CONTROL OF LESIONS IN TNFRp55-/- MICE INFECTED WITH  LEISHMANIA MAJOR LEISHMANIA MAJOR MAY BE PARTIALLY ACHIEVED WITH THALIDOMIDE.Seixas, V. A. R.[1,] Afonso, L. C. C [3]. , Arantes, R. M. E. [2,] Vieira, L. Q.[1] 1-Departamento de Bioquímica e Imunologia, 2- Departamento de Patologia Geral, ICB, UFMG , Belo Horizonte, MG 3- Departamento de Ciências Biológicas e Nupeb, UFOP, Ouro Preto, MG, Brazil.
Mice lacking the TNF receptor 1 (TNFRp55-/-) infected with *L. major* clear parasites but do not resolve lesions, which display an intense inflammatory infiltrate. TNFRp55-/- mice produce high levels of TNF and IFN-g sistemically, higher levels of chemokines and lower levels of apoptosis in lesions which, together, would lead to the unresolved cellular infiltrate. In this work we looked for ways to resolve lesions in TNFRp55-/- mice. Thalidomide is a candidate, since it inhibits TNF production. TNFRp55-/- mice received thalidomide orally from the 6$^{th}$ week of infection with *L. major* (30 mg/kg/day), for 30 days. Smaller lesions were found from 7 weeks of infection. No parasites were detected at the site of infection in either group. Macrophages harvested from thalidomide-treated mice at 24 weeks of infection showed higher NO production and lower arginase activity. In vitro treatment with thalidomide induced NO production by macrophages from thalidomide-treated mice. In addition, IL-10 production by lymph node and spleen cells was lower in thalidomide-treated mice. We conclude thalidomide induced increased inflammatory response, but effectively mediated resolution of lesions in TNFRp55-/- mice infected with *L. major*.
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