The *treA* gene contributes to expression of type I fimbriae and colonization of the urinary tract of extra-intestinal pathogenic *E. coli* strain MT78

Pavanelo, D. B.[1,2]; Houle, S.[2]; Matter, L. B.[3]; Dozois,C. M.[2]; Horn,F.[1]

1. UFRGS, Porto Alegre, RS, Brasil
2. INRS-IAF, Laval, QC, Canada
3. URI, Santo Ângelo,RS, Brasil

Pathogenic extraintestinal *Escherichia coli* (ExPEC) are responsible for various infections outside the gastrointestinal tract, including urinary tract infections, neonatal meningitis and sepsis in humans, infections in dogs and cats, and in farm animals. In the poultry industry, avian colibacillosis - as infections by ExPEC are collectively called - cause great economic losses worldwide. Screening a library of 1710 mutants of the ExPEC MT78 strain, generated by signature-tagged mutagenesis (STM), we identified a transposon mutant attenuated for adhesion to and invasion of non-phagocytic cells. The transposon mutation in this strain disrupted the gene *treA,* which encodes a periplasmic trehalase enzyme. A *treA* deletion mutant (MT78Δ*treA*) and a complemented mutant were created by the lambda red recombinase technique and this mutant was characterized for (1) its capacity of adhesion and invasion to avian fibroblasts (CEC-32 line); (2) yeast agglutination, to verify the level of type I fimbriae expression after growth in LB broth and human urine; and for (3) its virulence *in vivo* in a urinary tract infection model. MT78Δ*treA* presented a reduction of 36% of adhesion and 60% of invasion to avian fibroblasts compared to the wild type (MT78WT). This decrease can be explained by a reduction in type I fimbriae expression, since MT78Δ*treA* yeast agglutination titers were 22%, 36% and 45% reduced in LB broth statically and under agitation and in human urine compared to those of MT78WT, respectively. Regarding virulence in the mouse urinary tract, MT78Δ*treA* presented a 10-fold reduction in the bladder colonization ($p<0,001$, Man-Whitney test). The phenotype was recovered by the complemented mutant in all the tests. Altogether, our results suggest that the *treA* gene contributes to regulation of type I fimbriae expression in this particular ExPEC strain, and therefore consequently plays a role in cell adherence/invasion and colonization of the urinary tract during infection.