1828
AKADÉMIAI KIADÓ

Acta Microbiologica et Immunologica Hungarica

72 (2025) 2, 93–98

DOI:
10.1556/030.2025.02559
© 2025 The Author(s)

RESEARCH ARTICLE

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

Pandora Tsolakidou[1], Maria Anna Kyriazidi[2], Sotiris Varlamis[3], Fani Chatzopoulou[3], Ilias Frydas[3], Kyriazis Athanasios Kyriazidis[2], Kallirhoe Kalinderi[3], Stella Mitka[3], Petros Skepastianos[3] and Maria Chatzidimitriou[3]*

[1] Hospital of Volos, Polymeri 134, 38222, Volos, Greece

[2] Medical School, Faculty of Health Sciences, Aristotle University of Thessaloniki, Greece

[3] Department of Biomedical Sciences, School of Health Sciences, International Hellenic University, 5400, Thessaloniki, Greece

Received: February 21, 2025 • Accepted: March 14, 2025
Published online: March 24, 2025

ABSTRACT

The emergence of carbapenemase-producing *Klebsiella pneumoniae* poses a significant global health threat, particularly in hospital settings. This study reports on the first detection of a pandrug-resistant (PDR) high-risk ST15 *K. pneumoniae* strain co-producing NDM-1 and VIM-1 in Greece. The isolate was recovered from a blood culture of a male patient admitted to the Intensive Care Unit (ICU) of Volos Hospital in July 2024. Next generation Sequencing (NGS) confirmed the presence of $bla_{\text{NDM-1}}$ and $bla_{\text{VIM-1}}$ genes. Other beta-lactamase type (CTX-M-15) was detected in association with NDM and VIM enzymes. Furthermore, this isolate was resistant to other antimicrobial agents, including aminoglycosides [*aac(3)-II, aac(3)-IIe, aac(6′)-Ib, aadA1, aph(3″)-Ib, aph(6)-Id, aph(3′)-Ia),* chloramphenicol (*catB3*), fluoroquinolones (*qnrS1*) and sulfonamides (*sul1* and *sul2*). The Multilocus Sequence Typing revealed that the strain belonged to ST15. According to Kaptive the strain belonged to KL48. Our study provides new data about MBL producing *K. pneumoniae* in Greece. Thus, we report for the first time the co-expression of $bla_{\text{NDM-1}}$ and $bla_{\text{VIM-1}}$ in our country in ST15 *K. pneumoniae*. This study provides crucial epidemiological data on MBL-producing *K. pneumoniae* in Greece and highlights the urgent need for enhanced surveillance, infection control strategies, and access to last-resort antibiotics such as aztreonam-avibactam.

KEYWORDS

Klebsiella pneumoniae ST15, NDM-1, VIM-1, carbapenem resistance, metallo-beta-lactamase, antimicrobial resistance, ICU infections

INTRODUCTION

A significant challenge in treating infections caused by *Klebsiella pneumoniae* is the production of carbapenemase enzymes, which confer resistance to beta-lactam antibiotics [1]. The availability of effective treatment options is increasingly limited. Newer beta-lactamase inhibitors, such as avibactam and vaborbactam, are used for infections caused by KPC-producing *K. pneumoniae* [2]. However, for metallo-beta-lactamase (MBL)-producing

*Corresponding author.
E-mail: mchatzid952@gmail.com

AKJournals

Brought to you by MTA Könyvtár és Információs Központ olvasók | Unauthenticated | Downloaded 07/21/25 11:28 AM UTC

strains, treatment relies on aztreonam-avibactam and cefiderocol, which are available in certain countries but not in Greece [3, 4]. Additionally, aztreonam—potentially effective against such infections—is unavailable in many Greek hospitals due to financial constraints. Despite its relatively low cost, pharmaceutical companies in Greece are reluctant to distribute aztreonam nationwide, further limiting treatment options for patients with MBL-producing infections.

In addition, the co-existence of NDM-producing *K. pneumoniae* with other carbapenemases has been widely reported across the globe, highlighting the increasing complexity of antimicrobial resistance and the challenges in treatment strategies [5–10]. According to this phenomenon many studies from Greece have documented the double carbapenemase producers [11, 12]. An NDM-1 and VIM-1 co-producing *K. pneumoniae* strain belonging to sequence type (ST) 11 was first reported in Greece by Papagiannitsis et al. in 2017 [7].

