mechanism for cephalosporin resistance was mostly facilitated by *blaPDC11* in 2022-2023 while beta-lactam resistance among 2022-2023 isolates were mostly mediated by *blaOXA-486* gene.

The surveillance results showed that the sequence types of the 2022-2024 isolates were fairly diverse despite the fact that resistance genes detected among these colistin-resistant isolates were relatively uniform. Interestingly, many isolates were susceptible to empiric antibiotics, but showed resistance to colistin, on the other hand, there were also isolates with known colistin resistance that are susceptible to all other antibiotics. This could possibly imply that the bacterial features targeted by other antibiotics (e.g., cell wall synthesis, protein synthesis, DNA replication) [3] are still vulnerable to the empiric antibiotics and/ or the resistance genes for these drugs were not expressed. The primary mechanism of colistin-resistance involves mutations in chromosomal genes (e.g., *pmrAB, phoPQ, mgrB*) that regulate the modification of the bacterial outer membrane's lipopolysaccharide (LPS). [4] While chromosomal resistance itself isn't horizontally transferable like plasmid-mediated resistance, a bacterium that develops chromosomal colistin resistance and already possesses resistance to other drug classes can become extensively drug-resistant (XDR) or even pan-drug-resistant (PDR) when expressed. The ability of WGS to detect resistance mechanisms and its utilization in AMR surveillance underscores its role as an early warning system for emerging threats.

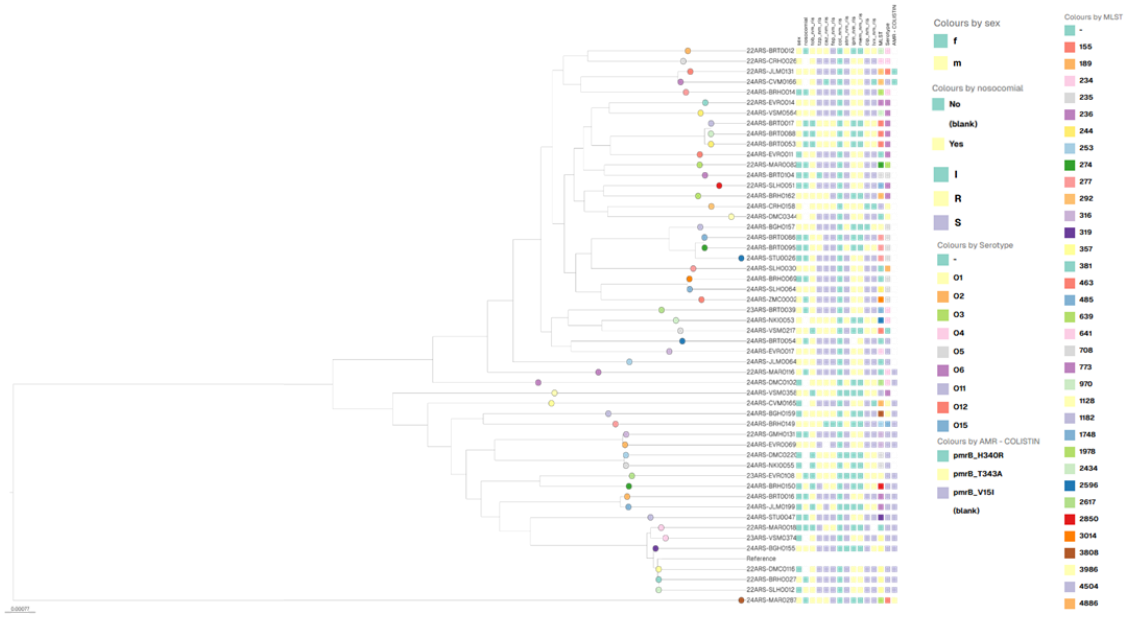


Figure 145. Microreact project of the demographic and genomic characteristics of the colistin-resistant *P. aeruginosa* isolates

References:

[1] Abd El-Baky RM, Masoud SM, Mohamed DS, Waly NG, Shafik EA, Mohareb DA, Elkady A, Elbadr MM, Hetta HF. Prevalence and Some Possible Mechanisms of Colistin Resistance Among Multidrug-Resistant and Extensively Drug-Resistant *Pseudomonas aeruginosa*. Infect Drug Resist. 2020 Feb 3;13:323-332. doi: 10.2147/IDR.S238811. PMID: 32099423; PMCID: PMC7006860.

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Structured Genomic Survey

A structured genomic survey was implemented to characterize the carbapenem resistance among local *Pseudomonas* aeruginosa isolates for 2024. A total of 67 *P. aeruginosa* isolates from 12 surveillance sites were included in the structured genomic survey for 2024 which involved n=52 (78%) carbapenem-resistant and n=15 (22%) carbapenem-susceptible isolates. Most (n=42, 62.69%) of the isolates were from the 20-64 age group, followed by the 65+ years old (n=15, 22.38%), 5-19 years old (n=8, 11.94%) and 0-4 age group (n=2, 2.96%). Sex distribution was relatively equal for males (n=34, 50.75%) and females (n=32, 47.40%), and most isolates were from tracheal aspirate (n=38, 56.71%).

There were 38 unique sequence types (ST) identified among the analyzed *P. aeruginosa* isolates. Many (n=7, 52%) of the isolates were observed to be ST235 and ST155 (n=3, 4.48%). It can be noted that ST235 was likewise the predominating sequence type among the resistant isolates, while ST1021 (n=2, 13.33%) and ST274 n=2, 13.33%) were the most common sequence types among the carbapenem-susceptible isolates.

Table 27. Carbapenemase genes of carba-R *P. aeruginosa* isolates by surveillance sites, ARSP 2024

	CARBAPENEMASE GENES					
Surveillance Site	NDM (n, %)	VIM (n, %)	IMP (n,%)	oprD (n,%)	total (n, %)	other (n, %)
BGH	0, 0%	0, 0%	0, 0%	2, 40%	2, 40%	0, 0%
BRH	0, 0%	0, 0%	1, 33%	0, 0%	1, 33%	0, 0%
BRT	2, 29%	2, 29%	1, 14%	0, 0%	5, 71%	0, 0%
CMC	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
CRH	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
CVM	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
DMC	0, 0%	1, 50%	0, 0%	1, 50%	2, 100%	0, 0%
EVR	0, 0%	1, 33%	0, 0%	0, 0%	1, 33%	0, 0%
FEU	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
GMH	0, 0%	0, 0%	0, 0%	2, 67%	2, 67%	0, 0%
JLM	0, 0%	1, 25%	2, 50%	0, 0%	3, 75%	0, 0%
LCP	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
MAR	2, 20%	0, 0%	0, 0%	3, 30%	5, 50%	0, 0%
MMH	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
NKI	0, 0%	0, 0%	1, 50%	0, 0%	1, 50%	1, 50%
NMC	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
ONP	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
PGH	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
RMC	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
RTH	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
RTM	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
SLH	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
STU	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
VSM	5, 20%	2, 8%	0, 0%	10, 40%	17, 68%	0, 0%
ZMC	0, 0%	0, 0%	0, 0%	1, 100%	1, 100%	0, 0%
ZPH	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%

Table 28. Number of resistant isolates to select antibiotics tested

Surveillance Site	amikacin %R	piperacillin-tazobactam %R	Colistin %R
BGH	0, 0%	3, 60%	5, 100%
BRH	1, 33%	2, 67%	3, 100%
BRT	1, 14%	7, 100%	7, 100%
CMC	0, 0%	0, 0%	0, 0%
CRH	0, 0%	0, 0%	1, 100%
CVM	0, 0%	0, 0%	0, 0%
DMC	0, 0%	2, 100%	2, 100%
EVR	0, 0%	2, 67%	3, 100%
FEU	0, 0%	0, 0%	0, 0%
GMH	0, 0%	1, 33%	3, 100%
JLM	0, 0%	3, 75%	4, 100%
LCP	0, 0%	0, 0%	0, 0%
MAR	0, 0%	6, 60%	10, 100%
MMH	0, 0%	0, 0%	0, 0%
NKI	1, 50%	2, 100%	2, 100%
NMC	0, 0%	0, 0%	0, 0%
ONP	0, 0%	0, 0%	0, 0%
PGH	0, 0%	0, 0%	0, 0%
RMC	0, 0%	0, 0%	0, 0%
RTH	0, 0%	0, 0%	0, 0%
RTM	0, 0%	0, 0%	0, 0%
SLH	0, 0%	0, 0%	0, 0%
STU	0, 0%	1, 100%	1, 100%
VSM	0, 0%	11, 44%	25, 100%
ZMC	0, 0%	0, 0%	1, 100%
ZPH	0, 0%	0, 0%	0, 0%

Resistance to carbapenem was mostly mediated by mutations in *oprD* gene (n=30) which was detected across 12 surveillance sites followed *blaNDM-1* (n=9), *blaVIM-2* (n=7) and *blaIMP-26* (n=5). The combination of *blaVIM-2* and *oprD_W277STOP* were seen for three carbapenem-resistant isolates from DMC and VSM. Notably, all carbapenem-susceptible *P. aeruginosa* isolates also carry a single carbapenem-resistance gene, and many (n=8, 53.33%) of these are *oprD_V359L*. AMR determinants for all other antibiotics are relatively uniform among carbapenem-resistant *P. aeruginosa* isolates while AMR genes are more variable among carbapenem-susceptible isolates.

