Akebia quinata Decaisne aqueous extract acts as a novel anti-fatigue agent in mice exposed to chronic restraint stress

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Background: Akebia quinata Decaisne extract (AQE) (Lardizabalaceae) is used in traditional herbal medicine for stress-fatigue-related depression, improved fatigue and mental relaxation.

Aim: To clarify the effects of AQE on stress-induced fatigue, we investigated the neuroprotective pharmacological effects of A. quinata Decaisne in mice exposed to chronic restraint stress.

Materials & Methods: Seven-week old C57BL/6 mice chronically stressed by immobilization for 3 hours daily for 15 days and non-stressed control mice underwent daily oral administration of AQE or distilled water. The open field, sucrose preference and forced swimming behavioral tests were carried out once weekly and immuno-histochemical analysis of NeuN, brain-derived neurotrophic factor (BDNF) and phosphorylated cAMP response element-binding (CREB) protein and BDNF receptor tropomyosin receptor kinase B (TrkB) in striatum and hippocampus were carried out at the end of the experimental period. Brain levels of serotonin, adrenaline and noradrenaline and serum level of corticosterone were measured.

Results: Behavioral tests showed that treatment with AQE improved all lethargic behaviors examined. AQE significantly attenuated the elevated levels of adrenaline, noradrenaline and serotonin in the brain and serum levels of corticosterone, alanine transaminase and aspartate transaminase. Histopathological analysis showed that AQE reduced liver injury and lateral ventricle size in restraint-stress mice through inhibition of neuronal cell death. Immuno-histochemical analysis showed increased phosphorylation of CREB and expression of BDNF and its receptor TrkB in striatum and hippocampus. Chlorogenic acid, isochlorogenic acid A, and isochlorogenic acid C were identified as the primary components of AQE. All three increased expression of BDNF in SH-SY5Y cells and PC12 cells with H2O2-induced neuronal cell damage.

Conclusions: AQE may have a neuroprotective effect and ameliorate the effects of stress-fatigue-associated brain damage through mechanisms involving regulation of BDNF-TrkB signaling.

Biography
Sun Haeng Park has completed her graduation from Korea Institute of Oriental Medicine (KIOM), South Korea. She had many researches on ethnopharmacological relevance, stress-induced fatigue and neuroprotective agents.

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