Communicable Diseases Intelligence

2025 Volume 49

cdc.gov.au/cdi • Electronic publication date: 18.11.2025 • doi.org/10.33321/cdi.2025.49.055

Australian Group on Antimicrobial Resistance (AGAR) Australian Gram-negative Surveillance Outcome Program (GnSOP) Bloodstream Infection Annual Report 2024

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Australian Government
Department of Health, Disability and Ageing

Interim Australian Centre for Disease Control

Communicable Diseases Intelligence

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ISSN: 2209-6051 Online

This journal is indexed by Index Medicus and Medline.

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Abstract

The Australian Group on Antimicrobial Resistance (AGAR) performs regular period-prevalence studies to monitor changes in antimicrobial resistance in selected enteric gram-negative pathogens. From 1 January 2024 to 31 December 2024, fifty-five hospitals across Australia participated in the Australian Gram-negative Surveillance Outcome Program (GnSOP).

A total of 10,340 isolates, comprising *Enterobacterales* (9,376; 90.9%), *Pseudomonas aeruginosa* (804; 7.7%) and *Acinetobacter* species (160; 1.4%), were tested using commercial automated methods. The results were analysed using European Committee on Antimicrobial Susceptibility Testing (EUCAST) breakpoints (January 2025). Key resistances reported are to the third-generation cephalosporin ceftriaxone in 14.9% of *Escherichia coli* and 10.5% of *Klebsiella pneumoniae* complex isolates. Resistance rates to ciprofloxacin were 15.4% for *E. coli*; 9.7% for the *K. pneumoniae* complex; 3.8% for the *Enterobacter cloacae* complex; and 8.8% for *P. aeruginosa*. Resistance rates to piperacillin–tazobactam were 7.5%, 10.3%, 25.2%, and 13.6% for the same four species/complexes, respectively. Thirty-nine *Enterobacterales* isolates from 38 patients were shown to harbour a carbapenemase gene: 21 with a bla_{NDM} gene (bla_{NDM-5} [8]; bla_{NDM-1} [7]; bla_{NDM-7} [6]); eight with bla_{IMP-4}; four with a $bla_{OXA-181}$-like gene ($bla_{OXA-181}$ [2]; $bla_{OXA-484}$ [1]; $bla_{OXA-1205}$ [1]); three with a bla_{OXA-48}-like gene (bla_{OXA-48} [2]; $bla_{OXA-244}$); two with bla_{KPC-2}; and one with bla_{NDM-5} + $bla_{OXA-484}$. Carbapenemase genes were also detected in two *P. aeruginosa* isolates (bla_{NDM-1} [1]; bla_{GES-5} [1]).

Keywords: Australian Group on Antimicrobial Resistance (AGAR); antimicrobial resistance; bacteraemia; gram-negative; *Escherichia coli*; *Enterobacter*; *Klebsiella*

Introduction

Emerging resistance in common pathogenic members of the *Enterobacterales* is a worldwide phenomenon and presents therapeutic problems, both in the community and in hospital practice. The Australian Group on Antimicrobial Resistance (AGAR) commenced surveillance of the key gram-negative pathogens, *Escherichia coli* and *Klebsiella* species, in 1992. Surveys were conducted biennially until 2008 when annual surveys began, alternating between community- and hospital-onset infections.[i] In 2004 *Enterobacter*, another genus of gram-negative pathogens in which resistance can be of clinical importance, was added. *E. coli* is the most common cause of community-onset urinary tract infection, while *Klebsiella* species are less common but are known to harbour important resistance genes. *Enterobacter* species are less common in the community but are of high importance due to intrinsic resistance to first-line antimicrobials used in that setting. Taken together, these three groups of species surveyed are valuable sentinels for multi-resistance and emerging resistance in enteric gram-negative bacilli. In 2013, AGAR commenced the *Enterobacterales* Sepsis Outcome Program (EnSOP) which focused on the collection of resistance data and some demographic data on all isolates collected prospectively from patients with bacteraemia. In 2015, *Pseudomonas aeruginosa* and *Acinetobacter* species were added, with the program then referred to as the Gram-negative Sepsis Outcome Program (GnSOP), since renamed the Gram-negative Surveillance Outcome Program.

