Australian Group on Antimicrobial Resistance (AGAR) Australian Gram-negative Sepsis Outcome Programme (GNSOP) Annual Report 2015

Jan M Bell, Thomas Gottlieb, Denise A Daley and Geoffrey W Coombs

# 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. The 2015 survey was the third year to focus on blood stream infections, and included Enterobacteriaceae, Pseudomonas aeruginosa and Acinetobacter species.

Seven thousand three hundred and thirty species, comprising Enterobacteriaceae (6,567, 89.6%), P. aeruginosa (660, 9.0%) and Acinetobacter species (103, 1.4%), were tested using commercial automated methods (Vitek® 2, BioMérieux; Phoenix™, BD) and results were analysed using Clinical and Laboratory Standards Institute (CLSI) and European Committee on Antimicrobial Susceptibility Testing (EUCAST) breakpoints (January 2016). Of the key resistances, non-susceptibility to the third-generation cephalosporin, ceftriaxone, was found in 10.6%/10.6% of E. coli (CLSI/EUCAST criteria) and 5.9%/5.9% of Klebsiella pneumoniae, and 8.4%/8.4% K. oxytoca. Non-susceptibility rates to ciprofloxacin were 12.6%/13.6% for E. coli, 3.9%/7.2% for K. pneumoniae, 0.4%/0.4% for K. oxytoca, 3.4%/4.0% for Enterobacter cloacae, and 6.3%/6.5% for Pseudomonas aeruginosa. Resistance rates to piperacillin-tazobactam were 2.8%/6.3%, 3.5%/6.4%, 8.9%/10.2%, 15.9%/20.6%, and 7.1%/13.9% for the same four species respectively. Twenty-two isolates were shown to harbour a carbapenemase gene, 14 blaIMP, four blaOXA-48, one blaKPC, one blaGES, one blaNDM+OXA-48, and one blaIMP+VIM.

Keywords: Australian Group on Antimicrobial Resistance (AGAR); antibiotic resistance; bacteraemia; Gram-negative; Escherichia coli; Enterobacter; Klebsiella

# Introduction

Emerging resistance in common pathogenic members of the Enterobacteriaceae is a world-wide phenomenon, and presents therapeutic problems for practitioners in both 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 have been conducted biennially until 2008 when annual surveys commenced alternating between community–and hospital–onset infections (http://www.agargroup.org/surveys). In 2004, another genus of Gram-negative pathogens in which resistance can be of clinical importance, Enterobacter species, 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 resistances. Enterobacter species are less common in the community, but of high importance due to intrinsic resistance to first-line antimicrobials used in the community. Taken together, the three groups of species surveyed are considered to be valuable sentinels for multi-resistance and emerging resistance in enteric Gram-negative bacilli. In 2013, AGAR commenced the Enterobacteriaceae Sepsis Outcome Programme (EnSOP) which focused on the collection of resistance and some demographic data on all isolates prospectively from patients with bacteraemia. In 2015, Pseudomonas aeruginosa and Acinetobacter species were added, and the program name was altered to GNSOP to reflect the additional species surveyed.

Resistances of particular interest include resistance to ß-lactams due to ß-lactamases, especially extended-spectrum ß-lactamases, which inactivate the third-generation cephalosporins that are normally considered reserve antimicrobials. Other resistances of interest are to agents important for treatment of these serious infections, such as gentamicin; and resistance to reserve agents such as ciprofloxacin, meropenem and colistin.

The objectives of the 2015 surveillance program were to:

1. monitor resistance in Enterobacteriaceae, P. aeruginosa and Acinetobacter species isolated from blood cultures taken from patients presenting to the hospital or already in hospital;
2. examine the extent of co-resistance and multidrug resistance in the major species;
3. detect emerging resistance to newer last-line agents such as carbapenems; and
4. characterise the molecular basis of resistance to third-generation cephalosporins, quinolones, amikacin and carbapenems.

# Methods

## Study Design

From 1st January to 31st December 2015, 31 laboratories across Australia collected either all or up to 200 isolates from different patient episodes of bacteraemia.

## Species identification

Isolates were identified using the routine method for each institution; Vitek®, Phoenix™ Automated Microbiology System, or where available mass spectrometry (MALDI-TOF).

