Parasitus

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Foto de Portada

Trichomonas vaginalis (azul) conectados por citonemas (naranja) observados por microscopía electrónica de barrido.

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trafficking, as demonstrated in human and yeast models. In previous research from our laboratory, we identified the *T. brucei* Vps32 ortholog (TbVps32) and observed that downregulation of TbVps32 leads to a reduction in endocytosis and affects intracellular trafficking. In our current work, we aim to further investigate the role of this protein in the PCF stage. To achieve this goal, we have established two different cell lines: one in which TbVps32 is overexpressed under a Tet-inducible regulatory system (HA-TbVps32) and another in which protein expression can be silenced using an inducible interference RNA system (TbVps32-iRNA). Using these cell lines, we have performed numerous assays to study endocytosis. Using Ultrastructure expansion microscopy (U-ExM), we have identified clear phenotypic differences between uninduced and induced parasites. In conclusion, we have shown that both overexpression and silencing of TbVps32 impairs cell proliferation and its results in severe abnormal nuclear-kinetoplast configurations.

BMC-096

Interplay between autophagy and metacyclogenesis in *Trypanosoma cruzi*, unravelling the role of TcVps34-Vps15 complex

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Autophagy is a ubiquitous eukaryotic process that also occurs in trypanosomatid parasites. Half of the known yeast and mammalian AuTophaGy (ATG) proteins were detected in trypanosomatids, although with low sequence conservation. Interestingly, autophagy is involved in differentiation of *T. cruzi* from epimastigotes to metacyclic trypomastigotes, a process called metacyclogenesis. In mammals, two kinases differentially regulate the process of autophagy: mTor and a phosphatidylinositol 3-kinase, Vps34, which interact with a regulatory subunit, Vps15. In this work, we demonstrate that parasites overexpressing TcVps34 or TcVps15 proteins enhance both, autophagy and metacyclogenesis. TcVps34 or TcVps15 overexpressing epimastigotes were able to differentiate to metacyclic forms in a higher proportion than wild type cells. Parasites overexpressing these proteins showed a more intense labeling with the autophagosome marker Atg8.1 and higher levels of monodansycadaverine (MDC) staining, a specific *in vivo* marker for autophagic vacuoles, in the intermediate forms of differentiated parasites, in comparison to control parasites. To extend this study we also performed assays with DQ-BSA, to evaluate degradative compartments. TcVps34 and TcVps15 overexpressing epimastigotes subjected to nutritional stress shown a significant increase in the number of lysosomes, as compared to controls. In addition, treatment with wortmannin, an inhibitor of autophagy, of parasites exposed to differentiation conditions impaired the autophagic response in less measure in overexpressing parasites. Finally, we are performing infection assays with these overexpressing parasites to assess whether this process is affected. Taken together, these data demonstrate the key role of phosphatidylinositol 3-phosphate pathway in autophagy, differentiation and cell cycle progression in *T. cruzi*.

BMC-097

Estudios de transcriptómica comparativa en amastigotas axénicos versus amastigotas celulares de *Trypanosoma cruzi*

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Trypanosoma cruzi es el agente causante de la enfermedad de Chagas, un serio problema de salud pública en gran parte de la población de