Species identification and molecular characterization of Acinetobacter spp. blood culture isolates from Norway

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Objectives: The study investigated the species distribution, antibiotic susceptibility patterns and genotypic resistance characteristics of 113 consecutive blood culture isolates of Acinetobacter species collected between 2005 and 2007 throughout Norway.

Methods: Species identification was performed by partial rpoB sequence analysis, and verified by 16S rDNA and recA sequence analyses. Susceptibility testing was performed by agar disc diffusion and Etest. Distribution of OXA carbapenemase genes and epidemic clonality of Acinetobacter baumannii isolates were detected by PCR assays. Analyses of blaOXA-51-like variants and quinolone resistance-determining regions (QRDRs) were done by sequencing.

Results: The most prevalent species in the collection were Acinetobacter genomic species (gen. sp.) 13TU (46.9%) and Acinetobacter gen. sp. 3 (19.5%), followed by A. baumannii (8.8%) and Acinetobacter lwoffii/Acinetobacter gen. sp. 9 (7.1%). Carbapenem resistance was observed in one blaOXA-23-like-positive A. baumannii isolate. Quinolone resistance was detected in five isolates from the Acinetobacter calcoaceticus–A. baumannii complex, of which two had point mutations in the QRDRs, including one novel ParC mutation. None of the A. baumannii isolates belonged to European/international clones I, II or III. Six blaOXA-51-like variants, including two novel variants, were identified.

Conclusions: Acinetobacter gen. sp. 13TU and Acinetobacter gen. sp. 3 were predominant in Norwegian blood cultures, in contrast to in other countries where A. baumannii has dominated. The study demonstrated the importance of genotypic identification to determine the exact epidemiology of non-baumannii Acinetobacter species.

Keywords: rpoB, Acinetobacter genomic species 13TU, Acinetobacter soli, parC

Introduction

Acinetobacter are strictly aerobic Gram-negative coccobacilli that are widely distributed in soil and water, but also commonly found in the hospital environment.1 Thirty-three genomic species (gen. sp.) of the Acinetobacter genus have so far been identified.1–4 Of these, Acinetobacter baumannii, Acinetobacter gen. sp. 3 and Acinetobacter gen. sp. 13TU have been considered the clinically most relevant species.1

Our knowledge on the ecology and epidemiology of many Acinetobacter species is limited, mostly due to the lack of accurate methods for routine identification of Acinetobacter isolates to the species level.5 While phenotypic identification of Acinetobacter species has been found insufficient,6 several molecular methods have been shown to be adequate for this purpose.1 Sequence analysis of the highly discriminative zone 1, between positions 2900 and 3250 of the rpoB gene, has been found to represent a reliable and rapid method for identification of Acinetobacter species.7 This method has recently been validated on both a collection of Acinetobacter reference strains and a collection of Acinetobacter clinical isolates.5 In addition, a modified rpoB zone 1, spanning 352 bp between positions 2916 and 3267, has been included

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in the sequence-based delineation of species within the genus Acinetobacter.3,8

The occurrence of class D carbapenemase genes in Acinetobacter represents vertical inheritance in specific species, but horizontal acquisition in other species.9 The genes blaOXA-51-like, blaOXA-23-like, and blaOXA-14, Ore, for instance, intrinsically present in A. baumannii, Acinetobacter radioresistens and Acinetobacter lwaffii, respectively.3,10

The aim of this study was to investigate the species distribution of a national blood culture collection of Acinetobacter spp. along with molecular characterization of the isolates.

Materials and methods

Bacterial isolates

The study included 113 consecutive blood culture isolates of Acinetobacter species collected between 2005 and 2007 from 111 patients by 19 diagnostic microbiology laboratories throughout Norway. Four isolates recovered from two patients were included in the study, since they belonged to different species.

