Oral Administration of Coenzyme Q10 has the Capacity to Stimulate Innate Lymphoid Cells Class Two during Experimental Cerebral Malaria

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Abstract

Cerebral malaria is a complex neurological syndrome, whose pathology is mediated by inflammatory processes triggered by the immune system of the host following infection with P. falciparum. Coenzyme Q10 (CoQ10) is an obligatory cofactor in the electron transport chain and a potent antioxidant which has been identified as a modulator of gene expression, inflammation and apoptosis. However, the modulatory effects of CoQ10 during Plasmodium berghei ANKA (PbA) infection process and risk occurrence of experimental cerebral malaria (ECM) have not been determined. In the present study we sought to determine the role of CoQ10 in regulation of innate lymphoid cells during pathogenic immune responses of ECM. We observed significant increase in the percentage of Innate lymphoid class two (ILC2) in the spleens of Co-Q 10 supplemented PbA-infected mice; whereas the frequency of Innate lymphoid class one (ILC1) and Innate lymphoid class three (ILC3) were comparable in the spleens upon PbA infection. The results also show Splenic ILC2 from CoQ 10 mice are avid co-producer of IL-13 (T h 2 phenotype cytokine) during ECM. Our data collectively demonstrates that Coenzyme Q10 administration was very effective in stimulating ILC2, which are known to play a protective role during ECM.

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