Lysophosphatidylcholine-induced cellular differentiation of the protozoan parasite *Herpetomonas samuelpessoai*: involvement of protein kinase CK2

Dutra, F. L.,[1] Vieira, D. P.,[1] Atella, G. C.,[2] Silva Neto, M. A. C.,[2] and Lopes, A. H.[1]
[1]*Instituto de Microbiologia Prof. Paulo de Góes - UFRJ*
[2]*Instituto de Bioquímica Médica - UFRJ*

Herpetomonas samuelpessoai is a trypanosomatid parasite of the insect *Zelus leucogramus*. Lysophosphatidylcholine (LPC) is a major bioactive compound of plasmatic lipoproteins like LDL. The presence of LPC in the saliva of *Rhodnius prolixus* and its anti-hemostatic activities have been demonstrated. The enzyme phospholipase A2 (PLA2) catalyzes the hydrolysis of the 2-ester bond of 3-sn-phosphoglyceride, transforming phosphatidylcholine (PC) into LPC. We have shown that LPC modulates some signaling pathways that lead to cell differentiation of *H. samuelpessoai* and that PLA2 probably rules this process by converting PC into LPC. The present study intends to confirm the involvement of protein kinase CK2 in this process. *H. samuelpessoai* parasites were grown for up to 96 hours in Roitman complex medium, in the absence or in the presence of PC, LPC, and CK2 inhibitors (DRB and TBB). The percentage of non-differentiated (promastigote) and differentiated forms (paramastigote and opisthomastigote) were daily determined by Giemsa stained preparations. The best results were obtained at 48 hours of growth: control (60% promastigotes, 40% differentiated); LPC (40% promastigotes, 60% differentiated); DRB + LPC (70% promastigotes, 30% differentiated); TBB + LPC (60% promastigotes, 40% differentiated). These results highly suggest the involvement of CK2 in cell differentiation of *H. samuelpessoai* triggered by LPC. Supported by: CNPq, FAPERJ, CNPq/PIBIC - UFRJ, CAPES.