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Persistent cognitive damage in cloroquine-treated mice with cerebral malaria

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Malaria remains the most important parasitic disease, causing 2–3 million deaths every year. Cerebral malaria (CM) is the most severe neurological complication of infection with *Plasmodium falciparum* and is the major cause of acute non-traumatic encephalopathy in tropical countries. Subsequent neurological impairments are most common and severe in children who survival from CM, and the persistent of cognitive deficits after healing suggests areas for further research. Male C57Bl/6 and Balb/c mice (20–28 g, n = 10/group) were infected on day 0 (200 μL, i.p.) with *Plasmodium berghei* Anka parasited erythrocytes (PRBC 10^6). On day 6 the animals were orally treated (200 μL) with chloroquine 25 mg/kg (p.o.) during 7 days (day 12 post-infection). As a control, one non parasited group (infected with 10^6 wealthy erythrocytes – RBC) was treated with the same dose of the standard drug and one received physiologic saline. On day 15 mice were submitted to behavioural task. Habituation to an open field was carried out in open field divided into 9 equal rectangles by black lines. Animals were gently placed on the left quadrant, and was allowed to explore the arena for 5 mins (training session) and 24 hrs later submitted again to a similar session (test session). Crossing of the black lines and rearing performed in both sessions were counted. No differences in the number of crossings and rearings was observed between groups in the habituation to the open-field training session in C57Bl/6 mice, it means, there was no difference in motor and exploratory activity between groups. In the test session, we did not observed significant reduction in both crossings and rearings in infected mice treated with chloroquine compared with the non-infected mice treated with chloroquine or saline. In Balb/c mice was observed a significant reduction in both crossings and rearings in infected mice treated with chloroquine group compared with the non-infected mice treat with chloroquine or saline ($p < 0.05$ Student's T test). The deficits demonstrated here is similar, at least in part, to the cognitive alterations observed in patients surviving cerebral malaria, particularly on memory impairment. In this way, we believe that the animal model of cerebral malaria will help us to investigate the biological mechanisms involved in the cognitive deficits associated with cerebral malaria and to suggest new therapeutics approaches to persistent neurological impairments.

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