# Hypoxia Preconditioning Attenuates the Severity of Seizures by Regulating the of BDNF and Its Signaling Pathway

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## Abstract

Hypoxia preconditioning (HPC) as an endogenous mechanism can resist hypoxia/ischemia injure and exhibit protective efforts on neurological function. It can regulates the expression of protective molecules by modulating brain-derive neurotrophic factor (BDNF). And BDNF has been implicated in the pathophysiology of epilepsy, However, the role of BDNF in epilepsy is largely unclear. Therefore, the study focused on the mechanism by which HPC attenuates the severity of seizures though regulating the role BDNF and its signaling pathway. Initially, the HPC model was established by hypoxia stimulations on ICR mice. And the model of epilepsy induced by pentylenetetrazole. The possible anticonvulsant and neuroprotective effects of HPC were compared by video monitoring of behavioral seizure activity (frequency, delay) and Immunofluorescence to examine changes in the morphology of hippocampal pyramidal neurons. We found that epilepsy-like behaviors induced by PTZ were ameliorated when HPC dealed. Then, BDNF expression in hippocampus was significantly decreased in epilepsy mice. Subsequently, down regulation of BDNF activated BDNF/TrkB/Akt signaling. However, after hypoxia preconditioning treatment, the situation was reversed and ultimately slowed down the generalized tonic-clonic seizure rate. Thus, we suggest that HPC may upregulate BDNF and then activate BDNF/TrkB/Akt signaling and reduce the oxidative stress reaction to improve severity of serious. And our findings indicate that HPC treatment has a protective effect on epilepsy via the BDNF/TrkB/Akt pathway. This may provide theoretical guidance for the clinical treatment of cognitive dysfunction caused by epilepsy disease.

## Keywords

HPC, BDNF, Epilepsy