29th ECCMID Amsterdam, Netherlands 13 – 16 April 2019

The congress of ESCMID

P1374 High frequency of plasmid-mediated colistin resistance genes in *Enterobacteriaceae* veterinary isolates in Portugal (2010-2015)

Lurdes Clemente[1], Vera Manageiro*[2,3], Ana Amaro[1], Ivone Correia[1], Teresa Albuquerque[1], Patrícia Themudo[1], Eugénia Ferreira[2,3], Manuela Caniça[2,3]

[1] INIAV – National Institute of Agrarian and Veterinary Research, Bacteriology and Mycology Laboratory, Oeiras, Portugal, [2] National Reference Laboratory of Antibiotic Resistances and Healthcare Associated Infections, National Institute of Health Dr. Ricardo Jorge, Lisbon, Portugal, [3] Centre for the Studies of Animal Science, Institute of Agrarian and Agri-Food Sciences and Technologies, University of Oporto, Oporto, Portugal

Background: Following the original report of plasmid-mediated colistin resistance (PMCR) in China, several studies in different countries reported a worldwide distribution of the *mcr* genes in *Enterobacteriaceae*. Thus, our aim was to understand the extension of the problem of PMCR in *Escherichia coli* and *Salmonella enterica* isolates from different animal origins, as colistin is the last resort to treat human infections caused by Gram negative bacteria resistant to all antibiotics, namely carbapenems.

Materials/methods: The antimicrobial susceptibility of 1206 *E. coli* and 634 *S. enterica* isolates from food-producing animals, meat and animal feed, was determined by Minimum Inhibitory Concentrations (MIC) and the results interpreted according to epidemiological cutoff values (EUCAST). All isolates with MIC>2µg/mL towards colistin were screened for the presence of PMCR-encoding genes (*mcr-1/mcr-2*), using a multiplex PCR, followed by sequencing. For all Mcr-positive isolates exhibiting an extended-spectrum β-lactamases (ESBL) or plasmid-mediated AmpC β-lactamases (PMAβ) phenotype, the respective *bla* gene was amplified by PCR and sequenced.

Results: We observed a high frequency (72.5%) of colistin-resistant *E. coli* and *S. enterica* isolates with *mcr-1*-like genes. Among 138 colistin-resistant isolates, *mcr*-1-like genes were detected in 97 *E. coli* (94.2%, 97/103) and in three *S. enterica* isolates (8.6%, 3/35). All amplicons excepting one, exhibited a sequence with 100% homology to the previously described *mcr-1*; one amplicon, hereafter named mcr-1.9 (KY780959), differed from *mcr-1* by one-point mutation (T1238C), leading to Val413Ala substitution. None of our isolates were positive for *mcr-2* gene. Forty-two *E. coli* isolates were ESBL/PMAβ co-producers: $bla_{\text{CTX-M-32}}$, n=13; $bla_{\text{CTX-M-1}}$, n=14; $bla_{\text{CTX-M-14}}$, n=5; $bla_{\text{CTX-M-8}}$, n=1; $bla_{\text{CTX-M-27}}$, n=1; $bla_{\text{SHV-12}}$, n=3; $bla_{\text{CMY-2}}$, n=3; bla_{ESAC}, n=2.

Conclusions: Of note is the high frequency of *mcr* positive *E. coli* isolates, particularly from turkeys (27.0%, 50/185), when comparing with other European countries. Selection pressure exerted by broad-spectrum cephalosporins and other antimicrobials may select and enhance the rapid dissemination of PMCR, e.g. the new *mcr-1.9* positive isolate co-harbouring a $bla_{\text{CTX-M-8}}$ gene. Globally, these results seem alarming in terms of public health evidencing that surveillance of this colistin resistance mechanism should continue, namely in other reservoirs (humans and environment), emphasizing the importance of the One Health perspective.