[PS1.58]

Effect of kinases, actin and dynamin inhibitors in *Toxoplasma gondii* egress from host cells

L. A. Caldas*, M. Attias, W. de Souza
Universidade Federal do Rio de Janeiro, Brazil

Toxoplasma gondii is capable of infecting almost all warm-blooded and nucleated cells. During *T. gondii* cellular cycle, egress is a very important step to enable another host cell invasion. Despite of it, egress of *T. gondii is* still poorly understood. However it is known that it is dependendt of an influx of calcium. For this reason, calcium ionophore is used to trigger the process. Since kinases are important for the invasion, at least in neutrophils, we decided to investigate their possible role in egress. In this work we show that parasite egress induction with calcium ionophore was only slightly affected by wortmanin and staurosporin, inhibitors of PI3-kinase and kinase C respectivelly. On the other hand the addition of the specific inhibitor of tyrosine kinase, genistein, efficiently blocked the exit of parasites to more than 50%. The actin polymerization inhibitor cytochalasin D also blocked the induced egress of *T. gondii*. Dynasore, which is known to block GTPase activity of dynamin had little or no effect in this step of *T. gondii* cellular cycle. In conclusion, phosphorilation of substrates by tyrosine kinases seem to be important for this process. Actin filaments also take part on this phenomenon, but membrane pinching off by dynamin is not necessary. Therefore, egress probably is an event that depends on multiple signaling pathways and requires the integrity, at least partial, of the cytoskeleton of the host cell to occurr.

Supported by: CNPq

Keywords: Toxoplasma gondii, egress, kinases, dynamin