7 clonal types and two of them were prevalent. B type was prevalent among the strains isolated up to September 2006, and A type among the strains isolated after this period. 16 out of the 17 B type strains were MDR and 50% of them harboured class 1 integrons. All the 30 A type strains harboured class 2 integrons and 29, out of them, were considered MDR isolates.

Conclusions: The incidence of MDR phenotype was high among the studied strains. Class 2 integrons were more prevalent than class 1 ones in Acinetobacter spp. isolates. Although both classes of integrons seem to be associated with MDR isolates, this phenotype was statistically related with A and B clonal types and it might be consequence of accumulation of different resistance mechanisms in specific clonal types.

#### Aminoglycoside resistance in a clinical isolate of Acinetobacter genomic species 13TU is associated with the up-regulation of its AdeABC-like efflux system

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**Objectives:** Acinetobacter genomic species (GS) 13TU is a member of the Acinetobacter calcoaceticus-Acinetobacter baumannii complex. Although commonly isolated from hospitalized patients, this species, unlike A. baumannii, is usually well susceptible to antibiotics. In a Czech hospital, two isolates of a GS 13TU strain differing in their susceptibilities to aminoglycosides were obtained from the same patient. The aim of this study was to assess whether the difference is associated with the up-regulation of the GS 13TU efflux system related to the AdeABC system in A. baumannii.

**Methods:** The two isolates, NIPH 952 and NIPH 953, were obtained, respectively, from the sputum and gastric juice of an ICU patient. Compared to NIPH 952, NIPH 953 showed elevated MICs to aminoglycosides, in particular to gentamicin (1 versus 8 mg/l) and netilmicin (2 versus 16 mg/l). The presence of the genes encoding putative efflux components was tested by PCR using primers derived from the AdeABC genes. The ability of NIPH 952 to produce aminoglycoside-resistant variants was assessed by challenging it with 4 or 8 mg/l of gentamicin. The adeB gene was partially sequenced and its expression level was examined by real-time reverse transcription PCR (RT-PCR).

**Results:** PCR amplicons of expected sizes were obtained with primers targeting adeA, adeB and adeD in both NIPH 952 and NIPH 953. The sequences of the adeB-like amplicons were identical in both isolates and were 84–89% identical to the known adeB sequences in A. baumannii. Variants with gentamicin MICs of more than 4 mg/l were obtained from NIPH 952 at frequencies of ~5 × 10^{-9}. Two of these variants were further investigated, i.e. NIPH 952-I (gentamicin MIC 8 mg/l) and NIPH 952-IV (gentamicin MIC 24 mg/l). Compared to NIPH 952, the susceptibility patterns of NIPH 952-I, NIPH 952-IV and NIPH 953 shared elevated MICs to aminoglycosides, tetracycline, tigecycline and ciprofloxacin, whereas no changes in MICs were observed for piperacillin, cefotaxime, meropenem or polymyxin B. Consistently, RT-PCR identified 27-fold, 214-fold and 38-fold increases in mRNA transcripts for adeB in NIPH 952-I, NIPH 952-IV and NIPH 953, respectively, as compared to NIPH 952.

**Conclusion:** The aminoglycoside resistance of the gastric GS 13TU isolate is likely to result from the up-regulation of its efflux system homologous to the AdeABC system in A. baumannii. Supported by grant 310/08/1747 of the Grant Agency of the Czech Republic.

#### Molecular characterization and outbreak analysis of multidrug-resistant Acinetobacter baumannii from German hospitals


**Objectives:** Emergence and dissemination of multidrug-resistant Acinetobacter baumannii are of special concern because of limited therapeutic options and increased mortality. In many cases colistin is the only antimicrobial substance for an adequate treatment. In Germany A. baumannii outbreaks were observed mainly in the summer months. Here we report on molecular-epidemiological analysis of A. baumannii from different German hospitals.

**Methods:** The strain collection includes 22 isolates from two outbreaks in one hospital (A) in 2007 as well as 42 outbreak isolates collected in a surgical ICU (hospital B) in 2006–2008. Furthermore 32 isolates (2005–2009) from two diagnostic laboratories were analysed. Molecular typing by PFGE and sequence-based multiplex PCR to identify isolates belonging to members of the European clonal complexes I-III were performed. Relevant resistance genes (blaOXA, blaVIM and blaIMP) were identified by PCR and sequencing.

**Results:** All above mentioned isolates were resistant to fluoroquinolones, aminoglycosides, sulphonamides and β-lactams including carbapenems. PCR and sequencing of resistance genes revealed the presence of OXA-β-lactamases in all isolates, with OXA-23 (n = 45) and OXA-58 (n = 47) as the most prevalent types. In several outbreak-independent isolates the genes blaOXA-72 (n = 1), blaOXA-58, blaOXA-23 (n = 1) or blaOXA-66 + insertion sequence ISAb1 were identified. In one single carbapenem-resistant isolate no carbapenemase gene was found. Multiplex-PCR analysis for identification of clonal lineages revealed that nearly all isolates are related to the European clones 1 (n = 19), II (n = 51) and III (n = 1) clonal complexes.