## Susceptibility testing

Testing was performed by two commercial semi-automated methods, Vitek® 2 (BioMérieux) or Phoenix™ (BD), which are calibrated to the ISO reference standard method of broth microdilution. Commercially available Vitek® AST-N246 and AST-N247, or Phoenix™ NMIC-203 cards were utilized by all participants throughout the survey period. The CLSI M1001 and EUCAST v5.02 breakpoints from January 2016 have been employed in the analysis. For analysis of cefazolin, breakpoints of ≤4 for susceptible, ≥8 for resistant were applied due to the restricted minimum inhibitory concentration (MIC) range available on the commercial cards, recognising that the January 2016 breakpoint is actually ≤2 mg/L for susceptible isolates.

## Multidrug resistance

For this survey, multidrug resistance was defined as acquired resistance to more than three antimicrobial classes. For each species, antimicrobials were excluded from the count if they were affected by intrinsic resistance mechanisms.

## Molecular confirmation of resistances

E. coli and Klebsiella isolates with ceftazidime or ceftriaxone MIC > 1 mg/L, or cefoxitin MIC > 8 mg/L; Enterobacter spp. with cefepime MIC > 1 mg/L; all isolates with ciprofloxacin MIC > 0.25 mg/L; all isolates with meropenem MIC > 0.25 mg/L; all isolates with amikacin MIC > 32 mg/L, and all isolates with colistin MIC > 2 mg/L were referred to a central laboratory (SA Pathology) for molecular confirmation of resistance.

All referred isolates were screened for the presence of the blaTEM, and blaSHV genes using a real-time polymerase chain reaction (PCR) platform (LC-480) and published primers.3,4 A multiplex real-time TaqMan PCR was used to detect CTX-M-type genes.5 Isolates were probed for plasmid-borne AmpC enzymes using the method described by Pérez-Pérez and Hanson6.and subjected to molecular tests for Metallo-Beta-Lactamase (MBL) (blaVIM, blaIMP, and blaNDM), blaKPC, and blaOXA-48-like genes using real-time PCR.7,8 Known plasmid mediated quinolone resistance (PMQR) mechanisms (Qnr, efflux [qepA, oqxAB]; aac(6’)-Ib-cr) were examined by PCR on all referred isolates with ciprofloxacin MIC >0.25 mg/L using published methods.9,10 All E. coli were examined for presence of the O25b-ST131 clone and its H30- and H30-Rx subclones.11-13

# Results

The species isolated, and the numbers of each are listed in Table 1. Enterobacteriaceae accounted for 89.6%, followed by P. aeruginosa (9.0%) and Acinetobacter species (1.4%). Of the Enterobacteriaceae, three genera - Escherichia (61.0%), Klebsiella (18.5%) and Enterobacter (7.4%) - contributed 86.9% of all isolates. Major resistances and non-susceptibilities for the top six ranked species are listed in Table 2. Non-susceptibility, (which includes both intermediately resistant and resistant strains), has been included for some agents because these figures provide information about important emerging acquired resistances. Multiple acquired resistances by species are shown in Table 3. Multidrug resistance was detected in 20.0% of Escherichia coli isolates, 8.6% of Klebsiella pneumoniae, and 15.2% of Enterobacter cloacae. A more detailed breakdown of resistances and non-susceptibilites by state and territory is provided in the online report from the group (http://www.agargroup.org/surveys).

Table 1. Number and proportion of species isolated, blood cultures, 2015

| Species | Total |  |
| --- | --- | --- |
| Escherichia coli | 4,006 | 54.7% |
| Klebsiella pneumoniae | 977 | 13.3% |
| Pseudomonas aeruginosa | 658 | 9.0% |
| Enterobacter cloacae complex | 326 | 4.4% |
| Klebsiella oxytoca | 238 | 3.2% |
| Proteus mirabilis | 223 | 3.0% |
| Serratia marcescens | 189 | 2.6% |
| Enterobacter aerogenes | 131 | 1.8% |
| Salmonella species (non-typhoidal) | 115 | 1.6% |
| Morganella morganii | 79 | 1.1% |
| Acinetobacter baumannii complex | 59 | 0.8% |
| Citrobacter koseri | 55 | 0.8% |
| Citrobacter freundii | 45 | 0.6% |
| Salmonella species (typhoidal) | 26 | 0.4% |
| Acinetobacter species | 20 | 0.3% |
| Pantoea agglomerans | 13 | 0.2% |
| Enterobacter species | 12 | 0.2% |
| Raoultella ornithinolytica | 12 | 0.2% |
| Providencia rettgeri | 11 | 0.2% |
| Proteus vulgaris | 11 | 0.2% |
| Other species (total n=40) | 124 | 1.7% |
| Total | 7,330 |  |