The ST15 lineage has been increasingly associated with multidrug resistance and nosocomial outbreaks worldwide. An ST15 KPC-2-producing *K. pneumoniae* outbreak was reported in Bulgaria in 2015 by Markovska R. et al. [9]. An NDM-1-producing ST15 *K. pneumoniae* strain was reported in Bulgaria in 2017 by Savov et al. [10]. In Greece, previous studies have reported ST15 strains carrying NDM-1 (Politi et al., 2019) but this is the first report of an ST15 strain harboring both NDM-1 and VIM-1. To the best of our knowledge, this is the first report of a pandrug-resistant (PDR) *K. pneumoniae* strain co-producing NDM and VIM belonging to the high-risk ST15 clone in Greece. The presence of such highly resistant strains in the Greek healthcare system is concerning, given the limited access to novel combination therapies such as aztreonam-avibactam. Additionally, the economic barriers limiting the availability of aztreonam in many Greek hospitals further exacerbate the challenges in treating such infections.

MATERIALS AND METHODS

The *K. pneumoniae* A436 strain was isolated from a positive blood culture of a male patient hospitalized in the Intensive Care Unit (ICU) of Volos Hospital, Greece, in July 2024.

The identification and antimicrobial susceptibility testing were conducted using the Vitek-2 automated system (Biomerieux, Marcy-l'Étoile, France). The susceptibility testing for newer beta-lactam/beta-lactamase inhibitor combinations was performed using gradient E-tests (Liofilchem). The determination of minimal inhibition concentration was performed according to Eucast guidelines (Eucast 2024, assessed on July 2024).

The detection of carbapenemase enzymes was carried out using immunochromatographic assay (NG Biotech).

Next Generation Sequencing was performed in a private laboratory in Greece. Libraries were prepared using Ion Torrent technology and Ion Chef workflows (Thermo Scientific). Sequencing was performed in the S5XLS system and analysis of primary data was conducted with Ion Torrent Suite v.5.10.0.

Genome assembly was performed with Spades (Galaxy Version 3.15.5+galaxy2). Quast Genome assembly Quality (Galaxy Version 5.3.0+galaxy0) was used in order to assess the quality of the assembly. Resistance profiling was performed with AMR Finder plus (Galaxy Version 3.12.8+galaxy0). Mlst via (Galaxy Version 2.22.0). Replicons were detected via PlasmidFinder (Galaxy Version 2.1.6+galaxy1). Integrons were detected via Integron Finder (Galaxy Version 2.0.5+galaxy0). Kaptive was used in order to find the K locus group of the strain (https://kaptive-web.erc.monash.edu/).

RESULTS

Antimicrobial susceptibility testing

The isolate demonstrated pandrug-resistance (PDR), exhibiting high-level resistance to all tested beta-lactams, aminoglycosides, fluoroquinolones, and colistin. The minimum inhibitory concentration (MIC) values and their respective interpretations are summarized in Table 1.

The isolate was resistant to all beta-lactams, including carbapenems and cephalosporins. It also showed high-level resistance to aminoglycosides, fluoroquinolones, colistin, and trimethoprim/sulfamethoxazole. The resistance to tigecycline (MIC = 1 μg mL^{-1}) was noted, despite its activity against certain carbapenem-resistant strains.

Genotypic characterization

Whole-genome sequencing (WGS) analysis revealed that the isolate belonged to the ST15 lineage, a high-risk clone associated with multidrug resistance and nosocomial outbreaks. The strain was found to harbor both $bla_{\text{VIM-1}}$ and $bla_{\text{NDM-1}}$ genes, encoding metallo-beta-lactamases responsible for carbapenem resistance. Additional beta-lactamase genes detected included $bla_{\text{CTX-M-15}}$, $bla_{\text{TEM-1}}$, $bla_{\text{OXA-1}}$, and $bla_{\text{SHV-28}}$ (Table 2).

Whole-genome sequencing identified mutations possibly associated with colistin resistance in the *K. pneumoniae* A436 isolate. Specifically, mutations in the *lpxM* (*S253G*) and *arnC* (*S30T*). The *lpxM*_S253G, a mutation in the *lpxM* gene, which is involved in the lipid A biosynthesis pathway, potentially altering the lipopolysaccharide (LPS) structure, leading to reduced colistin binding and resistance.

ArnC_S30T a mutation in the *arnC* gene, which plays a role in the addition of 4-amino-4-deoxy-L-arabinose (L-Ara4N) to lipid A, a modification known to reduce colistin susceptibility. These findings suggest that chromosomal modifications rather than plasmid-mediated *mcr* genes are responsible for the high-level colistin resistance observed in this isolate.

