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P0937 Genomic epidemiology of carbapenem- and colistin-resistant *Acinetobacter baumannii* from Greece

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Background: *Acinetobacter baumannii* is a worrisome healthcare-associated opportunistic pathogen that is naturally resistant to several antimicrobial agents and exhibits also a remarkable propensity to acquire new resistance traits. Colistin is used as last-resort agent for treatment of infections caused by carbapenem-resistant *A. baumannii*, but colistin resistance has also emerged.

Materials/methods: A total of 44 carbapenem- and colistin-resistant (col^R) *A. baumannii* isolated during the period 2015-2017 were obtained from 10 Greek hospitals . Antibiotic susceptibility testing was performed by using the Vitek2 automated instrument. Colistin minimum inhibitory concentrations (MICs) were obtained by reference broth microdilution method and interpreted according to the EUCAST susceptibility breakpoint of 2 mg/L. Whole genome sequencing was performed with an Illumina NovaSeq instrument.

Results: Antimicrobial susceptibility testing confirmed that all strains were carbapenem-resistant, and 41/44 were also col^R (colistin MIC range, 4-64 mg/L). Carbapenem resistance was associated to the production of OXA-23 by all strains. Analysis of the PmrA and PmrB proteins, involved in lipopolysaccharide modifications, revealed that all col^R strains had the A226V mutation in PmrB. This mutation has been previously described to be associated with the col^R phenotype. Strains carrying this mutation had colistin MICs ranging from 4 to 8 mg/L. In 5 strains, a second mutation in PmrB (either E140F or L178F) or in PmrA (either K172I or D10N) was detected. In these strains colistin MICs were 16 and 64 mg/L, respectively. Interestingly, the PmrB A226V mutation was present in strains of different lineages. Results from MLST analysis performed following the Pasteur scheme demonstrated the occurrence of two major Global Clones (GC1 and GC2), while results from the Oxford MLST scheme further differentiated the GC2 strains in 5 different STs (ST425, ST208, ST195, ST451 and ST436).

Conclusions: Genomic analysis of carbapenem-and colistin-resistant *A. baumannii* isolates from different Greek hospitals revealed a heterogenous population, all sharing the same alteration in PmrB, suggesting a convergent evolution towards the same col^R mechanism. This mutation was associated with low level col^R. In some strains additional mutations of PmrA or PmrB were likely responsible for higher colistin MICs.