PROINFLAMMATORY CYTOKINE PROFILE OF PORCINE INTESTINAL EPITHELIAL CELLS UPON STIMULATION WITH ENTEROTOXIGENIC ESCHERICHIA COLI AND HEAT LABILE TOXIN

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ABSTRACT

Enterotoxigenic Escherichia coli (ETEC) infection is of global importance because of its high morbidity and mortality rate in humans and animals. ETEC has been the principal agent causing the traveler’s diarrhea in humans and post-weaning diarrhea in pigs. Intestinal innate immunity is the first line of defense in enteric bacterial infections. The pathogenicity of ETEC is due to the fimbriae and heat labile (LT) and heat stable (ST) toxins. LT has two subunits A and B. Porcine intestinal epithelial cells (IPEC-J2) are a suitable in vitro model for studying ETEC infections. This study was conducted to analyze the changes in the gene expression of proinflammatory cytokines and chemokines upon stimulation of IPEC-J2 cells with different strains of ETEC and LT toxins for 3 hrs. We used wild type ETEC strains with K88ac fimbriae 3030-2 (O157:K87 LT+, STb+), 2534-86 (O8:K87: NM: LT-I+, STb+), 1836-2 (LT-, ST-, astA+), and G58-1, a non pathogenic, non-fimbriated, wild-type (O101:K28: NM, LT_), at multiplicity of infection 10:1. LT and LTb were used at 100 ng/ml. G58-1 and cells with media alone served as negative controls. The changes in the gene expression of proinflammatory cytokines such as IL-1α, IL-1β, TNF-α, IL-6 and chemokine, IL-8, were quantified relative to porcine specific cyclophilin-A. We found that the 3030-2 strain decreased IL-6 and increased TNF-α expression at 3h. Strain 1836-2 increased IL-8 and LTb decreased IL-8 gene expressions. Further investigations will help in understanding more about enteric mucosal innate immune responses to ETEC, which may potentially contribute to its prevention and treatment.