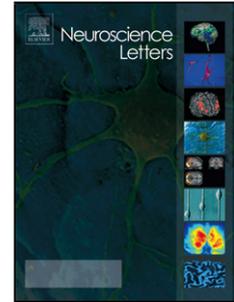


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## Overexpression of adiponectin alleviates intracerebral hemorrhage-induced brain injury in rats via suppression of oxidative stress

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### Highlights

- Oxidative stress have an important function in intracerebral hemorrhage(ICH).
- Adiponectin (APN) diminished ICH-induced oxidative stress injury via increasing the activities of SOD, GSH and the ratio of GSSG/GSH , and reducing the content of MDA.
- APN overexpression protectes against disruption of the blood-brain barrier, improved neurological functions.
- APN exerts its neuroprotection largely via inhibiting NOX2-mediated oxidative brain injury in ICH-induced stroke.
- APN may provide a new therapeutic strategy for ICH.

### ABSTRACT

Oxidative stress and blood-brain barrier (BBB) dysfunction contribute to brain injury after intracerebral hemorrhage (ICH). Adiponectin (APN) inhibits oxidative stress in the CNS, but the role of APN in ICH is not clear. Thus, we elucidated the possible neuroprotective effect of APN in ICH-induced brain injury in rats and investigated the neuroprotective mechanisms. A lentivirus-carrying APN gene was injected into rats 14 days before ICH induced via intracerebral injection of autologous blood. The effects of lentiviral overexpression of APN on brain injury were evaluated 24 h after ICH. Superoxide dismutase (SOD), glutathione (GSH), and the ratio of oxidized glutathione to reduced glutathione (GSSG/GSH) and malondialdehyde (MDA) were measured. Oxidative stress-related proteins were measured by Western blot and qRT-PCR. APN overexpression improved neurological function, reduced brain edema, preserved the BBB and increased the expression of APN and decreased the expression of NADPH oxidase-2 (NOX 2) compared with null vector controls ( $p < 0.01$ ). SOD, GSH, and GSSG/GSH increased, and MDA was reduced. Furthermore, tetrabromocinnamic acid (TBCA, a NADPH oxidase activator)

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