Alpha-tocopherol quinone inhibits beta-amyloid aggregation and cytotoxicity, disaggregates preformed fibrils and decreases the production of reactive oxygen species, NO and inflammatory cytokines. S. Yang, W. Wang, T. Ling, Y. Feng, X. Du, X. Zhang, X. Sun, M. Zhao, D. Xue, Y. Yang, R. Liu, *Neurochem. Int.*, **57**, 914-922 (2010).

Alzheimer's disease (AD) is a complex, multifactorial neurodegenerative disease. The aggregation of beta-amyloid (Aβ) into extracellular fibrillar deposition is a pathological hallmark of AD. The Aβ aggregate-induced neurotoxicity, inflammatory reactions and oxidative stress are linked strongly to the etiology of AD. The currently available hitting-one-target drugs are insufficient for the treatment of AD. Therefore, finding multipotent agents able to modulate multiple targets simultaneously is attracting more attention. Previous studies indicated that vitamin E or its constituent such as α-tocopherol (α-T) was able to attenuate the effects of several pathogenetic factors in AD. However, ineffective or detrimental results were obtained from a number of clinical trials of vitamin E. Here, we showed that naturally synthesized RRR-α-tocopherol quinone (α-TQ), a main derivative of α-T, could inhibit Aβ42 fibril formation dose-dependently. Further investigations indicated that α-TQ could attenuate Aβ42-induced neurotoxicity toward SH-SY5Y neuroblastoma cells, disaggregate preformed fibrils and interfere with natural intracellular Aβ oligomer formation. Moreover, α-TQ could decrease the formation of reactive oxygen species (ROS) and NO, and modulate the production of cytokines by decreasing TNF-α and IL-1β and increasing IL-4 formation in microglia. Taken together, α-TQ targeting multiple pathogenetic factors deserves further investigation for prevention and treatment of AD.