This genomic survey highlighted that mutations in *oprD* gene is the main driver of carbapenem resistance among the *P. aeruginosa* isolates for 2024. *OprD* is a porin protein in the outer membrane of *P. aeruginosa* that plays a crucial role in the entry of carbapenem antibiotics into the cell. In Singapore, oprD alterations or loss are common mechanisms contributing to carbapenem non-susceptibility in *P. aeruginosa*.[1] While, the *NDM-1* (New

Delhi metallo-β-lactamase 1) resistance is likewise a critical public health concern due to its ability to inactivate a broad spectrum of β-lactam antibiotics, including the last-resort carbapenems.[2]

Carbapenem-resistant *Pseudomonas aeruginosa* isolates tend to be quite similar in their genetic makeup (sequence type) and the specific antibiotic resistance genes they carry. In contrast, carbapenem-susceptible isolates show much more diversity in these characteristics. This homogeneity in resistant strains suggests that the ways *P. aeruginosa* develops carbapenem resistance might follow a more predictable and limited set of pathways. This understanding could lead to more focused and effective strategies for controlling and responding to outbreaks of carbapenem-resistant *P. aeruginosa*.

Genomic surveillance is paramount in combating carbapenem resistance in *Pseudomonas aeruginosa* by providing granular insights into resistance mechanisms, such as the acquisition of carbapenemase genes or mutations in porins like *OprD*. This detailed genetic information enables the precise tracking of resistant clones, like ST235, as they emerge and spread within healthcare settings and across geographic regions, facilitating rapid outbreak detection and containment. Furthermore, genomic data allow for improved comparability of surveillance findings across different laboratories and countries, fostering a unified global effort to monitor and mitigate the escalating threat of carbapenem-resistant *P. aeruginosa*. Ultimately, understanding the genomic underpinnings of resistance directly informs targeted interventions and the development of new therapeutic strategies against this critical pathogen.

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[1] Koh, T. H., et al. (2021). Genomic characterization of carbapenem-non-susceptible *Pseudomonas aeruginosa* in Singapore. Emerging Microbes & Infections, 10(1), 1968318.

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Acinetobacter baumannii

A total of **6,683** *A. baumannii* infections were reported for 2024.

6,683 infections

The largest contributors of *A. baumannii* data were PGH (15.88%), DMC (9.52%) and VSM (8.47%).

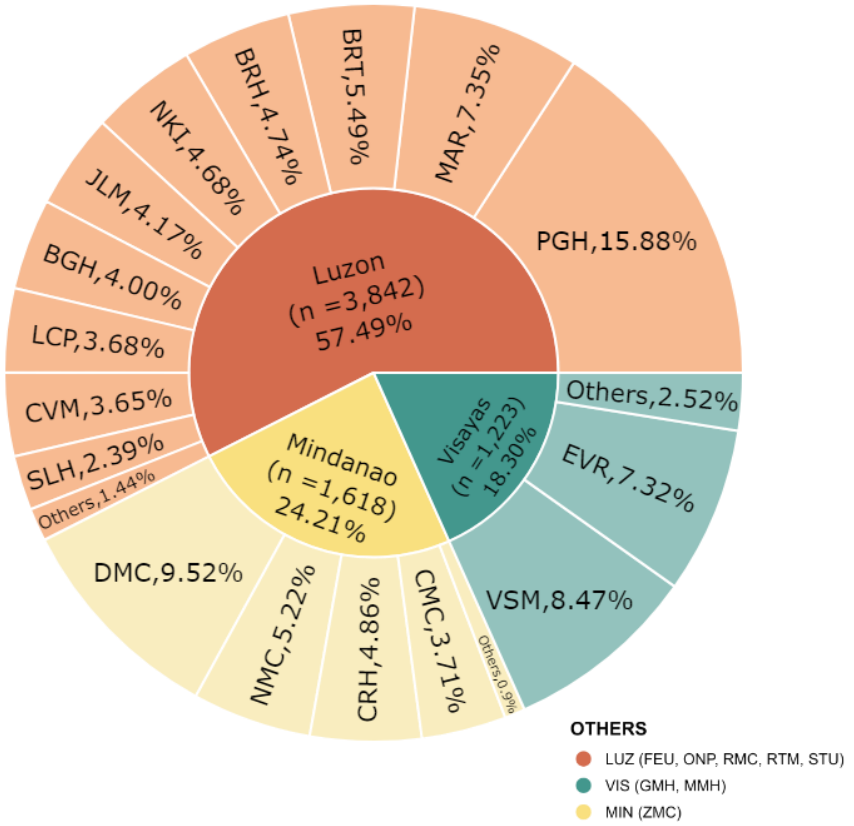


Figure 146. Distribution of *A. baumannii* infections, DOH-ARSP, 2024 (n=6,683)

Most (61.78%) of the infections were from the 20-64 age group and more than half (53.72%) were from male patients. Most (57.98%) infections were detected from respiratory samples and more than half (53.02%) were from presumptive community acquired infections.

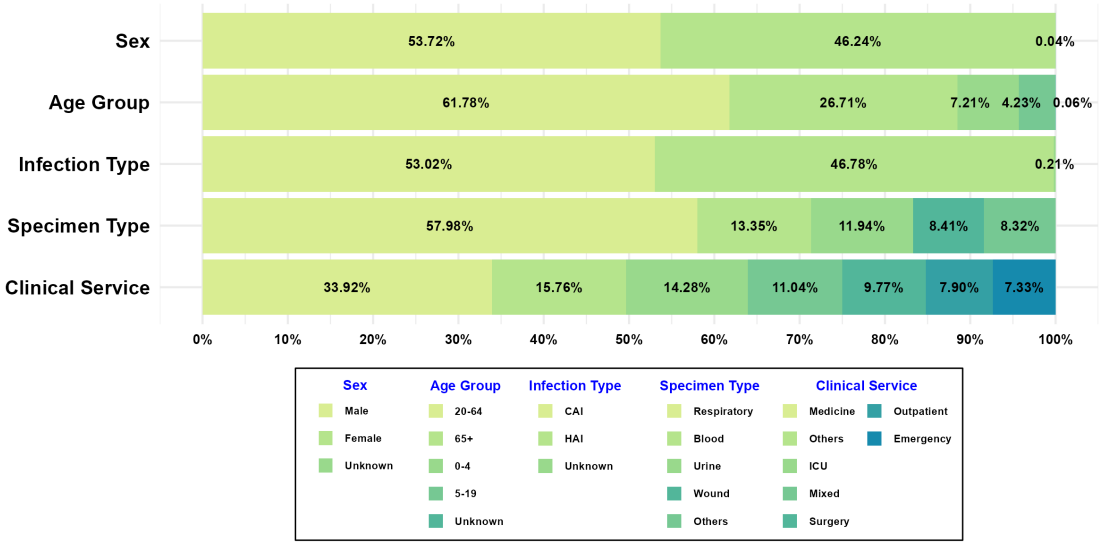


Figure 147. Patient characteristics in *A. baumannii* infections, DOH-ARSP, 2024 (n=6,683)

The percent positive of respiratory infections caused by *A. baumannii* for PGH, EVR, MAR, CRH, SLH and VSM were 26.36%, 15.77%, 14.97%, 13.09%, 11.32% and 10.98% respectively. The percent positive of respiratory infections due to *A. baumannii* for LCP, NMC, CMC, BRH, NKI, DMC and BRT were 5-10%. While the percent positive of the remaining surveillance sites ranged from 0.14-4.75%, and no *A. baumannii* causing respiratory infection was observed for FEU, RTH and ZPH.

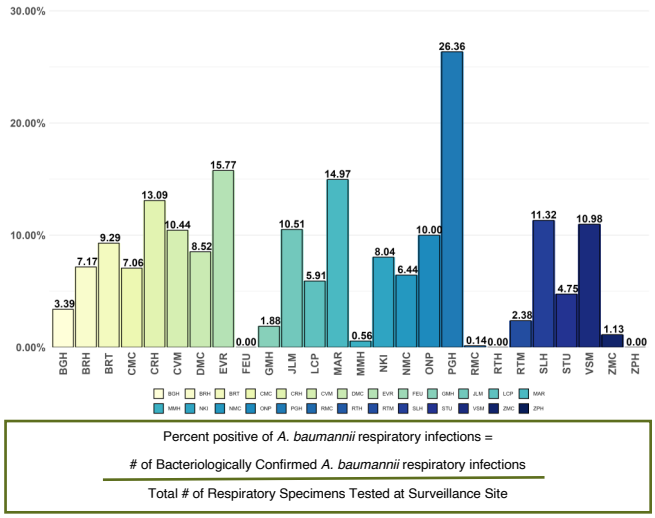


Figure 148. Percent positive of *A. baumannii* respiratory infections among all tested respiratory specimens per surveillance site, DOH-ARSP, 2024

All types of Infections

Figure 149 shows the cumulative resistance rates of all *A. baumannii* infections for 2024. Many were resistant to beta-lactam/beta-lactamase inhibitor combination antibiotics: ampicillin-sulbactam (42.03%) and piperacillin-tazobactam (51.01%), carbapenems: imipenem (49.64%) and meropenem (49.26%) cephalosporins: cefepime (49.42%) and ceftazidime (47.82%);and aminoglycoside tobramycin (40.63%). Resistance to minocycline was at 5.34% and colistin at 0.44%.

