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Australian Group on Antimicrobial Resistance (AGAR) Australian Enterococcal Surveillance Outcome Program (AESOP) Bloodstream Infection Annual Report 2024

Geoffrey W Coombs, Denise A Daley, Auriane Form, Xing Li, Christian Torres, Shakeel Mowlaboccus, on behalf of the Australian Group on Antimicrobial Resistance

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GPO Box 9848, Canberra ACT 2601

Website: **cdc.gov.au/cdi**

Email: **cdi.editor@health.gov.au**

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Abstract

From 1 January to 31 December 2024, fifty-five institutions across Australia participated in the Australian Enterococcal Surveillance Outcome Program (AESOP). The aim of AESOP 2024 was to determine the proportion of enterococcal bacteraemia isolates in Australia that were antimicrobial resistant, and to determine the molecular epidemiology of the reported *Enterococcus faecium* isolates. Of the 1,461 unique episodes of enterococcal bacteraemia investigated, 92.5% were caused by either *E. faecalis* (51.5%) or *E. faecium* (41.0%). Ampicillin and vancomycin resistance were not detected in *E. faecalis* but were detected in 96.8% and 44.5% of *E. faecium* respectively. Five linezolid-resistant *E. faecalis* isolates were identified, for which the linezolid minimum inhibitory concentrations (MICs) ranged from 6.0 mg/L to 8.0 mg/L. All five isolates harboured the linezolid resistance *optrA* gene and were vancomycin susceptible. One linezolid-resistant *E. faecium* was confirmed with an MIC of 6.0 mg/L. The isolate was vancomycin and teicoplanin resistant and harboured *vanA* and *optrA* genes.

Overall, 49.8% of *E. faecium* isolates harboured the *vanA* and/or the *vanB* gene: within these isolates, 40.2% harboured *vanA*, 58.8% harboured *vanB*, and 1.0% harboured *vanA* and *vanB*. The percentage of vancomycin-resistant *E. faecium* bacteraemia isolates in Australia remains substantially higher than that recorded in most European countries. The *E. faecium* isolates consisted of 56 multi-locus sequence types (STs); 85.7% of isolates were classified into eight STs, each containing ten or more isolates. The eight STs (ST17, ST78, ST80, ST117, ST555, ST796, ST1421 and ST1424) belonged to clonal complex (CC) 17, a global hospital-adapted polyclonal *E. faecium* CC, and were found in most Australian jurisdictions. Overall, 54.6% of *E. faecium* isolates belonging to the eight predominant STs harboured the *vanA* or *vanB* gene. AESOP 2024 has shown that enterococcal bacteraemia episodes in Australia continue to be frequently caused by polyclonal ampicillin-resistant high-level gentamicin-resistant *vanA*- or *vanB*-positive *E. faecium* which have limited treatment options.

Keywords: Australian Group on Antimicrobial Resistance (AGAR); antimicrobial resistance surveillance; *Enterococcus faecium*; *Enterococcus faecalis*; vancomycin resistant enterococci (VRE); bacteraemia

Background

Globally, *Enterococcus* accounts for approximately 10% of all bacteraemia cases and is the fourth and fifth leading cause of sepsis in North America and Europe, respectively.[1] In the 1970s, healthcare-associated enterococcal infections were primarily due to *Enterococcus faecalis*, but there has been a steadily increasing prevalence of *E. faecium* nosocomial infections.[2,3] Worldwide, the increase in nosocomial *E. faecium* infections has primarily been due to the expansion of polyclonal hospital-adapted clonal complex 17 (CC17) strains. Whilst innately resistant to many antimicrobial classes, *E. faecium* has demonstrated a remarkable capacity to evolve new antimicrobial resistances. In 2009, the Infectious Diseases Society of America highlighted *E. faecium* as one of the key problem bacteria or ESKAPE pathogens (*E. faecium*, *Staphylococcus aureus*, *Klebsiella pneumoniae*, *Acinetobacter baumannii*, *Pseudomonas aeruginosa*, and *Enterobacter* species) requiring new therapies.[4] In 2024, the World Health Organisation listed vancomycin-resistant *E. faecium* in its bacterial priority list of pathogens.[5]