Resistance to β-lactams due to β-lactamases, especially extended-spectrum β-lactamases that inactivate the third-generation cephalosporins normally considered reserve antimicrobials, is of particular interest. Also of interest is resistance to agents important for treatment of serious infections, such as gentamicin and piperacillin–tazobactam; to highly bioavailable oral agents such as ciprofloxacin; and to reserve agents such as meropenem.

The objectives of the 2024 surveillance program were:

- to monitor resistance in *Enterobacterales*, *P. aeruginosa* and *Acinetobacter* species isolated from blood cultures taken from patients presenting to hospital or already inpatients in hospital;
- to examine the extent of co-resistance and multidrug resistance in the major species;
- to detect emerging resistance to reserve agents such as carbapenems and colistin; and
- to examine the molecular basis of resistance to third-generation cephalosporins, quinolones and carbapenems.

i https://agargroup.org.au/reports/.

Methods

Study design

From 1 January to 31 December 2024, thirty-two laboratories servicing 55 hospitals across Australia, including six children's hospitals and 13 regional or district hospitals from north-west Western Australia, collected either all or up to 200 isolates from different patient episodes of bacteraemia. An episode was defined as community-onset (CO) if the first positive blood culture was collected 48 hours or less after admission, and as hospital-onset (HO) if collected greater than 48 hours after admission.

Species identification

Species were identified using the routine method at each institution: Vitek®; Phoenix™ automated microbiology systems; or, where available, matrix assisted laser desorption/ionisation – time of flight (MALDI-ToF) mass spectrometry.

Susceptibility testing

Testing was performed by two commercial semi-automated methods, Vitek® 2 (BioMérieux, France) or Phoenix™ (Becton Dickinson, USA), which are calibrated to the ISO reference standard method of broth microdilution. Commercially available Vitek (AST-N246, AST-N434, AST-N435, AST-N410) or Phoenix NMIC-422 cards were utilized by all participants throughout the survey period. EUCAST v15 breakpoints from January 2025 were used in the analysis.[1]

Multidrug resistance

The definitions used by Magiorakos et al. were applied in this survey,[2] where multidrug resistance (MDR) is defined as resistance to one or more agent in three or more antimicrobial categories. The antimicrobial categories (agents) included were aminoglycosides (gentamicin and/or tobramycin); antipseudomonal penicillins + β-lactamase inhibitor (piperacillin–tazobactam); carbapenems (meropenem); extended-spectrum cephalosporins (ceftriaxone and/or ceftazidime); fluoroquinolones (ciprofloxacin); folate pathway inhibitors (trimethoprim–sulfamethoxazole); non-extended-spectrum cephalosporins (cefazolin or cefuroxime); and aminopenicillins (ampicillin). Antimicrobials were excluded from these counts for any species with a natural resistance mechanism. For *K. pneumoniae* complex, aminopenicillins were excluded, and for *E. cloacae* complex, non-extended spectrum cephalosporins and aminopenicillins were excluded.

Whole genome sequencing

The following isolates were referred to a central laboratory (Centre for Infectious Diseases and Microbiology, The Westmead Institute for Medical Research):

- *E. coli*, *Klebsiella* spp., *Proteus* spp. and *Salmonella* spp. with ceftazidime or ceftriaxone minimum inhibitory concentration (MIC) > 1 mg/L, or cefoxitin MIC > 8 mg/L;
- any other *Enterobacterales* with cefepime MIC > 1 mg/L;
- *Salmonella* spp. with ciprofloxacin MIC > 0.25 mg/L;
- all *Enterobacterales* with meropenem MIC > 0.125 mg/L (> 0.25 mg/L if tested using Vitek®);
- all *P. aeruginosa* and *Acinetobacter* spp. with meropenem MIC > 4 mg/L;
- all isolates with amikacin MIC > 32 mg/L; and
- all isolates with colistin MIC > 4 mg/L.

All referred isolates underwent whole genome sequencing (WGS).

