Objective

• The recent increase in Acinetobacter baumannii resistance to carbapenems in the Czech Republic has been associated with the spread of strains belonging to European (E/U) clone II. Although multiresistance was a common property of these strains, they differed in resistance to particular agents.

• The aim of this study was to assess the genetic basis of this variation.

Conclusions

• The variation in antibiotic resistance in the strains results from the differences in the presence of acquired resistance genes and, possibly from the effect of ISAb1 on the expression of intrinsic genes.

• The high genetic versatility of EU clone II might contribute to its ability to develop resistance to nearly all clinically relevant antibiotics.

Strains

• Sixty-six isolates of EU clone II included in the present study were obtained during a recent prospective study aimed to analyse the emergence of carbapenem resistance in Acinetobacter in the Czech Republic (see abstract O 303 for details).

• The 66 isolates were from clinical specimens of patients hospitalised at 36 ICUs of 17 hospitals in 15 cities in the period 2005-6.

Results

• All isolates were positive for the genes encoding OXA-51, AmpC and the AdeABC efflux system while no strain tested positive for those encoding metallo-β-lactamases, OXA-23 and OXA-24 carbapenemases, or aminoglycoside-modifying enzymes AAC(3)-II, AAC(6')-I, and ANT(2')-I.

• The strains varied with respect to the presence of the genes encoding the following proteins (% positive strains): TEM-1 (80), Tet(B) (92), Tet(A) (5), AAC(3)-I (83), APH(3')-I (80), APH(3')-V1 (30), OXA-58 (3) and a class I integrase (83); ISAb1 was found in 95% strains and three integron variable regions were identified, differing only in the number of copies of the orfC cassette (Fig. 3).

• The presence of genes and the corresponding resistant phenotype were in good agreement (Fig. 4-6).

• Individual strains carried from 4 to 12 resistance genes in 17combinations (Table 1). Different combinations were also found in isolates from the same ward and having identical PFGE patterns (Fig. 2).

References


Table 1. Diversity of resistance genes among 66 EU clone II isolates.

<table>
<thead>
<tr>
<th>Gene Combination</th>
<th>No. of Isolates</th>
</tr>
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<tbody>
<tr>
<td>adeABC efflux</td>
<td>57</td>
</tr>
<tr>
<td>adeABC efflux +</td>
<td>9</td>
</tr>
<tr>
<td>adeABC efflux +</td>
<td>10</td>
</tr>
<tr>
<td>adeABC efflux +</td>
<td>5</td>
</tr>
<tr>
<td>adeABC efflux +</td>
<td>2</td>
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</table>

Fig. 1. Dendrogram of cluster analysis of AFLP fingerprints of 66 A. baumannii isolates belonging to EU clone II. Cut-off of 800 and 85% with respect to the proportion of banding of isolates of the same clone and the same strain, respectively. Numbers following the city name indicate different hospitals in the city. Capital letters indicate different ICUs in the same hospital. Capital is in the last column (A-Q) denote different combinations of data for genes (Table 1).

Fig. 2. A2p macrorestriction patterns of the EU clone II isolates.

Fig. 3. Structure of three class 1 integron variable regions found in the EU clone II isolate 53. U and V are conserved segments of an integron structure; orfX and orfX' are genes carried on Tn4140; orfC is carried on Tn1696. Three orfX derivatives are present in the right upper corner. Results of PCR detection of the variable regions are shown in the right upper corner.

Fig. 4. Relationship between minimum inhibitory concentration (MIC) of aminoglycosides (mg/L) and the presence of ISAb1 upstream of the blaoxa-51 gene.

Fig. 5. Relationship between aminoglycoside MIC (mg/L) and the presence of the orfC gene encoding phosphotransferase (APH(3')-V1).

Fig. 6. Relationship between aminoglycoside MIC (mg/L) and the presence of ISAb1 upstream of the orfC gene.