The Australian Group on Antimicrobial Resistance (AGAR) is a network of laboratories located across Australia that commenced surveillance of antimicrobial resistance in *Enterococcus* species in 1995.[6] In 2011, AGAR commenced the Australian Enterococcal Sepsis Outcome Program,[7] now known as the Australian Enterococcal Surveillance Outcome Program (AESOP). The objective of AESOP 2024 was to determine the proportion of *E. faecalis* and *E. faecium* bacteraemia isolates demonstrating antimicrobial resistance, with particular emphasis on:

1. resistance to ampicillin;
2. resistance to glycopeptides; and
3. the molecular epidemiology of *E. faecium*.

Methodology

Participants

Thirty-two laboratories servicing 55 institutions located in all Australian states and mainland territories.

Collection period

From 1 January to 31 December 2024, the 32 laboratories collected all enterococcal species isolated from blood cultures. Enterococci of the same species and antimicrobial susceptibility profiles isolated from a patient's blood culture within 14 days of the first positive blood culture were excluded. A new enterococcal bacteraemia episode in the same patient was recorded if the episode occurred more than 14 days after the initial positive culture. Data were collected on age, sex, dates of admission and discharge (if admitted), and mortality at seven and 30 days from date of blood culture collection. To avoid interpretive bias, no attempt was made to assign attributable mortality. Each episode of bacteraemia was designated as 'hospital-onset' if the first positive blood culture(s) in an episode was collected > 48 hours after admission.

Laboratory testing

Enterococcal isolates were identified to the species level by the participating laboratories using matrix-assisted laser desorption ionisation (MALDI) – MALDI Biotyper (Bruker Daltonics, USA) or Vitek-MS (bioMérieux, France) – or by the Vitek® 2 automated system (bioMérieux). Antimicrobial susceptibility testing was performed using the Vitek® 2 (bioMérieux) or the BD Phoenix™ (Becton Dickinson, USA) automated microbiology systems, according to the manufacturer's instructions. Minimum inhibitory concentration (MIC) data and isolates were referred to the AESOP reference laboratory at Murdoch University. The European Committee on Antimicrobial Susceptibility Testing (EUCAST) MIC breakpoints were utilised for interpretation.[8] Linezolid-resistant isolates, and vancomycin-susceptible isolates which harboured the *vanA* or *vanB* gene, were retested by Etest® (bioMérieux) using Mueller-Hinton agar and the method recommended by the manufacturer. The control strain used for the Etest® was *E. faecalis* ATCC® 29212. For all *E. faecium* isolates received, whole genome sequencing (WGS) was performed by the AESOP reference laboratory at Murdoch University on the Illumina NovaSeq™ platform. The multilocus sequence type (ST) was determined using the PubMLST website; *van* genes were identified using nucleotide sequences from the NCBI database and a BLAST interface. Genetic determinants for linezolid resistance were identified using the LREfinder tool.[9]

Confidence intervals for proportions, Fisher's exact test for categorical variables, and chi-square test for trend were calculated, if appropriate, using MedCalc for Windows, version 23.3.1 (MedCalc Software, Ostend Belgium).

Approval to conduct the prospective data collection was given by the research ethics committee associated with each participating laboratory.

Results

From 1 January to 31 December 2024, there were 1,461 unique episodes of enterococcal bacteraemia identified. Although thirteen different enterococcal species were identified, 752 isolates (51.5%) were *E. faecalis* and 599 isolates (41.0%) were *E. faecium*. One hundred and ten enterococci were identified as either *E. lactis* (42 isolates); *E. casseliflavus* (19 isolates); *E. gallinarum* (16 isolates); *E. avium* (12 isolates); *E. raffinosus* (9 isolates); *E. durans* (5 isolates); *E. hirae* (3 isolates); *E. malodoratus* (1 isolate); *E. mundtii* (1 isolate); *E. pallens* (1 isolate); or *Enterococcus* species (unidentified; 1 isolate).

