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Protective effects of kinetin against aluminum chloride and D-galactose induced cognitive impairment and oxidative damage in mouse

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Highlights

- Kinetin improved memory and spatial learning abilities, and attenuated brain histopathologic changes
- Kinetin inhibited Al level in cortex and hippocampus, depressed AChE activity and restored ACh content
- Kinetin elevated activities of anti-oxidative enzymes and increased the content of HO-1
- Kinetin inhibited oxidative damage
- Kinetin inhibited the expressions of APP, β-secretase, γ-secretase and $A\beta_{1-42}$

Abstract: Increasing evidence indicates that aluminum exposure and oxidative stress play crucial roles in the initiation and development of Alzheimer's disease (AD). Aluminum chloride ($AlCl_3$) and D-galactose (D-gal) combined treatment of mice is considered as an easy and cheap way to obtain an animal model of AD. Kinetin is a plant cytokinin, which is also reported to exert neuro-protective effects in vivo and in vitro. Thus, in this study, neuro-protective effects of kinetin were investigated in an AD model of mice induced by $AlCl_3$ and D-gal. The Morris water maze (MWM) test was performed to directly evaluate neuro-protective effects of kinetin on the memory and spatial learning abilities, while the histopathological changes were examined by hematoxylin and eosin (H & E) staining method. To further investigate mechanisms involved, Al content in cortex and hippocampus was determined. In addition, related detection kits were used to determine