1828
AKADÉMIAI KIADÓ

Shifting molecular epidemiology of carbapenem-resistant *Klebsiella pneumoniae* in a regional Greek hospital: Department-specific trends and national context (2022–2024)

Acta Microbiologica et Immunologica Hungarica

72 (2025) 3, 212–219

DOI:
10.1556/030.2025.02655

RESEARCH ARTICLE

Pandora Tsolakidou*, Georgios Tsikrikonis, Kontantina Tsaprouni, Martha Souplioti and Eumorfia Sxoina

Microbiology Department, Hospital of Volos, Volos, Greece

Received: June 17, 2025 • Accepted: July 4, 2025
Published online: July 17, 2025

ABSTRACT

Carbapenem-resistant *Klebsiella pneumoniae* (CRKP) poses a growing threat in Greek hospitals, with increasing reports of multidrug- and pandrug-resistant strains; however, molecular data from regional centers remain limited. This study aimed to investigate the molecular epidemiology, resistance mechanisms, and transmission dynamics of CRKP isolates collected at the General Hospital of Volos, Central Greece, between 2022 and 2024. Thirty-seven non-duplicate CRKP isolates were analyzed. Identification and antibiotic susceptibility testing were performed using VITEK® 2, disk diffusion, Etest®, and broth microdilution. Carbapenemase production was assessed using the NG-Test® Carba-5. Eight isolates underwent multilocus sequence typing (MLST). All isolates were resistant to carbapenems, cephalosporins, and fluoroquinolones; furthermore, 40% were colistin-resistant. The dominant carbapenemase genes were $bla_{\text{NDM-1}}$ (45.9%), $bla_{\text{KPC-2}}$ (18.9%), and $bla_{\text{VIM-1}}$ (27.0%), with co-expression of multiple carbapenemases in 30% of the isolates. MLST revealed the high-risk clones ST11, ST15, and ST323, and three intra-intensive care unit (ICU) transmission clusters. The emergence of dual-carbapenemase and colistin-resistant clones underscores the need for local genomic surveillance, improved infection control, and access to newer antimicrobials in non-tertiary settings.

KEYWORDS

Klebsiella pneumoniae, carbapenem resistance, New Delhi metallo-β-lactamase (NDM), *Klebsiella pneumoniae* carbapenemase (KPC), ST323, Intensive Care Unit (ICU), molecular epidemiology

1. INTRODUCTION

Carbapenem-resistant *Klebsiella pneumoniae* (CRKP) is a global health threat. This is particularly the case within healthcare settings, where it is associated with high mortality, prolonged hospitalization, and limited therapeutic options. As one of the most clinically significant multidrug-resistant pathogens among *Enterobacterales, K. pneumoniae* is frequently implicated in bloodstream infections, ventilator-associated pneumonia, urinary tract infections, and surgical site infections, especially among critically ill patients [1].

The primary mechanism of carbapenem resistance in *K. pneumoniae* is the production of carbapenemases, including *K. pneumoniae* carbapenemase (KPC), Verona integron-encoded metallo-β-lactamase (VIM), and New Delhi metallo-β-lactamase (NDM). According to the Ambler molecular classification, β-lactamases are grouped into four classes (A–D) based on amino acid sequence homology. Class A (e.g., KPC) and Class D (e.g., OXA-48) enzymes are serine-β-lactamases, while Class B enzymes (e.g., NDM, VIM, IMP) are metallo-β-lactamases that require zinc ions for their catalytic activity. This classification provides insight into both the structural and functional diversity of carbapenemases and their implications for

*Corresponding author.
E-mail: ptsolakidou@gmail.com

therapeutic strategies. These enzymes are often co-expressed with extended-spectrum β-lactamases (ESBLs) or other resistance determinants, leading to extensively drug-resistant (XDR) or even pandrug-resistant (PDR) phenotypes. Furthermore, many of these resistance genes are located on mobile genetic elements such as plasmids, enabling horizontal transmission across bacterial species and between patients [2].

Greece remains one of the European countries most affected by CRKP, with endemic transmission of high-risk clones such as ST11, ST258, ST147, and ST15. Surveillance data from recent years indicate a shift from predominantly KPC-producing strains toward NDM and VIM producers, often in complex co-resistance patterns. While national-level data have been well described, molecular and epidemiological surveillance in second-level hospitals—where resources and access to newer antimicrobial agents are often limited—remains under-represented [3–7].

