Video Article

Behavioral protocols to evaluate memory impairment during experimental cerebral malaria

Patricia A. Reis¹, Clarissa M. Comim², Patricia T. Bozza¹, Guy Zimmermam³, Valber S. Frutuoso¹, João Quevedo⁴, Hugo C. Castro-Faria-Neto¹

URL: http://www.jove.com/video/2902

DOI: doi:10.3791/2902

Keywords: Cerebral malaria, cognitive damage, Plasmodium berghei ANKA

Date Published: 6/15/2015

Citation: Reis, P.A., Comim, C.M., Bozza, P.T., Zimmermam, G., Frutuoso, V.S., Quevedo, J., Castro-Faria-Neto, H.C. Behavioral protocols to evaluate memory impairment during experimental cerebral malaria. *J. Vis. Exp.* (), e2902, doi:10.3791/2902 (2015).

Abstract

Neurological impairments are frequently detected in children surviving cerebral malaria (CM), the most severe neurological complication of infection with Plasmodium falciparum. The pathophysiology and therapy of long lasting cognitive deficits in malaria patients after treatment of the parasitic disease is a critical area of investigation. We have demonstrated recently that infection of C57BL/6 mice with Plasmodium berghei ANKA (PbA) resulted in documented CM and sustained persistent cognitive damage detected by a battery of behavioral tests after cure of the acute parasitic disease with chloroquine therapy. Strikingly, cognitive impairment was still present 30 days after the initial infection [1]. Memory function is vulnerable to a variety of pathological process. Several behavioral tasks have been used to identify cognitive impairment, and can predict witch brain areas have been mainly affected [2-4]. Here we show different models that evaluates learning and memory formation and consolidation. We use models of habituation to the open-field (elementary nonassociative learning tasks of behavioral during habituation to a novel environment and associated to hippocampus function), memory of recognition of objects (a critical component of declarative memory that is mainly dependent on the hippocampus) and inhibitory avoidance tasks (relies on the dorsal hippocampus, but also depends on the entorhinal and parietal cortex and is modulated by the amygdale)[3-9] to evaluated memory in mice surviving experimental cerebral malaria.

Disclosures

No conflicts of interest declared.

¹Laboratório de Imunofarmacologia, Instituto Oswaldo Cruz – FIOCRUZ

²Laboratório de Neurociências, Universidade do Extremo Sul Catarinense

³University of Utah, • Department of Medicine and Program in Human Molecular Biology and Genetics

⁴Laboratório de Neurociências, • Universidade do Extremo Sul Catarinense