The lectin Galectin-3 is important for survival of the host cell during infection by *Trypanosoma cruzi* modulating anti-apoptotic pathways.

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Introduction: Apoptosis can occur by an extrinsic pathway trough the "dead receptors" or/and by an intrinsic pathway that leads to the cytochrome c release from the mitochondria. Studies have shown evidences that galectin-3 participate in both pathways of apoptosis, and may influence the apoptosis induction in infected cells by *Trypanosoma cruzi*, the etiologic agent of Chagas disease **Objective**: Our experiments aim to investigate the role of galecin-3 over apoptosis signaling pathways in infected cells. **Methods**: HeLa, HeLa-shGal3 (depleted of Gal-3 by RNAi), HeLa-*scramble* (negative control of RNAi) cells were infected at different times with *T. cruzi* followed by functional analysis: (1) cell viability by MTT assay, (2) *western blot* analysis to determine the pro- e anti-apoptotic proteins levels, (3) colorimetric assay to measure caspase-3 activity, (4) and quantification of intracellular parasites by Giemsa staining. **Results and Perspectives:** HeLa-shGal3 infected cells presented the lowest viability rate. Furthermore, in the protein analysis Bax levels did not demonstrate a significant difference. However, HeLa-shGal3 presented a lower Bcl-2 level and an increased level of cleaved PARP was observed at 4 and 8 hours post-infection. More experiments have been developed to investigate the importance of Gal-3 nuclear-cytoplasm translocation on survival/death and the expression of pro- and anti-apoptotic molecules. We also aim to investigate the activation and processing of initiators and effectors caspases, unraveling these cellular signaling networks. Galectin-3 protein plays ubiquitous roles in many cellular events, the scientific contributions of its functions on Chagas disease can also be important in other protozoa disease that presents similar strategies of invasion and intracellular proliferation in the host.

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