Genomic DNA for WGS was extracted using the DNeasy® Blood & Tissue Kit (Qiagen) according to the manufacturer's instructions for gram-negative bacteria. WGS was performed by the Australian Genome Research Facility using the Illumina platforms. Data were analysed using a modification of the Nullarbor bioinformatic pipeline[3], incorporating searching contigs against the NCBI AMRFinder database[ii] using ABRicate[4] and AMRFinder,[5] followed by a custom AMR-specific pipeline which includes a read-based search using ARIBA[6] against the CARD[7] and NCBI databases. Ambiguities and potential multiple gene copies/variants were checked manually by mapping reads to reference genes[iii] using Geneious version 2025.03.3. Kleborate[8] was used to screen *K. pneumoniae* complex species for virulence loci and K (capsule) serotype.

ii https://www.ncbi.nlm.nih.gov/bioproject/PRJNA313047.

iii Reference gene source: https://www.ncbi.nlm.nih.gov/pathogens/isolates#/refgene/.

Results

The species isolated, and the numbers of each, are listed in Table 1. *Enterobacterales* accounted for 90.7% of isolates, followed by *P. aeruginosa* (7.8%) and *Acinetobacter* species (1.5%). In the *Enterobacterales*, 86.8% of all isolates belonged to three genera: *Escherichia* (59.8%), *Klebsiella* (21.3%) and *Enterobacter* (5.7%). Major resistances for the top six ranked species are listed in Table 2. For gram-negative species, 77.6% of all episodes were CO, with differences seen between *Enterobacterales* (77.4%), *Acinetobacter* species (65.6%) and *P. aeruginosa* (62.4%). The activity of antimicrobial agents tested against *E. coli* and *K. pneumoniae* complex by place of onset are shown in Table 3. A more detailed breakdown of resistance by state and territory is provided in the online GnSOP 2024 report.[iv]

Table 1: Number and proportion of species isolated, blood cultures, AGAR, 2024

Species	Percentage (*n*)	Place of onset, percentage within species (*n*)	
		Community onset	Hospital onset
Escherichia coli	54.2 (5,607)	83.4 (4,675)	16.6 (932)
Klebsiella pneumoniae complex	14.8 (1,527)	72.6 (1,108)	27.4 (419)
Pseudomonas aeruginosa	7.8 (804)	62.4 (502)	37.6 (302)
Enterobacter cloacae complex	5.1 (526)	51.5 (271)	48.5 (255)
Proteus mirabilis	3.1 (325)	78.5 (255)	21.5 (70)
Klebsiella oxytoca	2.9 (296)	62.2 (184)	37.8 (112)
Serratia marcescens	2.2 (228)	50.9 (116)	49.1 (112)
Klebsiella aerogenes	1.7 (171)	54.4 (93)	45.6 (78)
Salmonella species (non-typhoidal)	1.5 (150)	95.3 (143)	4.7 (7)
Morganella morganii	1.0 (106)	74.5 (79)	25.5 (27)
Citrobacter freundii complex	1.0 (105)	64.8 (68)	35.2 (37)
Citrobacter koseri	0.8 (87)	75.9 (66)	24.1 (21)
Acinetobacter baumannii complex	0.8 (84)	58.3 (49)	41.7 (35)
Salmonella species (typhoidal)	0.8 (79)	100.0 (79)	0.0 (0)
Acinetobacter species[a]	0.3 (26)	88.5 (23)	11.5 (3)
Raoultella ornithinolytica	0.2 (20)	80.0 (16)	20.0 (4)
Proteus vulgaris	0.2 (17)	82.4 (14)	17.6 (3)
Acinetobacter ursingii	0.2 (16)	50.0 (8)	50.0 (8)
Acinetobacter lwoffii	0.1 (15)	80.0 (12)	20.0 (3)
Hafnia alvei	0.1 (13)	23.1 (3)	76.9 (10)
Providencia rettgeri	0.1 (13)	69.2 (9)	30.8 (4)
Pantoea agglomerans	0.1 (12)	58.3 (7)	41.7 (5)
Proteus penneri	0.1 (10)	50.0 (5)	50.0 (5)
Other species (total *n* = 40)	1.0 (103)	69.0 (78)	22.1 (25)
Total	**10,340**	**76.0 (7,863)**	**24.0 (2,477)**

a Species not determined.

iv https://agargroup.org.au/reports/.

