Australian Group on Antimicrobial Resistance (AGAR) Australian Enterococcal Sepsis Outcome Programme (AESOP) Annual Report 2016

Geoffrey W Coombs, Denise A Daley, Yung Thin Lee and Stanley Pang on behalf of the Australian Group on Antimicrobial Resistance

# Abstract

From 1st January to 31st December 2016, 32 institutions around Australia participated in the Australian Enterococcal Sepsis Outcome Programme (AESOP). The aim of AESOP 2016 was to determine the proportion of enterococcal bacteraemia isolates in Australia that were antimicrobial resistant, and to characterise the molecular epidemiology of the E. faecium isolates. Of the 1,058 unique episodes of bacteraemia investigated, 95.2% were caused by either E. faecalis (56.2%) or E. faecium (39.0%) Ampicillin resistance was detected in 0.2% of E. faecalis and in 91.5% of E. faecium. Vancomycin non-susceptibility was reported in 0.3% and 47.7% of E. faecalis and E. faecium respectively. Overall, 49.3% of E. faecium harboured vanA or vanB genes. For the vanA/B positive E. faecium isolates, 55.2% harboured vanB genes and 42.8% vanA genes, 2% harboured vanA and vanB genes. The percentage of E. faecium bacteraemia isolates resistant to vancomycin in Australia is significantly higher than that seen in most European countries. E. faecium consisted of 48 multilocus sequence types (STs) of which 90.2% of isolates were classified into 13 major STs containing 5 or more isolates. All major STs belong to clonal cluster (C) 17, a major hospital-adapted polyclonal E. faecium cluster. Four of the 6 predominant STs (ST17, ST796, ST80 and ST203) were found across most regions of Australia. The most predominant clone ST1421 (previously known as M-type 1) does not have a pstS housekeeping gene and was found in NSW, the ACT and Victoria. This clone was first described in ASSOP 2015. Overall 74% of isolates belonging to the 6 predominant STs harboured vanA or vanB genes. The AESOP 2016 has shown enterococcal bacteraemias in Australia are frequently caused by polyclonal ampicillin-resistant high-level gentamicin resistant vanA or vanB 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, enterococci are thought to account for approximately 10% of all bacteraemias, and in North America and Europe is the fourth and fifth leading cause of sepsis respectively.1,2 Although, in the 1970s healthcare-associated enterococcal infections were primarily due to Enterococcus faecalis, there has been a steadily increasing prevalence of E. faecium nosocomial infections.3-5 Worldwide the increase in nosocomial E. faecium infections has primarily been due to the expansion of polyclonal hospital-adapted clonal complex (CC) 17 strains. While innately resistant to many classes of antibiotics, 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 (Enterococcus faecium, Staphylococcus aureus, Klebsiella pneumoniae, Acinetobacter baumannii, Pseudomonas aeruginosa, and Enterobacter species) pathogens requiring new therapies.6

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 1995.7 In 2011, AGAR commenced the Australian Enterococcal Sepsis Outcome Programme (AESOP).8 The objective of AESOP 2016 was to determine the proportion of E. faecalis and E. faecium bacteraemia isolates demonstrating antimicrobial resistance with particular emphasis on:

* Assessing susceptibility to ampicillin
* Assessing susceptibility to glycopeptides
* Molecular epidemiology of E. faecium

## Methodology

### Participants

32 laboratories from all 8 Australian states and territories.