A significant difference was observed in patient sex ($p < 0.01$), with 958 patients (65.6%) being male (95% confidence interval [95% CI]: 61.5–69.9). The average age of patients was 64 years, ranging from 0 to 99 years, with a median age of 68 years. Overall, isolates were approximately evenly divided by place of onset: 753/1,461 (51.5%) were community-onset and 708/1,461 (48.5%) were hospital-onset. However, a significant difference ($p < 0.01$) was observed between *E. faecium* and *E. faecalis* in place of onset: only 25.2% of *E. faecium* episodes (95% CI: 21.4–30.0) were community-onset, compared to 70.6% of *E. faecalis* episodes (95% CI: 64.7–76.9). All-cause mortality at 30 days, where outcome was known, was 23.6% (95% CI: 20.9–26.5). There was a significant difference in mortality between *E. faecalis* and *E. faecium* episodes (15.5% vs 34.0% respectively; $p < 0.01$). However, there was no significant difference in mortality between vancomycin-susceptible and vancomycin-resistant *E. faecium* episodes (33.3% vs 34.2% respectively; $p = 0.85$).

Enterococcus faecalis phenotypic susceptibility results

Acquired resistance was rare amongst the *E. faecalis* isolates (Table 1). Seven *E. faecalis* isolates (0.9%) were initially reported as linezolid resistant (EUCAST breakpoint > 4 mg/L). However, by Etest®, two of the seven referred isolates had linezolid MICs of 1.0 and 2.0 mg/L and were therefore considered linezolid susceptible. The remaining five isolates had linezolid MICs of 6.0 and 8.0 mg/L and were classified as linezolid resistant by EUCAST criteria. All five linezolid-resistant *E. faecalis* isolates were vancomycin susceptible and harboured the transferable linezolid resistance *optrA* gene. High-level gentamicin resistance was reported in 13.1% of *E. faecalis* isolates.

Table 1: The number and proportion of *E. faecalis* isolates resistant to ampicillin and the non-β-lactam antimicrobials, EUCAST breakpoints,[a] AGAR, Australia, 2024

Antimicrobial	Isolates (n)	Susceptible % (*n*)	Resistant % (*n*)
Ampicillin	748	100.0 (748)	0.0 (0)
Linezolid	747	99.3 (742)	0.7 (5)
High-level gentamicin	672	86.9 (584)	13.1 (88)
Teicoplanin	748	100.0 (748)	0.0 (0)
Vancomycin	748	100.0 (748)	0.0 (0)

a EUCAST: European Committee on Antimicrobial Susceptibility Testing.

Enterococcus faecium phenotypic susceptibility results

The majority of the *E. faecium* isolates were ampicillin resistant (Table 2). High-level gentamicin resistance was reported in 36.3% of isolates. Using the EUCAST interpretive breakpoints, 264 *E. faecium* isolates (44.5%) were phenotypically vancomycin resistant. Ninety-nine isolates (16.8%) were teicoplanin resistant. One isolate was reported as linezolid resistant by the Vitek® 2 (≥ 8 mg/L) and confirmed by Etest® (6.0 mg/L). The linezolid-resistant *E. faecium* isolate was also vancomycin and teicoplanin resistant and harboured the *optrA* and *vanA* genes.

Table 2: The number and proportion of *E. faecium* isolates resistant to ampicillin and the non-β-lactam antimicrobials, EUCAST breakpoints,[a] AGAR, Australia, 2024

Antimicrobial	Isolates (n)	Susceptible % (*n*)	Resistant % (*n*)
Ampicillin	591	3.2 (19)	96.8 (572)
Linezolid	593	99.8 (592)	0.2 (1)
High-level gentamicin	545	63.7 (347)	36.3 (198)
Teicoplanin	590	83.2 (491)	16.8 (99)
Vancomycin	593	55.5 (329)	44.5 (264)

a EUCAST: European Committee on Antimicrobial Susceptibility Testing.

