# Lipid mediators and vector infection: Trypanosoma rangeli inhibits Rhodnius prolixus hemocyte phagocytosis by modulation of phospholipase $A_2$ and PAF-acetylhydrolase activities.

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In this work we investigated the effects of Trypanosoma rangeli infection through a blood meal on the hemocyte phagocytosis in experiments using the 5th instar larvae of Rhodnius prolixus. Hemocyte phagocytic activity was strongly blocked by oral infection with the parasites. In contrast, hemocyte phagocytosis inhibition caused by T. rangeli infection was rescued by exogenous arachidonic acid (20ug/insect) or platelet activating factor (PAF; 1ug/insect) applied by hemocelic injection. Following the oral infection with the protozoan we observed significant attenuation of phospholipase $A_2$ ($PLA_2$) activities in R. prolixus hemocytes (cytosolic $PLA_2$: $cPLA_2$, 39±8% inhibition; $Ca^{+2}$-independent $PLA_2$: $iPLA_2$, 36 ± 6% inhibition) and enhancement of secreted $PLA_2$ ($sPLA_2$) activity in cell-free hemolymph (110±20%). At the same time, the PAF-acetyl hydrolase (PAF-AH) activity in the cell-free hemolymph increased considerably (9.2 times). Our results suggest that T. rangeli infection depresses eicosanoid and insect PAF analogous (iPAF) pathways giving support to the role of $PLA_2$ in the regulation of arachidonic acid and iPAF biosynthesis and of PAF-AH by reducing the concentration of iPAF in R. prolixus. This illustrates the ability of T. rangeli to modulate the immune responses of R. prolixus to favor its own multiplication in the hemolymph.

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