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Poster Presentation

The Role of Rho-Kinase (ROCK) in Microglia/Macrophage Polarization in Neuroinflammatory Diseases

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Abstract

Macrophage/microglia with heterogonous phenotype and function under physiological and pathological conditions are the main cell lineage involved in inducing immune responses in neuroinflammatory diseases which exhibit combined inflammatory and anti-inflammatory functions. An increase in the expression of iNOS triggers M1 phenotype that secrete high concentrations of inflammatory cytokines, while an elevation in the expression of Arg-1 triggers M2 phenotype which forms anti-inflammatory cytokines. Rho-kinase (ROCK) is a serine/threonine kinase and it expresses in both central nervous system and the periphery. ROCK inhibitors have been reported frequently to decrease the infiltration of leukocyte in some models of inflammation, including ischemic injury; the ROCK inhibitors changes M1 to M2 in neuroinflammatory diseases. Studies have found three possible mechanisms for M2 polarization by ROCK inhibitors, as follows: iNOS inhibition or Arg-1 enhancement, Change in multiple cytokines production lead to M2 activation likewise increased IL-10 proving M1 shift to M2 microglia. The present review aimed to investigate the role of Rho-kinase on M1 and M2 microglia and the effect of Rho-kinase inhibitors in shifting M1/M2 phenotypes which is significantly correlated with the neuroinflammatory diseases. The disease phases and severity might be involved in the microglial phenotype changes. Promising therapeutic purposes can be obtained by understanding the stage-specific switching of M1/M2 phenotypes.

Keywords: Rho-Kinase, Neuroinflammatory Diseases, Microglia, Macrophage

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