AVIAN FIBROBLAST INFECTION WITH APEC DID NOT RESULT IN CASPASE 3/7 ACTIVATION

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Avian pathogenic *Escherichia coli* (APEC) are the ethiological agent of colibacillosis, a disease in poultry that often culminates in septicemia. The first step in the establishment of colibacillosis is the colonization of the tracheal by APEC, which then spread to internal organs. Our previous work has shown that strains APEC17 and MT78 induced apoptosis in HD11 chicken macrophages. Here, we investigated if strains APEC17 and MT78 induced caspase 3/7 in avian fibroblasts CEC32, which are non-phagocytic cells. As a negative control, we used a non-pathogenic avian *E. coli* strain (IMT5104), and as a positive control, cells exposed to UV radiation. Cells were infected with 150 bacteria/cell for 1 h, washed and reincubated in medium containing 50 μg/ml gentamycin. At different time points, cell extracts were prepared and assayed for caspase 3/7 activity using Ac-DEVD-AMC substrate. Our results showed that caspase activation induced by UV irradiation (a known inducer of apoptosis) was 10 times lower in CEC32 fibroblast than in macrophages HD11; for both cell lines, activity was detected at 6 h post-infection; it was not detected at 2 h nor at 20 h post-infection. Infection with APEC17 or MT78 did not activated caspase 3/7 in CEC32, in agreement with previous results with chicken embryo fibroblasts, suggesting that fibroblasts do not respond to APEC infection as HD11 macrophages.

Financial support: BIC-Propesq UFRGS