EuroSciCon

EuroSciCon conference on

Protein, Proteomics and Computational Biology

December 06-07, 2018
Amsterdam, Netherlands

Biochem Mol biol J Volume:4
DOI: 10.21767/2471-8084-C5-021

MODELLING THE CYTOSOLIC [CA^{2+}] RESPONSES INDUCED BY *SHIGELLA* INVASION IN EPITHELIAL CELLS

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Shigellosis is an important problem of public health worldwide. *Shigella*, the causative agent of Shigellosis, invades the colon and causes an intense inflammatory reaction, leading to destruction of the epithelial tissue. During cell invasion, *Shigella* induces atypical Ca^{2+} signals, but its role in invasion has remained unclear. Every cell type uses Ca^{2+} as a second messenger to control a wide array of cellular functions. Thus, the perturbation of cellular Ca^{2+} homeostasis caused by *Shigella* facilitates the entrance of the bacteria and its dispersion to adjacent cells. This further leads to apoptosis and destruction of the intestinal epithelium. The bacterium induces local responses, described as an increase of Ca^{2+} localised in the invasion area, and global responses, that spread in the whole invaded cell. The local/global character of the responses is crucial in the cytotoxicity of the bacteria, as a high and sustained Ca^{2+} elevation could lead to cellular death and limit the dissemination of the bacteria. Preliminary work was carried out in order to analise the atypical Ca^{2+} responses induced by *Shigella* using modeling tools. Nevertheless, the models that have been proposed don't take into account the Ca^{2+} coming from the extracellular space, which has crucial contribution to the Ca^{2+} responses. Thus, in this work we present a partial differential equation (PDE) model that takes into account extracellular Ca^{2+} entry through Plasma Membrane Channels, as well as Ca^{2+} and InsP3 diffusion through the cytosol and the conditions caused by *Shigella*, in order to analise the global vs local character of the cytosolic [Ca^{2+}] responses during bacterial invasion. Numerical simulations show the impact of the plasma membrane channels in the local/global character of the [Ca^{2+}] responses, which implies that controlling extracellular Ca^{2+} entry to the cytosol could be crucial to find a mechanism that limit the dissemination of the bacteria.

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