Table 2: Antimicrobial resistance rates for the top six ranked gram-negative species isolated from blood, AGAR, 2024

Antimicrobial	Percentage resistant, EUCAST breakpoints (number)[a]					
	E. coli	*K. pneumoniae* complex	*P. aeruginosa*	*E. cloacae* complex	*P. mirabilis*	*K. oxytoca*
Ampicillin	54.2 (5,571)	b	na	b	17.4 (321)	b
Amoxicillin–clavulanic acid (2:1 ratio)[c]	9.7 (3,278)	5.1 (882)	na	b	2.6 (190)	16.3 (196)
Amoxicillin–clavulanic acid (fixed, oral)[c]	35.5 (2,296)	17.0 (634)	na	b	2.3 (132)	14.1 (99)
Amikacin	1.2 (5,577)	0.5 (1,520)	1.3 (782)	0.4 (524)	3.1 (320)	0.0 (295)
Cefazolin	24.2 (4,853)	13.2 (1,302)	na	b	na	na
Cefuroxime	23.3 (537)	21.8 (165)	na	b	na	na
Cefepime	5.8 (5,575)	5.6 (1,520)	6.4 (797)	5.2 (523)	1.9 (320)	2.0 (295)
Ceftazidime	9.3 (5,581)	9.5 (1,519)	8.5 (796)	24.7 (523)	1.6 (321)	4.1 (295)
Ceftriaxone	14.9 (5,581)	10.5 (1,520)	na	26.4 (523)	3.1 (321)	11.2 (295)
Ciprofloxacin	15.4 (5,579)	9.7 (1,519)	8.8 (795)	3.8 (521)	5.3 (322)	1.7 (295)
Gentamicin	9.7 (5,574)	3.9 (1,519)	na	4.8 (522)	6.5 (321)	2.7 (294)
Meropenem	0.2 (5,580)	0.5 (1,520)	3.0 (795)	1.1 (524)	0.0 (321)	1.4 (294)
Piperacillin–tazobactam	7.5 (5,471)	10.3 (1,425)	13.6 (773)	25.2 (512)	0.3 (311)	17.5 (285)
Tobramycin	10.1 (5,523)	3.7 (1,481)	0.8 (787)	4.9 (511)	4.4 (317)	2.8 (290)
Trimethoprim–sulfamethoxazole	30.2 (5,545)	17.7 (1,503)	na	16.5 (520)	16.7 (318)	5.8 (295)

a EUCAST: European Committee on Antimicrobial Susceptibility Testing; na: not applicable (testing not recommended).

b Considered largely intrinsically resistant.

c For susceptibility testing purposes, the Clinical and Laboratory Standards Institute (CLSI) uses a 2:1 ratio. EUCAST fixes the concentration of clavulanic acid at 2 mg/L; this formulation is only available on specific cards. Data for both formulations are shown.

Table 3: Number and antimicrobial resistance rates for *Escherichia coli* and *Klebsiella pneumoniae* complex isolates from blood, by place of onset, AGAR, 2024