Identification of Acinetobacter species

Species identification of isolates was performed by partial rpoB gene sequence analysis (zone 1, 352 bp).3,7 An rpoB sequence type (rpoB seqtype) was defined as a unique sequence of zone 1 (between positions 2916 and 3267) of the rpoB gene, in a comparable way to what has previously been described.5 A cut-off of ≥94%-95% nucleotide similarity was used for partial rpoB gene sequence-based identification of Acinetobacter spp.31 Sequence analysis of 1379 bp of the 16S rRNA gene was performed for A. baumannii and A. radioresistens isolates, and for all isolates with rpoB sequences showing <99% nucleotide identity to type/reference strains in GenBank (http://www.ncbi.nlm.nih.gov/Genbank).12 Sequence analysis of 382 bp of the recA gene was performed for one isolate with a 16S rDNA sequence most related to more than one Acinetobacter species.13 PCR products were purified by ExoSAP-IT (GE Healthcare Bio-Sciences AB, Uppsala, Sweden) treatment, according to the manufacturer’s instructions. Sequencing of PCR products was performed using BigDye 3.1 technology (Applied Biosystems, Foster City, CA, USA). Primers used for PCR amplifications and sequencing are listed in Table S1 (available as Supplementary data at JAC Online). Nucleotide sequence homology search was performed using BLAST (http://www.ncbi.nlm.nih.gov/BLAST). Sequences were aligned and compared with published sequences of Acinetobacter type strains (Table S2, available as Supplementary data at JAC Online), using the Lasergene software package (DNASTAR, Madison, WI, USA). PFGE on selected Acinetobacter gen. sp. 13TU and Acinetobacter gen. sp. 3 isolates was performed as previously described.14

Antimicrobial susceptibility testing

Antimicrobial susceptibility testing for meropenem, imipenem, gentamycin, amikacin, tobramycin, nalidixic acid, ciprofloxacin, levofloxacin and trimethoprim/sulfamethoxazole was performed by the agar disc diffusion method, as recommended by the European Committee on Antimicrobial Susceptibility Testing (EUCAST; http://www.eucast.org/). Etest (bioMerieux, Solna, Sweden) was used to confirm reduced susceptibilities. Results were interpreted using clinical breakpoints as defined by EUCAST (http://www.eucast.org/). The disc diffusion breakpoints used for nalidixic acid were those recommended by EUCAST for Enterobacteriaceae.

PCR assays for molecular characterization

PCR assays and sequencing were performed to: (i) investigate the distribution of four groups of OXA carbapenemase genes (blaOXA-23-like, blaOXA-24-like, blaOXA-51-like, and blaOXA-58-like);15 (ii) detect ISAba1 upstream of blaOXA-51-like and blaOXA-23-like genes;16 (iii) detect mutations in the quinolone resistance-determining regions (QRDRs) of gyrA and parC genes;17,18 (iv) determine full-length sequences of blaOXA-51-like genes;16,19 and (iv) determine the clonal lineage of A. baumannii isolates.20

GenBank accession numbers

The nucleotide sequences of blaOXA-179 from A. baumannii isolate K50-13, blaOXA-180 from A. baumannii isolate K50-71, zone 1 of the rpoB gene from Acinetobacter soli isolate K50-54 and the QRDR of parC from A. baumannii isolate K51-7 were deposited in the GenBank nucleotide database under accession numbers HM570035, HM570036, HM570037 and HM570038, respectively.

Ethics

The study was approved by the Regional Committee for Medical and Health Research Ethics, reference number 2010/371-4.

Results

Acinetobacter species isolates

Isolates were obtained from patients in: internal medicine wards (n=54); intensive care units (n=23); surgery wards (n=17); cancer wards (n=7); paediatric/neonatal wards (n=6); burns wards (n=2); ear, nose and throat ward (n=1); and orthopaedic ward (n=1). The age distribution of the patients was between <1 year and 93 years, with a median age of 69. Altogether, 67% of the patients were males and 33% were females.

Identification of Acinetobacter species

Sequence analysis of zone 1 of the rpoB gene assigned isolates into 35 rpoB seqtypes (Figure 1 and Table 1). One hundred and five isolates (rpoB seqtypes 1–28) showed between 97.7% and 100% nucleotide identity to Acinetobacter type/reference strains in GenBank, and were identified as: Acinetobacter gen. sp. 13TU (n=53); Acinetobacter gen. sp. 3 (n=22); A. baumannii (n=10); A. lwaffii/Acinetobacter gen. sp. 9 (n=8); Acinetobacter ursingii (n=3); A. radioresistens (n=3); Acinetobacter gen. sp. ‘close to 13TU’ (n=2); Acinetobacter calcoaceticus (n=1); Acinetobacter gen. sp. ‘between 1 and 3’ (n=1); Acinetobacter gen. sp. 14BJ (n=1); and Acinetobacter guillouiae (n=1). 16S rDNA sequence analysis confirmed the rpoB identification for all A. baumannii and A. radioresistens isolates, and for 11 isolates of other species showing >99% rpoB nucleotide identity to Acinetobacter type/reference strains (Table 1). Isolates showed ≥99.6% 16S rDNA nucleotide identity to corresponding Acinetobacter type/reference strains in GenBank. PFGE typing of 43 isolates of Acinetobacter gen. sp. 13TU (n=32) and Acinetobacter gen. sp. 3 (n=11) assigned them into 32 distinct pulsotypes, with each pulsotype comprising ≤3 isolates (data not shown).

