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Microglia Activation in Animal Model of Schizophrenia

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Abstract : Maternal immune activation (MIA) resulting from maternal viral infection during pregnancy is a known risk factor for schizophrenia. The neural mechanisms by which maternal infections increase the risk for schizophrenia remain unknown, although the prevailing hypothesis argues that an activation of the maternal immune system induces changes in the maternal-fetal environment that might interact with fetal brain development. It may lead to an activation of fetal microglia inducing long-lasting functional changes of these cells. Based on post-mortem analysis showing an increased number of activated microglial cells in patients with schizophrenia, it can be hypothesized that these cells contribute to disease pathogenesis and may actively be involved in gray matter loss observed in such patients. In the present study, we hypothesize that prenatal treatment with the inflammatory agent Poly(I:C) during embryogenesis at contributes to microglial activation in the offspring, which may, therefore, represent a contributing factor to the pathogenesis of schizophrenia and underlines the need for new pharmacological treatment options. Pregnant rats were treated with intraperitoneal injections a single dose of Poly(I:C) or saline on gestation day 17. Brains of control and Poly(I:C) offspring, were removed and into 20-µm-thick coronal sections were cut by using a Cryostat. Brain slices were fixed and immunostained with ba1 antibody. Subsequently, Iba1-immunoreactivity was detected using a secondary antibody, goat anti-rabbit. The sections were viewed and photographed under microscope. The immunohistochemical analysis revealed increases in microglia cell number in the prefrontal cortex, in offspring of poly(I:C) treated-rats as compared to the controls injected with NaCl. However, no significant differences were observed in microglia activation in the cerebellum among the groups. Prenatal immune challenge with Poly(I:C) was able to induce long-lasting changes in the offspring brains. This lead to a higher activation of microglia cells in the prefrontal cortex, a brain region critical for many higher brain functions, including working memory and cognitive flexibility. which might be implicated in possible changes in cortical neuropil architecture in schizophrenia. Further studies will be needed to clarify the association between microglial cells activation and schizophrenia-related behavioral alterations.

Keywords : Microglia, neuroinflammation, PolyI:C, schizophrenia
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