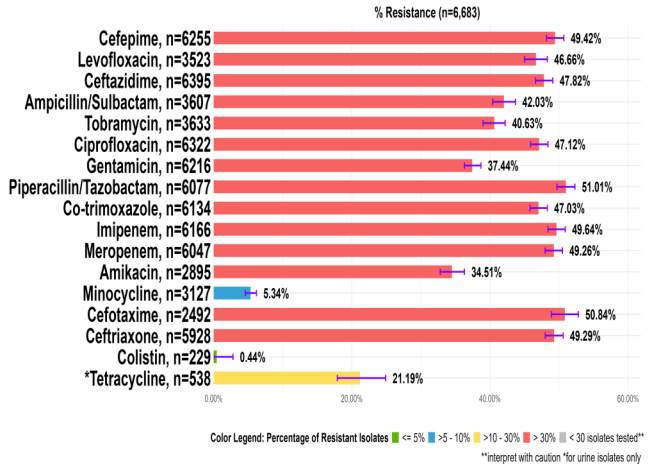
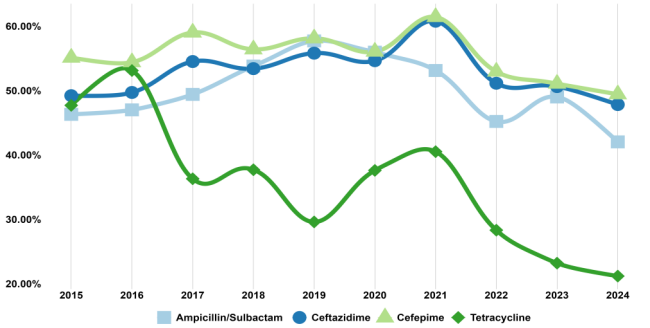
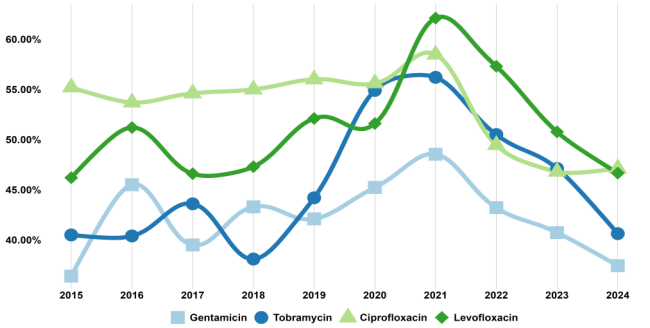


Figure 149. Proportion of all *A. baumannii* infections with resistance to tested antibiotics, DOH-ARSP, 2024

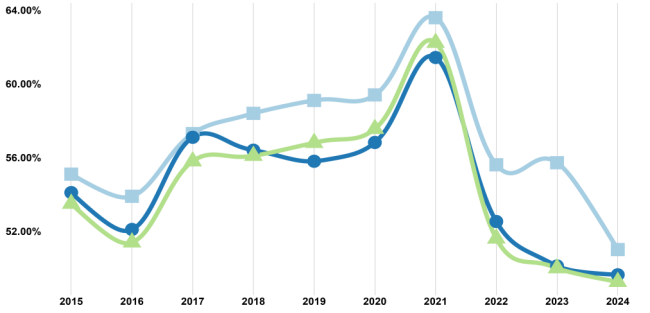
Figure 150 shows the yearly resistance rates of all *A. baumannii* infections. The decreasing pattern seen for colistin (p=0.0001), tetracycline (p=0.0000), and tobramycin (p=0.0000) were all significant. Although an increasing trend was observed for both amilkacin (p=0.6983) and cefotaxime (p=0.0000), it was only statistically significant for cefotaxime.



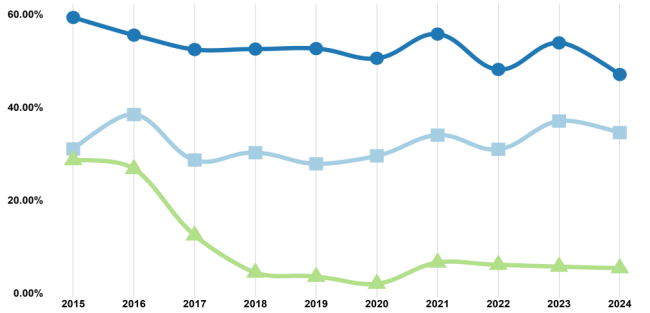
	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Ampicillin/Sulbactam	46.3%	47.0%	49.4%	53.8%	57.7%	56.0%	53.1%	45.2%	49.0%	42.0%
Ceftazidime	49.2%	49.7%	54.5%	53.4%	55.8%	54.6%	60.8%	51.1%	50.6%	47.8%
Cefepime	55.1%	54.4%	59.0%	56.4%	58.1%	56.0%	61.4%	52.9%	51.1%	49.4%
Tetracycline	47.7%	53.1%	36.3%	37.7%	29.6%	37.6%	40.5%	28.3%	23.2%	21.2%



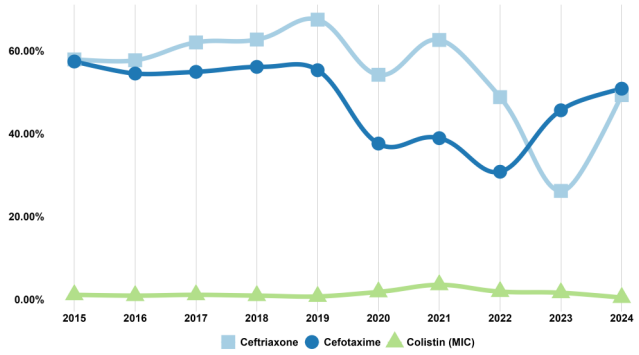
	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Gentamicin	36.4%	45.5%	39.5%	43.3%	42.1%	45.2%	48.5%	43.2%	40.7%	37.4%
Tobramycin	40.5%	40.4%	43.6%	38.1%	44.2%	54.9%	56.2%	50.5%	47.1%	40.6%
Ciprofloxacin	55.2%	53.7%	54.6%	55.0%	56.0%	55.6%	58.5%	49.5%	46.8%	47.1%
Levofloxacin	46.2%	51.2%	46.6%	47.3%	52.1%	51.6%	62.1%	57.3%	50.8%	46.7%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Piperacillin/Tazobactam	55.1%	53.9%	57.3%	58.4%	59.1%	59.4%	63.6%	55.6%	55.7%	51.0%
Imipenem	54.1%	52.1%	57.1%	56.4%	55.8%	56.8%	61.4%	52.5%	50.1%	49.6%
Meropenem	53.5%	51.4%	55.8%	56.1%	56.8%	57.6%	62.2%	51.6%	50.0%	49.3%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Amikacin	31.0%	38.4%	28.6%	30.2%	27.8%	29.5%	34.0%	30.9%	37.0%	34.5%
Co-trimoxazole	59.3%	55.5%	52.4%	52.5%	52.6%	50.5%	55.7%	48.1%	53.8%	47.0%
Minocycline	28.6%	26.7%	12.4%	4.4%	3.5%	2.0%	6.5%	6.1%	5.7%	5.3%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Ceftriaxone	57.9%	57.7%	62.0%	62.7%	67.5%	54.2%	62.6%	48.8%	26.2%	49.3%
Cefotaxime	57.4%	54.5%	54.9%	56.1%	55.3%	37.6%	38.9%	30.8%	45.6%	50.8%
Colistin (MIC)	1.1%	0.9%	1.1%	0.9%	0.7%	1.8%	3.5%	1.9%	1.6%	0.4%

Figure 150. Yearly resistance rates of all *A. baumannii* infections, DOH-ARSP, 2024

Figure 151 shows the carbapenem resistance rates of all *A. baumannii* infections across regions as represented by surveillance sites. Carbapenem resistance among many surveillance sites varies but most rates are high.

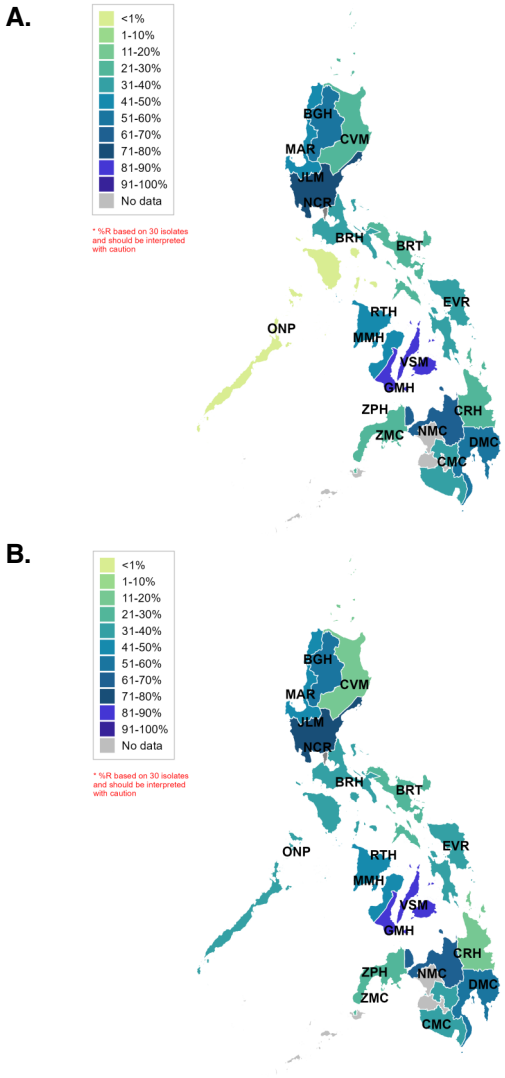


Figure 151. Resistance maps of all *A.baumannii* infections (A) Imipenem and (B) Meropenem, DOH-ARSP, 2024

Bloodstream Infections

Figure 152 shows the resistance rates of *A. baumannii* bloodstream infections. Most of the resistance rates were above 30%. Many were resistant to beta-lactam/beta-lactamase inhibitor combination antibiotics: ampicillin-sulbactam (35.12%) and piperacillin-tazobactam (41.21%). Resistance to carbapenem antibiotics, meropenem and imipenem were 35.33% and 36.09% respectively. Resistance to cephalosporin antibiotics, cefepime and ceftazidime were at 37.7% and 37.41% respectively. Compared to cumulative rates of resistance for all specimens, invasive isolates had relatively lower resistance rates for all antibiotics.

