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Microglia are the primary cells that exert immune function in the central nervous system (CNS), and they play an important role in the pathogenesis and progression of neuroinflammation-related diseases. Mesenchymal stem cells (MSCs) have been demonstrated to promote functional recovery in many neurological diseases. The mechanisms underlying this may be that MSCs can reduce inflammatory responses through various soluble factors. Among these factors, tumor necrosis factor- $\alpha$ -induced gene/protein 6 (TSG-6) is a key factor influencing MSCs immunomodulatory properties; however, the precise mechanisms underlying the anti-inflammatory effects are not fully understood. Here, we aim to investigate the potential effects of MSCs on neuroinflammation and to reveal the underlying mechanisms. First, we confirmed that administration of MSCs could inhibit the lipopolysaccharide (LPS)-induced neuroinflammatory responses in a mouse model. Then, we found that MSCs promoted M2 polarization and inhibited M1 polarization both in vivo and in vitro. Moreover, we demonstrated that the effect of MSCs on microglial polarization was dependent on TSG-6. This study demonstrated that MSCs promoted M2 polarization of microglia via TSG-6, thus conferring antineuroinflammatory effects  $^{1}$ .

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