Mitochondrial bioenergetics in *Leishmania infantum* devoid or overexpressing mitochondrial peroxiredoxin

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Leishmania infantum is the causative agent of visceral leishmaniasis in the Mediterranean. Like in other aerobic cells, mitochondria constitute an important source of reactive oxygen species (ROS) that can lead to mitochondria irreversible damage, but can also be involved in cell signaling pathways. We have previously shown that *L. infantum* promastigotes devoid of mitochondrial peroxiredoxin (*Lim*TXNPx) are capable of surviving and proliferating at wild type (WT) levels. ROS production by *Lim*TXNPx null mutants is higher than WT, although no significant differences were observed in ROS generation when cells were treated with antimycin A. Here we report on the determination of mitochondrial transmembrane potential (??) and on oxygen consumption by null mutants, obtained by a DNA recombination strategy, and *Lim*TXNPx overexpressing cells, transformed with pTEX. $Dioc_6(3)$, a cationic fluorescent probe, was used to measure the mitochondrial electrical transmembrane potential by flow cytometry and Oxygen consumption was determined using a Clark-type electrode. No significant differences in ?? among null mutants, overexpressing *Lim*TXNPx and WT cells was observed. Oxygen consumption was 20% lower than WT in null mutants, but overexpressing cells showed no difference when compared to WT cells. Together with our previous observations these results support the idea that in *L. infantum* promastigotes *Lim*TXNPx physiological functions may be compensated by other antioxidants and or / repair mechanisms.

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