ADHESION TO AND INVASION OF AVIAN FIBROBLASTS BY AVIAN PATHOGENIC *ESCHERICHIA COLI*

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Avian pathogenic *Escherichia coli* (APEC) are responsible for extraintestinal diseases in avian species. The infection usually starts in the upper respiratory tract and results in significant economic losses in poultry industry worldwide. Adhesion and colonization, promoted mainly by fimbriae, are important steps in the disease process. The aim of this work was to investigate if APEC strains: APEC17, UEL29, UEL31 and MT78 adhere to and invade fibroblasts of the CEC32 cell line. Monolayers of CEC32 were infected with bacteria at 20 CFU/cell for 1 h. For the adhesion assay, cells were washed, lysed with Triton X-100 1% and plated on LB agar for CFU determination. For the invasion assay, after cells were washed, they were reincubated with medium containing 50 µg/mL of gentamycin for further 3 h before being lysed and plated for CFU determination. *Salmonella typhimurium* SL 1344 and *E. coli* K12 were used as positive and negative controls, respectively. All pathogenic strains adhered to CEC32, in contrast to *E. coli* K12. *S. typhimurium* invaded CEC32 efficiently, followed by MT78 and UEL31; UEL29 and APEC17 presented no invasion, comparable to K12. Higher adhesion did not necessarily mean better invasion or internal viability. Since some APEC were capable of invading non-phagocytic cells *in vitro*, it is possible that they also invade epithelial cells of the respiratory tract *in vivo*.

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