

ORAL PRESENTATION

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Beneficial effects of tri-lithium pyrroloquinoline quinonein on behaviors and pathology in a mouse model of Alzheimer's disease

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Alzheimer's disease (AD) is a complex disease with characteristic pathological hallmarks of senile plaques and neurofibrillary tangles, the occurrence and development of which are involved in multiple neurodegenerative processes. Conventional AD therapies target only at single disease-causing mechanisms. However, these have been mostly shown to be ineffective in recent clinical trials. Here we tested a novel strategy using a synthetic organic lithium salt, tri-lithium pyrroloquinoline quinonein (Li₃PQQ), to modulate multiple pathways involved in AD pathogenesis. The study of acute toxicity with mice showed Li₃PQQ with a very low toxicity (Li₃PQQ ID₅₀ 5g/kg weight vs. LiCl ID₅₀ 2.5g/kg weight). We showed that 8 weeks of daily Li₃PQQ administration in the APP/PS1 mice significantly improved the learning and memory function in the Morris water maze test and facilitated long-term potentiation. Li₃PQQ significantly reduced the area and numbers of amyloid plaque and phosphorylated tau levels in cortical areas and mechanistically, it increased activities of A β -binding alcohol dehydrogenase but decreased activities of glycogen synthase kinase-3 in the transgenic mice. Therefore, Li₃PQQ exhibits profound beneficial effects on cognitive impairment and pathological alterations in the AD mouse model. Our study demonstrates the effectiveness of a novel therapeutic strategy for AD through targeting at multiple disease-causing mechanisms.

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