Artemisia argyi

Artemisia argyi Lev. et Vant., is a traditional Chinese herb, that has diverse therapeutic properties and is used to treat patients with skin diseases and oral ulcers.

Eupatilin is a major flavonoid from Artemisia plants such as Artemisia princeps and Artemisia argyi which has been reported to possess various beneficial biological effects including anti-inflammation, anti-tumor, anti-cancer, anti-allergy, and anti-oxidation activity.

Aging is accompanied by functional loss of many cellular pathways, creating an increased risk of many aging complications (ARC). Aging causes stem cell exhaustion with a concomitant increase in cellular dysfunction.

Senotherapy has been growing rapidly to promote healthy aging and as an intervention for aging complications. The research focused on screening the senomorphic properties of Artemisia argyi, as an emerging strategy for longevity, and prevention or treatment of aging complications. Ho et al. aimed to find the clinical efficacy of daily consumption of Artemisia argyi water extract (AAW) on aging. In vitro 0.1µM Doxorubicin induced senescent human adipose-derived mesenchymal stem cells was treated with different concentrations of AAW to show its anti-aging effect. 15 months old SHR rats (n=6) were treated with 7.9 mg/ml AAW for 4 weeks and the anti-aging effect was evaluated. In vitro study showed the protective effect of AAW in telomere shortening and helps in maintaining a balance in the expression of anti-aging protein Klotho and TERT. AAW effectively reduced mitochondrial superoxide and also provided a protective shield against senescence markers like over-expression of p21 and formation of double strand breaks, which is known to cause premature aging. Moreover, animal studies indicated that AAW promoted the expression of Klotho in naturally aging rats. In addition, AAW successfully restored the decline cardiac function and improved the grip strength and memory of aging rat. These findings showed that therapeutic targeting of senescent stem cells by AAW restored stem cell homeostasis and improves overall health [1)].

Wu et al. aimed to investigate the neuroprotective effects of A. argyi in promoting the TRPML1-mediated autophagy/mitophagy-enhancing effect. They used 1-methyl-4-phenyl-pyridinium (MPP+)-induced PD model established in an SH-SY5Y human neuroblastoma cell line as well as in a 1-methyl-4-phenyl-1,2,3,6-tetrahydro-pyridine (MPTP)-induced PD model in C57BL/6 J mice. MTT assay was conducted to measure the cell viability and further MitoSoX and DCFDA assay were used to measure the ROS. Western blot analysis was used to access levels of TRPML1, p-DRP1 (ser616), p-AKT, PI3K, and β-catenin, Additionally, IF and IHC analysis to investigate the expression of TRPML1, LC3B, β-catenin, TH+, α-synuclein. Mitotracker stain was used to check mitophagy levels and a lysosomal intracellular activity kit was used to measure the lysosomal dysfunction. Behavioral studies were conducted by rotarod and grip strength experiments to check motor functions.

In this in vitro study, A. argyi rescued the MPP+-induced loss of cell viability and reduced the accumulation of mitochondrial and total reactive oxygen species (ROS). Subsequently, it increased the expression of TRPML1 protein, thereby inducing autophagy, which facilitated the clearance of toxic accumulation of α-synuclein. Furthermore, A. argyi played a neuroprotective role by activating the PI3K/AKT/β-catenin cell survival pathway. MPP+-mediated mitochondrial damage was overcome by upregulation of mitophagy and downregulation of the mitochondrial fission regulator p-DRP1 (ser616) in SH-SY5Y cells. In the in vivo study, A. argyi ameliorated impaired motor function and rescued TH+ neurons in the SNpc region. Similar to the results of the in vitro study, TRPML1, LC3B,

and β-catenin expression was enhanced in the SNpc region in the A. argyi-treated mice brain.

These results first demonstrate that A. argyi can exert neuroprotective effects by stimulating TRPML1 and rescuing neuronal cells by boosting autophagy/mitophagy and upregulating a survival pathway, suggesting that A. argyi can further be exploited to slow the progression of PD. [2].

Eupatilin is a major flavonoid from Artemisia plants such as Artemisia princeps and Artemisia argyi which has been reported to possess various beneficial biological effects including anti-inflammation, anti-tumor, anti-cancer, anti-allergy, and anti-oxidation activity. Complete blockade of RANK-dependent osteoclastogenesis was accomplished upon stimulation prior to the receptor activator of nuclear factor κB (RANK)-ligand (RANKL) treatment or post-stimulation of bone marrow macrophages (BMCs) in the presence of RANKL with eupatilin. This blockade was accompanied by inhibition of rapid phosphorylation of Akt, GSK3β, ERK, and IκB as well as downregulation of c-Fos and NFATc1 at protein, suggesting that transcriptional suppression is a key mechanism for anti-osteoclastogenesis. Transient reporter assays or gain of function assays confirmed that eupatilin was a potent transcriptional inhibitor in osteoclasts (OC). Surprisingly, when mature osteoclasts were cultured on bone scaffolds in the presence of eupatilin, bone resorption activity was also completely blocked by dismantling the actin rings, suggesting that another major acting site of eupatilin is a cytoskeletal rearrangement. The eupatilin-treated mature osteoclasts revealed a shrunken cytoplasm and accumulation of multi-nuclei, eventually becoming fibroblast-like cells. No apoptosis occurred. Inhibition of phosphorylation of cofilin by eupatilin suggests that actin may play an important role in the morphological change of multinucleated cells (MNCs). Human OC similarly responded to eupatilin. However, eupatilin has no effects on osteoblast differentiation and shows cytotoxicity on osteoblast in the concentration of 50 μM. When eupatilin was administered to LPS-induced osteoporotic mice after the manifestation of osteoporosis, it prevented bone loss. Ovariectomized (OVX) mice remarkably exhibited bone protection effects. Taken together, eupatilin is an effective versatile therapeutic intervention for osteoporosis via; 1) transcriptional suppression of c-Fos and NFATc1 of differentiating OC and 2) inhibition of actin rearrangement of pathogenic MNCs [3]

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2)
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3)
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