The General Hospital of Volos—a 400-bed secondary care hospital in Central Greece—has reported an increasing burden of CRKP infections in recent years. However, there has been no detailed molecular characterization of the circulating clones, resistance mechanisms, or department-specific transmission patterns in this setting.

In this retrospective study, we analyzed 37 CRKP isolates collected between 2022 and 2024 at the General Hospital of Volos. Through species identification, antimicrobial susceptibility testing, carbapenemase screening, multilocus sequence typing (MLST), we aimed to achieve the following:

1. Characterize the molecular resistance mechanisms underlying circulating CRKP strains.
2. Identify predominant sequence types and transmission clusters within hospital departments.
3. Compare local trends with national epidemiological data.

2. MATERIALS AND METHODS

2.1. Ethical approval

The study protocol has been submitted to the Scientific Board of the General Hospital of Volos (Protocol No. 1200/17-6-2025), and final official approval has been obtained from the Scientific Council of the General Hospital of Volos, approval reference 7/23-6-25. All procedures involving clinical isolates were conducted in accordance with institutional and national ethical guidelines.

2.2. Study setting and sample collection

A total of 37 non-duplicate carbapenem-resistant *K. pneumoniae* (CRKP) isolates were recovered from clinical specimens collected at the General Hospital of Volos, a 400-bed regional secondary care center in Thessaly, Greece. The sampling period spanned from July 2022 to January 2024. The isolates were obtained from the following sample types: urine ($n = 16$), wound swabs ($n = 7$), bronchial secretions ($n = 7$), blood cultures ($n = 6$), and central venous catheter ($n = 1$). The departmental origin of the isolates included the Intensive Care Unit (ICU; $n = 21$), Surgical Department ($n = 4$), First and Second Internal Medicine Departments ($n = 9$), Emergency Department ($n = 1$), Urology Department ($n = 1$), and Chemotherapy Department ($n = 1$).

2.3. Bacterial identification and antimicrobial susceptibility testing

Species identification was performed using the VITEK® 2 automated system (BioMérieux, France). Antimicrobial susceptibility testing was conducted using the disk diffusion method (Oxoid, Thermo Fisher Scientific) and minimum inhibitory concentration (MIC) determination: disk diffusion testing covered a range of β-lactams, fluoroquinolones, aminoglycosides, and trimethoprim-sulfamethoxazole. MICs for ceftazidime–avibactam were determined using Etest® strips (Liofilchem). Colistin MICs were evaluated via broth microdilution using the SensiTest Colistin kit (SensiTest STC, Liofilchem). Interpretation followed EUCAST guidelines (versions 2021–2023).

2.4. Carbapenemase detection

Phenotypic detection of carbapenemase production was conducted using the NG-Test® CARBA 5 immunochromatographic assay (NG Biotech, France), identifying the presence of KPC, NDM, VIM, OXA-48, and IMP enzymes.

2.5. Molecular typing and Multi-Locus Sequence Typing

Polymerase chain reaction (PCR) was performed for beta-lactamase genes on all isolates. Extraction was performed with distilled water and the primers used for the amplification are shown in Table 1.

2.6. Multi-Locus Sequence Typing (MLST)

Eight isolates (IDs 4, 6, 13, 19, 22, 23, 24, 25) were subjected to MLST based on sequencing of seven housekeeping genes. The selection of these eight strains was done randomly. PCR amplification was performed in Biochemical Department of University Hospital of Larissa and sequencing were outsourced to Cemia Laboratories (Larissa, Greece).

Table 1. PCR primers used to detect common carbapenem-resistant genes in the isolated strains

Gene	Primer Name	Sequence (5′ → 3′)
NDM	NDMF	TGGCAGGACACTTCCTATC
	NDMR	AGATTGCCGAGCGAGCGACTTG
VIM	VIMF	AGTGGTGAGTATCCGACA
	VIMR	ATGAAAGTGCGTGGAGAC
KPC	KPCF	TCGCTAAACTCGAACAGG
	KPCR	TTAGTGCCCGTTGACGCCCAATCC
OXA-48	OXA-48F	TTGGTGGCATCGATTATCGG
	OXA-48R	GAGCACTTCTTTTGTGATGGC

3. RESULTS

3.1. Isolate identification and sample distribution

All 37 isolates collected between July 2022 and January 2024 were confirmed as *K. pneumoniae*. The majority originated from the Intensive Care Unit (ICU) (21/37), followed by Internal Medicine Departments (9/37) and the Surgical Department (4/37). Urine was the most common sample type (16/37), followed by wounds (7/37), bronchial secretions (7/37), blood cultures (6/37), and one central venous catheter.

