Campylobacter jejuni induces an anti-inflammatory response in human intestinal epithelial cells through activation of phosphatidylinositol 3-kinase/Akt pathway

Campylobacter jejuni is the most common cause of human acute bacterial gastroenteritis. Poultry is a major reservoir of C. jejuni and considered an important source of human infections, thus, it is important to understand the host response to C. jejuni from chicken origin. In this study, we demonstrated firstly that a chicken isolate SC11 colonized chicks faster than clinical isolate NCTC11168. Using the SC11, we further studied the host response to C. jejuni in terms of inflammatory response and involvement of cellular signaling pathways. Infection of C. jejuni SC11 was able to activate phosphatidylinositol 3-kinase (PI3K)/Akt pathway and induce pro-inflammatory interleukin-8 (IL-8) as well as anti-inflammatory cytokine IL-10 in human intestinal epithelial cell line Colo 205. The signalling pathways PI3K/Akt and mitogen-activated protein (MAP) kinases ERK and p38 were involved in C. jejuni-induced IL-8 and IL-10 expression. Inhibition of PI3K resulted in augmentation of C. jejuni-induced IL-8 production, concomitant with down-regulation of IL-10 mRNA, indicating an anti-inflammatory response was activated and associated with the activation of PI3K/Akt. Similar effect was observed for cytolethal distending toxin (CDT) deficient mutants. Moreover, we demonstrated that heat-killed bacteria were able to induce IL-8 and IL-10 expression to a lower level than live bacteria. We therefore conclude that C. jejuni activate a PI3K/Akt-dependent anti-inflammatory pathway in human intestinal epithelial cells which may benefit the intracellular survival of C. jejuni during infection.