Enriched environment reduces microglial activation after encephalitis induced by arbovirus Piry (Rhabdoviridae)

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Recent studies demonstrate that enriched environment increases the number of microglial cells that synthesizes neurotrofic factors, such as insulin growth factor I (IGF–I), inducing neuroprotection and neurogenesis in the murine dentate gyrus. However, branched microglial phenotype changes to aggressive amoeboid morphology with typical phagocytic activity during inflammatory response. This aggressive phenotype increases pro-inflammatory cytokines that seems to impair hippocampal-dependent tasks such as learning and memory. The aim of the present work is to study microglial activation along olfactory pathways and hippocampal formation after an acute encephalitis induced by intranasal infection with arbovirus Piry. Thirty infected albino Swiss female mice submitted either to enriched (EE, n = 15) or impoverished (IE, n = 15) environments by three months, were inoculated with 5 µL of normal or viral infected (10-7 v/v) brain homogenates, into each nostril and then perfused with aldehyde fixatives after 8 days post-infection. Brains were removed, cut and sections were immunoreacted for virus antigens or histochemically labeled for biotinylated Lycopersicum sculentum lectin, a specific marker for activated microglia. Dense immunolabeling for viral antigens and activated microglia were found along the olfactory pathways, CA1, CA2 and CA3 of ventral and dorsal hippocampus and these results confirm that viral encephalitis and inflammatory response follows anatomical tracts. Aggressive phenotypes were found in higher proportion in the IE group when compared to EE group, closer to the site of infection (bulb and olfactory tracts) and in the ventral hippocampus. Results suggest that enriched environment seems to reduce the activation of microglia after viral encephalitis. Cellular and molecular mechanisms associated with reduction of microglial activation remain to be investigated.

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