Among patients infected with carbapenem-resistant *K. pneumoniae*, the most common comorbidities were type 2 diabetes mellitus (15%), followed by infections (10%), cancer (10%), and trauma due to motor vehicle accidents (10%). Notably, 10% of records lacked a clear diagnostic label (ND). These findings reflect the vulnerability of immunocompromised, chronically ill, and critically injured patients to CRKP colonization and infection (Table 2).

3.2. Carbapenemase production and phenotypic resistance

Carba-5 testing identified 20 KPC-producing and 17 NDM-producing isolates. Seventeen isolates exhibited dual carbapenemase production (KPC + VIM or NDM + OXA-48), and one isolate co-produced KPC + NDM. All isolates were resistant to carbapenems, cephalosporins, and fluoroquinolones. Resistance to sulfamethoxazole/trimethoprim was noted in 89% of isolates, while 40% showed colistin resistance.

3.3. Molecular characterization of resistance genes

PCR was performed on all isolates. The most prevalent carbapenemase gene was bla_{NDM}, followed by bla_{KPC}, $bla_{\text{OXA-48}}$, and $bla_{\text{VIM-1}}$. Co-production of carbapenemase genes (e.g., bla_{NDM} + $bla_{\text{OXA-48}}$, bla_{KPC} + bla_{VIM}) was common.

3.4. Sequence types and clonal distribution

MLST analysis identified three main sequence types (Table 3):

ST11 (2/8 typed isolates). ST323 and ST15 (five and one isolate each)—associated with dual carbapenemase production. Two ST15 isolates and one ST11 isolate were pan-drug resistant (PDR).

4. DISCUSSION

This study provides a detailed molecular and phenotypic overview of *K. pneumoniae* isolates resistant to carbapenems, collected over a three-year period at a regional hospital in Central Greece. Our findings reveal a complex and evolving epidemiological landscape, with the predominance of high-risk clones, extensive resistance gene co-carriage, and evidence of intra-hospital transmission—especially in the ICU.

ST11 emerged as a sequence type in ICU departments, consistent with its well-established role in global and national CRKP dissemination. Notably, the ST11 isolates in our cohort frequently co-harbored carbapenemase genes [8–12].

The detection of ST15 and ST323 is also noteworthy [13]. ST15 has been increasingly reported across Europe as an emerging PDR clone, and our data support this trend, with one ST15 isolate exhibiting phenotypic pan-drug resistance and colistin resistance ST323, while globally not a high-risk clone, displayed an unusual combination of KPC, VIM, production, suggesting potential horizontal gene transfer events and a concerning resistance profile (3).

Interestingly, ST258, a dominant CRKP clone in many Greek hospitals according to ECDC data, was underrepresented in our cohort. This may reflect regional variation in clonal dynamics or the relatively small sample size for WGS. The absence of ST147—another high-risk clone often associated with CZA resistance—may be attributable to random isolate selection or local epidemiological factors (3).

Dual carbapenemase production (e.g., KPC + VIM, NDM + OXA-48, NDM + VIM) was observed in 11 isolates (29.7%), primarily within the ICU. Such combinations are clinically significant, as they are associated with reduced susceptibility to all β-lactams, including newer agents like ceftazidime–avibactam. Although ceftazidime–avibactam retained activity in the few tested isolates, their limited use in non-tertiary hospitals can reduce selection pressure and delay resistance, but may also limit therapeutic options.

Colistin resistance was detected in 40% of isolates. While colistin remains one of the few available treatments for CRKP in Greek regional hospitals, these data suggest emerging resistance may further restrict its clinical utility. The association of colistin resistance with ICU-derived isolates raises concerns about selective pressure due to frequent or prolonged use of intravenous and nebulized colistin in critical care settings.

Resistance to non-β-lactam antibiotics was also widespread while gentamicin susceptibility was preserved in some cases, although the possible spread of 16S rRNA methyltransferase genes could compromise this class as a treatment option. Similarly, all isolates indicate exhibited intrinsic resistance to fosfomycin.

