PUBLICATIONS ON OUR RESIST RANGE

NEW

Exploring New Delhi Metallo Beta Lactamases in *Klebsiella pneumoniae* and *Escherichia coli*: genotypic vs. phenotypic insights- 2025 - *Ann Clin Microbiol Antimicrob*

Early detection of OXA-48 producing *Klebsiella pneumoniae* with the use of rapid antibiotic susceptibility testing - 2024 - *Eur J Clin Microbiol Infect Dis*

RESIST ACINETO test for the rapid detection of NDM and OXA acquired carbapenemases directly from blood culture in Acinetobacter species - 2024 - *Microbiol Spectr.*

Performance evaluation of the newly developed *in vitro* rapid diagnostic test for detecting OXA-48-like, KPC-, NDM-, VIM- and IMP- type carbapenemases: the RESIST-5 O.K.N.V.I. Multiplex Lateral Flow Assay - 2021 - *Antibiotics (Basel)*

Comparison of three lateral flow immunochromatographic assays for the rapid detection of KPC, NDM, IMP, VIM and OXA-48 carbapenemases in Enterobacterales - 2022 - *J Antimicrob Chemother.*

Assessing O.K.N.V.I. RESIST-5 performance for post-mortem biological samples: A prospective pilot study - 2023 - *Exp Ther Med.*

Carbapenem-resistant organisms isolated in surgical site infections in Benin: A public health problem - 2022 - *Trop Med Infect Dis.*

RESIST Acineto rapid immunological test for the detection of acquired carbapenemase producers among Acinetobacter spp - 2023 - *Diagn Microbiol Infect Dis.*

Evaluation of RESIST ACINETO immunochromatographic assay from positive blood cultures - 2023 - *J Antimicrob Chemother.*

Comparison of two immunochromatographic tests for the detection of CTX-M ESBL on clinical isolates at the Belgian National Reference Centre - 2023 - *ECCMID*

AntiMicrobial Resistance

Pressbook

V5

A selection of the most recent publications on AMR

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HIGHLIGHTS

Targeted action and financing the fight against AMR in Asia - 2025 - *World Economic Forum report*

◊ Asia accounts for over half of global AMR-related deaths, a toll projected to exceed 4 million annually by 2050, with an economic burden of $550–700 billion per year

◊ Crisis driven by poor healthcare access, counterfeit/misused antibiotics, unsustainable farming, inadequate WASH, & climate change

◊ Coordinated action in education, prevention, monitoring, & treatment could save up to $50 billion annually via a One Health approach.

Prevalence and molecular epidemiology of carbapenem resistance in Asia - 2025 - *Syst. Rev.*

◊ Carbapenem resistance among Gram-negative pathogens in Asia is alarmingly high, with pooled prevalence estimates exceeding 50% for *A. baumannii* and over 20% for *K. pneumoniae* in several subregions

◊ Dominant carbapenemase genes vary geographically, with bla_{OXA-23} prevalent in *A. baumannii*, bla_{NDM} and $bla_{OXA-48-like}$ common in *K. pneumoniae*, and bla_{IMP} frequently detected in *P. aeruginosa*

◊ Significant inter-country heterogeneity reflects differences in surveillance coverage, infection control measures, and antimicrobial use practices. Urgent need for regionally coordinated AMR surveillance, molecular typing, and targeted interventions.

FOCUS ON *PSEUDOMONAS AERUGINOSA*

Multi-omics profiling of cross-resistance between CZA and meropenem identifies common strain-specific mechanisms in *P. aeruginosa* clinical isolates - 2025 - *J Clin Microbiol*

◊ Multi-omics analysis of *P. aeruginosa* clinical isolates resistant to both ceftazidime-avibactam (CZA) and meropenem revealed shared adaptive responses, including downregulation of OprD porin and overexpression of efflux systems

◊ Strain-specific resistance patterns were linked to variations in AmpC β-lactamase regulation, mutations in peptidoglycan recycling pathways, and distinct transcriptional profiles affecting cell envelope functions

◊ Cross-resistance was not solely explained by carbapenemase production, highlighting the role of non-enzymatic mechanisms and adaptive regulatory changes

◊ The findings underscore the complexity of resistance evolution in *P. aeruginosa* and support the need for molecular diagnostics that can detect both enzymatic and non-enzymatic determinants of multidrug resistance.

***In vitro* activity of cefiderocol against ESBL-producing and CRPA** - 2025 - *JAC Antimicrob Resist*

◊ Cefiderocol showed strong *in vitro* activity against ESBL-producing and carbapenem-resistant *P. aeruginosa*, including most MBL producers

◊ Reduced susceptibility was mainly linked to efflux pump overexpression or porin loss rather than β-lactamase hydrolysis

◊ Results support cefiderocol as a promising option for MDR *P. aeruginosa*, while underscoring the need for ongoing resistance monitoring.

