Letter to the Editor

The first clonal spread of vanA-positive Enterococcus raffinosus in a nursing home

Sir,

In a recent issue of this Journal, Jolivet and colleagues reported the first nosocomial outbreak of vanA-type vancomycin-resistant Enterococcus raffinosus in France [1]. We would like to report a vanA-positive E. raffinosus outbreak that is not only the first in Belgium but, to our knowledge, is also the first to be reported in a nursing home anywhere in the world. We also tabulate a literature search on previous E. raffinosus outbreaks worldwide.

Vancomycin-resistant enterococci (VRE) are important nosocomial pathogens and the treatment option for VRE infections is limited [2]. Invasive infections caused by VRE are associated with higher mortality than those caused by vancomycin-susceptible enterococci (VSE) [2]. Outbreaks of VRE usually occur in hospital settings caused by E. faecium and E. faecalis [2]. The most frequently reported resistance genotypes responsible for acquired resistance to vancomycin are vanA and vanB [2]. The vanA gene is responsible for high-level resistance to glycopeptides vancomycin and teicoplanin.

Outbreaks of VRE due to species other than E. faecium and E. faecalis are rarely reported. E. raffinosus is another species of Enterococcus that has been linked to severe infections such as endocarditis [3].

In the spring of 2015, the Belgian National Reference Center (NRC) for Enterococci received two E. raffinosus strains for confirmation of vancomycin resistance from two different hospitals within a distance of 30 km (18 miles). E. raffinosus was confirmed in the NRC using matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (Bruker Daltonik GmbH, Bremen, Germany). These isolates were also positive for vanA genes as detected using specific polymerase chain reaction primers with forward sequence AAAATGTGCA[T/G]CGGGGCTA [4].

The first strain originated from a screening sample from a patient in hospital 1. The VRE screening was performed in that hospital due to an outbreak of vanA-positive E. faecium. The second strain originated from a patient who received haemodialysis in hospital 2; this hospital had also implemented VRE screening due to the same vanA-positive E. faecium outbreak in hospital 1.

After contacting both hospitals, new information revealed that both patients lived in the same nursing home. The Agency for Care and Health was contacted and they performed VRE screening in a first circle of close contacts in the nursing home. This investigation resulted in the identification of an additional case. All three residents colonized with vanA-positive E. raffinosus lived in single rooms on the same floor in the nursing home and shared nursing and paramedical staff as well as a same dining room and physiotherapy room. The characteristics of the three patients and their isolates are presented in Table I. Residents from a second circle, having less contact with these three index cases, were screened but no additional cases were identified within this circle.

Pulsed-field gel electrophoresis (PFGE) confirmed the clonal spread of a vanA-positive E. raffinosus clone (Figure 1). PFGE was performed by digesting genomic DNA of E. raffinosus isolates with Smal (Life Technologies, Carlsbad CA, USA), embedded in agarose 0.75% w/v plugs and separated by using Pulsed Field-Certified Agarose. The following E. raffinosus strains were used as control strains: reference strain LMG 12888T, clinical isolates O8L1270 and 111-005886 (own strain collection) and 8991/64, UW 11260, UW 7358, UW 10887, C-31135 (kindly provided by R. Willems (UMC-Utrecht, The Netherlands), G. Werner (Robert Koch Institute, Germany), K. Hegstad (University Hospital of North-Norway, Norway) and P. Damborg (University of Copenhagen, Denmark), respectively). PFGE patterns were interpreted according to Tenover et al. [4].

To investigate the previous occurrence of E. raffinosus outbreaks, we searched Medline up to December 29th, 2016, using search terms ‘Enterococcus raffinosus’ without language restriction. After reading the abstracts of all retrieved references and obtaining the full text of possible outbreak reports, four studies on E. raffinosus outbreak were included (Table I) [1,5–7]. The first outbreak was reported in the USA in 1997 and the most recent one in a hospital Paris region in France in 2014 [1]. The outbreaks were mostly short-lived, lasting around two to three months. Even though the short duration of outbreaks is perhaps due to successful infection control measures, the possibility that the outbreaks were self-limiting cannot be excluded. All isolates involved in the outbreaks were resistant to vancomycin, teicoplanin and ampicillin but susceptible to linezolid. Unlike the present study, outbreaks published in other studies occurred only in hospital settings.
<table>
<thead>
<tr>
<th>Authors, year</th>
<th>Geographic location</th>
<th>Setting</th>
<th>No. of involved patients</th>
<th>Period of outbreak</th>
<th>MIC of antibiotics (mg/L)</th>
<th>Method used in identifying E. raffinosus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Present study</td>
<td>West Flanders, Belgium</td>
<td>Nursing home</td>
<td>Three (all screening isolates)</td>
<td>May to August 2015</td>
<td>Ampicillin: 32–128 (R) Vancomycin: 256 (R) Teicoplanin: 16 (R) Linezolid: 1–2 (S)</td>
<td>MALDI-TOF MS</td>
</tr>
<tr>
<td>Jolivet et al., 2016 [1]</td>
<td>Paris region, France</td>
<td>Hospital; intensive care unit</td>
<td>Four (one clinical (probably cholecystitis), three screening)</td>
<td>September to October 2014</td>
<td>Ampicillin: 32 (R) Vancomycin: &gt;256 (R) Teicoplanin: 32 (R) Linezolid: 2 (S)</td>
<td>MALDI-TOF MS</td>
</tr>
<tr>
<td>Samuel et al., 2008 [5]</td>
<td>Newcastle, UK</td>
<td>Hospital; haematology ward</td>
<td>17 (all screening)</td>
<td>August 2007 to February 2008</td>
<td>Ampicillin: &gt;8 (R) Vancomycin: &gt;32 (R) Teicoplanin: &gt;32 (R) Linezolid: 1–2 (S)</td>
<td>API 20 Strep Kit</td>
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<tr>
<td>Kawalec et al., 2007 [6]</td>
<td>Warsaw, Poland</td>
<td>Hospital; haematology, surgery and nephrology ward</td>
<td>11 (all screening)</td>
<td>March 2005 to June 2006</td>
<td>Cannot be derived since the data were presented together with E. faecium</td>
<td>VITEK2 compact version</td>
</tr>
<tr>
<td>Wilke et al., 1997 [7]</td>
<td>Iowa, USA</td>
<td>Hospital; internal medicine and vascular surgery wards</td>
<td>Four (all clinical, bloodstream infection)</td>
<td>April 1995 to June 1996</td>
<td>Ampicillin: no data available Vancomycin: &gt;16 (R) Teicoplanin: 16 (not interpreted) Linezolid: no data available</td>
<td>Reference biochemical method</td>
</tr>
</tbody>
</table>

MIC, minimum inhibitory concentration; S, susceptible; R, resistant; MALDI-TOF MS, matrix-assisted laser desorption/ionization time-of-flight mass spectrometry.

* Interpretation according to guidelines by the authors.
The vanA-positive E. raffinosus isolates mostly originated from screening, not from clinical samples. Considering that invasive infections due to VRE are associated with a higher mortality than those due to VSE, and because E. raffinosus may be an important reservoir of van genes and may contribute to the dissemination of vancomycin resistance, infection control measures should be taken to curb the spread of vanA-positive E. raffinosus [8]. Such strains ought therefore to be submitted to specialized centres for confirmation and surveillance.

Conflict of interest statement
None declared.

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References


