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Protective effects of a radical scavenger edaravone on oligodendrocyte precursor cells against oxidative stress

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Highlights:

- Edaravone protected OPCs in a mouse model of prolonged cerebral hypoperfusion.
- Edaravone did not induce cell death in OPC culture
- H₂O₂ exposure or starvation stress caused ROS accumulation in OPCs
- Edaravone protected OPCs against oxidative stress in vitro

Abstracts:

Oligodendrocyte precursor cells (OPCs) play critical roles in maintaining the number of oligodendrocytes in white matter. Previously, we have shown that oxidative stress dampens oligodendrocyte regeneration after white matter damage, while a clinically proven radical scavenger, edaravone, supports oligodendrocyte repopulation. However, it is not known how edaravone exerts this beneficial effect against oxidative stress. Using in vivo and in vitro experiments, we have examined whether edaravone exhibits direct OPC-protective effects. For in vivo experiments, prolonged cerebral hypoperfusion was induced by bilateral common carotid artery stenosis in mice. OPC damage was observed on day 14 after the onset of cerebral hypoperfusion, and edaravone was demonstrated to decrease OPC death in cerebral

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