Table 2. Proportion non-susceptible and resistant (CLSI and EUCAST) to antimicrobial agents for the top six ranked species, blood cultures, 2015

|   |  | *E.coli*(%) | *K.pneumoniae*(%) | *P.aeruginosa*(%) | *E.cloacae*(%) | *K.oxytoca*(%) | *P.mirabilis*(%) |
| --- | --- | --- | --- | --- | --- | --- | --- |
| Antimicrobial | Category\* | CLSI | EUCAST | CLSI | EUCAST | CLSI | EUCAST | CLSI | EUCAST | CLSI | EUCAST | CLSI | EUCAST |
| Ampicillin | I | 2.1 | - | † | † |  |  | † | † | † | † | 0.0 | - |
|   | R | 53.1 | 55.1 | † | † |  |  | † | † | † | † | 17.1 | 17.1 |
| Amoxycillinclavulanate ‡ | I | 13.7 | - | 4.8 | - |  |  | † | † | 4.2 | - | 8.6 | - |
|   | R | 8.7 | - | 4.2 | - |  |  | † | † | 7.6 | - | 1.8 | - |
| Piperacillintazobactam | R | 2.8 | 6.3 | 3.5 | 6.4 | 7.1 | 13.9 | 15.9 | 20.6 | 8.9 | 10.2 | 0.5 | 0.9 |
| Cefazolin | R | 21.8 | 21.8 | 10.9 | 10.9 |  |  | † | † | 62.3 | 62.3 | 25.4 | 25.4 |
| Cefoxitin | R | 3.4 | / | 4.8 | / |  |  | † | † | 2.5 | / | 0.5 | / |
| Ceftriaxone | NS | 10.6 | 10.6 | 5.9 | 5.9 |  |  | 25.8 | 25.8 | 8.4 | 8.4 | 2.3 | 2.3 |
| Ceftazidime | NS | 6.1 | 9.9 | 4.9 | 6.9 | 10.4 | 10.4 | 22.4 | 24.2 | 1.3 | 2.1 | 1.4 | 1.8 |
| Cefepime | NS | 5.7 | 8.7 | 2.6 | 4.7 | 2.6 | 8.0 | 5.8 | 12.9 | 0.8 | 1.3 | 1.4 | 1.4 |
| Meropenem | NS | <0.1 | 0.0 | 0.3 | 0.3 | 8.1 | 8.1 | 3.4 | 3.1 | 0.4 | 0.4 | 0.0 | 0.0 |
| Ciprofloxacin | NS | 12.6 | 13.6 | 3.9 | 7.2 | 6.3 | 6.5 | 3.4 | 4.0 | 0.4 | 0.4 | 4.1 | 4.5 |
| Gentamicin | NS | 7.8 | 7.9 | 4.2 | 4.5 | 2.4 | 3.4 | 6.7 | 7.4 | 0.8 | 1.3 | 0.5 | 1.8 |
| Trimethoprim | R | 31.0 | 31.3 | 15.4 | 16.1 |  |  | 16.0 | 16.0 | 3.4 | 3.4 | 18.1 | 19.0 |
| Nitrofurantoin | R | 1.3 | 1.3 | 32.4 | / |  |  | 20.6 | / | 2.1 | / | † | † |

\*R = resistant, I = intermediate, NS = non-susceptible (intermediate + resistant), using criteria as published by the CLSI [2014] and EUCAST [2014].
 †Considered largely intrinsically resistant due to natural β-lactamases; - no intermediate category; / no breakpoints defined
‡ For EUCAST interpretation, clavulanate is fixed at 2 mg/L, rather than a 2:1 ratio used in CLSI guidelines. As all susceptibility test cards used have a 2:1 ratio of clavulanate, no EUCAST category has been applied

Table 3. Number and proportion of acquired resistances (EUCAST) in Enterobacteriaceae, blood cultures, 2015