Detection of *van* genes in *E. faecalis* and *E. faecium*

For 349 of the 752 *E. faecalis* isolates (46.4%), *vanA*/*vanB* polymerase chain reaction (PCR) testing was performed by the referring laboratories. Neither *vanA* nor *vanB* was detected in *E. faecalis*.

The presence of the *vanA* or *vanB* gene was determined by PCR and/or WGS on 580 of the 599 *E. faecium* isolates (96.8%). Overall, 289 of the 580 isolates (49.8%) harboured a *vanA* and/or *vanB* gene. Of the vancomycin-resistant *E. faecium* isolates (Vitek® 2: vancomycin MIC > 4 mg/L), 105 harboured *vanA* and 150 harboured *vanB*. Two vancomycin-resistant isolates harboured both *vanA* and *vanB*. The *vanA* or *vanB* gene was detected in twenty-seven vancomycin-susceptible *E. faecium* isolates. Nine isolates, with vancomycin MICs ranging from ≤ 0.5 to 4.0 mg/L and teicoplanin MICs ranging from ≤ 0.5 to 2.0 mg/L, harboured *vanA*. Seventeen *vanB*-positive isolates had vancomycin MICs ranging from ≤ 0.5 mg/L to 2.0 mg/L. One isolate with a vancomycin MIC of 2.0 mg/L and teicoplanin MIC of 1.5 mg/L harboured both *vanA* and *vanB*. Vancomycin MICs were not available for five *vanA*/*B*-harbouring *E. faecium* isolates (two *vanA* and three *vanB*).

E. faecium molecular epidemiology

Of the 599 episodes, 530 *E. faecium* isolates (88.5%) were available for WGS. The 530 isolates were classified into 56 STs, including eight STs with ten or more isolates (Table 3). Of the 48 STs with fewer than ten isolates each, 35 were each represented by only one isolate. Overall, 454 of the 530 isolates (85.7%) were grouped into the eight major STs (greater than ten isolates). Using eBURST, all major STs were grouped into CC17.

Geographical distribution of the STs varied (Table 3). Amongst the eight major STs, ST78 (106 isolates) was identified in all regions except Queensland; ST1424 (94 isolates), ST17 (91 isolates) and ST80 (63 isolates) were identified in all regions except the Northern Territory; ST1421 (45 isolates) was identified in all regions except Western Australia, Tasmania and the Australian Capital Territory; ST117 (26 isolates) was identified in all regions except South Australia, the Northern Territory and the Australian Capital Territory; ST796 (17 isolates) was identified only in Western Australia; and ST555 (12 isolates) was identified only in Victoria, South Australia and Western Australia.

Table 3: The number and proportion of major *Enterococcus faecium* multilocus sequence types, AGAR, Australia, 2024, by jurisdiction

MLST[a]	Percentage % (*n*)[b]								
	ACT	NSW	NT[c]	Qld	SA	Tas.	Vic.	WA	Australia
ST78	31.3 (5)	23.2 (36)	— (4)	0.0 (0)	34.0 (16)	13.3 (4)	24.4 (33)	11.8 (8)	20.0 (106)
ST1424	25.0 (4)	20.0 (31)	— (0)	40.0 (28)	4.3 (2)	50.0 (15)	9.6 (13)	1.5 (1)	17.7 (94)
ST17	6.3 (1)	11.6 (18)	— (0)	14.3 (10)	19.1 (9)	20.0 (6)	9.6 (13)	50.0 (34)	17.2 (91)
ST80	31.3 (5)	4.5 (7)	— (0)	21.4 (15)	12.8 (6)	3.3 (1)	14.8 (20)	13.2 (9)	11.9 (63)
ST1421	0.0 (0)	21.3 (33)	— (3)	5.7 (4)	2.1 (1)	0.0 (0)	3.0 (4)	0.0 (0)	8.5 (45)
ST117	0.0 (0)	7.1 (11)	— (0)	1.4 (1)	0.0 (0)	3.3 (1)	8.9 (12)	1.5 (1)	4.9 (26)
ST796	0.0 (0)	0.0 (0)	— (0)	0.0 (0)	0.0 (0)	0.0 (0)	12.6 (17)	0.0 (0)	3.2 (17)
ST555	0.0 (0)	0.0 (0)	— (0)	0.0 (0)	14.9 (7)	0.0 (0)	3.0 (4)	1.5 (1)	2.3 (12)
Other types (n = 48)	6.3 (1)	10.3 (16)	— (2)	14.3 (10)	8.5 (4)	10.0 (3)	11.9 (16)	17.6 (12)	12.1 (64)
Total	**16**	**155**	**9**	**70**	**47**	**30**	**135**	**68**	**530**