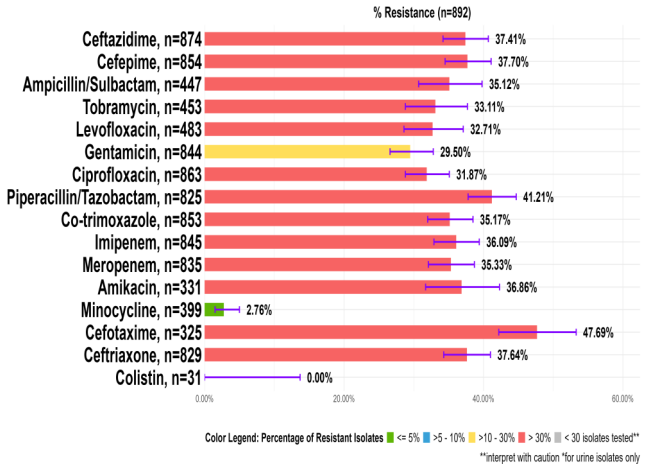
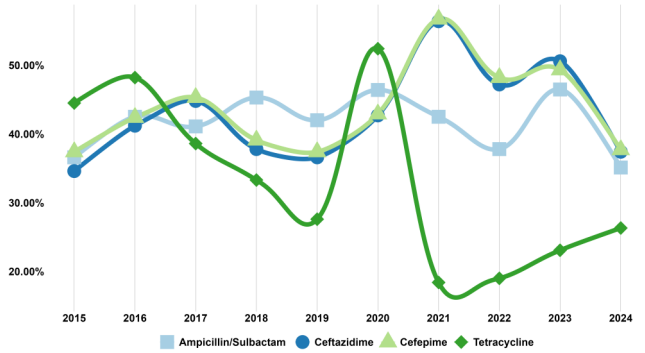
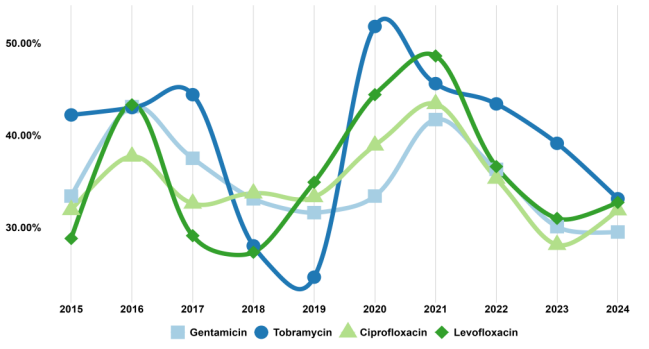


Figure 152. Proportion of *A. baumannii* bloodstream infections with resistance to tested antibiotics, DOH-ARSP, 2024

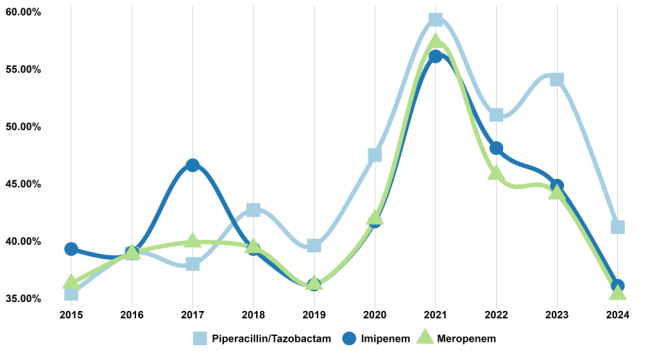
Figure 153 shows the yearly resistance rates of *A. baumannii* bloodstream infections. The decreasing trend seen among the following antibiotics were all statistically significant: levofloxacin, gentamicin, tobramycin, imipenem, meropenem, and colistin. While increasing resistance rates are observed for amikacin (p=0.7432) and tetracycline (p=0.0000).



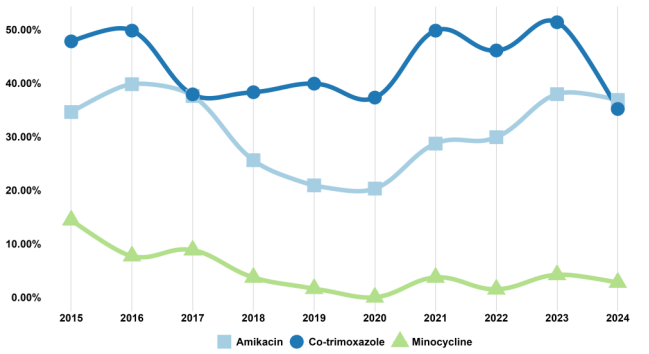
	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Ampicillin/Sulbactam	36.6%	42.5%	41.1%	45.3%	42.0%	46.4%	42.5%	37.8%	46.5%	35.1%
Ceftazidime	34.6%	41.2%	44.8%	37.8%	36.6%	42.7%	56.4%	47.2%	50.6%	37.4%
Cefepime	37.4%	42.4%	45.3%	39.1%	37.4%	42.9%	56.7%	48.2%	49.3%	37.7%
Tetracycline	44.5%	48.2%	38.6%	33.3%	27.6%	52.4%	18.4%	19.0%	23.1%	26.3%



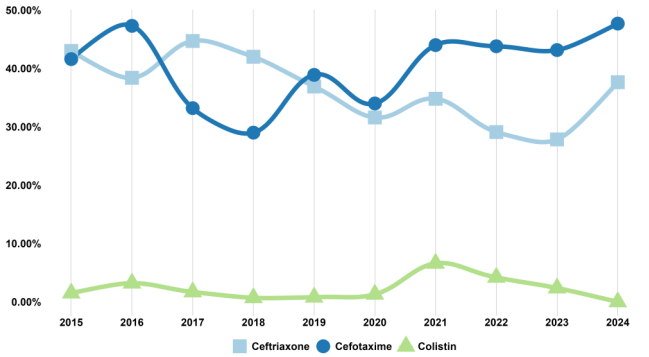
	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Gentamicin	33.4%	43.1%	37.5%	33.1%	31.6%	33.4%	41.7%	36.3%	30.1%	29.5%
Tobramycin	42.2%	43.0%	44.4%	28.0%	24.6%	51.8%	45.6%	43.4%	39.1%	33.1%
Ciprofloxacin	31.9%	37.7%	32.6%	33.7%	33.3%	38.9%	43.4%	35.3%	28.1%	31.9%
Levofloxacin	28.8%	43.3%	29.1%	27.3%	34.9%	44.4%	48.6%	36.6%	31.0%	32.7%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Piperacillin/Tazobactam	35.4%	38.9%	38.0%	42.7%	39.6%	47.5%	59.3%	51.0%	54.1%	41.2%
Imipenem	39.3%	39.0%	46.6%	39.3%	36.2%	41.7%	56.1%	48.1%	44.8%	36.1%
Meropenem	36.3%	38.9%	39.9%	39.4%	36.2%	41.9%	57.3%	45.8%	44.1%	35.3%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Amikacin	34.6%	39.8%	37.6%	25.6%	20.9%	20.3%	28.7%	29.9%	37.9%	36.9%
Co-trimoxazole	47.8%	49.8%	37.9%	38.3%	39.9%	37.3%	49.8%	46.1%	51.4%	35.2%
Minocycline	14.4%	7.7%	8.8%	3.7%	1.6%	0.0%	3.7%	1.5%	4.2%	2.8%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Ceftriaxone	43.0%	38.4%	44.7%	42.0%	36.9%	31.6%	34.8%	29.1%	27.8%	37.6%
Cefotaxime	41.6%	47.3%	33.2%	29.0%	38.9%	34.0%	44.0%	43.8%	43.1%	47.7%
Colistin	1.5%	3.2%	1.7%	0.7%	0.8%	1.3%	6.6%	4.2%	2.4%	0.0%

Figure 153. Yearly resistance rates of *A. baumannii* bloodstream infections, DOH-ARSP, 2024

Lower Respiratory Tract Infections

Figure 154 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 (71.32%) followed by imipenem (71.18%) and meropenem (71.06%). Minocycline resistance was at 7.61% and colistin resistance was at 0.70%.

