RESTRAIN PATHOGENIC RESPONSE BY *L. AMAZONENSIS* INFECTION IN PRE-INFECTED MICE WITH *L. MAJOR* ASSOCIATED WITH EARLY INDUCTION OF RESISTANCE MECHANISM.

González C.Z.L.[1], Santiago, Helton C.[1], Macedo J.P.[1], Seixas-Rego V.A.[1], Afonso L.C.C.[2], Vieira L.Q.[1]

[1]Departamento de Bioquímica e Imunologia, Instituto de Ciências Biológicas, UFMG , Belo Horizonte, MG, Brazil.
[2]Departamento de Ciências Biológicas, ICEB, and NUPEB, UFOP, Ouro Preto, MG, Brazil

Experimental *Leishmania* spp infection in mice has approximated to knowledge of several immunological events induced for protozoan parasites and the importance of the resistance mechanism in restrain parasite. We have demonstrated that C57BL/6 mice previously *L. major-infected*, restrain pathogenic responses induced by *L. amazonensis* infection despite of maintaining high parasite burden. These mice showed high TNF-α and iNOS mRNA expression in situ. Surprisingly, the regulatory response did not seem to be related to IL-10 production, as evidenced by lower levels of mRNA and protein expression of IL-10 in the *L. amazonensis* lesion from pre-infected mice. Early in infection, draining lymph node cells from *L. amazonensis* lesions from pre-infected mice had higher frequencies of $CD8^+CD25^+CD28^-$ T cells than mice infected only with *L. amazonensis,* 53 and 43 percent, respectively. We suggest that the control of lesion progression caused by *L. amazonensis* in C57BL/6 mice pre-infected with *L. major* is related with to early induction of regulatory T cell phenotype, the reduction of IL-10 levels and increase of some resistance mechanisms.

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