a MLST: multilocus sequence type.

b ACT: Australian Capital Territory; NSW: New South Wales; NT: Northern Territory; Qld: Queensland; SA: South Australia; Tas: Tasmania; Vic: Victoria; WA: Western Australia.

c Proportion by jurisdiction not calculated for jurisdictions with fewer than ten sequenced isolates in total.

The *vanA* gene was detected in five of the major STs: 97 isolates from ST17, ST80, ST117, ST1421 and ST1424 (Table 4). With the exception of ST1421, the *vanB* gene was detected in all major STs (149 isolates). Co-carriage of *vanA* and *vanB* was detected in two ST1424 isolates. Five minor STs (ST817, ST2771, ST1478, ST375 and ST2849) harboured at least one *vanA*-positive isolate; and seven minor STs (ST18, ST2842, ST2844, ST797, ST341 ST2846 and ST2850) harboured at least one *vanB*-positive isolate. One minor ST (ST203) harboured an isolate with both *vanA* and *vanB*.

Table 4: The number and proportion of major *Enterococcus faecium* multilocus sequence types harbouring *vanA*/*vanB* gene, AGAR, Australia, 2024

	Percentage[a] (*n*)				
MLST[b]	*vanA*	*vanB*	*vanA* and *vanB*	*vanA* or *vanB* not detected	Total, *n*
ST78	0.0 (0)	100.0 (106)	0.0 (0)	0.0 (0)	106
ST1424	53.2 (50)	2.1 (2)	2.1 (2)	42.6 (40)	94
ST17	2.2 (2)	6.6 (6)	0.0 (0)	91.2 (83)	91
ST80	4.8 (3)	9.5 (6)	0.0 (0)	85.7 (54)	63
ST1421	88.9 (40)	0.0 (0)	0.0 (0)	11.1 (5)	45
ST117	7.7 (2)	3.8 (1)	0.0 (0)	88.5 (23)	26
ST796	0.0 (0)	100.0 (17)	0.0 (0)	0.0 (0)	17
ST555	0.0 (0)	91.7 (11)	0.0 (0)	8.3 (1)	12
Other types	10.5 (8)	9.2 (7)	1.3 (1)	78.9 (60)	76
Total	**19.8 (105)**	**29.4 (156)**	**0.6 (3)**	**50.2 (266)**	**530**

a Percentage of total with *van* genes, for a given MLST category.

b MLST: multilocus sequence type.

Discussion

Enterococci are intrinsically resistant to a broad range of antimicrobials including the cephalosporins and sulfonamides. Because of their ability to acquire additional antimicrobial resistance (AMR) through the transfer of plasmids and transposons and to disseminate easily in the hospital environment, enterococci have become difficult to treat and provide major infection control challenges.

In AESOP 2024, a total of 41.0% of enterococcal bacteraemias were due to *E. faecium*, of which 44.5% (95% CI: 39.3–50.2) were phenotypically vancomycin resistant by Vitek® 2 or BD Phoenix™. However, 49.8% of all *E. faecium* isolates tested (289/580) harboured a *vanA* and/or *vanB* gene, of which 40.1% were *vanA*-positive. Overall, 119 *E. faecium* isolates (20.5%) harboured the *vanA* gene. Over the last five years (2020–2024), there has been a significant increasing trend in *vanA*-positive *E. faecium* in Australia (χ^2 for linear trend = 7.23; $p < 0.01$).[10–13] This trend is primarily due to an increase in the number of ST1424 and ST1421 isolates. The majority of *E. faecium* isolates were resistant to multiple antimicrobials.