The integron of *K. pneumoniae* ST15, harbors multiple resistance genes, contributing to its multidrug-resistant phenotype. The integron includes:

Brought to you by MTA Könyvtár és Információs Központ olvasók | Unauthenticated | Downloaded 07/21/25 11:28 AM UTC

Table 1. Susceptibility testing of *K. pneumoniae* A436 strain

Antimicrobial	MIC (μg mL^{-1})	Interpretation
Ertapenem	≥8	R
Ampicillin	≥32	R
Amoxicillin/Clavulanic Acid	≥32	R
Ampicillin/Sulbactam	≥32	R
Ticarcillin/Clavulanic Acid	≥128	R
Piperacillin	≥128	R
Piperacillin/Tazobactam	≥128	R
Cefalotin	≥64	IE
Cefuroxime	≥64	R
Cefuroxime Axetil	≥64	R
Cefoxitin	≥64	IE
Cefixime	≥4	R
Cefotaxime	≥64	R
Ceftazidime	≥64	R
Ceftazidime-avibactam	≥64	R
Ceftriaxone	≥64	R
Cefepime	≥64	R
Aztreonam	≥64	R
Imipenem	≥16	R
Meropenem	≥16	R
Amikacin	32	R
Gentamicin	≥16	R
Tobramycin	≥16	R
Nalidixic Acid	(−)	R
Ciprofloxacin	≥4	R
Levofloxacin	≥8	R
Moxifloxacin	≥8	R
Ofloxacin	≥8	R
Tigecycline	1	R
Chloramphenicol	≤2	IE
Colistin	≥16	R
Trimethoprim/ Sulfamethoxazole	≥320	R

- **Efflux pump gene (*qacEΔ1*)**, which may play a role in resistance to disinfectants and antiseptics.
- **Aminoglycoside resistance genes (*aadA1*, *aac(6′)-II*)**, encoding enzymes that modify aminoglycosides and confer resistance.
- **Trimethoprim resistance gene (*dfrA1*)**, which affects susceptibility to trimethoprim by encoding an alternative dihydrofolate reductase.
- **Carbapenemase gene (*bla*$_{\text{VIM-1}}$)**, encoding a Verona Integron-encoded Metallo-beta-Lactamase (VIM-1), a key enzyme conferring resistance to carbapenems.

This integron-mediated accumulation of resistance genes enhances *K. pneumoniae* ST15's ability to survive under antimicrobial pressure, making it a high-risk multidrug-resistant (MDR) clone with limited treatment options.

Virulence and plasmid analysis

Virulence factor analysis identified the presence of Type 1 and Type 3 fimbriae genes (mrk and fim clusters), the yersiniabactin siderophore, and genes associated with capsular polysaccharide production (Table 3).

Plasmid replicon typing identified the presence of IncA/C2, IncFIA(HI1), IncFIB(K), and IncFII(K) plasmids,

Table 2. Resistance genes of *K. pneumoniae* A436 strain

Resistance gene	Function	Targeted antibiotics
bla$_{\text{VIM-1}}$, *bla*$_{\text{NDM-1}}$	metallo-beta-lactamases (class B)	Carbapenems
oqxA, oqxB	Multidrug efflux RND transporter periplasmic adaptor subunit OqxA, OqxB	Multiple drug classes
bla$_{\text{SHV-28}}$, *bla*$_{\text{TEM-1}}$, *bla*$_{\text{OXA-1}}$	Broad-spectrum beta-lactamase SHV-28, TEM-1 (class A); oxacillin-hydrolyzing beta-lactamase OXA-1 (class D)	beta-lactams
bla$_{\text{CTX-M-15}}$	Extended-spectrum beta-lactamase CTX-M-15 (class A)	Third generation Cephalosporins, beta-lactams
*parC*_S80I, *gyrA*_D87A, *gyrA*_S83F	*K. pneumoniae* quinolone-resistant ParC, GyrA	Fluoroquinolones
qnrS1	Quinolone resistance pentapeptide repeat protein QnrS1	Fluoroquinolones
fosA	FosA5 family fosfomycin resistance glutathione transferase	Fosfomycin
sul1, sul2	Sulfonamide-resistant dihydropteroate synthase Sul1, Sul2	Sulfonamides
dfrA1, dfrA14	Trimethoprim-resistant dihydrofolate reductase DfrA1, DfrA14	Trimethoprim
aph(3″)-Ib, *aph(6)-Id*, *aph(3′)-Ia*	Aminoglycoside O-phosphotransferase APH(3″)-Ib, APH(6)-Id, APH(3′)-Ia	Aminoglycosides
aadA1	ANT(3″)-Ia family aminoglycoside nucleotidyltransferase AadA1	Aminoglycosides
aac(6′)-Il, *aac(3)-IIe*, *aac(6′)-Ib*	Aminoglycoside N-acetyltransferase AAC(6′)-Il, AAC(3)-IIe, AAC(6′)-Ib	Aminoglycosides
qacEdelta1	Quaternary ammonium compound efflux SMR transporter QacE delta 1	Disinfectants, biocides
mph(A)	*Mph(A)* family macrolide 2′-phosphotransferase	Macrolides
ble	Bleomycin binding protein Ble-MBL	Bleomycin
catB3	Type B-3 chloramphenicol O-acetyltransferase CatB3	Chloramphenicol

