Australian Group on Antimicrobial Resistance (AGAR) Australian Enterococcal Sepsis Outcome Programme (AESOP) Annual Report 2015

Geoffrey W Coombs, Denise A Daley, Yung Thin Lee, Stanley Pang, Jan M Bell and John D Turnidge for the Australian Group on Antimicrobial Resistance

# Abstract

From 1st January to 31st December 2015, 31 Australian institutions participated in the Australian Enterococcal Sepsis Outcome Programme (AESOP). The aim of AESOP 2015 was to determine the proportion of enterococcal bacteraemia isolates in Australia that were antimicrobial resistant, and to characterise the molecular epidemiology of the Enterococcus faecium isolates. Of the 1,009 unique episodes of bacteraemia investigated, 95.4% were caused by either E. faecalis (55.7%) or E. faecium (39.6%). Ampicillin resistance was detected in 0.2% of E. faecalis and in 86.0% of E. faecium. Vancomycin non-susceptibility was reported in 0.4% and 50.1% of E. faecalis and E. faecium respectively. Overall 56.2% of E. faecium harboured vanA or vanB genes. For the vanA/B positive E. faecium isolates, 61.0% harboured vanB genes and 36.8% vanA 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 49 multilocus sequence types (STs) of which 85.6% of isolates were classified into 11 major STs containing five or more isolates. All major STs belong to clonal cluster 17, a major hospital-adapted polyclonal E. faecium cluster. Four of the five predominant STs (ST796, ST555, ST203, and ST80) were found across most regions of Australia. The second most predominant clone was non-typable by multilocus sequence typing and found only in NSW and the ACT. Overall 73.9% of isolates belonging to the five predominant STs harboured vanA or vanB genes. In conclusion, the AESOP 2015 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 they are the fourth and fifth leading cause of sepsis respectively.1,2 Although 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 2015 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; and
* Molecular epidemiology of E. faecium.

# Methodology

## Participants

Thirty-one laboratories from all eight Australian states and territories.

## Collection Period

From 1 January to 31 December 2015, the participating 31 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 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.17

A chi-square test for comparison of two 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 1 January to 31 December 2015, 1,009 unique episodes of enterococcal bacteraemia were identified. Although nine Enterococcus species were identified, 55.7% (562 isolates) were E. faecalis and 39.6% (400 isolates) were E. faecium. Forty-seven enterococci were identified either as Enterococcus casseliflavus (16 isolates), E. avium (11), E. gallinarum (7), E. raffinosus (7), E. hirae (4) E. durans (1), and E. gilvus (1).

A significant difference was seen in patient sex (p<0.0001) with 613 (65.6%) being male (95% CI, 62.6 – 68.5). The average age of patients was 63 years ranging from 0 – 107 years with a median age of 67 years. Of the 1,006 episodes where onset was known, 493 (49.0%) were hospital onset (95% CI, 45.9 – 52.1). However, a significant difference was seen between E. faecium and E. faecalis , with 72.0% (95% CI, 62.7 – 76.4) of E. faecium episodes being hospital onset compared to 34.9% (95% CI, 31.0 – 39.0) for E. faecalis (p<0.0001). All-cause mortality at 30 days was 20.1% (95% CI, 14.4 – 26.6). There was no significant difference in mortality between E. faecalis and E. faecium episodes 15.5% vs 26.2% respectively (95%CI, -2.58 – 23.1), or between vancomycin susceptible and vancomycin non-susceptible E. faecium episodes 23.1% vs 29.3% respectively (95%CI, -13.15 – 24.2).

## 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 two isolates and two isolates were vancomycin non-susceptible. Thirty-nine (7.0%) E. faecalis, were initially reported as linezolid non-susceptible (CLSI breakpoint >2 mg/L). However by Etest® 12 of the 39 isolates available for MIC testing by Etest® had a linezolid MIC of ≤2 mg/L and were therefore considered linezolid susceptible. Twenty-five isolates with an MIC of 4 mg/L although non-susceptible by CLSI guidelines were considered susceptible by EUCAST guidelines. One isolate had an MIC of 6 mg/L and was non-susceptible and one isolate was not received for confirmation. All isolates were susceptible to daptomycin and teicoplanin.

