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Proteomic dynamics of Rhodnius prolixus under blood feeding conditions and its effect on *Trypanosoma cruzi* metacyclogenesis

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One of the most important endemic diseases in Latin America is Chagas disease. The etiologic agent of this disease is *Trypanosoma cruzi* which transmission is related to the blood feeding insect vectors, the triatomines *Rhodnius prolixus.* Triatomines are hematophagous insects, ingesting huge amounts of blood in a single meal. Inside the insects stomach, erythrocytes are lysed and water is absorbed. In the intestines, blood proteins are digested and nutrients are absorbed. Finally, in the rectum, blood remains are stored. In parallel with blood digestion, *T. cruzi* trypomastigotes ingested with the blood of the vertebrate host undergo three stages of transformations in the vector gut to complete their life cycle and become infectious. Since the first description of the disease, evidence in the literature has indicated that several factors are important to parasite establishment in the bug. We have thus started a large comparative proteomic study on *Rhodnius* gut tissues upon a blood meal and how this response is modulated by *T. cruzi* infection. Surprisingly, very few proteins, although directly linked to the gut physiology were observed responding to a blood meal and none to the presence of the parasite. Interestingly, we have identified the presence of the vertebrate host C4BP, a regulator of the complement which is at the junction between the classical and lectin pathways thereby preventing their processing. Moreover, two isoforms of a calcium-binding protein were found up-regulated after feeding. Since Ca^{+2} is required for C1q and lectins binding to their targets, its sequestration suggests an impact on the related complement pathways. The presence of these inhibitory molecules in the insects gut could as well benefit for the development of the parasite which insects development form is sensitive to the complement. Assuming that complement inhibitors may protect parasites, it is reasonable to suppose that inactivating them could impair parasites establishment.

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