STUDY OF MICROGLIA PROLIFERATION IN RESPONSE TO IL-4

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Microglia are the myeloid cell population that reside in the parenchyma of the central nervous system (CNS). Their dynamic ability to respond to diverse microenvironmental and pathological signals allow microglia to undertake biochemical and morphological transformations, that lead to destroy the damaging insult and trigger tissue repair. This process of microglia activation includes the classically activated M1 state, characterized by a proinflammatory phenotype and the alternatively activated M2 state, known to be associated with an anti-inflammatory activity. Together with activation, an increased proliferation of activated microglia has also been observed in association with acute CNS injury and chronic degenerative diseases, suggesting that dysregulated microglia proliferation might play a detrimental role on neural cells in some neurodegenerative disorders such as Alzheimer's disease and amyloid lateral sclerosis. Thus, targeting microglia proliferation may offer a new therapeutic strategy to selectively modulate disease outcome. On the other hand, an increased number of microglia with pro-resolving phenotype might have beneficial effects on disease progression; however, the ability of microglia to proliferate in response to M2 signals in brain is not yet understood. Since it has been demonstrated that interleukin-4 (IL4) is able to induce the proliferation of peripheral macrophages, the aim of the present study is to investigate microglia proliferation in response to the local delivery of IL4; to this purpose we used intracerebroventricular (icv) injections of IL4. Results of this study will be discussed.