

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*

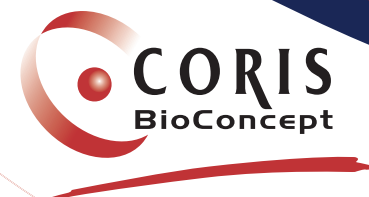


AntiMicrobial Resistance

Pressbook

V5

*A selection of the most
recent publications on AMR*



-AUGUST 2025-



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 ($\approx 30\%$) 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 ($\approx 41\%$ 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 ($\sim 63\%$ 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). KPCs' 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.