



In a db/db mouse model of Type-2 diabetes mellitus (T2DM), the diabetic condition worsens long-term functional deficits after distal middle cerebral artery occlusion (tMCAO). These functional deficits are robustly correlated with loss of structural and functional integrity of white matter. Furthermore, T2DM impairs the proliferation of oligodendrocyte precursor cells (OPCs) and the generation of new myelinating oligodendrocytes. T2DM also promotes a shift of microglia/macrophage phenotype toward the pro-inflammatory modality. Our data suggest that therapeutic modulation of microglia/macrophages towards an oligodendrogenesis-enhancing phenotype may be a fruitful strategy to promote white matter repair and functional recovery after stroke in diabetic patients.