Compared with national surveillance data, our findings show both overlap and divergence. While ST11 remains dominant, the frequent detection of dual-carbapenemase producers and the scarcity of ST258/ST147 suggest unique local dynamics. These patterns may be influenced by differences in antimicrobial usage, infection control practices, and access to advanced diagnostics.

5. LIMITATIONS

This study has several limitations. First, the sample size was modest, and isolate selection was not randomized. Second, most isolates were obtained from the ICU, potentially

Table 2. Isolates, phenotypes, sequence types (STs) and phenotypic resistance

ID	Department	Laboratory ID	Time collected	Underlying conditions	Sample	Carbapenemase Type (immunochromatographic assay)	ST	Carbapenem resistance gene (PCR)	Phenotypic Resistance
1	Surgical	1046	9–2022	Ulcerative colitis	Wound	NDM		bla_{NDM}	Fluoroquinolones, Carbapenems, Amikacin, Tobramycin, Trimethoprim-Sulfomethoxazole
2	Surgical	870	7–2022	T2DM, CAD (bypass, PCI), HTN, hyperuricemia, toe amputation (R foot gangrene)	Wound	NDM + OXA-48		bla_{NDM} + bla_{OXA-48}	Fluoroquinolones, Carbapenems, Aminoglycosides
3	ICU	96	10–2022	Fall (5 m), GCS 7, pupils iso, mid, reactive, Haemodynamically unstable	Bronchial	KPC		bla_{KPC}	Fluoroquinolones, Carbapenems, Aminoglycosides, Trimethoprim-Sulfomethoxazole
4	ICU	1355	8–2022	Resp distress	Bronchial	KPC + VIM	ST323	bla_{KPC} + bla_{VIM-1}	Fluoroquinolones, Carbapenems, Tobramycin, Trimethoprim-Sulfomethoxazole
5	Surgical	337	9–2022	T2DM, HTN, reported soft tissue infection of lower limbs	Wound	NDM + OXA-48		bla_{NDM} + bla_{OXA-48}	Fluoroquinolones, Carbapenems, Tobramycin
6	2nd Internal	68	8–2022	coma	Urine	KPC + VIM	ST323	bla_{KPC} + bla_{VIM}	Fluoroquinolones, Carbapenems, Tobramycin, Trimethoprim-Sulfomethoxazole
7	1st Internal	487	9–2022	foreign body	Urine	KPC		bla_{KPC}	Fluoroquinolones Carbapenems, Tobramycin, Trimethoprim-Sulfomethoxazole
8	ICU	1132	9–2022	Polytrauma	Central venous catheter	KPC		bla_{KPC}	Fluoroquinolones Carbapenems, Colistin, Tobramycin, Gentamicin, Trimethoprim-Sulfomethoxazole
9	Surgical	629	9–2022	Infected wound	Wound	NDM + VIM		bla_{NDM} + bla_{VIM}	Pan-drug resistant
10	Emergency	761	8–2022	Abd pain	Urine	NDM		bla_{KPC}	Fluoroquinolones, Carbapenems, Tobramycin, Gentamicin, Trimethoprim-Sulfomethoxazole Amikacin (intermediate)
11	ICU	1017	8–2022	Fever	Urine	KPC		bla_{KPC}	Fluoroquinolones, Carbapenems, Colistin, Tobramycin, Amikacin, Gentamicin, Trimethoprim-Sulfomethoxazole

(continued)

