upstream of the OXA-23 gene for all isolates except one. PFGE showed that the isolates' profile was related with the imipenem-resistant *A. baumannii* clones disseminated throughout the country, including the endemic OXA-40 producing *A. baumannii* clone.
Conclusions: Emergence of OXA-23 producing *A. baumannii* is occurring in several Portuguese hospitals associated to outbreaks and sporadic cases. Of interest is the relatdness with the endemic OXA-40 clone, although with an enlarged resistance profile, which supports the plasmidic acquisition of these two oxacillinases by a well adapted *A. baumannii* clone. It is of note the recovery of an OXA-23 producer from an ambulatory patient which can further promote community dissemination.

O302 Emergence during therapy of efflux-mediated tigecycline resistance in *Acinetobacter baumannii* belonging to a UK epidemic clone

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Objectives: Up-regulation of the RND-type efflux system AdeABC has been implicated in reduced susceptibility to tigecycline in *A. baumannii*. AdeABC expression is controlled by the two-component regulatory system AdeRS. We investigated the role of this pump in the emergence of tigecycline resistance using a pre- and post-treatment pair of clinical isolates.
Methods: Isolates were identified by API20NE profile, and susceptibilities were first determined by BSAC disc methodology, then by agar dilution and Etest on IsoSensitest agar. PFGE was used to determine relatedness. Expression of the efflux pump, AdeABC, and its regulatory proteins, AdeRS, was examined by real-time reverse transcriptase (RT)-PCR using primers specific for adeB and adeR, respectively, and quantified relative to the RNA polymerase beta subunit gene, rpoB, which was used as a reference.
Results: The two *A. baumannii* isolates were recovered from abdominal drain fluid of a 38-yr old woman who had undergone a cholecystectomy. Inter alia she had received a 14-day course of tigecycline, and a 32-fold difference in susceptibility was observed between pre- and post-treatment isolates (MICs, 0.5 mg/L and 16 mg/L, respectively). The patient has since made a full recovery. The isolates had identical PFGE profiles and belonged to a prevalent UK strain, OXA-23 clone 1, with OXA-23 carbapenemase and initial susceptibility only to tigecycline and polymyxin. Real-time RT-PCR identified a 24-fold increase in adeB gene expression in the post-treatment isolate. No concomitant difference in adeR expression was observed.
Conclusions: OXA-23 clone 1 is a widespread multi-resistant lineage and we report here the emergence of resistance to tigecycline during therapy in a representative. This resistance was associated with increased expression of the AdeABC efflux system, but not with altered expression of its known regulatory genes adeRS. This suggests that there may be 'cross-talk' between adeABC and other trans-acting regulatory factors.

O303 The recent increase in *Acinetobacter baumannii* resistance to carbapenems in the Czech Republic is associated with the spread of genotypically highly related strains of European clone II

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Objective: In 2003, a significant increase of *Acinetobacter* resistance to carbapenems has been noted in the Czech Republic. The aim of this study was to assess the prevalence and epidemiology of this resistance among hospital strains of *Acinetobacter* spp.
Methods: Isolates were collected prospectively via a network of diagnostic laboratories between January 2005 and April 2006. The laboratories were asked to send clinical isolates of *Acinetobacter* spp. from intensive care units (ICUs), with no more than one isolate per patient and 10 isolates per ICU. The isolates were identified to species by AFLP and assessed for relatedness using AFLP and PFGE. Susceptibility to 12 antimicrobial agents primarily effective against *A. baumannii* was tested by disk diffusion, and MICs were determined for carbapenems.
Results: A total of 150 *Acinetobacter* isolates were obtained from 56 ICUs of 20 hospitals in 15 cities. They were identified as *A. baumannii* (n = 108), genomic sp. 3 (n = 30), genomic sp. 13 TU (n = 8) or other species (n = 4). Using AFLP cluster analysis, *A. baumannii* isolates were allocated to EU clone II (n = 66), EU clone I (n = 5), 6 clusters with 2–5 isolates (n = 15) or 22 unique genotypes. Nearly all clone II isolates yielded identical or highly similar PFGE and AFLP patterns. A total of 24 (16%) isolates were resistant to at least one carbapenem. Seventy isolates were susceptible to all antimicrobials while 7 isolates showed resistance to 1–3 agents and 73 isolates were resistant to >3 agents. Resistance to >3 agents and/or carbapenem MICs >1 mg/l were found only in the EU clones and four unique strains while resistance to one or two carbapenems (MIC >8 mg/l) was confined to EU clone II. The 66 EU clone II isolates originated from 37 ICUs in 12 cities and showed the following susceptibility rates: piperacillin (0%), ceftazidime (5%), ampicillin-sulbactam (23%), imipenem (71%), meropenem (67%), gentamicin (15%), tobramycin (97%), amikacin (61%), netilmicin (52%), ofloxacin (0%), doxycycline (0%), co-trimoxazole (12%).
Conclusion: The increase in *Acinetobacter* resistance to carbapenems in the Czech Republic is associated with the spread of multidrug resistant *A. baumannii* strains belonging to EU clone II. The high genotypic similarity of the isolates suggests that they represent a recent subgroup within this clone.
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O304 Nosocomial multidrug-resistant *Acinetobacter baumannii* bloodstream infections: epidemiology, clinical features, treatment, and outcome, Bangkok, Thailand

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Objective: To determine the epidemiology, clinical features, antibiotic susceptibility, treatment, and outcome of nosocomial *Acinetobacter baumannii* bacteraemia.
Design: A retrospective study was conducted at KCMH in all hospitalised patients with microbiologically and clinically documented *A. baumannii* bacteraemia from January 2005 to February 2007.
Results: A total of 83 patients with nosocomial *A. baumannii* bacteraemia were identified. There were 51 males and 32 females with the mean age of 62 years (range: 19 to 99 years). Most patients had received antibiotics within three months before the diagnosis of *A. baumannii* bacteraemia. Third-generation cephalosporin was the most commonly prescribed antibiotic (78.9%). Ventilator-associated pneumonia (VAP) was the most common primary site of infection, followed by catheter-related blood stream infection (CRBSI), intraabdominal infection, and skin and soft tissue infection (45.8%, 20.5%, 7.2% and 2.4%, respectively). There were 16 patients (19.3%) with unknown source of infection. The mean duration of intubation in the patients with VAP and that of central venous catheter insertion in those with CRBSI before the diagnosis of *A. baumannii* infection were 9.46 days and 12.07 days, respectively. Most *A. baumannii* isolates were susceptible to cefoperazone-sulbactam (48.2%), followed by netilmicin (44.6%) and amikacin (31.3%). Of all isolates, the frequency of carbapenem-, multidrug-, and pan-drug-resistant *A. baumannii* was 69%, 45.8%, and 27.7%, respectively. The overall inhospital mortality rate was 69.9%, with high mortality rate in the patients in medical intensive care units (ICUs), compared to those in non-medical ICUs [92.6% and 58.9%, odds ratio (OR): 8.712, 95% confidence interval (CI): 1.876–40.457, p = 0.002]. The patients infected by multidrug- and pandrug-resistant strains had much higher mortality rate than those infected by non-multi- and pandrug-resistant strains (80.3% and 40.9%, OR: 5.898, 95% CI: 2.046–17.002, p = 0.001). Pneumonia had higher mortality rate than other source of infections (81.6% and 60%, OR: 2.952, 95% CI: 1.071–8.136, p = 0.03)