Brought to you by MTA Könyvtár és Információs Központ olvasók | Unauthenticated | Downloaded 07/21/25 11:28 AM UTC

Table 3. Virulence of the study strain *K. pneumoniae* A436

Category	Genes	Locus
Type 3 fimbriae	**mrk (B, C, D, F, H, I, J)**	**orf03846, orf03845, orf03844, orf03843, orf03840, orf03841, orf03842**
Type I fimbriae	fimA, fimB, **fim(C, D, E, F, G, H, I, K)**	orf04753, orf04756, **orf02699; orf04751, orf02698; orf03632; orf04750, orf04754, orf04749; orf05254, orf04748, orf04747; orf05253, orf04752, orf04746; orf05252**
Capsule	–	**orf00410 - orf00416; orf00427 - orf00433; orf04379**
AcrAB	acrA, acrB	orf01193, **orf01192; orf02899**
Aerobactin	iutA	**orf02105**
Ent siderophore	entA, entB, entC, entD, entE, entF, entS, **fep(A, B, C, D, G), fes**	orf02519, orf02518, orf02516, orf02505, orf02517, orf02509, orf02514, **orf01535; orf02506, orf02515, orf02510, orf02512, orf02511, orf02507**
Salmochelin	iroE, iroN	**orf03382, orf01702**
Yersiniabactin	fyuA, irp1, irp2, ybt(A, E, P, Q, S, T, U, X)	orf00515, orf00519, orf00520, **orf00521, orf00516, orf00522, orf00523, orf00525, orf00517, orf00518, orf00524**
RcsAB	rcsA, rcsB	orf00546, orf00273
T6SS-I	clpV/tssH, dotU/tssL, hcp/tssD, icmF/tssM, impA/tssA, ompA, sciN/tssJ, **tss(F, G), vasE/tssK, vgrG/tssI, vip(A, B)**	**orf04830, orf04833, orf04831, orf03111, orf03112, orf04832, orf03117, orf03115; orf03736, orf03116, orf04834, orf05465, orf04836, orf04835**
T6SS-II	clpV, dotU, icmF, imp(F, H, J), ompA, sciN, vgrG	orf02902, orf03729, orf03735, **orf03739, orf03737, orf03728, orf03730, orf03738, orf03731**
T6SS-III	dotU, icmF, imp(A, F, G, H, J), ompA, sciN	orf04605, orf02295, **orf02300, orf02299, orf02296, orf02297, orf04606, orf04604, orf02298**

Bold: Virulence of *K. pneumoniae* A436 strain.

which are commonly associated with carbapenemase-encoding genes and multidrug resistance.

Genome assembly and quality metrics

The genome assembly analysis yielded a total of 209 contigs, with a total genome length of 5,719,458 bp. The largest contig measured 689,525 bp, and the N50 value was 198,607 bp, indicating a well-assembled genome (Table 4).

DISCUSSION

ST15 *K. pneumoniae* is a high-risk lineage that has expanded across different regions. It has been associated with $bla_{\text{KPC-2}}$ gene in the past, but later on it has been associated in the Balkan region with $bla_{\text{NDM-1}}$, as lineage ST11. ST15 is recognized as a high-risk clone associated with multidrug resistance and has been implicated in various outbreaks globally. The KL48 capsular type, while less commonly reported in association with ST15, has been identified in certain studies. For instance, a population genomic analysis of clinical ST15 *K. pneumoniae* strains in China identified four clades, with one clade (C3) associated with the KL48 capsular type, although this clade represented a small proportion (0.7%) of the studied strains [13].

