# *TRYPANOSOMA CRUZI* SUBVERT THE AUTOPHAGIC PATHWAY TO INCREASE THE INFECTION IN THE HOST CELL.

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Autophagy is the cell process that serves to recycle cytoplasmic components and aged or damaged organelles. This process that is stimulated under starvation conditions, has been recently described as an innate immune response to intracellular pathogens. Although in several cases this response participates in the control of the infection, some microorganisms subvert autophagy for their own benefit. *Trypanosoma cruzi*, the etiologic agent of Chagas disease, invades a wide range of phagocytic and non-phagocytic cells by means of the infective trypomastigote form. Using CHO cells overespressing GFP-LC3, the best characterized autophagosome marker, we observed by confocal microscopy a colocalization between the parasitophorous vacuole and LC3 whereas no localization was observed in CHO cells overexpressing only GFP. This interaction with autophagosomes was then confirmed by double labeling of the parasites and the endogenous LC3 protein. Quantification studies show that induction of autophagy by starvation significantly increased the percentage of infected cells at 1-3 h after infection. Interestingly, this percentage was markedly reduced in the absence of the protein Atg5 necessary for the first steps of autophagic pathway, as demonstrated by using Atg5-deficient mouse embryonic fibroblasts. We conclude that *Trypanosoma cruzi* take advantage of the autophagic response to improve the colonization of the host cell.