Macrophage antioxidant mechanisms modulation in Leishmanis infection

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INTRODUCTION. Leishmaniasis represents a class of neglected tropical diseases prevalent in 98 countries in 5 continents. Leishmaniasis is endemic in Brazil, especially on the Northeast region. The etiological agent of leishmaniosis, *Leishmania spp.*, infects human macrophage to complete its life cycle. The Leishmania mechanisms involved in evading macrophage oxidative burst are still not clear, since this class of parasite is poor in endogenous antioxidant defenses. A possible mechanism might involve the activation of host antioxidant defenses to survive inside macrophage. **OBJECTIVE:** Here, we investigate macrophage antioxidant defenses modulation during *in vitro* Leishmania infection. **MATERIAL AND METHODS:** We retrieved transcriptional data from Gene Expression Omnibus, GSE42088. This dataset involves a time-course transcriptional investigation in dendritic cells infected with *Leishmania major*. Microarray data was normalized using Affy R package. Differential expression was conducted using limma R package and functional enrichment was made using topGO R package. We also experimentally investigated macrophage oxidative parameters such as lipoperoxidation, protein carbonylation, thiol content, as well as SOD and CAT activity in RAW 264.7 cell line infected with *Leishmania infantum*. **RESULTS AND DISCUSSION:** We found *L. major* infection significantly alters dendritic cells gene expression. The Gene Ontology term GO:1901700 (response to oxygen-containing compound) was significantly enriched among differentially expressed genes (adjusted pvalue = 4.43e-17) after 8h of *L. major* infection. Additionally, *L. infantum* infection was able to modulate oxidative parameters in RAW 264.7 cell line. **CONCLUSION:** Leishmania infection was able to modulate macrophage oxidative response *in vitro*.

Keywords: Leishmaniasis, antioxidant, transcriptome.

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