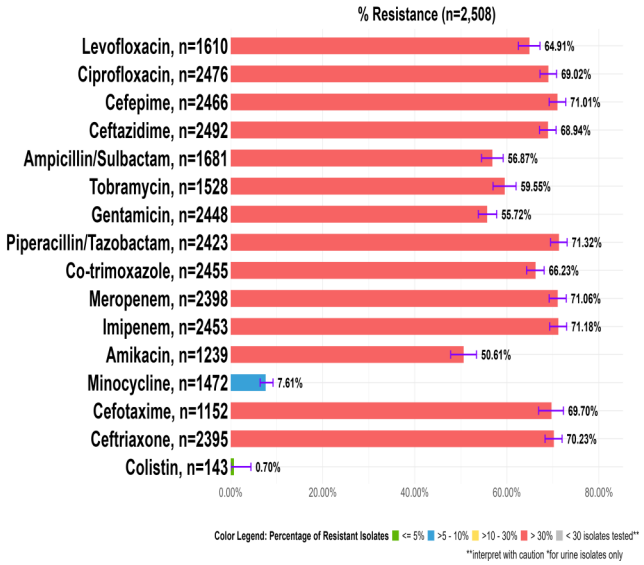
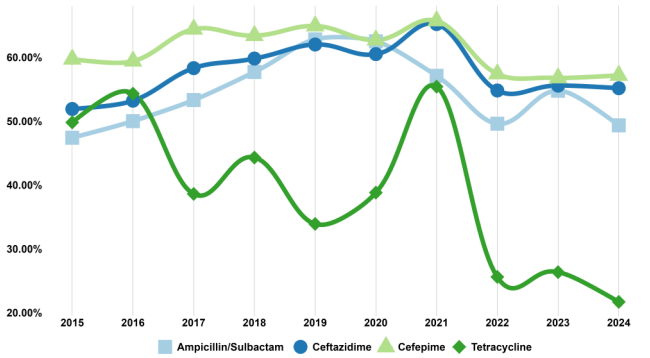
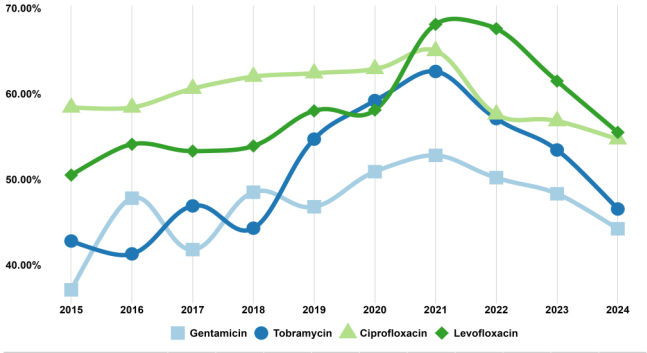


Figure 154. Proportion of *A. baumannii* lower respiratory tract infections with resistance to tested antibiotics, DOH-ARSP, 2024

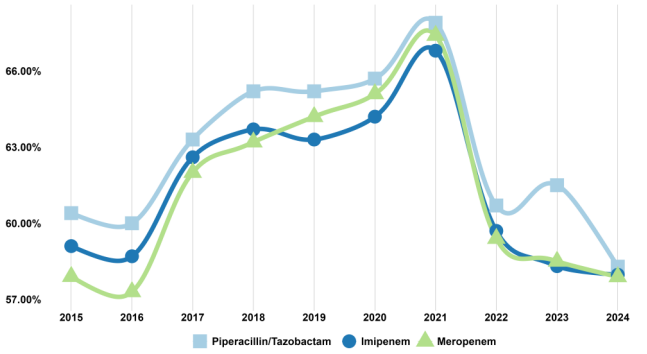
Figure 155 shows the yearly resistance rates of A. *baumannii* respiratory infections. The observed decreasing trend for ciprofloxacin, levofloxacin, tobramycin and gentamicin were all significant.



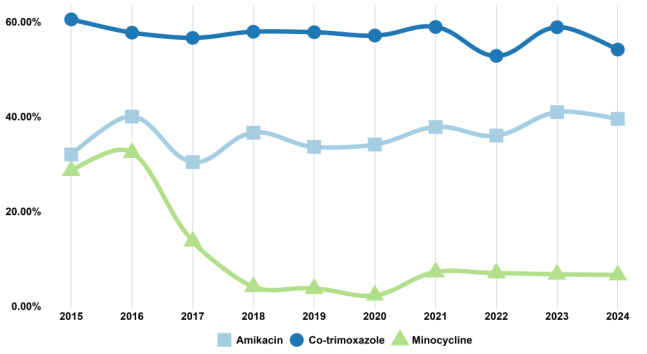
	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Ampicillin/Sulbactam	47.4%	50.0%	53.3%	57.7%	62.8%	62.5%	57.1%	49.6%	54.8%	49.3%
Ceftazidime	51.9%	53.2%	58.3%	59.8%	62.0%	60.5%	65.2%	54.8%	55.6%	55.2%
Cefepime	59.7%	59.4%	64.4%	63.4%	64.9%	62.7%	65.7%	57.4%	56.8%	57.2%
Tetracycline	49.8%	54.3%	38.6%	44.3%	33.9%	38.8%	55.4%	25.6%	26.4%	21.7%



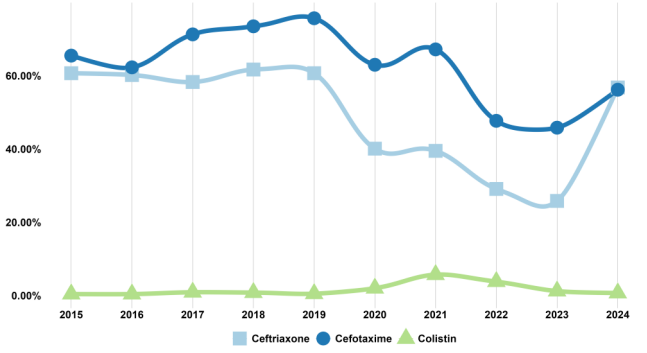
	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Gentamicin	37.1%	47.8%	41.8%	48.5%	46.8%	50.9%	52.8%	50.2%	48.3%	44.2%
Tobramycin	42.8%	41.3%	46.9%	44.3%	54.7%	59.2%	62.6%	57.1%	53.4%	46.5%
Ciprofloxacin	58.4%	58.4%	60.6%	62.0%	62.4%	62.9%	65.0%	57.6%	56.8%	54.7%
Levofloxacin	50.5%	54.1%	53.3%	53.9%	58.0%	58.1%	68.1%	67.6%	61.5%	55.5%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Piperacillin/Tazobactam	60.4%	60.0%	63.3%	65.2%	65.2%	65.7%	67.9%	60.7%	61.5%	58.3%
Imipenem	59.1%	58.7%	62.6%	63.7%	63.3%	64.2%	66.8%	59.7%	58.3%	58.0%
Meropenem	57.9%	57.3%	62.0%	63.2%	64.2%	65.1%	67.4%	59.4%	58.5%	57.9%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Amikacin	32.0%	40.0%	30.4%	36.6%	33.6%	34.1%	37.8%	36.0%	40.9%	39.5%
Co-trimoxazole	60.5%	57.7%	56.6%	57.9%	57.8%	57.1%	58.9%	52.8%	58.9%	54.1%
Minocycline	28.6%	32.4%	13.7%	4.1%	3.7%	2.3%	7.2%	7.0%	6.7%	6.6%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
Ceftriaxone	60.7%	60.2%	58.3%	61.7%	60.7%	40.1%	39.5%	29.1%	25.8%	56.8%
Cefotaxime	65.5%	62.3%	71.3%	73.5%	75.7%	63.0%	67.2%	47.7%	45.8%	56.2%
Colistin	0.4%	0.4%	0.9%	0.8%	0.5%	2.0%	5.7%	3.8%	1.2%	0.7%

Figure 155. Yearly resistance rates of *A. baumannii* respiratory infections, DOH-ARSP, 2024

Healthcare-associated Infections

The resistance rates of *A. baumannii* isolates from healthcare-acquired infections are shown in **Figure 156**. Resistance to beta-lactam/beta-lactamase combination antibiotics exceeded 40%, with ampicillin-sulbactam and piperacillin-tazobactam were at 46.33% and 59.12% respectively, cephalosporins: ceftazidime (55.51%) and cefepime (57.77%); carbapenems: imipenem (58.32%) and meropenem (58.30%). Colistin resistance was at 0.59% and minocycline at 2.84%.

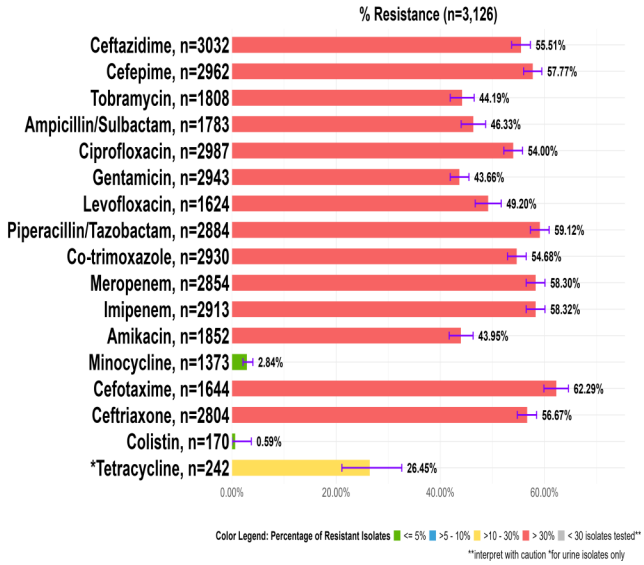


Figure 156. Proportion of presumptive healthcare-associated *A. baumannii* infections with resistance to tested antibiotics, DOH-ARSP, 2024

Emerging Resistance

Colistin resistance in *Acinetobacter baumannii* poses a significant threat to global health due to the limited treatment options available for infections caused by this multidrug-resistant bacterium. As colistin is often considered a last-resort antibiotic, its increasing ineffectiveness leads to higher rates of morbidity and mortality, particularly in hospital settings.[1] The development of resistance mechanisms, such as modifications to the bacterial outer membrane, further complicates treatment strategies. This resistance can spread rapidly, exacerbating the challenge of managing *A. baumannii* infections worldwide. Consequently, the rise of colistin-resistant *A. baumannii* necessitates urgent research into novel therapeutic approaches and improved infection control measures.[2]

Table 29. Patient demographics and clinical characteristics of colistin-resistant *A. baumannii* isolates

Accession number	Age	Sex	Specimen Type	Ward	Infection type
22ARS-VSM0139	56	f	ta	w11	HAI
22ARS-VSM0377	33	f	bl	icu	HAI
22ARS-VSM0382	70	f	ta	w11	HAI
24ARS-BGH0160	63	f	bl	opm	CAI
24ARS-JLM0065	77	m	ta	mwd	HAI
24ARS-STU0027	59	m	pf	mwd	CAI
24ARS-STU0071	31	m	sf	mcu	CAI

