XIII

SIMPÓSIO BRASILEIRO DE MICROBIOLOGIA APLICADA

ANAIS

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POINT MUTATIONS IN FimH ADHESIN IN NEONATAL-MENINGITIS *Escherichia coli* STRAINS

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Extraintestinal pathogenic *Escherichia coli* (ExPEC) are responsible for various infections outside the gastrointestinal tract, including urinary tract infections (UTI), neonatal meningitis and sepsis in humans, infections in dogs and cats, and colibacillosis in poultry. While there is a vast literature on the epidemiology and virulence of uropathogenic (UPEC) and avian pathogenic (APEC) *E. coli*, neonatal meningitis is a rather rarer disease and thus much less is known about the epidemiology of neonatal meningitis-causing *E. coli* (NMEC). We have been assembling and characterizing a collection of Brazilian NMEC strains isolated from liquor; we have sequenced the genome of 11 NMEC strains, 10 from the city of São Paulo (including a PFGE-clonal strain isolated twice 4 years apart in distinct hospitals) isolated between 1985 and 1990, and 1 strain from Espírito Santo isolated in 2018. Type 1 fimbriae-mediated adherence to mucosa is central for the pathogenesis of UTI and likely so of avian colibacillosis, and is believed to be required for a bloodstream *E. coli* to adhere to human brain microendothelial cells and eventually cross the blood-brain barrier. Among the 11 NMEC strains, all except for IAL31 and IAL39 harbor the full *fim* operon in its genome; yet 4 of these (IAL 34, IAL36, IAL38 and IAL42) did not display a functional type 1 fimbriae, evaluated by yeast agglutination. We thus analysed the aminoacid sequence of the FimH adhesin in the *fim* operon-positive NMEC strains to look for possible mutations that could explain the absence of yeast agglutination in the above-mentioned 4 strains. Compared with the FimH sequence of K-12, we found the following mutations: V27A in all of them, like other ExPEC strains of human and avian origin; N70S and S78N in IAL41 and IAL42, also a common mutation among ExPEC strains, such as NMECs RS218 and NMEC O18; A119V in IAL44 and IAL39, like NMEC S88; and a Q269K mutation unique to IAL34, IAL36 and IAL38. This latter mutation could explain the lack of yeast agglutination observed for these strains, a hypothesis that deserves further investigation. IAL42, in contrast, doesn't carry any mutation that distinguished it from other yeast agglutination-positive IAL strains, such as IAL41. Finally, IAL39 harbors the *fimH* gene despite lacking the full *fim* operon. Interestingly, IAL39 FimH sequence displayed 3 unique mutations: P173S, A234T and G273A, in addition to the A119V, making its *fimH* the most mutated among all IAL strains.

Keywords: ExPEC; neonatal meningitis-causing *E. coli*; NMEC; whole genome sequencing; ExPEC virulence

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