Species and antimicrobial	Community-onset[a]				Hospital-onset[a]			
	No.	S, %	S-IE, %	R, %	No.	S, %	S-IE, %	R, %
Escherichia coli								
Ampicillin	4,650	47.4	—[b]	52.6	921	37.8	—[b]	62.2
Amoxicillin–clavulanic acid (2:1 ratio)[c]	2,719	81.2	10.4	8.4	559	72.5	11.8	15.7
Amoxicillin–clavulanic acid (fixed, oral)[c]	1,934	—[b]	65.9	34.1	362	—[b]	57.2	42.8
Amikacin	4,655	98.8	—[b]	1.2	922	98.7	—[b]	1.3
Cefazolin	4,078	—[b]	77.7	22.3	775	—[b]	65.8	34.2
Cefepime	4,652	88.4	6.0	5.6	923	86.5	6.6	6.9
Ceftazidime	4,658	84.9	6.4	8.7	923	77.7	9.8	12.6
Ceftriaxone	4,657	85.9	0.0	14.1	923	80.9	0.2	18.9
Cefuroxime	429	0.0	77.2	22.8	108	0.0	75.0	25.0
Ciprofloxacin	4,657	78.8	6.5	14.7	922	74.7	6.4	18.9
Gentamicin	4,653	90.8	—[b]	9.2	921	87.3	—[b]	12.7
Meropenem	4,657	99.8	0.0	0.2	923	99.5	0.2	0.3
Piperacillin–tazobactam	4,576	93.6	—[b]	6.4	895	87.2	—[b]	12.8
Tobramycin	4,615	90.3	—[b]	9.7	908	87.7	—[b]	12.3
Trimethoprim–sulfamethoxazole	4,630	71.5	0.1	28.4	915	60.5	0.3	39.1
***Klebsiella pneumoniae* complex**								
Amoxicillin–clavulanic acid (2:1 ratio)[c]	615	93.5	3.6	2.9	267	85.8	4.1	10.1
Amoxicillin–clavulanic acid (fixed, oral)[c]	485	—[b]	85.8	14.2	149	—[b]	73.8	26.2
Amikacin	1,103	99.5	—[b]	0.5	417	99.5	—[b]	0.5
Cefazolin	943	—[b]	89.3	10.7	359	—[b]	80.2	19.8
Cefepime	1,103	92.7	2.5	4.8	417	87.8	4.6	7.7
Ceftazidime	1,103	90.3	2.3	7.4	416	81.0	3.8	15.1
Ceftriaxone	1,103	91.2	0.0	8.8	417	84.2	0.7	15.1
Cefuroxime	123	0.0	84.6	15.4	42	0.0	59.5	40.5
Ciprofloxacin	1,103	87.7	4.4	8.0	416	78.8	6.7	14.4
Gentamicin	1,102	97.3	—[b]	2.7	417	93.0	—[b]	7.0
Meropenem	1,103	99.5	0.1	0.4	417	99.0	0.0	1.0
Piperacillin–tazobactam	1,080	91.6	—[b]	8.4	401	84.8	—[b]	15.2
Tobramycin	1,085	96.9	—[b]	3.1	406	92.4	—[b]	7.6
Trimethoprim–sulfamethoxazole	1,090	83.9	0.5	15.7	413	77.0	0.0	23.0

a No.: number of isolates; S: susceptible; S-IE: susceptible, increased exposure; R: resistant.

b No category defined.

c For susceptibility testing purposes, the Clinical and Laboratory Standards Institute (CLSI) uses a 2:1 ratio. The European Committee on Antimicrobial Susceptibility Testing (EUCAST) fixes the concentration of clavulanic acid at 2 mg/L; this formulation is only available on specific cards. Data for both formulations are shown.

Escherichia coli

The moderately high levels of resistance to ampicillin (and therefore amoxicillin) observed in *E. coli* were similar to those in the 2023 survey (2024: 54.2%; versus 2023: 52.3%). Resistance to third generation cephalosporins increased compared with 2023 (ceftriaxone 2024: 14.9%; versus 2023: 12.9%; ceftazidime 2024: 9.3%; versus 2023: 6.5%). An extended spectrum β-lactamase (ESBL) phenotype was significantly more prevalent among HO than CO episodes of *E. coli* (23.9% versus 16.2%, $p < 0.0001$). Moderate levels of resistance to cefazolin (24.2%) and trimethoprim–sulfamethoxazole (30.2%) were detected. There was a slight increase in ciprofloxacin resistance in 2024 (15.4%) compared with 2023 (14.5%). Resistance to gentamicin (9.7%), piperacillin–tazobactam (7.5%) and cefepime (5.8%) increased but remained low. Twenty isolates (0.4%) had an elevated meropenem MIC (≥ 0.5 mg/L). For isolates with an ESBL phenotype, 51.3% and 29.7% were resistant to ciprofloxacin and gentamicin, respectively. Just over one-quarter of *E. coli* isolates (26.6%) would be considered multi-drug resistant.