### Collection Period

From 1st January to 31st December 2016, the 32 laboratories collected all enterococcal species isolated from blood cultures. Enterococci with the same species and antimicrobial susceptibility profiles isolated from a patient’s blood culture within 14 days of the first positive culture were excluded. A new enterococcal sepsis episode in the same patient was recorded if it was confirmed by a further culture of blood taken more than 14 days after the initial positive culture. Data were collected on age, sex, date of admission and discharge (if admitted), and mortality at 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 one of the following methods: API 20S (bioMérieux), API ID32Strep (bioMérieux), Vitek2® (bioMérieux), Phoenix™ (BD), matrix-assisted laser desorption ionization (MALDI) Biotyper (Bruker Daltonics), Vitek-MS (bioMérieux), PCR, or conventional biochemical tests. Antimicrobial susceptibility testing was performed by using the Vitek2® (bioMérieux, France) or the Phoenix™ (BD, USA) automated microbiology systems according to the manufacturer’s instructions. Minimum inhibitory concentration (MIC) data and isolates were referred to the Antimicrobial Resistance and Infectious Diseases (AMRID) Research Laboratory, at Murdoch University. Clinical and Laboratory Standards Institute (CLSI) and European Committee on Antimicrobial Susceptibility Testing (EUCAST) breakpoints were utilised for interpretation.9,10 Isolates with either a resistant or an intermediate category were classified as non-susceptible. Linezolid and daptomycin non-susceptible isolates and selected vancomycin susceptible isolates were retested by Etest® (bioMérieux, France) using the Mueller-Hinton agar recommended by the manufacturer. E. faecalis ATCC®29212 was used as the control strain. Molecular testing was performed by whole genome sequencing using the MiSeq® platform (Illumina, San Diego, USA). Sequencing results were analysed using the Nullarbor pipeline.11

A chi-square test for comparison of 2 proportions was performed and 95% confidence intervals (95% CI) were determined using MedCalc for Windows, version 12.7 (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 1st January to 31st December 2016, 1,058 unique episodes of enterococcal bacteraemia were identified. Although 7 Enterococcus species were identified, 56.2% (595 isolates) were E. faecalis and 39.0% (413 isolates) were E. faecium. Fifty enterococci were identified either as E. casseliflavus (25 isolates), E. gallinarum (11), E. avium (8), E. hirae (3) and E. raffinosus (3).

A significant difference was seen in patient sex (p<0.0001) with 701 (66.3%) being male (95% CI, 63.4– 69.2). The average age of patients was 64 years ranging from 0 – 101 years with a median age of 68 years. The majority of episodes 53.0% (561/1,058) were hospital-onset (95% CI, 49.9 – 56.0). However, a significant difference (p<0.0001) was seen between E. faecium and E. faecalis, with 72.4% (95% CI, 67.8 – 76.7) of E. faecium episodes being hospital-onset compared to 31.3% (95% CI, 28.0 – 35.2) for E. faecalis. All-cause mortality at 30 days where data was known was 19.3% (95% CI, 16.8 – 22.0). There was a significant difference (p<0.0001) in mortality between E. faecalis and E. faecium episodes 12.9% vs 27.2% respectively, and between vancomycin susceptible and vancomycin non-susceptible E. faecium episodes 17.0% vs 28.3% respectively (p=0.0008).

## *E. faecalis* Phenotypic Susceptibility Results

Apart from erythromycin, tetracycline, ciprofloxacin and high-level gentamicin, acquired resistance was rare amongst E. faecalis (Table 1). Ampicillin resistance was detected in one isolate and 2 isolates were vancomycin non-susceptible. Twenty-two (3.7%) E. faecalis, were initially reported as linezolid non-susceptible (CLSI breakpoint >2 mg/L). However, by Etest® seven of the 22 isolates had a linezolid MIC of ≤2 mg/L and were therefore considered linezolid susceptible. Fifteen isolates with an MIC of 4 mg/L although non-susceptible by CLSI guidelines were considered susceptible by EUCAST guidelines. Two isolates had an MIC of 8 mg/L and were non-susceptible. All isolates were susceptible to teicoplanin and daptomycin.

Table 1: The number and proportion of *E. faecalis* non-susceptible to ampicillin and the non-β-lactam antimicrobials, Australia, 2016

| Antimicrobial | Tested | Breakpoint (mg/L) | Non-Susceptible | |
| --- | --- | --- | --- | --- |
| n | % |
| Ampicillin | 592 | >8\* | 1 | 0.2 |
| >4† | 1 | 0.2 |
| Vancomycin | 592 | >4‡ | 2 | 0.3 |
| Erythromycin | 579 | >0.5\* | 522 | 90.2 |
| Tetracycline/Doxycycline | 579 | >4\* | 384 | 71.0 |
| Ciprofloxacin | 559 | >1\* | 86 | 15.4 |
| Daptomycin | 542 | >4\* | 0 | 0 |
| Teicoplanin | 558 | >8\* | 0 | 0 |
| >2† | 0 | 0 |
| Linezolid | 591 | >2\* | 17 | 2.9 |
| >4† | 2 | 0.3 |
| Nitrofurantoin | 591 | >32\* | 3 | 0.5 |
| >64† | 0 | 0 |
| High Level Gentamicin | 589 | >128† | 143 | 24.3 |

