Telmisartan inhibits the NLRP3 inflammasome by activating the PI3K pathway in neural stem cells injured by oxygen-glucose deprivation (OGD)

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- Angiotensin II receptor blockers (ARBs) have been shown to exert neuroprotective effects by suppressing inflammatory and apoptotic responses.
- Effects of the telmisartan on the NLRP3 (nod-like receptor – NLR family pyrin domain containing 3) inflammasome induced by OGD in neural stem cells (NSCs) were investigated in this study.
- OGD reduced the viability of NSCs in a time-dependent manner whereas treatment with telmisartan increased the viability and proliferation of OGD-injured NSCs.
- Telmisartan promoted the expression of survival-related proteins and mRNA while inhibiting the expression of death-related proteins induced by OGD.

Telmisartan attenuated OGD-dependent activation of the NLRP3 inflammasome and its related signaling proteins by triggering the PI3K pathway, thereby contributing to neuroprotection.