Table 2. Continued

ID	Department	Laboratory ID	Time collected	Underlying conditions	Sample	Carbapenemase Type (immunochromatographic assay)	ST	Carbapenem resistance gene (PCR)	Phenotypic Resistance
12	1st Internal	1333	7–2022	Liver failure	Urine	KPC		bla_{KPC}	Fluoroquinolones, Carbapenems, Tobramycin, Amikacin, Trimethoprim-Sulfomethoxazole
13	ICU	497	8–2022	Fall (~2 m)	Bronchial	KPC	ST323	bla_{KPC}	Fluoquinolones, Carbapenems, Tobramycin, Amikacin, Trimethoprim-Sulfomethoxazole
14	2nd Internal	540	9–2022	Diverticulum	Urine	KPC + VIM		bla_{KPC} + bla_{VIM}	Fluoroquinolones, Carbapenems, Colistin, Tobramycin, Gentamicin, Trimethoprim-Sulfomethoxazole
15	ICU	156	9–2022	LBO	Bronchial	KPC + VIM		bla_{KPC} + bla_{VIM}	Fluoroquinolones, Carbapenems, Tobramycin, Amikacin, Trimethoprim-Sulfomethoxazole
16	Urology	1227	8–2022	Hematuria	Urine	KPC		bla_{KPC}	Fluoroquinolones, Carbapenems, Tobramycin, Amikacin, Trimethoprim-Sulfomethoxazole
17	ICU	298	9–2022		Blood	KPC		bla_{KPC}	Fluoroquinolones, Carbapenems, Colistin, Tobramycin, Amikacin, Gentamicin, Trimethoprim-Sulfomethoxazole
18	2nd Internal	213	8–2022	Urinary tract infection	Blood	NDM + OXA-48		bla_{NDM} + bla_{OXA-48}	Fluoroquinolones, Carbapenems, Tobramycin, Amikacin
19	ICU	745	7–2022	MVA	Wound	KPC + VIM	ST11	bla_{KPC} + bla_{VIM}	Fluoroquinolones, Carbapenems, Tobramycin, Trimethoprim-Sulfomethoxazole
20	Chemotherapy	1083	9–2022	C67 – Bladder cancer C18 – Colon cancer	Urine	NDM + OXA-48		bla_{NDM} + bla_{OXA-48}	Fluoroquinolones, Carbapenems, Tobramycin, Amikacin
21	ICU	457	9–2022	C22 – Liver/ intrahepatic bile duct CA	Urine	KPC + VIM		bla_{KPC}	Fluoroquinolones, Carbapenems, Tobramycin, Amikacin, Gentamicin, Trimethoprim-Sulfomethoxazole
22	ICU	22	9–2022	MVA (motorcycle)	Wound	NDM + VIM	ST15	bla_{NDM} + bla_{VIM}	Pan-drug resistant
23	ICU	1305	7–2022	Chronic cerebellar degeneration, alcoholism, seizures	Wound	KPC + VIM	ST11	bla_{KPC} + bla_{VIM}	Fluoroquinolones, Carbapenems, Tobramycin, Trimethoprim-Sulfomethoxazole
24	ICU	160	7–2022	Shock, hypoxemia	Blood	KPC + VIM	ST323	bla_{KPC} + bla_{VIM}	Fluoroquinolones, Carbapenems, Tobramycin, Amikacin, Gentamycin, Trimethoprim-Sulfomethoxazole
25	ICU	283	7–2022	Acute ischemic stroke	Blood	KPC + VIM	ST323	bla_{KPC} + bla_{VIM}	Fluoroquinolones, Carbapenems, Tobramycin, Amikacin, Gentamicin, Trimethoprim-Sulfomethoxazole

(continued)

Table 2. Continued

ID	Department	Laboratory ID	Time collected	Underlying conditions	Sample	Carbapenemase Type (immunochromatographic assay)	ST	Carbapenem resistance gene (PCR)	Phenotypic Resistance
26	ICU	582	3–2022		Bronchial	NDM + VIM		bla_{NDM} + bla_{VIM}	Pan-drug resistant
27	ICU	1218	10–2023	COVID-19, CAD, HF, CKD, T2DM, PH, anemia	Urine	NDM		bla_{NDM}	Pan-drug resistant
28	ICU	912	12–2023	Intertrochanteric fracture	Bronchial	NDM			Pan-drug resistant
29	2nd Internal	550	1–2023	HBV (+), T2DM, ESRD (HD Tue-Thu-Sat), dyslipidemia, cholecystectomy Covid-19	Urine	KPC		bla_{KPC}	Fluoroquinolones, Carbapenems, Tobramycin, Trimethoprim-Sulfomethoxazole
30	ICU	18	10–2023		Blood	KPC		bla_{KPC}	Fluoroquinolones, Carbapenems, Tobramycin, Amikacin, Gentamicin, Trimethoprim-Sulfomethoxazole
31	2nd Internal	512	1–2023	Ischemic stroke (2015), carotid stents	Urine	OXA-48		bla_{KPC}	Fluoroquinolones, Carbapenems, Tobramycin, Amikacin, Trimethoprim-Sulfomethoxazole
32	1st Internal	1037	5–2023		Urine	NDM + KPC		bla_{NDM} + bla_{KPC}	Fluoroquinolones, Carbapenems, Tobramycin, Amikacin, Gentamicin, Trimethoprim-Sulfomethoxazole
33	ICU	43	1–2024	ICH	Urine	NDM		bla_{NDM}	Pan-drug resistant
34	ICU	38	1–2024		Urine	NDM		bla_{NDM}	Pan-drug resistant
35	ICU	51	1–2024	Polytrauma post-MVA, GCS 15 (reported)	Bronchial	NDM		bla_{NDM}	Pan-drug resistant
36	ICU	147	1–2024	occipital skull fracture	Blood	NDM		bla_{NDM}	Pan-drug resistant
37	1st Internal	123	1–2024		Urine	NDM		bla_{NDM}	Fluoroquinolones, Carbapenems, Tobramycin, Amikacin, Colistin

