Taenia crassiceps cysticerci antigens modulate the inflammatory response in the experimental neurotoxoplasmosis

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Toxoplasma gondii is a pathogenic agente capable o causing both local and systemic disease in immunocompetent and immunocompromised individuals. It can be agressive inducing lesions in the central nervous system, viscera, eye globe and/or lymohatic ganglia. Neurocysticercosis is the most severe form of cysticercosis. It is the one of the main helminthiasis of the central nervous system leasing to varied symptoms. During toxoplasmosis infection the inflammatory response is typically pro-inflammatory. When there is co-infection between these two agents, this typical pro-inflammatory response may lead to the death of the parasites resulting in the release of antigens. Therefore an experimental model using *T. gondii* cysts and *Taenia crassiceps* antigens was developed. The aim of this study was to evaluate the influence of *T. crassiceps* cysticerci antigens in the modulation of the inflammatory response of the experimental neurotoxoplasmosis. BALB/c mice were inoculated with *T. gondii* cysts and/or *T. crassiceps* cysticerci antigens. The animals were euthanized 60 or 90 days after the inoculation. The histopathologic analysis and the cytokine dosage from spleen cell culture were performed. The animals from the neurotoxoplasmosis group at 90 DAI (NT90) presented greater intensity of the lesions such as vasculitis, meningitis and microgliosis alongside with a Th1 immune profile with high dosages of IFNγ. While in the neurocysticercosis group at 60 DAI (NCC60) the lesions were more discrete with high dosages of IL4 displaying a Th2 immune profile. In the co-infected group the parenchyma lesions were more discrete. Also in the co-infected group there were lower dosages of IFNγ and higher dosages of IL4 in comparison to the NT90 group. It is possible to conclude that the *T. crassiceps* cysticerci antigens decreased the intensity of the lesions caused by the *T. gondii* infection inducing a Th2 immune response.