\*CLSI non-susceptible breakpoint  
†EUCAST non-susceptible breakpoint  
‡CLSI and EUCAST non-susceptible breakpoint

## E. faecium Phenotypic Susceptibility Results

The majority of E. faecium were non-susceptible to multiple antimicrobials (Table 2). Most isolates were non-susceptible to ampicillin, erythromycin, tetracycline, ciprofloxacin, nitrofurantoin and high-level gentamicin. Overall, 197 (47.7%) were phenotypically vancomycin non-susceptible (MIC >4 mg/L). Seventy-eight (18.9%) and eighty-one (19.6%) isolates were teicoplanin non-susceptible by CLSI and EUCAST guidelines respectively. Four (1.0%) isolates were initially reported as linezolid non-susceptible (CLSI breakpoint >2 mg/L). However, by Etest® 2 of the 4 isolates had a linezolid MICs of 2 mg/ and 1.5mg/L and therefore were considered susceptible. Two isolates had MICs of 4mg/L which was considered susceptible by EUCAST guidelines but non-susceptible by CLSI guidelines.

Table 2: The number and proportion of *E. faecium* non-susceptible to ampicillin and the non-β-lactam antimicrobials, Australia, 2016

| Antimicrobial | Tested | Breakpoint (mg/L) | Non-Susceptible | |
| --- | --- | --- | --- | --- |
| n | % |
| Ampicillin | 412 | >8\* | 377 | 91.5 |
| >4† | 380 | 92.2 |
| Vancomycin | 413 | >4‡ | 197 | 47.7 |
| Erythromycin | 410 | >0.5\* | 389 | 94.9 |
| Tetracycline/Doxycycline | 411 | >4\* | 256 | 62.3 |
| Ciprofloxacin | 404 | >1\* | 372 | 92.1 |
| Teicoplanin | 413 | >8\* | 78 | 18.9 |
| >2† | 81 | 19.6 |
| Linezolid | 399 | >2\* | 2 | 0.5 |
| >4† | 0 | 0 |
| Nitrofurantoin | 398 | >32\* | 319 | 77.8 |
| >64† | 196 | 47.8 |
| High Level Gentamicin | 385 | >128\* | 230 | 59.7 |

\*CLSI non-susceptible breakpoint  
†EUCAST non-susceptible breakpoint  
‡CLSI and EUCAST non-susceptible breakpoint

## Genotypic Vancomycin Susceptibility Results

vanA/vanB PCR results were available for 302 of the 595 E. faecalis isolates. Three of the 302 isolates harboured a vanB gene and one isolate harboured a vanA gene.

The presence of vanA/B genes was determined by PCR or whole genome sequencing on 408 of the 413 E. faecium isolates. Overall, 201 (49.3%) of the 408 isolates harboured a vanA and/or vanB gene. Eighty-three of the vancomycin non-susceptible E. faecium isolates harboured vanA (Vitek® vancomycin MIC >4mg/). A further 110 E. faecium vancomycin non-susceptible isolates harboured vanB). Four isolates harboured vanA and vanB genes.

vanA or vanB genes were detected in 6 vancomycin susceptible E. faecium isolates. Three isolates harboured vanA (Vitek® vancomycin MIC ≤ 0.5mg/L [one isolate], MIC = 1 mg/L [2 isolates], teicoplanin ≤1mg/L [3 isolates]). Three isolates harboured vanB (Vitek® vancomycin MIC = 1 mg/L, 2mg/L and 4mg/L)

