Diverse role of macrophages in experimental autoimmune encephalomyelitis: a controversy

T. Shin[1], M. Ahn
[1]*Department of Veterinary Medicine, Jeju National University, South Korea*

Organ specific central nervous system (CNS) autoimmune diseases including experimental autoimmune encephalomyelitis (EAE), a model of human multiple sclerosis, has been known to be caused by autoreactive T cells and bystander macrophages. The inflammatory lesions are characterized by the infiltration of T cells and monocyte originated macrophages, followed by reactive microgliosis and astrogliosis. There is a general agreement that classically activated M1 macrophages play an important role in the initiation of CNS tissue damages. Recently alternatively activated M2 macrophages has been found in the EAE lesions with concurrent remission of paralysis in Lewis rats. The source of M2 macrophages in rat EAE lesions remains controversial whether they are originated from either monocytes or microglial cells, or both. As for the phenotypic switch of macrophage or microglia, in vivo EAE study shows that phenotypic switch occur in Iba-1 positive macrophages (monocyte and/or microglia) because either inducible nitric oxide synthase^{+}(M1 marker), arginase-1^{+} (M2 marker), or both –positive macrophages were found in EAE lesions. It is postulated that spontaneous recovery of EAE paralysis in rats is closely related with the relative prevalence of M2 milieu of inflammatory lesions, in which M2 macrophages secrete tissue protective molecules including heat shock protein and TGF beta. The control of macrophage phenotypes would be an alternative therapeutic strategy in organ specific autoimmune diseases. This research was supported by the Basic Science Research Program of the National Research Foundation of Korea (NRF), funded by the Ministry of Education (Grant number: NRF-2014R1A1A2055965).