Table 1: The number and proportion of E. faecalis non-susceptible to ampicillin and the non-β-lactam antimicrobials, Australia, 2015

| Antimicrobial | Number of isolates tested | Breakpoint (mg/L) | Non-Susceptible |
| --- | --- | --- | --- |
| n | % |
| Ampicillin | 561 | >8\* | 1 | 0.2 |
| >4† | 2 | 0.4 |
| Vancomycin | 561 | >4‡ | 2 | 0.4 |
| Erythromycin | 547 | >0.5\* | 475 | 86.8 |
| Tetracycline | 489 | >4\* | 384 | 78.5 |
| Ciprofloxacin | 521 | >1\* | 90 | 17.3 |
| Daptomycin | 542 | >4\* | 0 | 0 |
| Teicoplanin | 558 | >8\* | 0 | 0 |
| >2† | 0 | 0 |
| Linezolid | 561 | >2\* | 26 | 4.6 |
| >4† | 1 | 0.2 |
| Nitrofurantoin | 558 | >32\* | 13 | 2.3 |
| >64† | 2 | 0.4 |
| High-Level Gentamicin | 554 | >128† | 151 | 27.3 |

\*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 200 (50%) were phenotypically vancomycin non-susceptible (MIC >4 mg/L). Seventy (17.5%) and 71 (17.8%) isolates were teicoplanin non-susceptible by CLSI and EUCAST guidelines respectively. This is an increase from 2014, where 31 (8.2%) and 33 (8.8%) isolates were teicoplanin non-susceptible by CLSI and EUCAST guidelines respectively. Thirteen (3.3%) isolates were initially reported as linezolid non-susceptible (CLSI breakpoint >2 mg/L). However, by Etest® seven of the nine isolates had a linezolid MIC of ≤2 mg/L. Six 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, 2015

| Antimicrobial | Number tested | Breakpoint (mg/L) | Non-Susceptible |
| --- | --- | --- | --- |
| n | % |
| Ampicillin | 398 | >8\* | 343 | 86.2 |
| >4† | 345 | 86.3 |
| Vancomycin | 400 | >4‡ | 200 | 50.0 |
| Erythromycin | 391 | >0.5\* | 374 | 95.7 |
| Tetracycline | 342 | >4\* | 199 | 58.2 |
| Ciprofloxacin | 372 | >1\* | 342 | 91.9 |
| Teicoplanin | 399 | >8\* | 70 | 17.5 |
| >2† | 71 | 17.8 |
| Linezolid | 399 | >2\* | 6 | 1.5 |
| >4† | 0 | 0 |
| Nitrofurantoin | 398 | >32\* | 306 | 76.9 |
| >64† | 150 | 37.7 |
| 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 was performed on 420 of the 562 E. faecalis isolates. Overall, two (0.4%) of the 420 isolates harboured a vanA or vanB gene. The two vancomycin non-susceptible E. faecalis isolates (Vitek® vancomycin MIC ≥32 mg/Land 8mg/L) harboured the vanB gene.

The presence of vanA / B genes was determined by PCR or whole genome sequencing on 397 of the 400 E. faecium isolates. Overall, 223 (56.2%) of the 397 isolates harboured a vanA and/or vanB gene. Seventy-four of the vancomycin non-susceptible E. faecium isolates harboured vanA (Vitek® vancomycin MIC = >16mg/). A further 121 E. faecium vancomycin non-susceptible isolates harboured vanB (Vitek® vancomycin MIC = 8 mg/L [two isolates] and >16 mg/L [119 isolates]). Five isolates harboured both vanA and v anB genes (Vitek® vancomycin MIC >16 mg/L).

vanA or vanB genes were detected in 23 vancomycin susceptible E. faecium isolates. Eight isolates harboured vanA (Vitek® vancomycin MIC ≤ 0.5mg/L [three isolates], MIC = 1 mg/L [five isolates], teicoplanin ≤1mg/L [8 isolates]). Fifteen isolates harboured vanB (Vitek® vancomycin MIC ≤ 0.5mg/L [seven isolates], MIC = 1 mg/L [eight isolates].

Of the 136 vanB E. faecium isolates, three were teicoplanin resistant (MIC >32 mg/L).

## *E. faecium* Molecular Epidemiology

Of the 400 episodes, 390 E. faecium isolates were available for typing by whole genome sequencing. The 390 isolates were classified into 49 sequence types (STs) including 11 STs with five or more isolates (Table 3). Based on six of the seven MLST housekeeping genes, 53 pstS- negative isolates were considered the same clone. Of the 38 STs with <5 isolates, 25 had only one isolate. Overall, 334 (85.6%) of the 390 isolates were grouped into the 11 major STs. Using eBURST, the eleven STs were grouped into CC 17.

