XIII

SIMPÓSIO BRASILEIRO DE MICROBIOLOGIA APLICADA

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GENOMIC CHARACTERIZATION OF AVIAN *ESCHERICHIA COLI* STRAINBEN2908 AND ANALYSIS OF POINT MUTATIONS IN FimH ADHESIN.

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Avian pathogenic *Escherichia coli* (APEC) are a subpathotype of extraintestinal *Escherichia coli* (ExPEC) strains. APEC cause diseases of varying symptomatology in poultry and respond to major economic losses in the poultry industry worldwide. In this study, we evaluated the APEC strain MT78 based on data from whole-genome sequencing (WGS) and *in vitro* assays. MT78 (serovar O2:K1:H5, ST95, phylogenetic group B2), is invasive to avian fibroblasts and hepatocytes, human pneumocytes and brain microendothelial cells. We analysed the complete genome of MT78 and compared it with other ExPEC genomes from NCBI. The WGS of MT78 was performedon Oxford Nanopore Technologies and Illumina by the Genome and Transcriptome Facility of Bordeaux, France. Reads were assembled with Canu. Contigs were polishedby Illumina reads using Pilon, the annotation was performed with Prokka and chromosome and plasmid have been deposited in GenBank under the accessionnumbers LR740776.1 and LR740777.1, respectively. A comparative ring of MT78against other ExPEC was composed in the BLAST Ring Image Generator software andused to compare the common virulence genes. The ring showed MT78 genome is mostsimilar to IMT5155, followed by APECO1 and χ7122, all well characterized APEC strains. We identified a region that corresponded to a possible viral island, and a region that corresponded to GimB and GimA, both genomic islands related to neonatal meningitis, sugar metabolism and virulence factors for adherence, invasion, toxins, iron uptake and protectins. Identification of orthogroups shared among the strains was made using the software Orthofinder with RAxML; 3,113 orthologous proteins were found. A phylogenetic tree was composed and showed MT78 clustered with other ExPEC ST95 strains. We also performed an alignment of the FimH protein sequence inthe Clustal software: three mutations were found in the lectin domain of MT78 (V58A, N91S and S99N) and one relevant mutation in the linker chain (G180S). In urinary ExPEC, mutations in FimH linker chain are important due to an alternation between lowand high-affinity binding states. Additionally, the type 1 fimbria phenotype will beassessed through yeast agglutination and the *fim* operon orientation will be verified to complement our *in silico* findings. The analysis of *fimH* sequences can be useful to predict the ExPEC virulence potential and to identify new therapeutic strategies againstboth avian and human ExPEC.

Keywords: Avian pathogenic *Escherichia coli*, whole-genome sequencing, type 1 fimbria, invasion

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