## E. faecium Molecular Epidemiology

Of the 413 episodes, 400 E. faecium isolates were available for typing by whole genome sequencing. The 400 isolates were classified into 48 sequence types (STs) including 13 STs with 5 or more isolates (Table 3). A clone first described in AESOP 2015 (non-typable pstS housekeeping gene absent) was re-named M-type 1. In 2016, there were 4 M-type single locus variants (M-type 1 [64 isolates], M-type 2 [5 isolates], M-type 3 [15 isolates] and M-type 4 [1 isolate]). These have now been classified as ST1421, ST1422, ST1424 and ST1423 respectively. Of the 35 STs with <5 isolates, 30 had only one isolate. Overall 359 (89.8%) of the 400 isolates were grouped into the 13 major STs. Using eBURST, the 13 STs were grouped into CC 17.

Table 3: The number and proportion of *major Enterococcus faecium* sequence types, Australia, 2016, by region

| ST | ACT | | NSW | | NT | | Qld | | SA | | Tas | | Vic | | WA | | Aus | |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
| n | % | n | % | n | % | n | % | n | % | n | % | n | % | n | % | n | % |
| 1,421 | 15 | 68.2 | 40 | 34.8 |  |  |  |  |  |  |  |  | 9 | 8.3 |  |  | 64 | 16.0 |
| 17 | 1 | 4.5 | 5 | 4.3 |  |  | 22 | 52.4 | 2 | 4.9 |  |  | 8 | 7.3 | 21 | 39.6 | 59 | 14.8 |
| 796 |  |  | 2 | 1.7 | 1 | 25.0 | 1 | 2.4 | 2 | 4.9 | 6 | 42.9 | 45 | 41.3 |  |  | 57 | 14.3 |
| 80 | 3 | 13.6 | 19 | 16.5 |  |  | 3 | 7.1 | 1 | 2.4 |  |  | 15 | 13.8 | 10 | 18.9 | 51 | 12.8 |
| 555 |  |  |  |  | 2 | 50.0 | 1 | 2.4 | 20 | 48.8 | 3 | 21.4 |  |  | 13 | 24.5 | 39 | 9.8 |
| 203 | 1 | 4.5 | 6 | 5.2 |  |  | 4 | 9.5 | 3 | 7.3 | 1 | 7.1 | 10 | 9.2 | 1 | 1.9 | 26 | 6.5 |
| 1,424 |  |  | 15 | 13.0 |  |  |  |  |  |  |  |  |  |  |  |  | 15 | 3.8 |
| 78 |  |  | 7 | 6.1 |  |  | 6 | 14.3 |  |  |  |  |  |  |  |  | 13 | 3.3 |
| 262 |  |  |  |  |  |  |  |  | 12 | 29.3 |  |  |  |  |  |  | 12 | 3.0 |
| 117 |  |  | 4 | 3.5 |  |  | 1 | 2.4 |  |  | 1 | 7.1 |  |  | 1 | 1.9 | 7 | 1.8 |
| 192 |  |  |  |  |  |  |  |  |  |  | 2 | 14.3 | 4 | 3.7 |  |  | 6 | 1.5 |
| 18 |  |  | 1 | 0.9 |  |  | 1 | 2.4 |  |  |  |  | 1 | 0.9 | 2 | 3.8 | 5 | 1.3 |
| 1,422 |  |  | 5 | 4.3 |  |  |  |  |  |  |  |  |  |  |  |  | 5 | 1.3 |
| Other | 2 | 9.1 | 11 | 9.6 | 1 | 25.0 | 3 | 7.1 | 1 | 2.4 | 1 | 7.1 | 17 | 15.6 | 5 | 9.4 | 41 | 10.3 |
| Total | 22 | 100 | 115 | 100 | 4 | 100 | 42 | 100 | 41 | 100 | 14 | 100 | 109 | 100 | 53 | 100 | 400 | 100 |

\*The pstS housekeeping gene is absent in the M-type isolates. ACT = Australian Capital Territory; NSW = New South Wales; NT = Northern Territory; Qld = Queensland; SA = South Australia; Tas1 = Tasmania; Vic = Victoria; WA = Western Australia; Aus = Australia