Footnote: **T2DM**: Type 2 Diabetes Mellitus; **CAD**: Coronary Artery Disease; **PCI:** Percutaneous Coronary Intervention; **HTN**: Hypertension; **R**: Right **GCS**: Glasgow Coma Scale; **iso**: isocoric; **mid**: mid-position; **Abd**: Abdominal **LBO**: Large Bowel Obstruction; **MVA**: Motor Vehicle Accident; **HBV (+)** → Hepatitis B virus positive; **ESRD:** End-Stage Renal Disease; **HD:** Hemodialysis; **ICH**: Intracerebral hemorrhage; **CKD**: Chronic Kidney Disease; **PH**: Pulmonary Hypertension; **HF**: Heart Failure.

Table 3. Multilocus sequence typing of eight *K. pneumoniae* strains

Laboratory ID	ID	Sequence Type
1355	ID 4	ST323
68	ID 6	ST323
497	ID13	ST323
745	ID 19	ST11
22	ID 22	ST15
1305	ID 23	ST11
160	ID 24	ST323
283	ID 25	ST323

over-representing hospital-acquired rather than community-acquired CRKP. Third, access to molecular tests such as Next Generation Sequence was impossible due to limited funding, and susceptibility to newer agents (e.g., cefiderocol, meropenem–vaborbactam) could not be assessed.

6. CONCLUSION

The shifting molecular landscape of CRKP in a non-tertiary hospital in Greece is marked by high clonal diversity, multidrug resistance, and ICU-centered transmission. The emergence of PDR strains represents a growing threat to regional healthcare systems. Ongoing genomic surveillance, expanded diagnostic capacity, and tailored antimicrobial stewardship programs are urgently needed to contain the spread of high-risk clones and preserve therapeutic options.

Author contributions: T.G, K.T., M.S., and E.S. collected and processed the samples. P.T. wrote and edited the manuscript. All authors have read and approved the final version.

Funding: This research received no external funding.

Institutional review board statement: This study was approved by the Scientific Board of Volos Hospital (Protocol 5/22-3-24).

Informed consent statement: Not applicable.

Data availability statement: The data presented in this study are available on request from the corresponding author due to privacy and ethical restrictions, as the dataset contains sensitive patient information that cannot be shared publicly.

Conflicts of interest: The authors declare no conflicts of interest.

ACKNOWLEDGMENTS

The authors would like to thank the staff of the General Hospital of Volos for their technical support. In addition, we would like to thank Professors Petinaki Efthimia and Papagiannitsis Costas of University of Thessaly, Medical School, Department of Microbiology for their guidance and support.