Table 3: The number and proportion of major *E. faecium* sequence types, Australia, 2015, by region

| Sequence Types | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | Aus |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
| n | % | n | % | n | % | n | % | n | % | n | % | n | % | n | % | n | % |
| 17 |  |  | 7 | 6.4 |  |  | 1 | 3.2 |  |  |  |  | 2 | 1.7 | 9 | 17.3 | 19 | 4.9 |
| 18 |  |  | 2 | 1.8 |  |  |  |  | 1 | 2.3 |  |  | 1 | 0.9 | 1 | 1.9 | 5 | 1.3 |
| 78 |  |  | 12 | 10.9 |  |  | 9 | 29 |  |  |  |  |  |  | 1 | 1.9 | 22 | 5.6 |
| 80 | 6 | 27.3 | 15 | 13.6 | 1 | 12.5 | 3 | 9.7 | 1 | 2.3 |  |  | 14 | 12 | 6 | 11.5 | 46 | 11.8 |
| 117 | 1 | 4.5 | 5 | 4.5 |  |  | 2 | 6.5 |  |  |  |  | 2 | 1.7 |  |  | 10 | 2.6 |
| 192 |  |  |  |  |  |  |  |  |  |  |  |  | 9 | 7.7 |  |  | 9 | 2.3 |
| 203 | 4 | 18.2 | 6 | 5.5 |  |  | 7 | 22.6 | 7 | 16.3 | 2 | 28.6 | 10 | 8.5 | 3 | 5.8 | 39 | 10 |
| 262 |  |  |  |  |  |  |  |  | 11 | 25.6 |  |  | 1 | 0.9 |  |  | 12 | 3.1 |
| 555 |  |  | 1 | 0.9 | 6 | 75 |  |  | 17 | 39.5 | 1 | 14.3 | 3 | 2.6 | 21 | 40.4 | 49 | 12.6 |
| 796 |  |  | 2 | 1.8 |  |  | 3 | 9.7 | 1 | 2.3 | 1 | 14.3 | 63 | 53.8 |  |  | 70 | 17.9 |
| Non-typable\* | 9 | 40.9 | 44 | 40 |  |  |  |  |  |  |  |  |  |  |  |  | 53 | 13.6 |
| Other | 2 | 9.1 | 16 | 14.5 | 1 |  | 6 | 19.4 | 5 | 11.6 | 3 | 42.9 | 12 | 10.3 | 11 | 21.2 | 56 | 14.4 |
| Total | 22 | 100 | 110 | 100 | 8 |  | 31 | 100 | 43 | 100 | 7 | 100 | 117 | 100 | 52 | 100 | 390 | 100 |

\*The non-typable group were a single clone missing the pstS gene
ACT = Australian Capital Territory; NSW = New South Wales; NT = Northern Territory; Qld = Queensland; SA = South Australia; Tas = Tasmania; Vic = Victoria; WA = Western Australia; Aus = Australia

Geographical distribution of the STs varied (Table 3). For the five most prominent STs, ST796 (70 isolates) was identified primarily in Victoria; the non-typable clone (53 isolates) primarily in New South Wales; ST555 (49 isolates) across most of Australia except Queensland and the Australian Capital Territory; ST80 (46 isolates) found in all mainland regions; and ST203 (39 isolates) found in all regions except the Northern Territory. For the remaining six STs, ST78 (22 isolates) was found in New South Wales, Queensland and Western Australia, ST17 (19 isolates) in New South Wales, Queensland, Victoria and Western Australia, ST262 (12 isolates) predominantly in South Australia, ST117 (10 isolates) in New South Wales, the Australian Capital Territory, Queensland and Victoria, ST192 (9 isolates) found exclusively in Victoria and ST18 (5 isolates) in New South Wales, South Australia, Victoria and Western Australia.

vanA was detected in five major STs (79 isolates, the non-typable clone, ST80, ST203, ST78 and ST117). vanB was detected in six major STs (135 isolates, ST796, ST555, ST203, ST78, ST17 and ST117) (Table 4). ST78, ST117 and ST203 harboured vanA and vanB genes. Four minor STs (5 isolates) also harboured vanB genes.

Table 4: The number and proportion of major *Enterococcus faecium* sequence types harbouring vanA/B genes, Australia, 2015

