The Involvement of Replication Protein A-1 and RAD51 with Telomere Damage Response in *Leishmania*

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Replication protein A is a complex of single-stranded DNA-binding proteins implicated in DNA metabolism, including DNA repair and telomere maintenance. In *Leishmania* the subunit RPA-1 (LaRPA-1) binds and co-localizes *in vivo* with telomeres. In yeast and humans RPA also works as a telomerase recruiter and as a DNA damage sensor at telomeres. Its involvement with the DNA damage repair triggers its hyperphosphorylation and the recruitment of proteins from the RAD51 group. We thought to determine if in *Leishmania* RPA is also required to protect chromosome ends from being detected by the DNA damage. Thus, we checked the expression of LaRPA-1 and the repair protein RAD51 in parasites treated and non-treated with sub-lethal doses of the DNA-damaging agent phleomycin. We also tested if native LaRPA-1 undergoes phosphorylation upon DNA damage. Here we show that phosphorylation of native LaRPA-1 is independent on DNA damage and occurs naturally during parasite growth, although hyperphosphorylation appears to increase upon damage. Phleomycin also induced G1/S cell cycle arrest and culture synchronization. In addition, the expression of LaRPA-1 slightly diminished in parasites treated with phleomycin whereas a gradual increase in the expression of RAD51 occurred probably in response to DNA damage. Moreover, more LaRPA-1 was immediately recruited to telomeres upon phleomycin-induced damage, suggesting that the presence of LaRPA-1 may prevent loss of single-stranded telomeric DNA and also elicit activation of a local response.