Most of the referred *E. coli* with an ESBL phenotype (867/925; 93.7%) harboured an Ambler class A ESBL gene (664/867, 76.6%); a plasmid-borne class C gene (pAmpC) (153; 17.6%); or a carbapenemase gene alone (3; 0.3%); or an ESBL plus a pAmpC gene (38; 4.4%); or a carbapenemase gene plus either an ESBL gene or a pAmpC gene (9, 1.0%). The dominant β-lactamase genes in *E. coli* continue to be $bla_{\text{CTX-M}}$ types. Of 706 *E. coli* isolates with a confirmed ESBL gene, 694 (98.3%) had one or more $bla_{\text{CTX-M}}$ genes detected by WGS, predominantly $bla_{\text{CTX-M-15}}$ ($n = 316$) or $bla_{\text{CTX-M-27}}$ ($n = 291$). *E. coli* with pAmpC harboured $bla_{\text{DHA-1}}$ (154/198; 77.8%); a $bla_{\text{CMY-2}}$-like gene (43/198; 21.7%); or $bla_{\text{DHA-1}}$ + a $bla_{\text{CMY-2}}$-like gene (1/198; 0.5%).

Klebsiella pneumoniae complex

K. pneumoniae complex isolates showed slightly higher levels of resistance to piperacillin–tazobactam than did *E. coli*, but lower rates of resistance to cefazolin, ceftriaxone, ciprofloxacin, gentamicin, and trimethoprim–sulfamethoxazole. An ESBL phenotype was higher among HO than CO episodes (20.4% versus 10.1%, $p < 0.0001$). Nineteen *K. pneumoniae* complex isolates (1.3%) had an elevated meropenem MIC (see below), up from 0.8% in 2023. In 2024, most of the referred *K. pneumoniae* complex isolates with an ESBL phenotype (167/189; 88.4%) harboured an ESBL gene (124/167; 74.3%); a pAmpC gene (30/167; 18.0%); or a carbapenemase gene (4; 2.4%) alone; or an ESBL gene and a pAmpC gene (3; 1.8%); or a carbapenemase gene with either an ESBL gene or a pAmpC gene (6; 3.6%). Almost all the ESBL genes (125/133; 94.4%) were $bla_{\text{CTX-M}}$ types, mostly $bla_{\text{CTX-M-15}}$ (108/125; 94.7%). *K. pneumoniae* complex isolates with pAmpC harboured either $bla_{\text{DHA-1}}$ (31/34; 91.2%) or a $bla_{\text{CMY-2}}$-like gene (3/34). In 2024, a total of 11.4% of *K. pneumoniae* complex isolates would be considered multi-drug resistant.

In GnSOP 2024, seven *K. pneumoniae* isolates (and one *K. oxytoca*) would be classified as hypervirulent (virulence score ≥ 3) by Kleborate.[8] Two isolates had a K1 or K2 capsule serotype, the most common types in hvKp. Both had a virulence score of 3; one was ST23-K1 with no ESBL or carbapenemase genes, and one was ST25-K2 with $bla_{\text{OXA-181}}$. Carbapenemase genes were detected in two other isolates (ST11-K64 with $bla_{\text{KPC-2}}$ and $bla_{\text{CTX-M65}}$; ST4922-K21 with $bla_{\text{NDM-5}}$).

Enterobacter cloacae complex

Acquired resistance was common among *E. cloacae* complex isolates, to piperacillin–tazobactam (25.2%); ceftriaxone (26.4%); or ceftazidime (24.7%). There was a moderate level of resistance to trimethoprim–sulfamethoxazole (16.5%); cefepime, ciprofloxacin and gentamicin resistance all remain at less than 5%. Although *E. cloacae* complex isolates are generally more resistant to β-lactam antimicrobials than *E. coli*, resistance rates to non-β-lactams tend to be lower. Nineteen *E. cloacae* complex isolates (3.6%) had an elevated meropenem MIC. In 2024, a total of 6.7% of *E. cloacae* complex isolates would be considered multi-drug resistant.

