Exogenous Calreticulin, incorporated onto non-infective Trypanosoma cruzi epimastigotes, promotes their internalization into mammal host cells

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 Chagas disease is an endemic pathology in Latin America, now emerging in developed countries, caused by the intracellular protozoan Trypanosoma cruzi, whose life cycle involves three stages: amastigotes, epimastigotes, and trypomastigotes. T. cruzi Calreticulin (TcCRT), an endoplasmic reticulum resident chaperone, translocates to the external cellular membrane, where it captures complement component C1, ficolins and MBL, thus inactivating the classical and lectin pathways. Trypomastigote-bound C1 is detected as an ?eat me? signal by macrophages and promotes the infective process. Unlike infective trypomastigotes, non-infective epimastigotes either do not express or express only marginal levels of TcCRT on their external membrane. We show that epimastigotes bind exogenous rTcCRT to their cellular membrane and, in the presence of C1q, this parasite form is internalized into normal fibroblasts. On the other hand, Calreticulin (CRT)-deficient fibroblasts show impaired