Geographical distribution of the STs varied (Table 3). For the six most prominent STs, ST1421 (64 isolates) was identified in the Australian Capital Territory, New South Wales and Victoria; ST17 (59 isolates) in all regions except Western Australia and the Australian Capital Territory; ST80 (51 isolates) found in all regions except the Northern Territory and Tasmania; ST555 (39 isolates) across most of Australia except the Australian Capital Territory, New South Wales and Victoria; and ST203 (39 isolates) found in all regions except the Northern Territory.

vanA was detected in seven major STs (85 isolates, ST1421, ST17, ST80, ST555, ST203, ST1424, and ST78). vanB was detected in nine major STs (111 isolates, ST1421, ST17, ST796, ST80, ST555, ST203, ST78, and ST262) (Table 4). ST780 harboured vanA and vanB genes. One minor ST (1 isolate) harboured vanB genes and one minor ST (1 isolate) harboured vanA and vanB genes.

Table 4: The number and proportion of major *Enterococcus faecium* sequence types harbouring vanA/B genes, Australia, 2016

| ST | n | vanA | | vanB | | vanA and vanB | | Not Detected | |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
| n | % | n | % | n | % | n | % |
| 1,421 | 64 | 51 | 79.7 | 1 | 1.6 |  |  | 12 | 18.8 |
| 17 | 59 | 1 | 1.7 | 4 | 6.8 |  |  | 54 | 91.5 |
| 796 | 57 |  |  | 55 | 96.5 |  |  | 2 | 3.5 |
| 80 | 51 | 17 | 33.3 | 5 | 9.8 | 2 | 3.9 | 27 | 52.9 |
| 555 | 39 | 1 | 2.6 | 17 | 43.6 |  |  | 21 | 53.8 |
| 203 | 26 | 3 | 11.5 | 14 | 53.8 |  |  | 9 | 34.6 |
| 1,424 | 15 | 11 | 73.3 |  |  |  |  | 4 | 26.7 |
| 78 | 13 | 1 | 7.7 | 11 | 84.6 |  |  | 1 | 7.7 |
| 262 | 12 |  |  | 2 | 16.7 |  |  | 10 | 83.3 |
| 117 | 7 |  |  |  |  |  |  | 7 | 100 |
| 192 | 6 |  |  |  |  |  |  | 6 | 100 |
| 18 | 5 |  |  |  |  |  |  | 5 | 100 |
| 1,422 | 5 |  |  |  |  |  |  | 5 | 100 |
| Other | 41 |  |  | 1 | 2.4 | 1 | 2.4 | 39 | 95.1 |
| Total | 400 | 85 | 21.3 | 110 | 27.5 | 3 | 0.8 | 202 | 50.5 |

\*The pstS housekeeping gene is absent in the M-type isolates.

# Discussion

Enterococci are intrinsically resistant to a broad range of antimicrobials including the cephalosporins and sulphonamides. By their ability to acquire additional resistance 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.

As the AGAR programs are similar to those conducted in Europe (http://www.ecdc.europa.eu/en/healthtopics/antimicrobial\_resistance/database/Pages/database.aspx) comparison of Australia antimicrobial resistance data with other countries is possible.

In the 2016 European Centre for Disease Prevention and Control and Prevention (ECDC) Enterococci surveillance program, the European Union/European Economic Area (EU/EEA) population-weighted mean percentage of E. faecium resistant to vancomycin was 11.8% (95% CI, 11 – 13), ranging from 0.0% in Estonia (95% CI, 0 – 6), Finland (95% CI, 0 – 1), Iceland, (95% CI, 0 – 21) Luxembourg (95% CI, 0 – 11) and Slovenia (95% CI, 0 – 3) to 45.8% (95% CI, 31 – 63) in Cyprus. (http://ecdc.europa.eu/en/publications/Publications/antimicrobial-resistance-surveillance-europe-2016.pdf).

