LYSOPHOSPHATIDYLCHOLINE IS A MODULATOR OF *TRYPANOSOMA CRUZI* INFECTION

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Rhodnius prolixus is a *Trypanosoma cruzi* vector, transmitted by its feces. One of the routes of host cell invasion occurs through the wound produced by the insect bite. Parasite thus faces a cell environment within the wound previously stimulated by saliva. In the present work we tested the role of bug saliva on parasite transmission. Firstly, saliva injection on mice skin induced dose-dependent cell recruitment. Secondly, treatment of macrophages with saliva triggered the phosphorylation of almost all proteins on phosphotyrosine. We showed that *R. prolixus* saliva stores lysophosphatidylcholine (LPC) during their growth. LPC is a candidate molecule in saliva able to trigger the above mentioned effects. To clarify the molecular mechanism underlying LPC signaling, we studied the effect of commercial LPC and saliva on the intracellular free calcium concentration $[Ca^{2+}]_i$ and NO production of murine peritoneal macrophages. LPC, in the same way as saliva, induced an elevation of $[Ca^{2+}]_i$ in a concentration-dependent manner and was able to reduce the NO production of macrophages stimulated by lipopolysaccharide. Moreover, mice that received saliva or LPC showed increased parasitemia levels in acute phase of infection. This is the first demonstration of involvement of a triatominae-derived molecule as an enhancing factor of Chagas disease transmission. Supported by CNPq, FAPERJ, IFS, OMS.