The study isolate A436 exhibited resistance to all tested beta-lactams, aminoglycosides, fluoroquinolones, and colistin, underscoring the critical limitation of available treatment options. The detection of both bla_{NDM-1} and bla_{VIM-1} indicates a dual metallo-beta-lactamase (MBL) production, a rare but increasingly reported mechanism in multidrug-resistant *K. pneumoniae* strains [5–7]. This co-production of MBLs renders all carbapenems ineffective, with aztreonam-avibactam remaining as the only potential treatment option, which, however, is not currently available in Greece. The absence of *mcr* genes suggests that colistin resistance is likely due to chromosomal mutations in the *lpxM* and *arnC* genes, leading to modifications in lipid A and reducing colistin binding.

Given the rapid evolution and dissemination of carbapenemase-producing *K. pneumoniae*, enhanced infection control measures are urgently needed. Routine surveillance, strict antimicrobial stewardship programs,

Table 4. Genome Assembly Metrics for *K. pneumoniae* A436 strain

Statistic	Value
# Contigs	103
# Contigs (≥0 bp)	209
# Contigs (≥1,000 bp)	83
Largest Contig	689,525
Total Length	5,685,559
Total Length (≥0 bp)	5,719,458
Total Length (≥1,000 bp)	5,672,811
N50	198,607
N90	62,718
auN	242,994
L50	10
L90	29
GC (%)	56.96
Per Base Quality	–
#N's per 100 kbp	0
#N's	0

Brought to you by MTA Könyvtár és Információs Központ olvasók | Unauthenticated | Downloaded 07/21/25 11:28 AM UTC

and rapid molecular diagnostics should be prioritized to limit the spread of PDR pathogens. Furthermore, the monitoring of integron-carrying strains is essential, as they represent a major reservoir for the horizontal gene transfer of resistance determinants. Whole-genome sequencing (WGS) and epidemiological tracking of high-risk clones like ST15 can provide valuable insights into their transmission dynamics and guide targeted containment strategies.

A major limitation of the study is the absence of phylogenic correlation with other strains ST15 of the Hospital of Volos. Such comparative analyses are crucial for understanding the genetic relationships, potential transmission pathways, and evolutionary dynamics of this high-risk lineage within the hospital setting. Thus, incorporating phylogenetic comparisons with local ST15 strains would enhance the study's findings by providing a clearer picture of the genetic landscape and transmission dynamics of *K. pneumoniae* within the Hospital of Volos.

CONCLUSION

This study highlights the first detection of a PDR *K. pneumoniae* ST15 strain co-producing NDM-1 and VIM-1 in Greece, emphasizing the growing threat of metallo-beta-lactamase-producing *K. pneumoniae* in hospital settings. The integron-mediated accumulation of resistance genes in this strain further complicates treatment options and underscores the need for urgent infection control measures, antimicrobial stewardship programs, and access to last-resort antibiotics. Given the global dissemination of ST15 high-risk clones, continuous surveillance is critical to prevent the establishment and spread of such extensively resistant pathogens.

Author contributions: CM: conceptualization methodology and design of the study, resources, data curation, writing—original draft preparation, writing—review and editing. TP: writing- original draft preparation. MAK: review and editing. SV: laboratory testing. FC: laboratory testing. IF: laboratory testing and analysis. KAK: software, validation, formal analysis. KK: editing. MS: writing—review and editing. PS: editing. All authors have read and agreed to the published version of the manuscript.

Funding: This study received no external funding.

Institutional review board statement: Not applicable.

Informed consent statement: Not applicable.

Data availability statement: The whole genome of *K. pneumoniae* has been deposited at DDBJ/ENA/GenBank under the accession Number PRJNA1222132.

