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# New antiparkinsonian agent Diol restores cognitive function, enhances neurotrophic function, and diminishes neuroinflammation in a MPTP-induced model of Parkinson's disease in mice

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*Motivation and Aim*: Experimental therapy for cognitive deficits in neurodegenerative disorders including Parkinson's disease (PD) is area of high priority in current research. Recently synthesized monoterpenoid (1R,2R,6S)-3-methyl-6-(prop-1-en-2-yl)cyclohex-3-ene-1,2-diol 1 (Diol) alleviates motor manifestations of PD in animal models [1]. This study was aimed at evaluating the effects of Diol on cognitive deficits in mice with MPTP-induced PD-like pathology and the related mechanisms.

*Methods and Algorithms*: C57BL6 mice were injected with a single dose of MPTP (30 mg/kg) or saline intraperitoneally; Diol was given orally one time (20 mg/kg) on the next day of the experiment or chronically (20 mg/kg, daily, 31 days). Behavioral testing started 5 days after the first Diol administration. Biosamples for immunohistochemical analysis of the brain were taken 12 days after the first Diol administration.

*Results*: MPTP reduced learning and accelerated extinction of the conditioned response of passive avoidance that were associated with the increased microglia activation in the frontal cortex and hippocampus while the expression of neurotrophic factor BDNF was significantly decreased in the frontal cortex, hippocampus, and substantia nigra. Both types of Diol treatment completely reversed cognitive deficits in MPTP-treated mice. Moreover, Diol prevented neuroinflammatory response in the frontal cortex and hippocampus. Chronic Diol treatment restored the BDNF levels in the hippocampus.

*Conclusion*: The beneficial effect of Diol on MPTP-induced cognitive deficits, neuroinflammatory response, and neurotrophic function in the brain has been demonstrated in mice.

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