# DNA repair BER pathway inhibition increases cell death caused by oxidative DNA damage in Trypanosoma cruzi

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Trypanosoma cruzi, a parasitic protozoan, is the etiological agent of Chagas disease, an endemic and neglected pathology in Latin America. It presents a life cycle that involves a hematophagous insect and man as well as domestic and wild mammals. The parasitic infection is not eliminated by the immune system of mammals; thus, the vertebrate host serves as a parasite reservoir. Additionally, chronic processes leading to dysfunction of the cardiac and digestive systems are observed. To establish a chronic infection some parasites should resist the oxidative damage to its DNA exerted by oxygen and nitrogen free radicals (ROS/RNS) generated in host cells. Till date there are no reports directly showing oxidative DNA damage and repair in T. cruzi. We establish that ROS/RNS generate nuclear and kinetoplastid DNA damage in T. cruzi that may be partially repaired by the parasite. Furthermore, we determined that both oxidative agents diminish T. cruzi cell viability. This effect is significantl