

302**INCREASED TAU PHOSPHORYLATION AND RECEPTOR FOR ADVANCED GLYCATION ENDPRODUCTS (RAGE) IN THE BRAIN OF MICE INFECTED WITH LEISHMANIA AMAZONENSIS**

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Leishmaniasis is a parasitosis caused by several species of the genus *Leishmania*, an obligate intramacrophagic parasite. Although neurologic symptoms have been largely observed in human cases of the disease, the manifestation of degenerative processes associated to the central nervous system (CNS) in leishmaniasis is poorly studied. The aim of the present work was to investigate if peripheral infection of BALB/c mice with *Leishmania amazonensis* affects tau phosphorylation and RAGE protein content in the brain, which represent biochemical markers of neurodegenerative processes observed in diseases with a pro-inflammatory component, including Alzheimer's disease and Down syndrome. Four months after a single right hind footpad subcutaneous injection of *L. amazonensis*, the brain cortex of BALB/c mice was isolated. Western blot analysis indicated an increase in tau phosphorylation (Ser396) and RAGE immunocontent in infected animals. Brain tissue TNF- α and IL-1 β levels were not different from control animals; however, increased protein carbonylation and impairment in enzymatic and non-enzymatic antioxidant defenses were detected. These data, altogether, indicate an association between impaired redox state, tau phosphorylation and RAGE up-regulation in the brain cortex of animals infected with *L. amazonensis*. In this context, it is possible that neurologic symptoms associated to chronic leishmaniasis are associated to disruptions in the homeostasis of CNS proteins, such as tau and RAGE, as consequence of oxidative stress. This is the first demonstration of alterations in biochemical parameters of neurodegeneration in an experimental model of *Leishmania* infection. Palavra-chave: Leishmaniasis; oxidative stress; neurodegeneration.