Selenoproteins are characterized by the incorporation of at least one amino acid selenocysteine (Sec-U) encoded by in-frame UGA stop codons. Although not a ubiquitous pathway in all organisms, it was also identified in several protozoa, including the Kinetoplastida. Previous reports demonstrated that *Trypanosoma brucei* selenophosphate synthetase (SPS), central enzyme in the selenocysteine synthesis, is required under sub-optimal growth conditions, suggesting probable role in oxidative stress protection of the parasite and its absence severely hampers the parasite survival in the presence of an oxidizing environment. The presenting work demonstrated growth defects for the both forms of *T. brucei* under normal growth conditions and treatment with stressors of endoplasmic reticulum, tunicamycin and DTT, reduced the viability of tetracycline-induced RNAi lineages, although no increase of BiP expression was observed. Complex purification using SPS fused to PTP-tag demonstrated no stable interaction with other proteins and Indirect Immunofluorescence assay showed cytoplasmic localization, but is possible to observe not conventional pattern, with presence of dots along of the cytoplasm. Selenoprotein T (SelT) is not required to procyclic growth and it lack in bloodstream form presented slight growth reduction. Treatment with ER stressors also reduced SelT RNAi lineage and no increase of BiP expression was observed. The data suggest which selenoproteins participation in the ER defense, but not direct activation of the Unfolded Protein Response (UPR). SelT single knock-out in *Leishmania amazonensis* no presented decrease of promastigote viability nor increase of cell sensibility of stressors of ER. Small reduction of amastigote growth inside of macrophages was observed. New experiments are being planned to evaluate the participation of selenoproteins in other steps of endoplasmic stress response, such as Ca$^{2+}$ homeostasis.