Survival of *Corynebacterium pseudotuberculosis* within macrophages and induction of phagocytes death

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Abstract

Since *C. pseudotuberculosis* is a facultative intracellular pathogen the aim of this study was focused on evaluating mechanisms that allowed these bacteria to survive in macrophages and determining their influence on induction of cell death. The influence of *Corynebacteria* on the programmed cell death of macrophages was determined on the basis of induction the autophagy and apoptosis in the cultures of murine macrophage cell lines J774 infected with bacteria. *Corynebacterium pseudotuberculosis* strains could survive within macrophages more than 48 hours. During that time bacteria were released as a result of the process that lead to death of phagocytes. This property varied among studied strains. There was no increase of microtubule-associated protein I light chain 3 (MAP I LC3) activity in macrophages infected with examined strains comparing with uninfected cultures and cultures treated with autophagy inducer (rapamycin) that served as negative and positive controls, respectively. The study with confocal microscopy did not show the increasing of caspase-3 activity in the infected macrophages and their nucleus did not reveal the fragmentation.

Key words: *Corynebacterium pseudotuberculosis*, pathogenicity, macrophages, apoptosis, autophagy

Introduction

*Corynebacterium pseudotuberculosis* (*C. pseudotuberculosis*) is a gram-positive facultative intracellular pathogen. It is the etiological agent of caseous lymphadenitis (CLA), a chronic, common, economically important disease of small ruminants characterized by caseous abscessation of lymph nodes and/or internal organs (Williamson 2001). The pathogenesis of infection caused by these bacteria is not well defined, but many investigations have shown that two virulence factors of *C. pseudotuberculosis* play crucial roles in the development of the disease. The first is the exotoxin phospholipase D, that mediates dissemination of the pathogen within the host by increasing local vascular permeability. The second are the cell wall surface lipids, that allow bacteria to resist digestion by phagocytes (Brown and Olander 1987, Dorella et al. 2006).