Expression, functionality, and localization of apurinic/apyrimidinic endonucleases in replicative and non-replicative forms of Trypanosoma cruzi

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Trypanosoma cruzi is the etiological agent of Chagas disease. The parasite has to overcome oxidative damage by ROS/RNS all along its life cycle to survive and to establish a chronic infection. We propose that T. cruzi is able to survive, among other mechanisms of detoxification, by repair of its damaged DNA through activation of the DNA base excision repair (BER) pathway. BER is highly conserved in eukaryotes with apurinic/apirimidinic endonucleases (APEs) playing a fundamental role. Previous results showed that T. cruzi exposed to hydrogen peroxide and peroxinitrite significantly decreases its viability when co-incubated with methoxyamine, an AP endonuclease inhibitor. In this work the localization, expression and functionality of two T. cruzi APEs (TcAP1, Homo sapiens APE1 orthologous and TcAP2, orthologous to Homo sapiens APE2 and to Schizosaccaromyces pombe Apn2p) were determined. These enzymes are present and active in the two replicative parasite forms (epimastigotes and amastigo