

# Study on Microglial Immune Response and Neuronal Regulatory Mechanisms Mediated by EMCV Infection

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## Abstract

Encephalomyocarditis virus (EMCV) infection can trigger central nervous system (CNS) immune inflammation and neuronal dysfunction, yet the underlying mechanism by which EMCV modulates microglial immune responses and subsequent neuronal functional regulation remains poorly elucidated. This study established a consistent viral titer infection paradigm with a standardized 150 TCID (converted to corresponding PFU via the classical conversion coefficient of 0.69) to conduct integrated in vivo and in vitro experiments. For the in vivo assay, 2–3-month-old mice were subjected to intracranial EMCV injection to induce CNS infection. We dynamically observed the activation state, phenotypic characteristics, and cytokine secretion profile of microglia post infection, and further explored the functional regulatory effect of activated microglia on cerebral neurons. Meanwhile, kidney tissues were collected as a peripheral organ reference to comparatively analyze the differential immune responses between central and peripheral systems upon EMCV infection. For the in vitro assay, RAW264.7 macrophages were infected with the same TCID viral titer to monitor cellular phenotypic changes and immune activation characteristics, which assisted in verifying the microglia-mediated immune response pattern induced by EMCV. By virtue of the unified TCID infection dosage bridging in vivo and in vitro models, this study focuses on clarifying the cascade mechanism of EMCV directly triggering microglial immune activation, releasing inflammatory cytokines, and further regulating neuronal functions. The findings are expected to provide new experimental evidence and mechanistic insights for understanding EMCV-induced neuroinflammation and CNS immune injury.

## Keywords

EMCV, Microglia, Neuroinflammation, Neuronal Dysfunction, Immune Activation