|   | Number of acquired resistances (EUCAST breakpoints) |
| --- | --- |
|   | Non-multidrug resistant |  | Multidrug resistant |  |
| **Species** | Total | 0 | 1 | 2 | 3 | Cumulative% | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | Cumulative% |
| **E. coli** | 3,743 | 1,497 | 580 | 561 | 358 |  | 217 | 149 | 93 | 144 | 53 | 53 | 26 | 12 | 0 | 0 |  |
|  | % | 40.0 | 15.5 | 15.0 | 9.6 | 80.0% | 5.8 | 4.0 | 2.5 | 3.8 | 1.4 | 1.4 | 0.7 | 0.3 | 0.0 | 0.0 | 20.0% |
| **K. pneumoniae\*** | 909 | 667 | 101 | 42 | 21 |  | 25 | 5 | 9 | 12 | 9 | 16 | 1 | 1 |  |  |  |
|  |  | 73.4 | 11.1 | 4.6 | 2.3 | 91.4% | 2.8 | 0.6 | 1.0 | 1.3 | 1.0 | 1.8 | 0.1 | 0.1 |  |  | 8.6% |
| **E. cloacae complex †** | 277 | 187 | 17 | 7 | 24 |  | 15 | 11 | 9 | 7 | 0 | 0 | 0 |  |  |  |  |
|  |  | 67.5 | 6.1 | 2.5 | 8.7 | 84.8% | 5.4 | 4.0 | 3.2 | 2.5 | 0.0 | 0.0 | 0.0 |  |  |  | 15.2% |
| **P. mirabilis** | 205 | 109 | 56 | 11 | 14 |  | 9 | 3 | 1 | 0 | 0 | 2 | 0 | 0 | 0 |  |  |
|  |  | 53.2 | 27.3 | 5.4 | 6.8 | 92.7% | 4.4 | 1.5 | 0.5 | 0.0 | 0.0 | 1.0 | 0.7 | 0.0 | 0.0 |  | 7.3% |
| **S. marcescens †** | 142 | 127 | 6 | 4 | 3 |  | 1 | 1 | 0 | 0 | 0 | 0 | 0 |  |  |  |  |
|  |  | 89.4 | 4.2 | 2.8 | 2.1 | 98.6% | 0.7 | 0.7 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |  |  |  | 1.4% |
| **K. oxytoca\*** | 217 | 72 | 111 | 4 | 12 |  | 15 | 2 | 0 | 1 | 0 | 0 | 0 | 0 |  |  |  |
|  |  | 33.2 | 51.2 | 1.8 | 5.5 | 91.7% | 6.9 | 0.9 | 0.0 | 0.5 | 0.0 | 0.0 | 0.0 | 0.0 |  |  | 8.3% |
| **E. aerogenes †** | 130 | 68 | 6 | 4 | 43 |  | 3 | 2 | 1 | 3 | 0 | 0 | 0 |  |  |  |  |
|  |  | 52.3 | 4.6 | 3.1 | 33.1 | 93.1% | 2.3 | 1.5 | 0.8 | 2.3 | 0.0 | 0.0 | 0.0 |  |  |  | 6.9% |
| **Salmonella spp. (non Typhi)** | 106 | 97 | 5 | 1 | 1 |  | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 |  |  |
|   |  | 91.5 | 4.7 | 0.9 | 0.9 | 98.1% | 0.0 | 0.0 | 1.9 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |  | 1.9% |

\* Antibiotics included: amoxycillin-clavulanate, piperacillin-tazobactam, cefazolin, cefoxitin, ceftriaxone, ceftazidime, cefepime, gentamicin, amikacin, ciprofloxacin, nitrofurantoin, trimethoprim, meropenem;Antibiotics excluded: ampicillin (intrinsic resistance), ticarcillin-clavulanate, tobramycin, norfloxacin, nalidixic acid, sulfamethoxazole-trimethoprim, due to high correlation with antibiotics in the included list.
† Antibiotics included: piperacillin-tazobactam, ceftriaxone, ceftazidime, cefepime, gentamicin, amikacin, ciprofloxacin, nitrofurantoin, trimethoprim, meropenem
Antibiotics excluded: ampicillin, amoxycillin-clavulanate, cefazolin, and cefoxitin, (all four due to intrinsic resistance); also excluded were ticarcillin-clavulanate, tobramycin, norfloxacin, nalidixic acid, sulfamethoxazole-trimethoprim, due to high correlation with antibiotics in the included list.

## Escherichia coli

Moderately high levels of resistance to ampicillin (and therefore amoxycillin) were maintained (53.1%/55.1%, CLSI/EUCAST criteria), with lower rates for amoxycillin-clavulanate (13.7%/- intermediate, 8.7%/- resistant). Non-susceptibility to third-generation cephalosporins was low ceftriaxone 10.6%/10.6%, ceftazidime 6.1%/9.9%). Moderate levels of resistance were detected to cefazolin (21.8%/21.8%) and trimethoprim (31.0%/31.4%). Ciprofloxacin non-susceptibility was found in 12.6%/13.6% of E. coli isolates. Resistance to gentamicin (7.8%/7.9%), piperacillin-tazobactam (2.8%/6.3%), cefepime (3.7%/4.8%) were low. Four isolates had elevated meropenem MICs (≥ 0.5 mg/L). For the extended-spectrum β-lactamase (ESBL)-producing strains, ciprofloxacin and gentamicin resistance was found in 60.7%/61.5% and 32.4%/32.4% respectively.