One isolate (rpoB seqtype 29) showed 98.9% nucleotide identity to the reference strain of Acinetobacter gen. sp. ‘close to 13TU’ (Table 1). However, according to the 16S rDNA sequence,
Figure 1. Dendrogram representing phylogenetic relationships between clinical seqtypes and type/reference strains of Acinetobacter species. The tree was derived from sequences of 352 bp of the *rpoB* gene between positions 2916 and 3267. The Lasergene MegAlign software (DNASTAR, Madison, WI, USA) was used to construct the tree using the Clustal W method. One thousand replicates were used for bootstrap analysis. The bootstrap support of branches representing our *rpoB* seqtypes is indicated by values at the nodes. A broken line on the tree indicatives a negative branch length.
### Table 1. Results of partial rpoB gene and near-complete 16S rDNA sequence analyses

<table>
<thead>
<tr>
<th>rpoB seqtype</th>
<th>No. of isolates</th>
<th>rpoB identification</th>
<th>16S rDNA identification</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>first nearest species (nucleotide similarity with type/reference strains)</td>
<td>second nearest species (nucleotide similarity with type/reference strains)</td>
</tr>
<tr>
<td>1</td>
<td>16</td>
<td>Acinetobacter gen. sp. 13TU (100%)</td>
<td>Acinetobacter gen. sp. ‘close to 13TU’ (96%)</td>
</tr>
<tr>
<td>2</td>
<td>2</td>
<td>Acinetobacter gen. sp. 13TU (99.7%)</td>
<td>Acinetobacter gen. sp. ‘close to 13TU’ (95.7%)</td>
</tr>
<tr>
<td>3</td>
<td>3</td>
<td>Acinetobacter gen. sp. 13TU (99.7%)</td>
<td>Acinetobacter gen. sp. ‘close to 13TU’ (95.7%)</td>
</tr>
<tr>
<td>4</td>
<td>19</td>
<td>Acinetobacter gen. sp. 13TU (99.4%)</td>
<td>Acinetobacter gen. sp. ‘close to 13TU’ (95.5%)</td>
</tr>
<tr>
<td>5</td>
<td>11</td>
<td>Acinetobacter gen. sp. 13TU (99.4%)</td>
<td>Acinetobacter gen. sp. ‘close to 13TU’ (96%)</td>
</tr>
<tr>
<td>6</td>
<td>2</td>
<td>Acinetobacter gen. sp. 13TU (98.6%)</td>
<td>Acinetobacter gen. sp. ‘close to 13TU’ (95.2%)</td>
</tr>
<tr>
<td>7</td>
<td>13</td>
<td>Acinetobacter gen. sp. 3 (100%)</td>
<td>A. baumannii (94.9%)</td>
</tr>
<tr>
<td>8</td>
<td>5</td>
<td>Acinetobacter gen. sp. 3 (99.7%)</td>
<td>A. baumannii (95.2%)</td>
</tr>
<tr>
<td>9</td>
<td>1</td>
<td>Acinetobacter gen. sp. 3 (99.4%)</td>
<td>A. baumannii (94.3%)</td>
</tr>
<tr>
<td>10</td>
<td>3</td>
<td>Acinetobacter gen. sp. 3 (99.1%)</td>
<td>A. baumannii (94.6%)</td>
</tr>
<tr>
<td>11</td>
<td>2</td>
<td>A. baumannii (99.1%)</td>
<td>A. baumannii gen. sp. 13TU (96.9%)</td>
</tr>
<tr>
<td>12</td>
<td>1</td>
<td>A. baumannii (98.6%)</td>
<td>A. baumannii gen. sp. 13TU (96.6%)</td>
</tr>
<tr>
<td>13</td>
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<td>A. baumannii (98.3%)</td>
<td>A. baumannii gen. sp. 13TU (96.3%)</td>
</tr>
<tr>
<td>14</td>
<td>4</td>
<td>A. baumannii (98.3%)</td>