Table 30. Antimicrobial resistance profile of colistin-resistant *A. baumannii* isolates

Accession Number	Aminoglycosides			B-lactam Inhibitors		Cephalosporins				Folate Pathway Inhibitor	Lipo-peptide	Carbapenem		Fluoro-quinolone
	AMK	GEN	TOB	SAM	TZP	CAZ	CRO	CTX	FEP	SXT	COL	IPM	MEM	CIP
22ARS-VSM0139	R	S	S	R	R	R	R	R	R	R	R	R	R	R
22ARS-VSM0377	R	R	R	R	R	R	R	R	R	R	R	R	R	R
22ARS-VSM0382	R	R	R	R	R	R	R	R	R	R	R	R	R	R
24ARS-BGH0160	R	S	S	R	R	R	R	R	R	R	R	R	R	S
24ARS-JLM0065	R	R	R	R	R	R	R	R	R	R	R	R	R	R
24ARS-STU0027	S	S	S	S	S	I	I	-	S	S	R	S	S	S
24ARS-STU0071	S	S	S	S	S	S	S	R	S	S	R	S	S	S

Molecular characterization was done for four colistin-resistant *A. baumannii* isolates submitted by three surveillance sites to the reference laboratory in 2024. All isolates were from adult patients aged 31 to 63 years old. The colistin-resistant isolates were from four unique specimen types including blood, cerebrospinal fluid, pleural fluid and tracheal aspirate. Three of these isolates were presumptive community acquired infection while one isolate was presumed to be a hospital acquired infection (24ARS-JLM0065).

As seen in **Table 30**, these 2024 isolates have varying resistance profiles. The colistin-resistant *A. baumannii* isolate from the tracheal aspirate of a 77-year-old male in JLM showed non-susceptibility to 13 other antibiotics tested from 6 antibiotics classes, while the other MDR *A. baumannii* isolate from the blood specimen of a 63-year-old female in BGH showed susceptibility to aminoglycoside and quinolone. The two other colistin-resistant isolates from STU were mostly susceptible to all other tested antibiotics. The first isolate (24ARS-STU0027) from a 59-year-old male showed intermediate resistance to ceftazidime and ceftriaxone while the second isolate from a 31-year-old male (24ARS-STU0071) was observed to be cefotaxime-resistant.

Table 31. Sequence type and AMR genes detected from colistin-resistant *A. baumannii* isolates

Accession number	Sequence Type	K Locus	O Locus	Colistin	Carbapenem	Aminoglycoside	Quinolone	Macrolide	Cephalosporin	Chloramphenicol	Folate Pathway Inhibitor
22ARS-VSM0139	2	KL2	OCL1		blaOXA-23, blaOXA-66	ant(3'')-IIa, aph(3'')-Ib, aph(6)-Id, aph(3')-Via	gyrA_S81L, parC_S84L	msr(E), mph(E)	blaADC-30	cxpE	sul2
22ARS-VSM0377	2	KL3	OCL1	pmrB_Q270P	blaOXA-23, blaOXA-66, ftsI_A515V	ant(3'')-IIa, aph(3'')-Ib, aph(6)-Id, armA	gyrA_S81L, parC_S84L	msr(E), mph(E)	blaADC-73	cxpE	sul2
22ARS-VSM0382	2	KL32	OCL1	pmrB_P233S	blaOXA-23, blaOXA-66, ftsI_A515V	ant(3'')-IIa. aph(3'')-Ib, aph(6)-Id, armA	gyrA_S81L, parC_S84L	msr(E), mph(E)	blaADC-73	cxpE	sul2
24ARS-BGH0160	198	KL223	OCL1		blaNDM-1, blaOXA-670	aph(3'')-Ib, aph(6)-Id, aph(3')-Via, aac(6')-Ij		msr(E), mph(E)			sul1, dfrA19
24ARS-JLM0065	2	KL2	OCL1		blaOXA-23, blaOXA-66, ftsI_A515V	ant(3'')-IIa, aph(3'')-Ib, ash(6)-Id, armA	gyrA_S81L, parC_S84L	msr(E), mph(E)	blaADC-73	cxpE	sul2
24ARS-STU0027	-	KL189	OCL1		blaOXA	ant(3'')-IIa			blaADC	cxpE	
24ARS-STU0071	-	KL35	OCL3		blaOXA-879	ant(3'')-IIa			blaADC	cxpE	

Genomics analysis revealed that isolates belong to sequence type (ST)198 (24ARS-BGH0160) and ST 2 (24ARS-JLM0065), however, sequence types of the two colistin-resistant isolates from STU were not categorized. Furthermore, isolates showed unique K locus while OCL1 was seen for 3 of the 4 isolates for 2024. This may imply that colistin resistance were not seen from a single sequence type but rather with multiple STs. As seen in **Table 31**, no AMR gene for colistin resistance was detected among the 2024 *A. baumanni* isolates, however, mutation in the *pmrA-pmrB* genes (pmrBQ270P) was observed in the previously described isolates in 2022 from VSM (22ARS-VSM0377 and 22ARS-VSM0382) and are indicative of colistin resistance.[3] Carbapenem resistance among 2024 isolates on the other hand was mostly mediated by multiple *blaOXA* genes and a single *blaNDM1* observed in isolate 24ARS-BGH0160. The gene *ant(3'')-IIa* that confers resistance to aminoglycoside antibiotics was observed on 3 of 4 2024 *A. baumanni* isolates, *armA* was also seen on 24ARS-JLM0065. Quinolone resistance seen on isolate from JLM was conferred by the presence of the *gyrA_S81L, parC_S84L* mutations. Moreover, macrolide resistance was mediated by msr (E) and mph (E), while resistance to cephalosporin, chloramphenicol and folate pathway inhibitor were potentially conferred by *blaADC, cxpE* and *sul2* respectively. Concordance between phenotypic and genotypic resistance was mostly concordant for the two MDR-resistant isolates, while it is otherwise for the two pan-susceptible isolates from STU. Interestingly, these isolates from STU The potential non-expression of these AMR genes drives the characteristic phenotypic susceptibility of these isolates against tested antibiotics.

The number of characterized colistin-resistant *A. baumannii* isolates in 2024 is higher than the 2022 isolates, and no isolates of the similar phenotype was seen in 2023. In the previous year, all isolates were ST2 and were seen from VSM, and interestingly, these isolates share a common mechanism of colistin resistance (pmrBQ270P) which may suggest a low-level dissemination. On the other hand, 2024 *A. baumannii* isolates were seen from three surveillance sites, from various regions, however, the exact colistin-resistance mechanism among these isolates was not yet recognized. Limitations of the current genomic pipeline used potentially contributed to the non-detection of the other underlying resistance mechanism such as hetero-resistance and post-transcriptional mutations.[4] Nevertheless, the utilization of whole genome sequencing in AMR surveillance is of high value to elaborate on the potential mechanisms of colistin resistance.

References:

[1] Bostanghadiri, N., Narimisa, N., Mirshekar, M. et al. Prevalence of colistin resistance in clinical isolates of *Acinetobacter baumannii*: a systematic review and meta-analysis. Antimicrob Resist Infect Control 13, 24 (2024). https://doi.org/10.1186/s13756-024-01376-7

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[3] Sun B, Liu H, Jiang Y, Shao L, Yang S, Chen D. New Mutations Involved in Colistin Resistance in *Acinetobacter baumannii*. mSphere. 2020 Apr 1;5(2):e00895-19. doi: 10.1128/mSphere.00895-19. PMID: 32238571; PMCID: PMC7113586.

[4] Charretier Y, Diene SM, Baud D, Chatellier S, Santiago-Allexant E, van Belkum A, Guigon G, Schrenzel J. 2018. Colistin heteroresistance and involvement of the PmrAB regulatory system in *Acinetobacter baumannii*. Antimicrob Agents Chemother 62:e00788-18. https://doi.org/10.1128/AAC .00788-18.

Structured Genomic Survey

A structured genomic survey was implemented to characterize carbapenem resistance among local *Acinetobacter baumannii* isolates for 2024. A total of 50 *A. baumannii* isolates from 12 surveillance sites were included in the structured genomic survey for 2024 which involved n=36 (72%) carbapenem-resistant and n=14 (28%) carbapenem-susceptible isolates. More than half (n=29, 58%) of the isolates were from the 20-64 age group, followed by 65+ years old (n=13, 26%), 0-4 years old (n=5, 10%) and 5-19 age group (n=3, 6%). There were more male (n=28, 56%) than female (n=22, 44%) patients included in the analyzed population and majority (n=37, 74%) of the isolates were from tracheal aspirate.

There were 20 unique sequence types (ST) identified among the analyzed *A.baumannii* isolates, more than half (n=26, 52%) of the isolates were observed to be ST 2 followed by ST 164 (n=4, 8%). It can be noted that ST2 was likewise the predominating sequence type among the resistant isolates, while interestingly, all carbapenem-susceptible isolates are of distinct STs.