As the AGAR programs are similar to the AMR surveillance programs conducted in Europe, comparison of Australian AMR data with other European countries is possible.

In the 2023 European AMR Surveillance Network (EARS-Net) program, the national percentages of vancomycin-resistant *E. faecium* ranged from 0.0% in Iceland to 60.9% in Lithuania.[14,15] The AESOP 2024 survey confirms that the incidence of vancomycin-resistant *E. faecium* bacteraemia in Australia continues to be significantly higher than that recorded in most European countries.

Seventeen (10.0%) of the 170 *vanB*-positive *E. faecium*, and nine (7.8%) of the 116 *vanA*-positive *E. faecium* isolates had a vancomycin MIC at or below the EUCAST susceptible breakpoint (≤ 4 mg/L) and therefore would not have been identified using routine phenotypic antimicrobial susceptibility methods.

WGS demonstrates that *E. faecium* in Australia is polyclonal, consistent with the known plasticity of the enterococcal genome. The eight major *E. faecium* STs identified form part of CC17, a global hospital-derived lineage that has successfully adapted to hospital environments. CC17 is characteristically ampicillin and quinolone resistant and subsequent acquisition of *vanA*- or *vanB*-containing transposons by horizontal transfer has resulted in multi-resistant CC17 enterococci with pandemic potential.

In AESOP 2024, eight *E. faecium* STs predominated: ST78 (100% harboured *vanB*); ST1424 (53.2% *vanA*, 2.1% *vanB*); ST17 (2.2% *vanA*, 6.6% *vanB*); ST80 (4.8% *vanA*, 9.5% *vanB*), ST1421 (88.9% *vanA*) ST117 (7.7% *vanA*, 3.8% *vanB*), ST796 (100% *vanB*); and ST555 (91.7% *vanB*).

Conclusions

The AESOP 2024 study has shown that, although predominately caused by *E. faecalis*, enterococcal bacteraemia in Australia is frequently caused by ampicillin-resistant, high-level gentamicin-resistant and vancomycin-resistant *E. faecium*. Furthermore, the percentage of *E. faecium* bacteraemia isolates resistant to vancomycin in Australia (44.5%) remains significantly higher than that seen in most European countries. In addition to being a significant cause of healthcare-associated bacteraemia, the emergence of multiple multi-resistant hospital-adapted *E. faecium* strains has become a major infection control issue in Australian hospitals.

Ongoing studies on the enterococcal genome will contribute to our understanding of the rapid and ongoing evolution of enterococci in the hospital environment and will assist in preventing their nosocomial transmission.

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St John Of God Hospital, Murdoch, Sudha Pottumarthy-Boddu and Alicia Robinson

Author details

Prof. Geoffrey W Coombs,[1,2,3]

Ms Denise A Daley,[2,3]

Ms Auriane Form,[1]

Ms Xing Li,[1]

Mr Christian Torres[1]

Dr Shakeel Mowlaboccus,[1,3]

on behalf of the Australian Group on Antimicrobial Resistance

1. School of Medical, Molecular and Forensic Sciences, Murdoch University, Murdoch, Western Australia, Australia
2. Department of Microbiology, PathWest Laboratory Medicine-WA, Fiona Stanley Hospital, Murdoch, Western Australia, Australia
3. Australian Group on Antimicrobial Resistance, Fiona Stanley Hospital, Murdoch, Western Australia, Australia

Corresponding author

Prof. Geoffrey Coombs

School of Medical, Molecular and Forensic Sciences, Murdoch University, Murdoch, Western Australia, Australia

Telephone: +61 8 6152 2397

Email: g.coombs@murdoch.edu.au

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