P3. *Cordyceps Cicadae*-Fermented Product Cultured with Deep Ocean Water Improves Memory Deficit in Amyloid Beta 40 and Streptozotocin-Induced Alzheimer's Disease Rat via Inhibiting Inflammatory Factors Expression

Yen-Chung Wu and ◦Chun-Lin Lee

(Department of Life Science, National Taitung University)

1.Introduction

Alzheimer's disease (AD) is a common neurodegenerative disease characterized by continuous accumulation of β-amyloid (Aβ) in the brain, resulting in neuron damage, formation of senile plaque and neurofibrillary tangles, as well as memory and cognitive dysfunction.

Deep ocean water (DOW) with rich inorganic salts and minerals can be used as a source of mineral supplements for the human body. In the past, it was discovered that the imbalance of mineral elements in the body was thought to cause diseases. DOW rich with magnesium and calcium ions was proven to regulate lipid metabolism and improve liver damage.

Cordyceps cicadae NTTU 868, a functional fungi, can produce higher anti-oxidant and anti-inflammatory compounds including adenosine, polysaccharide and N(6)-(2-Hydroxyethyl) adenosine. Using DOW as culture water can increase the functional compounds and functional effect of *C. cicadae* NTTU 868.

2.Materials and Methods

This study used *C. cicadae* NTTU 868 as an experimental strain to culture with ultrapure water and deep ocean water for producing UPW-CC and DOW-CC, respectively. We further compared and investigated the effects on and mechanisms related to improving memory deficit and repressing risk factor expressions in a Aβ40 and streptozotocin (STZ) -induced Alzheimer's disease rat model.

3.Results

The results indicated that DOW-CC has more effect on the improvement of memory deficit than UPW-CC. Furthermore, the decreased magnesium levels in hippocampus and cortex were recovered as a result of DOW-CC but not UPW-CC. Daily feeding DOW-CC effectively reduced the accumulation of Aβ40 in the brain, and further reduced the expression of AD-related risk factors BACE. Furthermore, DOW-CC showed increased preventative action against Aβ40-induced inflammatory response than UPW-CC via repression of microglial cell activation factor sRAGE, and proinflammatory factor expression (TNF-α, IL-1β, IL-6).