Carbapenemase genes

Overall, 41 isolates (40 patients) from 23 hospitals from five states were found to harbour an acquired carbapenemase gene. A bla_{NDM} gene was detected in 23 isolates: eight *E. coli* (bla_{NDM-5} [6]; bla_{NDM-1} [1]; bla_{NDM-5}+$bla_{OXA\ 484}$ [1]); five *K. pneumoniae* complex (bla_{NDM-1} [2]; bla_{NDM-5} [2]; bla_{NDM-7} [1]); four *K. oxytoca* (bla_{NDM-7} [3]; bla_{NDM-1} [1]); three *C. freundii* complex (bla_{NDM-1} [2]; bla_{NDM-7} [1]); two *E. cloacae* complex (bla_{NDM-1} [1], bla_{NDM-7} [1]), and one *P. aeruginosa* (bla_{NDM-1}). $blaI_{MP-4}$ was detected in eight isolates: *E. cloacae* complex (three); *K. pneumoniae* complex (two); *Serratia marcescens* (two); and *Pluralibacter gergoviae* (one). A $bla_{OXA-181}$-like gene was detected in four isolates: two *E. coli* ($bla_{OXA-484}$ [1]; $bla_{OXA-1205}$ [1]); and two *K pneumoniae* complex ($bla_{OXA-181}$). A bla_{OXA-48}-like gene was detected in three *E. coli* isolates (bla_{OXA-48} [2]; $bla_{OXA-244}$ [1]). A bla_{KPC-2} gene was detected in two *K. pneumoniae* complex and bla_{GES-5} in one *P. aeruginosa* isolate. An additional isolate initially reported as a carbapenem-resistant *P. aeruginosa* was found on sequencing to be *P. otitidis*, which carries the intrinsic bla_{POM-1} gene.

Plasmid-borne colistin determinants

Four isolates with bla_{NDM} carbapenemase genes also harboured *mcr-9.1* (*E. hormaechei* bla_{NDM-1}, *E. coli* bla_{NDM-5}, and two *K. oxytoca* bla_{NDM-7}). Two *Enterobacter cloacae* with bla_{IMP-4} also harboured *mcr-9.1* or *mcr-9.2*. Nine additional isolates (*E. hormaechei*, *n* = 5; *E. coli*, *n* = 2; *K. pneumoniae*, *n* = 1; *K. oxytoca*, *n* = 1) that did not carry a carbapenemase gene had *mcr-9.1* (*n* = 7), *mcr-8.2* (*n* = 1), or *mcr-10.1* (*n* = 1). The *mcr-9* gene has recently been found among several species of *Enterobacterales*. It is not associated with a resistant phenotype,[9] but is typically carried on HI2 plasmids.[10,11]

Discussion

AGAR has been tracking antimicrobial resistance in sentinel enteric gram-negative bacteria since 1992. From 2008, surveillance was separated into HO versus CO infections. The last year of HO-only surveillance was 2011.[12] In 2013, the first survey of antimicrobial resistance among *Enterobacterales* isolates from bacteraemic patients throughout Australia was conducted using an approach similar to the European EARS-Net program.[13] The 2024 survey was the twelfth of antimicrobial resistance among *Enterobacterales*, and the tenth for *P. aeruginosa* and *Acinetobacter* spp. from bacteraemic patients through Australia.

In 2024 in *E. coli*, there were significant increases in the percentages of resistant isolates compared to 2023, notably for ceftazidime, ceftriaxone, cefepime, meropenem, and amoxicillin–clavulanic acid. For the *K. pneumoniae* complex, the percentage of resistant isolates in 2024 increased for ceftriaxone, ceftazidime, cefepime, and trimethoprim–sulfamethoxazole compared to 2023.

Previous AGAR reports showed a longitudinal trend of increasing resistance to key anti-gram-negative antimicrobial agents, such as ceftriaxone and ciprofloxacin, in *E. coli*.[14,15] Resistance to both agents stabilised from 2018 to 2020 (ceftriaxone 13.4–13.3%, ciprofloxacin 15.2–16.1%); the levels of resistance declined to 12.4% and 12.3%, respectively, in 2021. In 2024, the levels of resistance increased (14.9% and 15.4%). The steady rise in resistance to ciprofloxacin is more striking in HO bacteraemia, with a change from 13.7% to 21.8% between 2013 and 2020. In 2021 the level of resistance fell to 16.7%, increasing to 17.8–17.7% (2022–2023), and 18.9% in 2024. In the *K. pneumoniae* complex, rates of resistance to ciprofloxacin were lower than for *E. coli*. Resistance in this species complex peaked in 2018 (11.0%), falling to 7.3–7.8% in 2021–2023, and was 9.7% in 2024.