Most of the E. coli strains with ESBL genes harboured genes of the CTX-M type (349/408 = 86%). Over 65% of E. coli with CTX-M group 1 types were found to belong to sequence type 131 (O25b-ST131). ST131 accounted for 74% of E. coli ESBL phenotypes that were ciprofloxacin resistant (MIC >1 mg/L), and only 6% of ciprofloxacin susceptible ESBL phenotypes. Ninety-five percent and 64% of O25b-ST131 with an ESBL phenotype were associated with the H30 and H30-Rx subclones, respectively, consistent with their reported association with more antibiotic resistances and greater virulence potential.12

## Klebsiella pneumoniae

K. pneumoniae showed slightly higher levels of resistance to piperacillin-tazobactam and ceftazidime compared with E. coli, but lower rates of resistance to amoxycillin-clavulanate, cefazolin, ceftriaxone, ciprofloxacin, gentamicin, and trimethoprim. Nine K. pneumoniae isolates had elevated meropenem MICs (see below). ESBLs were present in 53 of 71 (75%) presumptively ESBL-positive isolates of K. pneumoniae, 44 (83%) of which proved to be of the CTX-M type.

## Enterobacter species

Acquired resistance was common to piperacillin-tazobactam (15.9%/20.6% and 27.7%/40.0%), ceftriaxone (25.8%/25.8% and 42.0%/42.0%), ceftazidime (22.4%/24.2% and 37.4%/39.7%) and trimethoprim (16.0%/16.0% and 6.2%/6.2%) for E. cloacae and E. aerogenes, respectively. Cefepime, ciprofloxacin, and gentamicin resistance were all less than 10%. Twenty-one E. cloacae strains had elevated meropenem MICs.

## Carbapenemase resistance

Overall, 22 isolates (20 patients) in eleven institutions from five states/territories were found to harbour a carbapenemase gene. blaIMP-4 was detected in E. cloacae (8 isolates, from 7 patients), Citrobacter freundii (2), and in one K. pneumoniae, K. oxytoca, Raoultella ornithinolytica and Serratia marcescens; blaOXA-48 was detected in K. pneumoniae (4 isolates, from 3 patients); blaKPC was detected in one K. pneumoniae; blaGES-5 was detected in one P. aeruginosa; blaNDM+OXA-48 was detected in one K. pneumoniae; and blaNDM-4+VIM-2 in one P. aeruginosa.

# Discussion

AGAR has been tracking resistance in sentinel enteric Gram-negative bacteria since 1992. From 2008, surveillance was segregated into hospital versus community onset infections. The last year of hospital-onset only surveillance was 2011.14 In 2013, the first survey of antimicrobial resistance among Enterobacteriaceae isolates from bacteraemic patients through Australia was conducted using an approach similar to that conducted by the European Antimicrobial Resistance Surveillance Network (EARS-Net) program. 2015 was the third survey of antimicrobial resistance among Enterobacteriaceae, and the first to include P. aeruginosa and Acinetobacter spp. from bacteraemic patients through Australia.

CTX-M-producing E. coli and Klebsiella species and gentamicin- and ciprofloxacin-resistant E. coli continued to be a problem in patients with bacteraemia. Of concern is the high proportion of E. coli that belong to the ST131 H30-Rx subclone, and its recognised association with greater antibiotic resistance, in particular to ciprofloxacin, and greater virulence potential.12 Carbapenem resistance attributable to acquired carbapenemases is still uncommon in patients with bacteraemia in Australia, although six different types of carbapenemase (IMP, KPC, NDM, OXA-48, VIM, and GES) were detected from eleven of the participating institutions. Compared with many other countries in our region, resistance rates in Australian Gram-negative bacteria are still relatively low15, but similar to those observed in 2015 in many Western European countries.22

Multidrug resistance is being increasingly observed, especially in E. coli and E. cloacae, both of which now have multidrug resistance rates (as defined by AGAR) above 15%. Despite national efforts to improve antimicrobial stewardship, increasing multi-resistance is likely to be a driver for broad-spectrum antibiotic use, and will increase the resistance selection pressure for important reserve classes, especially the carbapenemases.

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This study was funded by a grant from the Australian Commission on Safety and Quality in Health Care.

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This journal is indexed by Index Medicus and Medline

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Communicable Diseases Intelligence is produced by Health Protection Policy Branch, Office of Health Protection, Australian Government, Department of Health, GPO Box 9848, (MDP 6) CANBERRA ACT 2601;

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