Transcriptome analysis of mice hearts infected with two strains of Trypanosoma cruzi: Insights into the parasite effects on the host gene expression

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Chagas disease is a parasitic infection, caused by the kinetoplastid protozoan *Trypanosoma cruzi* (*T. cruzi*). Both, mammal host and *T. cruzi* parasites are highly genetically polymorphic, and therefore many variables come into play, making the disease outcome difficult to predict. For example, even more than a century after Chagas disease discovery, the mechanisms underlying the tissue tropism of *T. cruzi* has still to be elucidated. These complex interactions between host and pathogens comprise many still poorly understood surface and soluble glycoproteins as well as intracellular mediators. They are responsible to permit the survival of *T. cruzi* in the blood stream, adhesion to the host cellular membrane, as well as evasion to reactive oxygen species and immune system. On the other side, distinct MHC haplotypes in the mammal host may also change the disease course. In the present work, we seek to better understand the global host response or gene expression, to different *T. cruzi* strain parasites. To do so, we performed a RNA-Seq analysis of BALB/c mice hearts single or double-infected with two strains until day 15º, representing the acute phase of infection. Here, we demonstrate a clear distinction between the host gene expression against these two *T. cruzi* strains. Col1.7G2 (*T. cruzi* I), a known virulent strain, strongly activates Th1-polarized immune response genes. Whereas JG (*T. cruzi* II), a known non-virulent strain showed weaker expression of immunological genes while strongly inhibiting ribosomal proteins and oxidative pathways. Interestingly, enrichment pathway analysis of mice hearts infected with the mixture of the two strains, showed both phenotypes simultaneously. Altogether, our data aid to better understand the complex host-pathogen interactions in the context of Chagas disease, and the effect of different *T. cruzi* strains over mouse gene expression.