REFERENCES

1. Huy TXN. Overcoming *Klebsiella pneumoniae* antibiotic resistance: new insights into mechanisms and drug discovery. Beni-Suef Univ J Basic Appl Sci 2024; 13: 13. https://doi.org/10.1186/s43088-024-00470-4.
2. Ackley R, Roshdy D, Meredith J, Minor S, Anderson WE, Capraro GA, et al. Meropenem-vaborbactam versus ceftazidime-avibactam for treatment of carbapenem-resistant *Enterobacteriaceae* infections. Antimicrob Agents Chemother 2020; 64(5): e02313–19. https://doi.org/10.1128/AAC.02313-19.
3. Sader HS, Castanheira M, Kimbrough JH, Kantro V, Mendes RE. Aztreonam/avibactam activity against a large collection of carbapenem-resistant Enterobacterales (CRE) collected in hospitals from Europe, Asia and Latin America (2019–21). JAC Antimicrob Resist 2023; 5(2): dlad032. https://doi.org/10.1093/jacamr/dlad032.
4. Kimbrough JH, Maher JM, Sader HS, Castanheira M, Mendes RE. *In vitro* activity assessment of cefiderocol against Enterobacterales, *Pseudomonas aeruginosa,* and *Acinetobacter* spp., including β-lactam nonsusceptible molecularly characterized isolates, collected from 2020 to 2021 in the United States and European hospitals. Microbiol Spectr 2024; 12(11): e0147424. https://doi.org/10.1128/spectrum.01474-24.
5. Flores C, Bianco K, de Filippis I, Clementino MM, Romão CMCPA. Genetic relatedness of NDM-producing *Klebsiella pneumoniae* Co-occurring VIM, KPC, and OXA-48 enzymes from surveillance cultures from an intensive care unit. Microb Drug Resist 2020; 26(10): 1219–26. https://doi.org/10.1089/mdr.2019.0483.
6. Thapa S, Adhikari N, Shah AK, Lamichhane I, Dhungel B, Shrestha UT, et al. Detection of NDM-1 and VIM genes in carbapenem-resistant *Klebsiella pneumoniae* isolates from a tertiary health-care center in Kathmandu, Nepal. Chemotherapy 2 December 2021; 66(5–6): 199–209. https://doi.org/10.1159/00051825.
7. Papagiannitsis CC, Malli E, Florou Z, Sarrou S, Hrabak J, Mantzarlis K, et al. Emergence of sequence type 11 *Klebsiella pneumoniae* coproducing NDM-1 and VIM-1 metallo-β-lactamases in a Greek hospital. Diagn Microbiol Infect Dis 2017; 87(3): 295–7. https://doi.org/10.1016/j.diagmicrobio.2016.12.008.
8. Politi L, Gartzonika K, Spanakis N, Zarkotou O, Poulou A, Skoura L, et al. Emergence of NDM-1-producing *Klebsiella pneumoniae* in Greece: evidence of a widespread clonal outbreak. J Antimicrob Chemother 2019; 74(8): 2197–202. https://doi.org/10.1093/jac/dkz176.
9. Markovska R, Stoeva T, Schneider I, Boyanova L, Popova V, Dacheva D, et al. Clonal dissemination of multilocus sequence type ST15 KPC-2 producing *Klebsiella pneumoniae* in Bulgaria. APMIS 2015; 123: 887–94.
10. Savov E, Politi L, Spanakis N, Trifonova A, Kioseva E, Tsakris A. NDM-1 hazard in the Balkan States: evidence of the first outbreak of NDM-1-producing *Klebsiella pneumoniae* in Bulgaria. Microb Drug Resist 2018; 24(3): 253–9. https://doi.org/10.1089/mdr.2017.0230.
11. Chatzidimitriou M, Tsolakidou P, Voulgaridis A, Kyriazidi MA, Chatzopoulou F, Mavridou M, et al. NDM-1 and KPC-3 co-producing *Klebsiella pneumoniae* ST512 in bronchial secretion from a patient in an intensive care unit of a Greek Tertiary Care Hospital.

Brought to you by MTA Könyvtár és Információs Központ olvasók | Unauthenticated | Downloaded 07/21/25 11:28 AM UTC

Acta Microbiol Immunol Hung 2024; 71(4): 289–94. https://doi.org/10.1556/030.2024.02464.

12. Chatzidimitriou M, Tsolakidou P, Panagiota C, Mylona E, Mitka S. KPC-2 and VIM-1 producing *Klebsiella pneumoniae* ST39 high-risk clone isolated from a clinical sample in Volos, Greece. Acta Microbiol Immunol Hung 2024; 71(1): 43–51. https://doi.org/10.1556/030.2024.02226.
13. Feng L., Zhang M., Fan Z. Population genomic analysis of clinical ST15 *Klebsiella pneumoniae* strains in China. Front Microbiol 2023; 14: 1272173. https://doi.org/10.3389/fmicb.2023.1272173.

Open Access statement. This is an open-access article distributed under the terms of the Creative Commons Attribution-NonCommercial 4.0 International License (https://creativecommons.org/licenses/by-nc/4.0/), which permits unrestricted use, distribution, and reproduction in any medium for non-commercial purposes, provided the original author and source are credited, a link to the CC License is provided, and changes – if any – are indicated.

Brought to you by MTA Könyvtár és Információs Központ olvasók | Unauthenticated | Downloaded 07/21/25 11:28 AM UTC