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Hydrogen-rich water attenuates amyloid β-induced cytotoxicity through upregulation of Sirt1-FoxO3a by stimulation of AMP-activated protein kinase in SK-N-MC cells

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Abstract

Amyloid β (Aβ) peptides are identified in cause of neurodegenerative diseases such as Alzheimer's disease (AD). Previous evidence suggests Aβ-induced neurotoxicity is linked to the stimulation of reactive oxygen species (ROS) production. The accumulation of Aβ-induced ROS leads to increased mitochondrial dysfunction and triggers apoptotic cell death. This suggests antioxidant therapies may be beneficial for preventing ROS-related diseases such as AD. Recently, hydrogen-rich water (HRW) has been proven effective in treating oxidative stress-induced disorders because of its ROS-scavenging abilities. However, the precise molecular mechanisms whereby HRW prevents neuronal death are still unclear. In the present study, we evaluated the putative pathways by which HRW protects against Aβ-induced cytotoxicity. Our results indicated that HRW directly counteracts oxidative damage by neutralizing excessive ROS, leading to the alleviation of Aβ-induced cell death. In addition, HRW also stimulated AMP-activated protein kinase (AMPK) in a sirtuin 1 (Sirt1)-dependent pathway, which upregulates forkhead box protein O3a (FoxO3a) downstream antioxidant response and diminishes Aβ-induced mitochondrial potential loss and oxidative stress. Taken together, our findings suggest that HRW may have potential therapeutic value to inhibit Aβ-induced neurotoxicity.

Keywords: AMP-activated protein kinase; Amyloid β; Forkhead box protein O3a; Hydrogen-rich water; Sirtuin 1.

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