<td>A. baumannii gen. sp. 13TU (96.9%)</td>
</tr>
<tr>
<td>15</td>
<td>1</td>
<td>A. baumannii (97.7%)</td>
<td>A. baumannii gen. sp. 13TU (96.9%)</td>
</tr>
<tr>
<td>16</td>
<td>1</td>
<td>A. lwofii/Acinetobacter gen. sp. 9 (100%)</td>
<td>A. lwofii/Acinetobacter gen. sp. 9 (99.6%)</td>
</tr>
<tr>
<td>17</td>
<td>1</td>
<td>A. lwofii/Acinetobacter gen. sp. 9 (99.4%)</td>
<td>A. lwofii/Acinetobacter gen. sp. 9 (99.8%)</td>
</tr>
<tr>
<td>18</td>
<td>4</td>
<td>A. lwofii/Acinetobacter gen. sp. 9 (98.3%)</td>
<td>A. lwofii/Acinetobacter gen. sp. 9 (98.5%)</td>
</tr>
<tr>
<td>19</td>
<td>2</td>
<td>A. lwofii/Acinetobacter gen. sp. 9 (98.9%)</td>
<td>A. lwofii/Acinetobacter gen. sp. 9 (98.9%)</td>
</tr>
<tr>
<td>20</td>
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<td>A. ursingii (100%)</td>
<td>Acinetobacter venetianus/Acinetobacter gerneri (85.5%)</td>
</tr>
<tr>
<td>21</td>
<td>1</td>
<td>A. ursingii (99.7%)</td>
<td>A. venetianus/A. gerneri (85.2 %)</td>
</tr>
<tr>
<td>22</td>
<td>3</td>
<td>A. radioresistens (99.1%)</td>
<td>Acinetobacter gen. sp. 15TU (84.1%)</td>
</tr>
<tr>
<td>23</td>
<td>1</td>
<td>A. calcoaceticus (98%)</td>
<td>Acinetobacter gen. sp. ‘between 1 and 3’ (96.3%)</td>
</tr>
<tr>
<td>24</td>
<td>1</td>
<td>Acinetobacter gen. sp. ‘between 1 and 3’ (100%)</td>
<td>A. calcoaceticus (96.9%)</td>
</tr>
<tr>
<td>25</td>
<td>1</td>
<td>Acinetobacter gen. sp. 14BJ (98.6%)</td>
<td>A. venetianus/Acinetobacter gen. sp. 14BJ (96%)</td>
</tr>
<tr>
<td>26</td>
<td>1</td>
<td>A. guillouiae (98.9%)</td>
<td>Acinetobacter bouvetii (89.8%)</td>
</tr>
<tr>
<td>27</td>
<td>1</td>
<td>Acinetobacter gen. sp. ‘close to 13TU’ (99.4%)</td>
<td>A. bouvetii (99.6%)</td>
</tr>
<tr>
<td>28</td>
<td>1</td>
<td>Acinetobacter gen. sp. ‘close to 13TU’ (99.4%)</td>
<td>A. bouvetii (99.6%)</td>
</tr>
<tr>
<td>29</td>
<td>1</td>
<td>Acinetobacter gen. sp. ‘close to 13TU’ (98.9%)</td>
<td>A. bouvetii (99.6%)</td>
</tr>
<tr>
<td>30</td>
<td>1</td>
<td>A. towneri (90.6%)</td>
<td>A. venetianus (88.9%)</td>
</tr>
<tr>
<td>31</td>
<td>1</td>
<td>A. towneri (89.8%)</td>
<td>A. venetianus (88.9%)</td>
</tr>
<tr>
<td>32</td>
<td>2</td>
<td>A. baylyi (87.2%)</td>
<td>Acinetobacter tandoii (86.6%)</td>
</tr>
</tbody>
</table>

Continued
the isolate was most related to Acinetobacter gen. sp. 13TU (99.4% nucleotide identity) and Acinetobacter gen. sp. ‘close to 13TU’ (99.4% nucleotide identity). Of note, the isolate showed 99.9% 16S rDNA nucleotide identity to Acinetobacter sp. RUH 1139, for which a nosocomial outbreak of infection has previously been reported. Partial recA sequence analysis confirmed the rpoB identification, showing 100% nucleotide identity to the reference strain of Acinetobacter gen. sp. ‘close to 13TU’ compared with 93.2% nucleotide identity to the reference strain of Acinetobacter gen. sp. 13TU.