Antimicrobial resistance and mortality in CRPA infections in Southern Thailand - 2025 - *Antibiotics*

◊ CRPA showed high 30-day mortality (≈30%) with risk concentrated in patients with high comorbidity, sepsis/septic shock; timely active therapy was protective

◊ Mechanisms clustered around MexAB-OprM overexpression, OprD downregulation, MBL production, and AmpC overexpression - implicating both permeability/efflux and enzyme-mediated routes

◊ Regional signal: higher MBL prevalence than other Thai regions, prompting earlier consideration of MBL-active strategies (e.g., ATM-AVI) and rapid mechanism-aware diagnostics.

Genomic characterization of CRPA from ICU admission screening in Hanoi, Vietnam - 2025 - *J. Glob. Antimicrob. Resist.*

◊ Genomic analysis of Carbapenem-resistant *P. aeruginosa* from ICU admission screening in Hanoi revealed a predominance of high-risk clones ST235 and ST357 carrying bla_{IMP-15} and bla_{NDM-1}

◊ Multiple isolates harbored additional resistance determinants, including aminoglycoside-modifying enzymes and fluoroquinolone resistance mutations, indicating extensive multidrug resistance

◊ Findings underscore the role of active admission screening in detecting and controlling the spread of high-risk CRPA clones in healthcare settings.

Risk factors and outcomes of MDR-PA in Kelantan, Malaysia: a multicenter case-control study - 2025 - *Saudi J. Med. Med. Sci.*

◊ Nearly half of MDR isolates carried bla_{NDM-1}; ceftolozane-tazobactam retained the highest activity among β-lactams (≈41% susceptible), guiding empirical choices when BL/BLI options are limited

◊ Independent risk factors for MDR acquisition included genito-urinary disease and central venous catheter; MDR associated with markedly higher treatment failure (41%) and mortality (40%)

◊ Practical implication: early mechanism-aware therapy and device stewardship are central to outcome gains.

FOCUS ON KPC-31 & KPC-33

Evaluation of phenotypic and genotypic methods for detecting KPC variants - 2025 - *Antimicrob Agents Chemother*

◊ The study assessed phenotypic and PCR-based methods for detecting carbapenemases in Enterobacterales and Pseudomonas aeruginosa, with a specific focus on distinguishing KPC enzymes with true carbapenemase activity from ESBL-like KPC variants

◊ Phenotypic methods showed reduced sensitivity for detecting ESBL-like KPC variants, which remain susceptible to carbapenems but resistant to CZA, potentially leading to misclassification and suboptimal treatment decisions

◊ PCR correctly identified both KPC and KPC-like variants at the gene level, but without additional sequencing or targeted assays, it could not determine carbapenemase activity versus ESBL-like behaviour

◊ For CZA resistance detection, molecular methods were more reliable in identifying underlying resistance genes, whereas some phenotypic assays missed isolates with non-enzymatic CZA resistance mechanisms

KPC variants resistant to CZA: an evolutionary overview - 2022 - *Antimicrob Agents Chemother*

◊ After CZA rollout, KPC evolved along three mutation hot spots, with an unusually high share of insertions/deletions (~63% of CZA–resistant clinical variants), often as small duplications, underpinning its rapid, convergent adaptation

◊ These adaptations carry a consistent evolutionary trade-off: many variants lose carbapenemase efficiency, shifting to ESBL-like phenotypes (restored susceptibility to meropenem/imipenem or selective loss limited to ertapenem/meropenem). KPC's exceptionally high thermostability (higher melting temperature than TEM/CTX-M) cushions the fitness cost, explaining why KPC tolerates large indels and evolves fast under CZA pressure, including on-therapy emergence and occasional reversion

◊ Diagnostic/therapy takeaway: use of variant-aware algorithms - PCR/LFIA first line, complemented by targeted media or hydrolysis where needed - to separate true carbapenemase from ESBL-like KPC and pivot promptly to MVB or I-R when indicated.

Meropenem-Vaborbactam as salvage therapy for Ceftazidime-Avibactam - 2021 - *OFID*

◊ KPC-31 is a D179Y mutation of KPC-3 that acts as an ESBL-like enzyme: it confers resistance to ceftazidime-avibactam (CZA) but restores susceptibility to meropenem

◊ The reported isolate was resistant to both CZA and cefiderocol, limiting options; clinicians avoided colistin due to nephrotoxicity risk

◊ Meropenem-vaborbactam (MVB) was fully active against the isolate, led to clinical recovery, and may be preferable to meropenem alone to reduce resistance risk in KPC-31 cases

Clinical and microbiological characteristics of patients with CZA-resistant KPC-producing *K. pneumoniae* strains - 2025 - *Ann. clin. microbiol. antimicrob.*

◊ Most CZA-resistant isolates carried KPC variants with mutations in the Ω-loop (notably D179Y in KPC-3, i.e. KPC-31), which reduce avibactam binding but often restore carbapenem susceptibility

◊ CZA resistance frequently emerged during therapy, particularly in patients with prolonged exposure and high bacterial burden, underscoring the risk of resistance selection *in vivo*

◊ Many CZA-resistant isolates remained susceptible to meropenem-vaborbactam, supporting its role as a salvage therapy for KPC-variant–mediated CZA resistance.