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A recombinant form of *Trypanosoma cruzi* P21 upregulates different pathogens cell invasion

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We have recently characterized a new component from *T. cruzi* (P21). The recombinant P21 (P21-His_6) adhered to host cell in a dose dependent manner and treatment of non phagocytic host cell with P21-His_6 during invasion of extracellular amastigotes and metacyclic trypomastigotes increased cell invasion by both forms. Here, we proposed to verify the effect of P21-His_6 on cell invasion by amastigotes from *T. cruzi*, promastigotes from *Leishmania amazonensis* and taquizoites from *Toxoplasma gondii* into inflammatory peritoneal macrophages. Our results showed that host cell treatment with P21-His_6 upregulated cell invasion by *T. cruzi*, *L. amazonensis* and *T. gondii*. The mechanism beneath the increased cell invasion due to P21-His_6 cell treatment seems to be related to cortical actin polymerization. In this context, inflammatory peritoneal macrophages treatment with P21-His_6 during 24hs showed enhanced cortical actin polymerization and cells acquired a ring shaped form in contrast with control cell which showed a slight cortical actin staining and non polymerized actin in the cytosol. In conclusion, P21-His_6 may be considered an unspecific phagocytosis inducer that acts by regulating cortical actin polymerization. Financial Support: FAPEMIG/UFU/CNPq.

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