Carbapenem resistance attributable to acquired carbapenemase genes is still uncommon in patients with bacteraemia in Australia. Seven different gene profiles (bla_{NDM} [22], bla_{IMP-4} [8], a $bla_{OXA\ 181}$-like gene [4], a bla_{OXA-48}-like gene [3], bla_{KPC-2} [2], bla_{GES-5} [1], or bla_{NDM-5} + $bla_{OXA-484}$ [1]) were detected in 41 isolates from 23 of the participating hospitals. Compared with many other countries in our region, antimicrobial resistance rates in Australian gram-negative bacteria are still relatively low,[16,17] but similar to those observed in 2023 in many Northern European countries.[18,19] Resistance to third generation cephalosporins in *E. coli* from bacteraemic patients in Australia is similar to the European Union and European Economic Area average.[19] Although we see rates of ceftriaxone and ciprofloxacin resistance in *E. coli* that parallel Northern Europe, rates in the *K. pneumoniae* complex are lower in Australia (< 10%), compared to rates of resistance > 25% in parts of Europe. Some of this is explained by the relatively greater predisposition for *Klebsiella* species to carry carbapenemase gene types found in Europe (such as bla_{KPC}) and to the unregulated fluoroquinolone use compared to Australia, where use of this antimicrobial class has been under greater scrutiny and regulation both in the human and animal husbandry sectors. Nonetheless, the high rates of resistance observed in many other regions illustrates the potential for greater increases in resistance rates over time and the need for ongoing surveillance.

Just over one-quarter (26.6%) of *E. coli* would be classed as MDR, up from 23.2% in the 2023 survey. The proportion of *K. pneumoniae* complex isolates classed as MDR fell from 11.1% in 2019 to 7.5% in 2021 and 7.2% in 2022. The MDR proportion increased to 8.2% in 2023, and to 11.4% in 2024.

The SARS-CoV-2 pandemic has been reported to have impacted antimicrobial resistance. In Australia, a combination of COVID-19-related travel restrictions on incoming travellers throughout much of 2020 and 2021,[20] and an increasing awareness of and utilization of antimicrobial stewardship as part of the Australia-wide implementation and accreditation of National Safety and Quality Health Service Standards,[21] may have reduced some resistance rates, particularly for ESBLs. There are also changes in the relative proportions of different carbapenemase genes after removal of travel restrictions. There was an increase in the number of isolates with a bla_{NDM} gene reported from patients with bacteraemia in 2024.[22,23] In 2024, over one-half of all CPE (21/39; 53.9%) carried a bla_{NDM} gene, compared with 7/29 (24.1%) in 2022, and 20.5% carried $bla_{IMP\ 4}$ (n = 8); the latter compared with 62.1% (18/29) CPE in 2022. Almost three-quarters of all CPE in 2024 (29/39; 74.4%) were from New South Wales (n = 19; 48.7%) or Victoria (n = 10; 25.6%).

The 2024 survey suggests that resistance rates have now returned to pre-COVID-19 levels. Future AGAR surveys will help determine if this observed increase in resistance rates is sustained.

Acknowledgments

This study was funded by the Australian Government Department of Health, Disability and Ageing.

AGAR gratefully acknowledges Jenny Draper for processing WGS data, and the Genomics Facility at WIMR (Joey Lai, David Zheng) and the Australian Genome Research Facility for performing whole genome sequencing.

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Liverpool Hospital, Michael Maley and Helen Ziochos

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Alfred Hospital, Adam Jenney and Jacqueline Williams

Austin Health, Marcel Leroi and Elizabeth Grabsch

Dandenong Hospital, Tony Korman and Kathryn Cisera

Monash Children's Hospital, Tony Korman and Despina Kotsanas

Royal Melbourne Hospital, Katherine Bond and Rose Cotronei

Royal Women's and Children's Hospital, Andrew Daley and Gena Gonis

St Vincent's Hospital, Amy Crowe and Lisa Brenton

Western Australia

Fiona Stanley Hospital, Shakeel Mowlaboccus and Denise Daley

Joondalup Hospital, Shalinie Perera and Gemma Groves

Perth Children's Hospital, Christopher Blyth

Regional hospitals in north-west Western Australia, Michael Leung

Royal Perth Hospital, Owen Robinson and Geoffrey Coombs

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