In AESOP 2016, approximately 40% of enterococcal bacteraemia were due to E. faecium of which 47.7% (95% CI, 42.8 – 52.6) were phenotypically vancomycin non-susceptible by Vitek2® or Phoenix™. However, 49.3% of E. faecium isolates tested (201/408) harboured vanA/vanB genes, of which 55% were vanB. Overall, 21.6% (88/408) of E. faecium isolates harboured a vanA gene. There has been a significant increase in vanA E. faecium in Australia over the last 3 surveys from 6% (8/310) in AESOP 2013, 9.5% (35/370) in 2014 and 20.7% (82/397) in 2015.12,13 The majority of E. faecium isolates were also non-susceptible to multiple antimicrobials including ampicillin, erythromycin, tetracycline, ciprofloxacin and high level gentamicin. In AESOP 201114, 201312, 201413 and 201515 37.0%, 48.6%, 51.1% and 49.3% of E. faecium respectively harboured vanA/vanB confirming the incidence of vancomycin resistant E. faecium bacteraemia in Australia is a significant problem.

Three (2.7%) of the 110 vanB E. faecium and 3 of the vanA E. faecium isolates had a vancomycin MIC at or below the CLSI and the EUCAST susceptible breakpoint (≤4 mg/L) and therefore would not have been identified using routine phenotypic antimicrobial susceptibility methods.

By whole genome sequencing, E. faecium was shown to be very polyclonal, consistent with the known plasticity of the enterococcal genome. The 13 major E. faecium STs formed 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 in CC17 clones has resulted in VRE with pandemic potential.

In AESOP 2016, six E. faecium STs predominated: ST1421 (of which 79.7% of isolates harboured vanA genes, 1.6% vanB genes); ST17 (6.8% vanB, 1.7% vanA); ST796 (96.5% vanB); ST80 (33.3% vanA, 9.8% vanB, 3.9% vanA and vanB), ST555 (43.6% vanB, 2.6% vanA) and ST203 (53.8% vanB, 11.5% van).

# Conclusions

The AESOP 2016 study has shown, although predominately caused by E. faecalis, enterococcal bacteraemia in Australia is frequently caused by ampicillin-resistant high-level gentamicin-resistant vanB E. faecium. Furthermore, the percentage of E. faecium bacteraemia isolates resistant to vancomycin in Australia is significantly higher than that seen in almost all European countries. Although the vanB operon continues to be the predominant genotype, the number of vanA E. faecium identified in AESOP 2016 has increased when compared to AESOP 2013, 2014 and 2015. In addition to being a significant cause of healthcare-associated sepsis, the emergence of multiple multi-resistant hospital-adapted E. faecium strains has become a major infection control issue in Australian hospitals. Further studies on the enterococcal genome will contribute to our understanding of the rapid and ongoing evolution of enterococci in the hospital environment and assist in preventing their nosocomial transmission.

# Acknowledgments

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

Members of the AGAR in 2016 were:

## Australian Capital Territory

Peter Collignon and Susan Bradbury, The Canberra Hospital

## New South Wales

Thomas Gottlieb and Graham Robertson, Concord Hospital

James Branley and Donna Barbaro, Nepean Hospital

Peter Huntington, Royal North Shore Hospital

Sebastian van Hal and Alicia Beukers, Royal Prince Alfred Hospital

Jon Iredell and Andrew Ginn, Westmead Hospital

Rod Givney and Ian Winney, John Hunter Hospital

Peter Newton and Melissa Hoddle, Wollongong Hospital

## Northern Territory

Rob Baird and Jann Hennessy, Royal Darwin Hospital

James McLeod, Alice Springs Hospital

## Queensland

Enzo Binotto and Bronwyn Thomsett, Pathology Queensland Cairns Base Hospital

Graeme Nimmo and Narelle George, Pathology Queensland Central Laboratory, Royal Brisbane and Women’s Hospital

Sam Maloney and Cheryl Curtis, Pathology Queensland Gold Coast Hospital

Robert Horvath and Laura Martin, Pathology Queensland Prince Charles Hospital

Naomi Runnegar and Joel Douglas, Pathology Queensland Princess Alexandra Hospital

Jenny Robson and Georgia Peachey, Sullivan Nicolaides Pathology, Greenslopes Hospital