REFERENCES

1. Hafiz TA, Alanazi S, Alghamdi SS, Mubaraki MA, Aljabr W, Madkhali N, et al. *Klebsiella pneumoniae* bacteraemia epidemiology: resistance profiles and clinical outcome of King Fahad Medical City isolates, Riyadh, Saudi Arabia. BMC Infect Dis 2023 Sep 5; 23 (1): 579. PMID: 37670240; PMCID: PMC10481561. https://doi.org/10.1186/s12879-023-08563-8
2. Asokan S, Jacob T, Jacob J, AlSosowaa AA, Cherian T, Peijnenburg WJGM, et al. A growing threat in the era of antimicrobial resistance. The Microbe 2025; 7: 100333. ISSN 2950-1946. https://doi.org/10.1016/j.microb.2025.100333
3. European Centre for Disease Prevention and Control. Carbapenem- and/or colistin-resistant *Klebsiella pneumoniae* in Greece: molecular follow-up survey 2022. Stockholm: ECDC; 2023.
4. Karampatakis T, Tsergouli K, Behzadi P. Carbapenem-resistant *Klebsiella pneumoniae:* virulence factors, molecular epidemiology and latest updates in treatment options. *Antibiotics (*Basel) 2023 Jan 21; 12(2): 234. PMID: 36830145; PMCID: PMC9952820. https://doi.org/10.3390/antibiotics12020234
5. Galani I, Karaiskos I, Souli M, Papoutsaki V, Galani L, Gkoufa A, et al. Outbreak of KPC-2-producing *Klebsiella pneumoniae* endowed with Ceftazidime-avibactam resistance mediated through a VEB-1-mutant (VEB-25), Greece, September to October **2019**. Eurosurveillance 2020; 25: 2000028.
6. Galani I, Karaiskos I, Souli M, Papoutsaki V, Gkoufa A, Antoniadou A, et al. Outbreak of high-risk clone ST323 *Klebsiella pneumoniae* resistant to ceftazidime–avibactam due to acquisition of blaVEB-25 and to cefiderocol due to mutated fiu gene. Antibiotics 2025; 14: 223. https://doi.org/10.3390/antibiotics14030223
7. Galani I, Nafplioti K, Adamou P, Karaiskos I, Giamarellou H, Souli M., Study Collaborators. Nationwide epidemiology of carbapenem resistant *Klebsiella pneumoniae* isolates from Greek hospitals, with regards to plazomicin and aminoglycoside resistance. BMC Infect Dis Feb 15 2019; 19(1): 167. Erratum in: BMC Infect Dis. **2019** Mar 7; 19(1): 230. 10.1186/s12879-019-3854-1. PMID: 30770727; PMCID: PMC6377745. https://doi.org/10.1186/s12879-019-3801-1
8. Karampatakis T, Antachopoulos C, Iosifidis E, Tsakris A, Roilides E. Molecular epidemiology of carbapenem-resistant *Klebsiella pneumoniae* in Greece. Future Microbiol 2016 Jun; 11: 809–23. Epub 2016 May 20. PMID: 27206024. https://doi.org/10.2217/fmb-2016-0042
9. Kontopoulou K, Meletis G, Pappa S, Zotou S, Tsioka K, Dimitriadou P, et al. Spread of NDM-producing *Klebsiella pneumoniae* in a tertiary Greek hospital. Acta Microbiol Immunol Hung 2021; 68(3): 162–8. https://doi.org/10.1556/030.2021.01400
10. Meletis G, Skoura L. Polymyxin resistance mechanisms: from intrinsic resistance to mcr genes. Recent Pat Antiinfect Drug Discov 2018; 13(3): 198–206. https://doi.org/10.2174/1574891X14666181126142704

11. Chatzidimitriou M, Tsolakidou P, Kyriazidi MA, Chatzopoulou F, Varlamis S, Mavridou M, et al. Identification of NDM-1 producing and colistin resistant *Klebsiella pneumoniae* ST11: a highly drug-resistant strain detected in intensive care unit of a Greek tertiary care hospital. Acta Microbiol Immunol Hung 2025; 72(1): 16–22. https://doi.org/10.1556/030.2025.02499
12. Tsolakidou P, Papadimitriou A, Kyriazidis KA, Ilias P, Mitka S, Chatzidimitriou M. Characterization of a carbapenemase-producing *Klebsiella pneumoniae* isolate of a patient in an intensive care unit in Greece: a study of resistome, virulome, and mobilome. Acta Microbiol Immunol Hung 2024; 71(4): 295–8. https://doi.org/10.1556/030.2024.02468
13. Tsolakidou P, Kyriazidi MA, Varlamis S, Chatzopoulou F, Frydas I, Kyriazidis KA, et al. NDM-1 and VIM-1 dual metallo-beta-lactamase producing *Klebsiella pneumoniae* ST15 high-risk clone from a blood culture of a patient at Intensive Care Unit in a Greek Tertiary Care Hospital. Acta Microbiol Immunol Hung 2025; 72(2): 93–8. https://doi.org/10.1556/030.2025.02559