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Genetic integration of colistin resistance mcr-1 gene in Enterobacteriaceae from human and animal samples from different countries

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Background: Worldwide emergence of multidrug resistant bacteria is a major concern. Resistance of Gram negative bacteria to almost all antibiotics used in antibiotherapy led to the use of old drugs such as colistin. Unfortunately, colistin re-use has led to the emergence of sporadic resistance cases mediated by chromosomic alterations of target genes. Plasmid-mediated colistin resistance by *mcr-1* gene has been recently reported (*LiuYY*, et al). This gene encoding for a transferable phosphoethanolamine transferase is usually carried on plasmids leading to a possible high dissemination and endemic resistance to colistin.

Material/methods: We investigate here a total of 32 *mcr-1* strains including 25 *E. coli* and 7 *K. pneumoniae* isolated from Laos, Thailand, France, Algeria and Hajj pilgrims from animals, and humans. Antibiotic susceptibility testing and multilocus sequence typing were performed. Plasmid presence was checked by conjugation and transformation methods, plasmid typing was also realized. Whole genome sequence of 4 *E. coli* strains were done to analyze genetic environment of *mcr-1* gene.

Results: Colistin minimum inhibitory concentrations of strains harboring *mcr-1* gene ranged from 3 to 32µg/mL. 91% of strains were susceptible to third generation cephalosporins. Three strains were resistant to most beta-lactams tested and one of them was also resistant to nitrofuran and to ertapenem. None of the strain was resistant to tazobactam-piperacillin, imipenem or amikacin. Several clones were found with 6 STs recurrent in *E. coli* strains including ST4015, ST3997, ST10, ST93, ST48, and ST648. Plasmid carrying *mcr-1* gene was detected in 22 *E. coli* strains (88%) and 2 *K. pneumoniae* strains (28.6%). Predominance of IncI2 type plasmid (82.6%) and Inc P (17.4%) were observed. Preliminary analysis of genetic environment of *mcr-1* gene by WGS of 4 *E. coli* strains

showed the loss of insertion sequence ISApl1 and an insertion sequence not described in *mcr-1* transposon. Further analysis are planned to investigate the full integration mechanism of the *mcr-1* gene in plasmid and chromosome among Enterobacteriaceae isolates. We described here for the first time a case of asymptomatic human carrying *mcr-1* gene with a chromosomic location.

Conclusions: *Mcr-1* gene diffusion is principally due to a transposon located in plasmid Inc2 type but some *E. coli* clones may harbor *mcr-1* gene in their chromosome. Genetic environment of *mcr-1* gene has evolved over time resulting to a wide diversity of insertion sequences allowing this gene to be translocated into the chromosome. This variability facilitates quickly dissemination of this gene in Gram negative bacteria. Emergence of endemic colistin resistant strains with chromosomic location of *mcr-1* gene must be monitored to avoid a scenario similar to NDM-1.