| Sequence Types | n | vanA | vanB | vanA and vanB | Not Detected |
| --- | --- | --- | --- | --- | --- |
| n | % | n | % | n | % | n | % |
| 17 | 19 |  |  | 2 | 1.5 |  |  | 17 | 9.9 |
| 18 | 5 |  |  |  |  |  |  | 5 | 2.9 |
| 78 | 22 | 1 | 1.3 | 15 | 11.1 | 3 | 60 | 3 | 1.8 |
| 80 | 46 | 23 | 29.1 |  |  |  |  | 23 | 13.5 |
| 117 | 10 | 1 | 1.3 | 2 | 1.5 |  |  | 7 | 4.1 |
| 192 | 9 |  |  |  |  |  |  | 9 | 5.3 |
| 203 | 39 | 8 | 10.1 | 18 | 13.3 | 1 | 20 | 12 | 7 |
| 262 | 12 |  |  |  |  |  |  | 12 | 7 |
| 555 | 49 |  |  | 24 | 17.8 |  |  | 25 | 14.6 |
| 796 | 70 |  |  | 69 | 51.1 | 1 | 20 |  |  |
| Non-typable\* | 53 | 46 | 58.2 |  |  |  |  | 7 | 4.1 |
| Other | 56 |  |  | 5 | 3.7 |  |  | 51 | 29.8 |
| Total | 390 | 79 | 100 | 135 | 100 | 5 | 100 | 171 | 100 |

  \*The non-typable group were a single clone missing the pstS gene

# 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 programmes are similar to those conducted in Europe18 comparison of Australia antimicrobial resistance data with other countries is possible.

In the 2015 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 8.3% (95% CI, 8 – 9), ranging from 0.0% (95% CI, 0 – 17) in Estonia, Luxembourg, Iceland, Norway and Sweden to 45.8% (95% CI, 41 – 51) in Ireland.19

In AESOP 2015, approximately 40% of enterococcal bacteraemia were due to E. faecium of which 50% (95% CI, 45.0 – 55.0) were phenotypically vancomycin non-susceptible by Vitek2® or Phoenix™. However, 56.2% of E. faecium isolates tested (223/397) harboured vanA/vanB genes, of which 61% were vanB . Overall, 20.7% 82/397 of E. faecium isolates harboured a vanA gene. There has been a significant increase over the last two surveys from 9.5% (35/370) in 2014 and 6% (8/310) in AESOP 2013.14 15 The majority of E. faecium isolates were also non-susceptible to multiple antimicrobials including ampicillin, erythromycin, tetracycline, ciprofloxacin and high-level gentamicin. In AESOP 201116 , 201314 and 201415 , 37.0%, 48.6% and 51.1% of E. faecium harboured vanA/vanB respectively confirming the incidence of vancomycin resistant E. faecium bacteraemia in Australia is increasing.

Fifteen (11%) of the 136 vanB E. faecium isolates had a vancomycin MIC at or below the CLSI and the EUCAST susceptible breakpoint (≤4 mg/L) and 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 11 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 2015, five E. faecium STs predominated: ST796 (of which 98.6% of isolates harboured vanB genes, one isolate had both vanA and vanB genes); the non-typable clone (100% harboured vanA ); ST555 (100% harboured vanB ); ST80 (100% harboured vanA ) and ST 203 (46.2% harboured vanB, 20.5% harboured vanA and one isolate had both vanA and vanB genes).

# Conclusions

The AESOP 2015 study has shown that, 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 2015 has significantly increased when compared to AESOP 2013 and 2014. 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 Health Care.

**Members of the AGAR in 2015 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

George Kotsiou and Peter Huntington, Royal North Shore Hospital

Sebastian van Hal and Bradley Watson, 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

Petra Derrington and Sharon Dal-Cin, 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

## 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

## Victoria

Denis Spelman and Amanda Dennison, The Alfred Hospital

Benjamin Howden and Peter Ward, 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 Queen Elizabeth II 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, St John of God Pathology - Murdoch Hospital

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 , Ms Jan M Bell4 and Prof John D Turnidge5 for the Australian Group on Antimicrobial Resistance

1. Antimicrobial Resistance and Infectious Disease Laboratory, School of Veterinary and Life Sciences, Murdoch University, Murdoch, Western Australia, Australia
2. Department of Microbiology and Infectious Diseases, PathWest Laboratory Medicine-WA, Fiona Stanley Hospital, Murdoch, Western Australia, Australia
3. Australian Group on Antimicrobial Resistance, Fiona Stanley Hospital, Murdoch, Western Australia, Australia
4. School of Biological Sciences, University of Adelaide, Adelaide, South Australia
5. Department of Molecular and Cellular Biology, University of Adelaide, Adelaide, South Australia, Australia

## Corresponding Author

Prof Geoffrey Coombs

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

**Telephone:** +61 8 6152 2397

**Email:** g.coombs@murdoch.edu.au

# References

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. http://www.ecdc.europa.eu/en/healthtopics/antimicrobial\_resistance/database/Pages/database.aspx
19. http://ecdc.europa.eu/en/publications/Publications/antimicrobial-resistance-surveillance-europe-2015.pdf).



© 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