Table 32. Carbapenemase genes detected among *A. baumannii* isolates from ARSP surveillance sites

	CARBAPENEMASE GENES																			
Surveillance Site	Oxa-23 (n, %)	Oxa-51 (n, %)	Oxa-58 (n, %)	Oxa-64 (n, %)	Oxa-65 (n, %)	Oxa-66 (n, %)	Oxa-68 (n, %)	Oxa-91 (n,%)	Oxa-94 (n,%)	Oxa-120 (n,%)	Oxa-121 (n,%)	Oxa-144 (n,%)	Oxa-259 (n,%)	Oxa-431 (n,%)	Oxa-500 (n,%)	Oxa-670 (n,%)	Oxa-735 (n,%)	OXA	NDM (n, %)	other (n, %)
BGH	3, 75%	0, 0%	0, 0%	0, 0%	0, 0%	3, 75%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0.01, 0%	0, 0%	0, 0%	1, 25%	0, 0%	0, 0%	1, 25%	3, 75%
BRH	5, 56%	0, 0%	3, 33%	0, 0%	3, 33%	1, 11%	0, 0%	3, 33%	0, 0%	0, 0%	0, 0%	1, 11%	0, 0%	0, 0%	0, 0%	0, 0%	1, 11%	0, 0%	5, 56%	1, 11%
BRT	3, 30%	0, 0%	1, 10%	0, 0%	1, 10%	3, 30%	0, 0%	1, 10%	0, 0%	1, 10%	1, 10%	0, 0%	1, 10%	0, 0%	1, 10%	0, 0%	0, 0%	0, 0%	2, 20%	3, 30%
CMC	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
CRH	1, 100%	0, 0%	0, 0%	0, 0%	0, 0%	1, 100%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	1, 100%
CVM	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
DMC	10, 91%	0, 0%	0, 0%	0, 0%	0, 0%	10, 91%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	1, 9%	0, 0%	10, 91%
EVR	1, 33%	0, 0%	0, 0%	0, 0%	0, 0%	1, 33%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	1, 33%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	1, 33%
FEU	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
GMH	1, 100%	0, 0%	0, 0%	0, 0%	0, 0%	1, 100%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	1, 100%
JLM	1, 100%	0, 0%	0, 0%	0, 0%	0, 0%	1, 100%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	1, 100%
LCP	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
MAR	0, 0%	1, 0%	0, 0%	1, 33%	0, 0%	0, 0%	1, 33%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
MMH	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
NKI	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
NMC	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
ONP	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
PGH	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
RMC	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
RTH	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
RTM	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
SLH	4, 80%	0, 0%	0, 0%	0, 0%	0, 0%	4, 80%	0, 0%	0, 0%	0, 0%	1, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	4, 80%
STU	1, 100%	0, 0%	0, 0%	0, 0%	0, 0%	1, 100%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	1, 100%
VSM	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%
ZMC	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	1, 100%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	1, 100%	0, 0%
ZPH	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%	0, 0%

Table 33. Number of resistant isolates to select antibiotics tested

Surveillance Site	Ampisulbactam %R	Piperacillin-tazobactam %R	Colistin %R
BGH	4, 100%	4, 100%	4, 100%
BRH	6, 67%	7, 78%	9, 100%
BRT	5, 50%	5, 50%	10, 100%
CMC	0, 0%	0, 0%	0, 0%
CRH	1, 100%	1, 100%	1, 100%
CVM	0, 0%	0, 0%	0, 0%
DMC	10, 91%	10, 91%	11, 100%
EVR	1, 33%	1, 33%	3, 100%
FEU	0, 0%	0, 0%	0, 0%
GMH	1, 100%	1, 100%	1, 100%
JLM	1, 100%	1, 100%	1, 100%
LCP	0, 0%	0, 0%	0, 0%
MAR	0, 0%	0, 0%	3, 100%
MMH	0, 0%	0, 0%	0, 0%
NKI	0, 0%	0, 0%	0, 0%
NMC	0, 0%	0, 0%	0, 0%
ONP	0, 0%	0, 0%	0, 0%
PGH	0, 0%	0, 0%	0, 0%
RMC	0, 0%	0, 0%	0, 0%
RTH	0, 0%	0, 0%	0, 0%
RTM	0, 0%	0, 0%	0, 0%
SLH	4, 80%	4, 80%	5, 100%
STU	1, 100%	1, 100%	1, 100%
VSM	0, 0%	0, 0%	0, 0%
ZMC	1, 100%	1, 100%	1, 100%
ZPH	0, 0%	0, 0%	0, 0%

Resistance to carbapenem was mostly mediated by *blaOXA-23* (n=30) which was detected across ten surveillance sites followed by *blaOXA-66* (n=26) and *blaNDM-1* (n=9). The combination of *blaOXA-23* and *blaOXA-66* genes were apparent for 26 carbapenem-resistant isolates, while there were three isolates from BRH that harbors blaNDM-1 and three other *blaOXA* genes, moreover, *blaNDM-1* gene was commonly seen in combination with *blaOXA-91* (n=4). Notably, all carbapenem-susceptible *A. baumannii* isolates also carries a single carbapenem-resistance gene, and many (n=4, 28.57%) of these are *blaOXA-65*. In addition, it can be observed that across all carbapenem-resistant isolates, AMR determinants for all other antibiotics are relatively uniform, while AMR genes are more variable among carbapenem-susceptible isolates.

The genomic survey revealed that the key players for carbapenem resistance among local cases of *A. baumannii* infections are mostly facilitated by *blaOXA-23* (n=30) and *blaNDM-1* genes, this was consistent with the reports from many Asian regions.[1] Studies in countries like Thailand, China, and India have reported this gene as highly prevalent among *Acinetobacter baumannii* isolates. In Thailand, *blaOXA-23* like was found in 68.31% of isolates,[2] while in China, roughly 80.6% of CRAB isolates carried the *blaOXA-23* gene,[3] and across India, *blaOXA-23* was the predominant OXA group associated with carbapenem resistance, found in 97% of isolates.[4] AMR genes might be present in an isolate, but not actively expressed, which suggest the chances of susceptibility among these isolates, in addition, under specific conditions, these genes can be reactivated, potentially leading to therapeutic failure. The sequence type and AMR genes composition are more homogenous among carbapenem-resistant isolates and more heterogenous among carbapenem-susceptible isolates. This may suggest that the mechanism of carbapenem resistance in *A. baumannnii* is relatively conserved which may imply a more targeted and specific containment and response.

Genomic surveys can directly identify the specific genes and mutations that confer antimicrobial resistance, leveraging from simply knowing if a bacterium is resistant and tells us how it's resistant, which is crucial for understanding its potential to spread and for guiding targeted interventions. Moreover, genomic analysis allows for better comparability of surveillance data among different laboratories, nationally, and internationally, which facilitates global efforts to monitor and combat AMR.

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

Table 34. Multidrug-resistant, extremely drug-resistant and pan-drug resistant bacteria in an international expert proposal interim standard definitions for acquired resistance

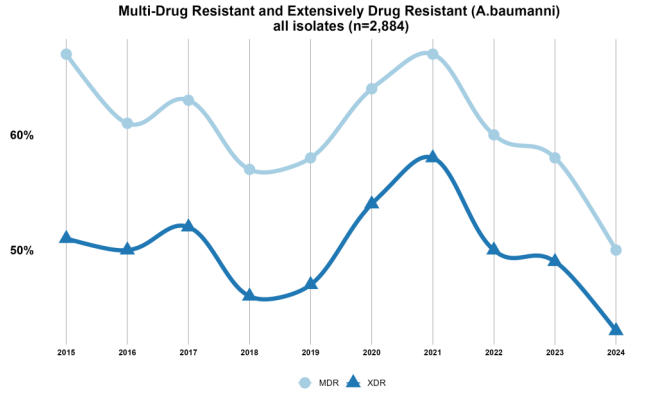
Term	Definition
MDR Multidrug-resitant	Acquired non-susceptibility to at least one agent in three or more antimicrobial categories
XDR Extensively drug-resitant	Non-susceptibility to at least one agent in all but two or fewer antimicrobial categories
PDR Pandrug-resitant	Non-susceptibility to all agents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

More than half of *E. coli* and *K. pneumoniae* BSI in 2024 were MDR with 11% of *E. coli* BSI being possible XDR and 35% of K. pneumoniae BSI being possible XDR (**Table 34**).

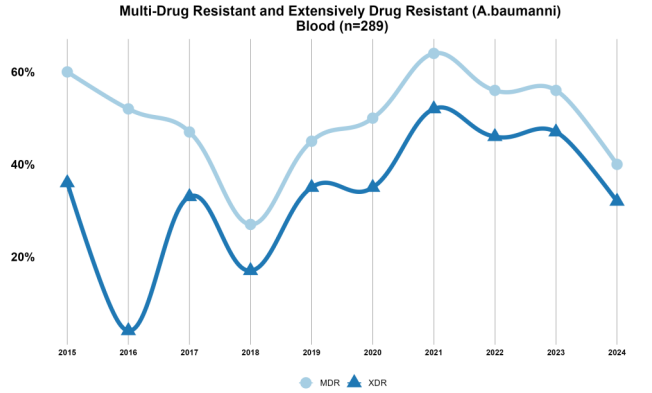
Many of *A. baumannii* BSIs are MDR (40%) and possible XDR (32.4%). Likewise, many of the *P. aeruginosa* BSIs are MDR (18.4%) and possible XDR (12.0%) (**Table 34**). It appears that this trend had persisted in the past decade (**Figure 157 and 158**).

