**INCREASED EXPRESSION OF IRON SUPEROXIDE DISMUTASE-A (FESOD-A) AND TRYPAREDOXIN PEROXIDASE (TXNPx) ENZYMES IN *Trypanosoma cruzi* POPULATION RESISTANT TO BENZNIDAZOLE.**

Nogueira FB, Romanha AJ and Murta SMF
Lab. de Parasitologia Celular e Molecular, CPqRR, FIOCRUZ, Belo Horizonte, MG, Brazil

Iron-Superoxide Dismutase-A (FeSOD-A) removes excess superoxide radicals via dismutation to oxygen and hydrogen peroxide. The tryparedoxin peroxidase (TXNPx) catalyzes the reduction of hydrogen peroxide or small-chain organic hydroperoxides to water or alcohols, respectively. In this work, FeSOD-A and TXNPx was characterized in 8 *T. cruzi* populations susceptible, naturally resistant or with *in vitro*-induced (17LER), or *in vivo*-selected (BZR) resistance to Benznidazole (BZ). The northern blot profile of total RNA from *T. cruzi* samples, hybridized with specific probes, revealed that the levels of TcFeSOD-A and TcTXNPx mRNA were 3 and 2-fold higher in the 17LER *T. cruzi* population, compared with counterpart 17WTS. This difference of expression was confirmed by real-time RT-PCR. The results of real-time PCR and southern blot, showed that 17LER has 2-fold more TcFeSOD-A gene copies than 17WTS. However, the TcTXNPx gene is not amplified in the *T. cruzi* genome. Western blot analysis of *T. cruzi* protein extracts probed with a rabbit anti-recombinant TcFeSOD-A and anti-TcTXNPx polyclonal serum revealed one polypeptide of 23 KDa of same size for both genes. The intensity these polypeptides were similar in all samples analyzed, except 17LER, which displayed a band 2-fold more intense for both genes. Thus, we observed the increased expression of FeSOD-A and TXNPx in *Trypanosoma cruzi* population with *in vitro*-induced resistance to BZ.

Supported by CNPq, FAPEMIG and CPqRR/FIOCRUZ.
Key words: *Trypanosoma cruzi*, resistance, benznidazole, antioxidant defense, differential expression.