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The effect of a gallic acid derivative on encephalitogenic cells

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This study aimed to evaluate the effects of a synthetic gallic acid (GA) derivative in the central nervous system (CNS) autoimmunity, *i.e.* in experimental autoimmune encephalomyelitis (EAE), an animal model of multiple sclerosis. EAE was induced in DA rats by injection of autologous spinal cord homogenate, with a gallic acid derivative being applied subcutaneously (20 mg/kg, day 7-22 post-immunization). GA derivative ameliorated EAE. Cells from lymph nodes draining the site of immunization (DLNC), isolated in the inductive phase of the disease, and spinal cord immune cells (SCIC), isolated at the peak of disease, were exposed to GA derivative *in vitro*. Encephalitogenic cytokines, interferon (IFN)- γ and interleukin (IL)-17, were decreased in SCIC and DLNC under the influence of GA derivative. The proportion of IL-17-producing CD4⁺ T cells was reduced in SCIC (flow cytometry). Treatment of microglial BV2 cells with GA derivative led to inhibition of NO, IL-6, and tumor necrosis factor release. These results imply that the synthesized GA derivative is a potent immunomodulator, able to ameliorate EAE. Its effects on the CNS autoimmunity are related to the inhibition of encephalitogenic T cells and macrophage/microglia activity in our study.

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