## South Australia

Kelly Papanaoum and Nicholas Wells, SA Pathology (Flinders Medical Centre)

Morgyn Warner and Kija Smith, SA Pathology (Royal Adelaide Hospital and Women’s and Children’s Hospital)

## Tasmania

Louise Cooley and David Jones, Royal Hobart Hospital

Pankaja Kalukottege and Kathy Wilcox, Launceston General Hospital

## Victoria

Denis Spelman and Rose Bernhard, The Alfred Hospital

Paul Johnson and Frances Hurren, Austin Hospital

Tony Korman and Despina Kotsanas, Monash Medical Centre

Andrew Daley and Gena Gonis, Royal Women’s Hospital

Mary Jo Waters and Lisa Brenton, St Vincent’s Hospital

## Western Australia

David McGechie and Denise Daley, PathWest Laboratory Medicine – WA Fiona Stanley Hospital

Ronan Murray and Jacinta Bowman, PathWest Laboratory Medicine – WA Sir Charles Gairdner Hospital

Michael Leung and Jacinta Bowman, PathWest Laboratory Medicine – Northwest WA

Owen Robinson and Geoffrey Coombs, PathWest Laboratory Medicine – WA Royal Perth Hospital

Sudha Pottumarthy-Boddu and Fay Kappler, Australian Clinical Laboratories, St John of God Hospital, Murdoch

Shalinie Perera and Ian Meyer, Western Diagnostic Pathology, Joondalup Hospital

# Author Details

Prof Geoffrey W Coombs1,2, Ms Denise A Daley3, Ms Yung Thin Lee1, Dr Stanley Pang1,2, on behalf of the Australian Group on Antimicrobial Resistance

1. Antimicrobial Resistance and Infectious Disease (AMRID) Research Laboratory, School of Veterinary and Life 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

Antimicrobial Resistance and Infectious Disease (AMRID) Research Laboratory, School of Veterinary and Life Sciences, Murdoch University, Murdoch, Western Australia, Australia