Table 35. MDR and Possible XDR *E. coli, K. pneumoniae, P. aeruginosa and A. baumannii* infections, DOH-ARSP, 2024

	Number of isolates tested	Percentage MDR	Percentage Possible XDR
Escherichia coli			
All isolates	14,895	57.1%	13.7%
Blood isolates	1,369	56.5%	11.4%
Klebsiella pneumoniae			
All isolates	18,378	56.5%	27.8%
Blood isolates	1,744	59.6%	35.0%
Acinetobacter baumannii			
All isolates	6,683	50.2%	43.2%
Blood isolates	892	40.0%	32.4%
Pseudomonas aeruginosa			
All isolates	10,375	22.7%	15.6%
Blood isolates	681	18.4%	12.0%

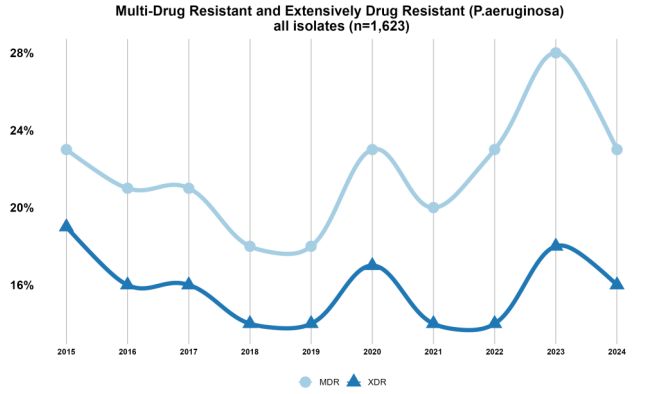


	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
XDR	51.0%	50.0%	52.0%	46.0%	47.0%	54.0%	58.0%	50.0%	49.0%	43.0%
MDR	67.0%	61.0%	63.0%	57.0%	58.0%	64.0%	67.0%	60.0%	58.0%	50.0%

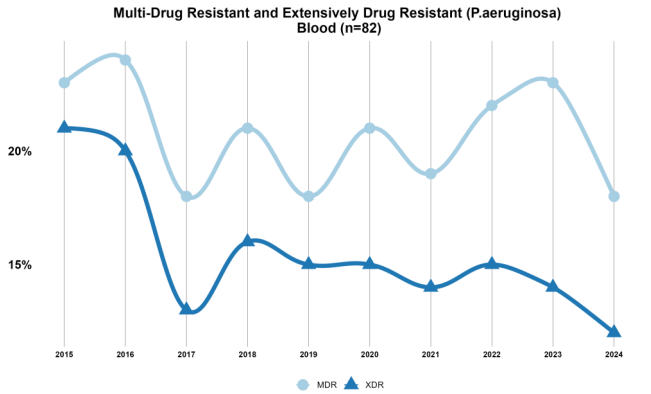


	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
XDR	36.0%	4.0%	33.0%	17.0%	35.0%	35.0%	52.0%	46.0%	47.0%	32.0%
MDR	60.0%	52.0%	47.0%	27.0%	45.0%	50.0%	64.0%	56.0%	56.0%	40.0%

Figure 157. Yearly percentage of MDR and XDR *A. baumannii* infections from all specimens and blood specimen, DOH-ARSP, 2015-2024



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
XDR	19.0%	16.0%	16.0%	14.0%	14.0%	17.0%	14.0%	14.0%	18.0%	16.0%
MDR	23.0%	21.0%	21.0%	18.0%	18.0%	23.0%	20.0%	23.0%	28.0%	23.0%



	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024
XDR	21.0%	20.0%	13.0%	16.0%	15.0%	15.0%	14.0%	15.0%	14.0%	12.0%
MDR	23.0%	24.0%	18.0%	21.0%	18.0%	21.0%	19.0%	22.0%	23.0%	18.0%

Figure 158. Yearly percentage of MDR and XDR *P. aeruginosa* infections from all specimens and blood specimen, DOH-ARSP, 2015-2024

Conclusions, Recommendations and Future Directions

Ongoing surveillance of antimicrobial resistance (AMR) among priority bacterial pathogens is critical for preventing disease and informing appropriate treatment strategies. Based on the 2024 data, the following recommendations are proposed:

Penicillin may still be an effective treatment for non-meningeal *Streptococcus pneumoniae* infections, provided local resistance patterns are monitored. In pneumococcal meningitis, increasing penicillin resistance based on meningitis-specific breakpoints highlights the importance of susceptibility-guided therapy. Third-generation cephalosporins have maintained consistently low resistance rates over the past five years and are recommended for empiric treatment pending susceptibility results. Enhancing AMR surveillance to include pneumococcal serotyping will improve monitoring and assessment of the government's vaccination strategy against this vaccine-preventable pathogen.

Ampicillin resistance in *Haemophilus influenzae* continues to increase, limiting its current use. Third-generation cephalosporins remain the preferred treatment for invasive *H. influenzae* infections. In non-life-threatening infections, co-amoxiclav and cefuroxime may be effective treatment options.

When selecting empiric antibiotic therapy for enteric fever, travel history and local antibiotic resistance patterns should be carefully considered. Locally, no multidrug-resistant (MDR) Salmonella infections were detected and resistance rates to tested antibiotics remained below 5%.

Due to increasing AMR, particularly to fluoroquinolones, ceftriaxone, and co-trimoxazole, antibiotic susceptibility testing is essential for guiding treatment of *Shigella* infections. Azithromycin resistance remains below 10% and may be considered a first-line option for patients who can tolerate oral therapy. Strengthening surveillance by encouraging clinicians to submit stool specimens for culture is recommended to monitor resistance trends more effectively.

Tetracycline, chloramphenicol, and co-trimoxazole remain effective treatment options for cholera. No azithromycin resistance has been reported in the past two years. Ongoing AMR surveillance of *Vibrio cholerae* infections is essential to guide treatment strategies and prevent the spread of resistant strains.

Ceftriaxone remains the empiric antibiotic of choice for gonococcal infections. However, emerging resistance to third-generation cephalosporins in neighboring Southeast Asian countries underscores the need for enhanced surveillance. Clinicians should be encouraged to submit specimens for culture, and surveillance sites should forward isolates to the reference laboratory for confirmatory testing to monitor resistance patterns effectively.

Despite a statistically significant decrease in MRSA rates from 2015 to 2024, high rates of oxacillin resistance continue to limit its effectiveness as an empiric treatment option for *Staphylococcus aureus* infections. Given the relatively lower resistance rates to clindamycin and macrolides, these antibiotics could be considered as alternative treatment options. However, the judicious use of reserve antibiotics like vancomycin and linezolid remains crucial to preserve their effectiveness in treating *S. aureus* infections.

Multidrug resistance in bacteria like *Escherichia coli, Klebsiella pneumoniae*, *Pseudomonas aeruginosa*, and *Acinetobacter baumannii* continues to be a major public health concern. This is primarily due to the limited treatment options available and the significant challenges in containing these infections. To combat the spread of these "superbugs," a comprehensive strategy is essential. Real-time data analysis and genotyping are crucial for establishing linkages between cases, enabling timely patient isolation and the implementation of effective infection control measures. Furthermore, developing specific and stratified antibiograms will empower clinicians to select the most appropriate empiric antibiotic treatments for suspected infections caused by these resistant organisms.

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 bioinformatics 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.

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Annexes

Annex A: Software and Version Used

WHONET 64 bit	
Build Date	2025-04-10
Build Number	25.4.10
R	
R software	4.5.0
R Studio	2025.05.0+496
Workflow	
bactopia	3.2.0
Nextflow	24.10.5
BACTOPIA Assembler Module	
any2fasta	0.4.2
assembly-scan	1.0.0
bwa	0.7.17-r1188
dragonflye	1.2.0
flash	1.2.11
medaka	1.11.3
megahit	1.2.9
miniasm	0.3-r179
minimap2	2.27-r1193
nanoq	0.10.0
pigz	2.8
racon	1.5.0
rasusa	0.8.0
raven	1.8.3
samclip	0.4.0
samtools	1.18
shovill	1.1.0
shovill-se	1.1.0
skesa	2.5.1
spades	3.15.5
unicycler	0.5.0
velvetg	1.2.10
velveth	1.2.10
BACTOPIA Gather Module	
art	2.5.8
fastq-dl	2.0.4
fastq-scan	1.0.1
ncbi-genome-download	0.3.3
pigz	2.6
BACTOPIA QC Module	
bbduk	39.08
fastp	0.23.4
fastq-scan	1.0.1
fastqc	0.12.1
lighter	1.1.13
nanoplot	1.43.0
nanoq	0.10.0
pigz	2.8
porechop	0.2.4
rasusa	2.1.0

BACTOPIA Annotator Module	
makeblastdb	2.14.1+
prokka	1.14.6
BACTOPIA Sketcher Module	
mash	2.3
sourmash	4.8.2
BACTOPIA MLST Module	
mlst	2.23.0
mlst-database	2025-02-25 13:58:13
BACTOPIA AMRFINDERPLUS	
amrfinderplus	4.0.19
amrfinderplus-database	4.0.19
Custom Dump Software Versions	
python	3.12.6
yaml	6.0.2
BACTOPIA Tools Snippy	
gubbins	3.4
iqtree	2.4.0
pigz	2.8
snippy	4.6.0
bedtools	2.31.1
vcf-annotator	0.7
snp-dists	0.8.2
BACTOPIA Tools	
clermontyping	24.02
ectyper	1.0.0
kleborate	3.1.3
plasmidfinder	2.1.6
csvtk	0.31.0
pasty	2.2.1
agrvate	1.0.2
sccmec	1.2.0
spatyper	0.2.1
mykrobe	0.13.0
seqsero	1.3.1
sistr	1.1.3
seroba	1.0.2
Bioinformatics Tools - Other	
amrfinder	4.0.19
amrfinder-database	2024-12-18.1
kaptive	3.1.0
checkm2	1.1.0

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Annexes

Annex B: List of ARSRL Staff 2024

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Polle Krystle V. Macaranas, RMT	Ma. Cecilia G. Alea	
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Mariane A. Magbanua, RMT		
June Janice B. Borlasa, RMT		
Ma. Cristina F. Rempillo, RMT		
Rose M. Marasigan, RMT		
Carl Joey D. Divino		
Flordeliza C. Timajo		
Joey N. Legayada		
Levi L. Calixto		

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