**Edible Bracken Extract and Its Active Compounds Reduce Beta-Amyloid Production *In Vitro* and Ameliorate Scopolamine-Induced Memory Impairment *In Vivo***

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**Background and aims.** Alzheimer’s disease (AD) is caused by the death of nerve cells due to excessive accumulation of beta-amyloid (Aβ) in brain neurons. Therefore, inhibiting Aβ production could be a potential strategy to prevent AD. Previously, an extract from the rhizome of *Dryopteris crassirhizoma* significantly inhibited Aβ production, but its application was limited due to cytotoxicity. In this study, we investigated the ethanol extract of an edible bracken, *Pteridium aquilinum var. latiusculum* (PAE), for its effects on Aβ production and memory improvement.

**Methods.** Chinese hamster ovary cells stably transfected with Amyloid Precursor Protein (APP-CHO) were used for *in vitro* studies, and scopolamine-administered mice were used for *in vivo* experiments. The effects of PAE on Aβ production were assessed by measuring the levels of sAPPβ and β-secretase in APP-CHO cells via western blot analysis. The active constituents of PAE were isolated and identified by NMR data. The passive avoidance and Morris water maze test were performed in scopolamine-administered mice. The levels of acetylcholine (ACh), acetylcholine esterase (AChE), Brain-derived neurotrophic factor (BDNF), Phosphor c-AMP response element-binding protein (p-CREB), and Aβ were determined by ELISA and immunohistochemistry.

**Results.** PAE significantly reduced Aβ production dose-dependently. Eight compounds were isolated, and astragalin (**2**), tiliroside (**3**), and 5-*O*-caffeoylshikimic acid (**5**) were the most active. Moreover, PAE ameliorated scopolamine-induced memory impairment in Morris water maze test in accordance with reduced Aβ deposition. PAE treatment significantly increased ACh levels and decreased AChE activity, along with upregulated BDNF and p-CREB.

**Conclusion/Discussion.** PAE and its active constituents significantly inhibited Aβ production and ameliorated scopolamine-induced memory impairment in mice. These findings suggest that PAE and its active constituents have potential as a safe and effective preventive agent for AD.

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**References:**

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