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Under transient middle cerebral artery occlusion (tMCAO) and oxygen-glucose deprivation/reoxygenation (OGD/Re) condition in vivo and in vitro, astrocyte-derived VEGFD activates the VEGF receptor 3 (VEGFR3) located on the membrane of astrocytes and microglia, respectively, disrupting astrocytic interleukin-3 (IL-3)/microglial IL-3 receptor α (IL-3R α) crosstalk, leading to the activation of A1 astrocytes and M1 microglia and elevated neuro-inflammatory cytokines. The disruption of this crosstalk between IL-3 and IL-3R α induces lipid metabolic reprogramming of microglia evidenced by upregulating CPT1A expression, a rate-limiting enzyme for the mitochondrial β -oxidation, causing accumulated lipid droplets (LDs), activated microglia and neuroinflammation, reduced glycerophospholipids levels, microglial phagocytosis dysfunction and necrosis, as well as neuronal cell death. The therapeutic interventions by VEGFR antagonist axitinib or exogenous IL-3 can reestablish the IL-3/IL-3R α crosstalk, reducing astrocytes and microglia activation, LDs accumulation and neuroinflammation, and restoring microglial phagocytotic function, ultimately contributing to brain recovery from tMCAO insult. See the article in pages 292–307.

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