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The role of mitochondrial dysfunction in NLRP3 inflammasome pathway during cerebral ischemia/reperfusion injury

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The nod-like receptor protein 3 (NLRP3) inflammasome is the crucial factor in mediating inflammatory responses after cerebral ischemia/reperfusion (I/R), but its pathogenesis has not yet come to a conclusion, and there is still no specific evidence to state the relationship between mitochondria and the NLRP3 inflammasome in ischemic stroke. We detected cellular localization of NLRP3 inflammasome pathway in transient middle cerebral artery occlusion (tMCAO) rat model at different time points and verified it in BV2 cells and transwell co-cultured system of BV2 and PC12 cells or BV2 and bEnd3 cells under oxygen-glucose deprivation and reoxygenation (OGD/R) conditions. Then we investigated the relationship of mitochondria and the NLRP3 inflammasome in different cells after OGD/R. The results showed that microglia was the main source of activated NLRP3 inflammasome at early stage after cerebral ischemia-reperfusion onset, and then the NLRP3 in microglia was transferred to neurons and microvascular endothelial cells over time, and mainly gathered in neurons at late stage. Furthermore, mitochondrial dysfunction was the activator of NLRP3 inflammasome in microglia, but in neurons, mitochondrial dysfunction was observed after the activation of NLRP3 inflammasome under OGD/R conditions. Our findings may provide a novel insight into the specific expression of NLRP3 inflammasome in cerebral ischemia reperfusion injury at different stages and the role of mitochondrial dysfunction in the activation of NLRP3 inflammasome pathway

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