Four isolates (rpoB seqtypes 30–32) were most related to Acinetobacter towneri (n=2) and Acinetobacter baylyi (n=2). However, the isolates showed ≤90.6% rpoB nucleotide identities to the corresponding type strains in GenBank (Table 1). 16S rDNA sequence analysis confirmed the rpoB genotypic closeness of the four isolates to A. towneri (n=2) and A. baylyi (n=2) species, though with ≤99% 16S rDNA nucleotide identities to the corresponding type strains. Two isolates (rpoB seqtypes 30 and 31) were recognized in this article as ‘close to towneri’. However, for the other two isolates (rpoB seqtype 32), 16S rDNA sequence analysis showed ≥99.9% nucleotide identity to the type strain of A. soli (GenBank accession number EU290155) and these isolates were accordingly assigned into this species. No rpoB sequence for the type strain of A. soli was found in GenBank.

Three isolates (rpoB seqtypes 33–35) were most related to more than one species (Table 1) and were identified as ‘unclassified’. The three isolates showed ≤97.7% rpoB and ≤99.1% 16S rDNA nucleotide identities to Acinetobacter spp. type strains in GenBank. One of the three isolates showed 99.9% 16S rDNA nucleotide identity to Acinetobacter sp. phenon 8 strain LUH 4713 (GenBank accession number AJ633634).

**Antimicrobial susceptibility testing**

Five out of the 113 (4.4%) isolates showed reduced susceptibility to one or more of the antimicrobial agents: A. baumannii (n=2); Acinetobacter gen. sp. 13TU (n=2); and Acinetobacter gen. sp. 3 (n=1). One A. baumannii isolate was resistant to all the investigated antimicrobial agents, whereas the other A. baumannii isolate was resistant to ciprofloxacin, nalidixic acid, trimethoprim/ sulfamethoxazole and gentamicin, and immediately susceptible to amikacin. One Acinetobacter gen. sp. 13TU isolate was resistant to all aminoglycosides and ciprofloxacin, whereas the other Acinetobacter gen. sp. 13TU isolate and the Acinetobacter gen. sp. 3 isolate were resistant only to nalidixic acid.

**Molecular characterization**

The gene blaOXA-51-like was detected in all 10 A. baumannii isolates, while it was absent in all other species. None of the isolates harboured an ISAba1 element upstream of the blaOXA-51-like genes. The gene blaOXA-23-like was present in all three A. radioreisistens isolates and in the carbapenem-resistant A. baumannii isolate. An ISAba1 element was detected upstream of blaOXA-23-like only in the A. baumannii isolate and not in the A. radioreisistens isolates. The blaOXA-24-like and blaOXA-58-like genes were absent in all isolates.

Sequence analysis of the QRDRs of the five isolates showing resistance to fluoroquinolones and/or nalidixic acid identified double mutations (Ser-83→Leu in GyrA and Ser-80→Tyr in ParC) in one A. baumannii isolate and a single mutation (Ser-83→Leu in GyrA) in the Acinetobacter gen. sp. 3 isolate, whereas no mutations were detected in the other three isolates.

None of the A. baumannii isolates belonged to any of the three previously described European/international clones of A. baumannii. Six different variants of blaOXA-51-like genes were detected: blaOXA-65 (n=5); blaOXA-64 (n=1); blaOXA-78 (n=1); blaOXA-69 (n=1); and the novel variants blaOXA-179 (n=1) and blaOXA-180 (n=1).

**Discussion**

Partial sequence analysis of the rpoB gene assigned Acinetobacter spp. isolates of the Norwegian blood culture collection into ≥13 different species (Table 1). Interestingly, the most prevalent species were Acinetobacter gen. sp. 13TU and Acinetobacter gen. sp. 3. The clinical significance of these two species has been indicated before, both in individual case reports and in epidemiological studies. The isolates were collected by 19 diagnostic microbiology laboratories throughout Norway, indicating a low probability of large-scale cross-transmission among patients. Furthermore, PFGE indicated that it is unlikely that a major outbreak involving Acinetobacter gen. sp. 13TU or Acinetobacter gen. sp. 3 has taken place in Norway.

Although A. baumannii has been considered the most clinically relevant Acinetobacter species, only 8.8% of the isolates in our collection belonged to this species. The finding that Acinetobacter species—other than Acinetobacter gen. sp. 13TU, Acinetobacter gen. sp. 3 and A. baumannii—together accounted...
for 24.8% of the isolates (about three times more than A. baumannii). Further demonstration of the limited contribution of A. baumannii. Two of our isolates were assigned by 16S rDNA sequencing into A. soli species. To our knowledge, this study is the first to report the clinical relevance of A. soli, since this species has formerly been isolated only from forest soil.2

A recently published study has described the identification of three isolates that did not cluster closely enough with any of the currently described Acinetobacter species.25 According to rpoB sequencing, the closest currently described species for the three isolates was A. towneri.25 Similarly, two of our isolates showed low rpoB and 16S rDNA nucleotide identities to any of the known species, and the two isolates were found to be most related to A. towneri species. All or part of the five isolates from the two studies may represent a new species genotypically close to A. towneri.