Telephone: +61 8 6152 2397

Email: g.coombs@murdoch.edu.au

# Reference

1. Pinholt M, Ostergaard C, Arpi M et al. Incidence, clinical characteristics and 30-day mortality of enterococcal bacteraemia in Denmark 2006-2009: a population-based cohort study. Clin Microbiol Infect 2014;20(2):145-151.
2. Deshpande LM, Fritsche TR, Moet GJ et al. Antimicrobial resistance and molecular epidemiology of vancomycin-resistant enterococci from North America and Europe: a report from the SENTRY antimicrobial surveillance program. Diagn Microbiol Infect Dis 2007; 58: 163-70.
3. Murray BE. The life and times of the Enterococcus. Clin Microbiol Rev 1990; 3: 46-65.
4. Simonsen GS, Smabrekke L, Monnet DL et al. Prevalence of resistance to ampicillin, gentamicin and vancomycin in Enterococcus faecalis and *Enterococcus faecium* isolates from clinical specimens and use of antimicrobials in five Nordic hospitals. J Antimicrob Cchemother 2003; 51: 323-31.
5. Treitman AN, Yarnold PR, Warren J et al. Emerging incidence of *Enterococcus faecium* among hospital isolates (1993 to 2002). J clin Microbiol 2005; 43: 462-3.
6. Boucher HW, Talbot GH, Bradley JS et al. Bad bugs, no drugs: no ESKAPE! An update from the Infectious Diseases Society of America. Clin Infect Dis 2009; 48: 1-12.
7. Christiansen KJ, Turnidge JD, Bell JM et al. Prevalence of antimicrobial resistance in Enterococcus isolates in Australia, 2005: report from the Australian Group on Antimicrobial Resistance. Commun Dis Intell 2007; 31: 392-7.
8. Coombs GW, Daley D, Pearson JC et al. A change in the molecular epidemiology of vancomycin resistant enterococci in Western Australia. Pathol 2014; 46: 73-5.
9. CLSI. Performance standards for antimicrobial susceptibility testing. Twenty-fourth informational supplement M100-S24. Villanova, PA, USA, 2014.
10. European Committee on Antimicrobial Susceptibility Testing. Clinical breakpoints. 2015.
11. Kulski JK, Wilson RD, Bending R et al. Antibiotic resistance and genomic analysis of enterococci in an intensive care unit and general wards. Pathol 1998; 30: 68-72.
12. Tenover FC, Arbeit RD, Goering RV et al. Interpreting chromosomal DNA restriction patterns produced by pulsed-field gel electrophoresis: criteria for bacterial strain typing. J Clin Microbiol 1995; 33: 2233-9.
13. Homan WL, Tribe D, Poznanski S et al. Multilocus sequence typing scheme for *Enterococcus faecium*. J Clin Microbiol 2002; 40: 1963-71.
14. Coombs GW, Pearson JC, Daly DA et al. Australian Enterococcal Sepsis Outcome Programme annual report, 2013. Commun Dis Intell 2014; 38: E320-6.
15. Coombs GW, Daley DA, Thin Lee Y, Pang S, Pearson JC, Robinson JO, Johnson PD, Kotsanas D, Bell JM, Turnidge JD; Australian Group on Antimicrobial Resistance. Australian Group on Antimicrobial Resistance Australian Enterococcal Sepsis Outcome Programme annual report, 2014.Commun Dis Intell Q Rep. 2016 Jun 30;40(2):E236-43.
16. Coombs GW, Pearson JC, Daley DA et al. Molecular epidemiology of enterococcal bacteremia in Australia. J Clin Mmicrobiol 2014; 52: 897-905.
17. Seemann T, Goncalves da Silva A, Bulach DM, Schultz MB, Kwong JC, Howden BP. Nullarbor. San Francisco; Github. [Accessed: 03 Jun 2016]. Available from: https://github.com/tseemann/nullarbor
18. Coombs GW, Daley DA, Lee YT, Pang S, Bell JM, Turnidge JD for the Australian Group on Antimicrobial Resistance. Australian Group on Antimicrobial Resistance (AGAR) Australian Enterococcal Sepsis Outcome Programme (AESOP) Annual Report 2015 – submitted for publication. Commun Dis Intell



© Commonwealth of Australia 2018 - ISSN: 2209-6051 (Online)

This work is copyright. You may download, display, print and reproduce the whole or part of this work in unaltered form for your own personal use or, if you are part of an organisation, for internal use within your organisation, but only if you or your organisation do not use the reproduction for any commercial purpose and retain this copyright notice and all disclaimer notices as part of that reproduction. Apart from rights to use as permit­ted by the Copyright Act 1968 or allowed by this copyright notice, all other rights are reserved and you are not allowed to reproduce the whole or any part of this work in any way (electronic or otherwise) without first being given the specific written permission from the Commonwealth to do so. Requests and inquiries concerning reproduction and rights are to be sent to the Online, Services and External Relations Branch, Department of Health, GPO Box 9848, Canberra ACT 2601, or by email to copyright@health.gov.au

Communicable Diseases Intelligence aims to disseminate information on the epidemiology and control of communicable diseases in Australia. Communicable Diseases Intelligence invites contri­butions dealing with any aspect of communicable disease epidemiology, surveillance or prevention and control in Australia. Submissions can be in the form of original articles, short reports, surveil­lance summaries, reviews or correspondence. Instructions for authors can be found in Commun Dis Intell 2016;40(1):E189–E193.

Communicable Diseases Intelligence contributes to the work of the Communicable Diseases Network Australia

http://www.health.gov.au/cdna

This journal is indexed by Index Medicus and Medline

**Disclaimer:** Opinions expressed in Communicable Diseases Intelligence are those of the authors and not necessarily those of the Australian Government Department of Health or the Communicable Diseases Network Australia. Data may be subject to revision.

**Editor:** Cindy Toms  
**Deputy Editor:** Phil Wright  
**Editorial and Production Staff:** Leroy Trapani, Kasra Yousefi  
**Editorial Advisory Board:** Peter McIntyre (Chair), David Durrheim, Mark Ferson, John Kaldor, Martyn Kirk  
**Website:** http://www.health.gov.au/cdi

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;

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