In vitro treatment of lipopolysaccharide increases invasion of Pasteurella multocida serotype B:2 into bovine aortic endothelial cells

ABSTRACT

Pasteurella multocida serotype B:2 causes hemorrhagic septicemia in cattle and buffalo. The invasion mechanism of the bacterium when invading the bloodstream is unclear. This study aimed to characterize the effects of immunomodulatory molecules, namely dexamethasone and lipopolysaccharide, on the invasion efficiency of P. multocida serotype B:2 toward bovine aortic endothelial cells (BAECs) and the involvement of actin microfilaments in the invasion mechanism. The results imply that treatment of BAECs with lipopolysaccharide at 100 ng/mL for 24 h significantly increases the intracellular bacteria number per cell ($p < 0.01$) compared with those in untreated and dexamethasone-treated cells. The lipopolysaccharide-treated cells showed a significant decrease in F-actin expression and an increase in G-actin expression ($p < 0.001$), indicating actin depolymerization of BAECs. However, no significant differences were detected in the invasion efficiency and actin filament reorganization between the dexamethasone-treated and untreated cells. Transmission electron microscopy showed that P. multocida B:2 resided in a vacuolar compartment of dexamethasone-treated and untreated cells, whereas the bacteria resided in cellular membrane of lipopolysaccharide-treated cells. The results suggest that lipopolysaccharide destabilizes the actin filaments of BAECs, which could facilitate the invasion of P. multocida B:2 into BAECs.

Keyword: Pasteurella multocida; Actin cytoskeleton; Bovine aortic endothelial cells; Invasion; Lipopolysaccharide