The capability of rpoB zone 1 sequencing for accurate species allocation of Acinetobacter clinical isolates was supported by bootstrap values ≥86.7% for 13 branches (comprising 32 rpoB seqtypes and 110 isolates) in the rpoB phylogenetic tree (Figure 1) and, furthermore, by the low nucleotide identities of their 16S rDNA sequences to type/reference strains in GenBank (Table 1). Moreover, the parallel results of 16S rDNA (Table 1) and/or recA sequence analyses maintained the validity of partial rpoB sequencing for the identification of 25/25 (100%) isolates of the A. calcoaceticus – A. baumannii complex, A. lwaffii/ Acinetobacter gen. sp. 9, A. radioresistens, Acinetobacter gen. sp. 14BJ and A. guillouiae species.

On the other hand, partial rpoB sequencing was not able to precisely assign three isolates (seqtypes 33–35). This could initially be anticipated by the low bootstrap values for their corresponding branches in the rpoB phylogenetic tree (Figure 1) and, furthermore, by the low nucleotide identities of their 16S rDNA sequences to type/reference strains in GenBank (Table 1). Further study is needed to determine precise species identification of these three isolates.

The blaOXA-51-like gene was present in all A. baumannii isolates, whereas blaOXA-23-like was present in all A. radioresistens isolates and in 1 out of 10 A. baumannii isolates. This maintained the proposal that blaOXA-51-like and blaOXA-23-like are intrinsic genes in A. baumannii and A. radioresistens, respectively, and that A. radioresistens represents the source for dissemination of blaOXA-23-like into other Acinetobacter species.26,27

The occurrence of specific amino acid substitutions in the QRDRs of DNA gyrase and topoisomerase IV has represented a major mechanism of bacterial resistance to quinolones.28 A novel mutation, Ser-80→Tyr, in the QRDR of ParC was identified in one A. baumannii isolate with high levels of resistance to ciprofloxacin, levofloxacin and nalidixic acid. Further investigations are required to determine the exact role of the newly detected ParC mutation in conferring resistance to quinolones.

One Acinetobacter gen. sp. 3 isolate showing a high level of resistance to nalidixic acid (MIC 128 mg/L) but susceptibility to ciprofloxacin (MIC 1 mg/L) and levofloxacin (MIC 0.5 mg/L) contained only a single amino acid substitution, Ser-83→Leu, in the QRDR of GyrA. The occurrence of this mutation in A. baumannii isolates has generally resulted in ciprofloxacin MICs of ≥4 mg/L.17,29,30 However, consistent with our finding, a study conducted by Wisplinghoff et al.29 has reported the occurrence of this mutation in one A. baumannii isolate with a ciprofloxacin MIC of 1 mg/L. The MIC of nalidixic acid for that isolate was not reported.29

Based on partial sequences of ompA, csuE and blaOXA-51-like genes, the majority of epidemic strains of A. baumannii have been found to belong to three major European/international clones (I, II and III).20 None of our A. baumannii isolates belonged to any of these three prevalent groups. The sporadic nature of our isolates was predictable, since most of them were susceptible to all antibiotics tested, whereas multidrug resistance has generally been a signal feature of A. baumannii European/international clones I, II and III.31 In accordance with a recently published study,32 the blaOXA-65-positive isolate in our collection did not belong to European/international clone I, even though it yielded a positive blaOXA-51-like amplicon in the group II multiplex PCR. However, in contrast to the same study,32 blaOXA-65-positive isolates in our collection did not belong to European/international clone II, since they did not yield csuE amplicons in the group 1 multiplex PCR.

In conclusion, the present study revealed the predominance of Acinetobacter gen. sp. 13TU and Acinetobacter gen. sp. 3 in Norwegian blood cultures, indicating the significance of molecular identification of Acinetobacter species clinical isolates for precise determination of the epidemiology of non-baumannii species.

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Transparency declarations
None to declare.
Supplementary data

Tables S1 and S2 are available as Supplementary data at JAC Online (http://jac.oxfordjournals.org/).

References