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Phytopharmacological Communications

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Editor's Choice

Editor's Selection of the Important Research Investigations in the Field of Phytopharmacological Communications from Around the World

Editorial Staff

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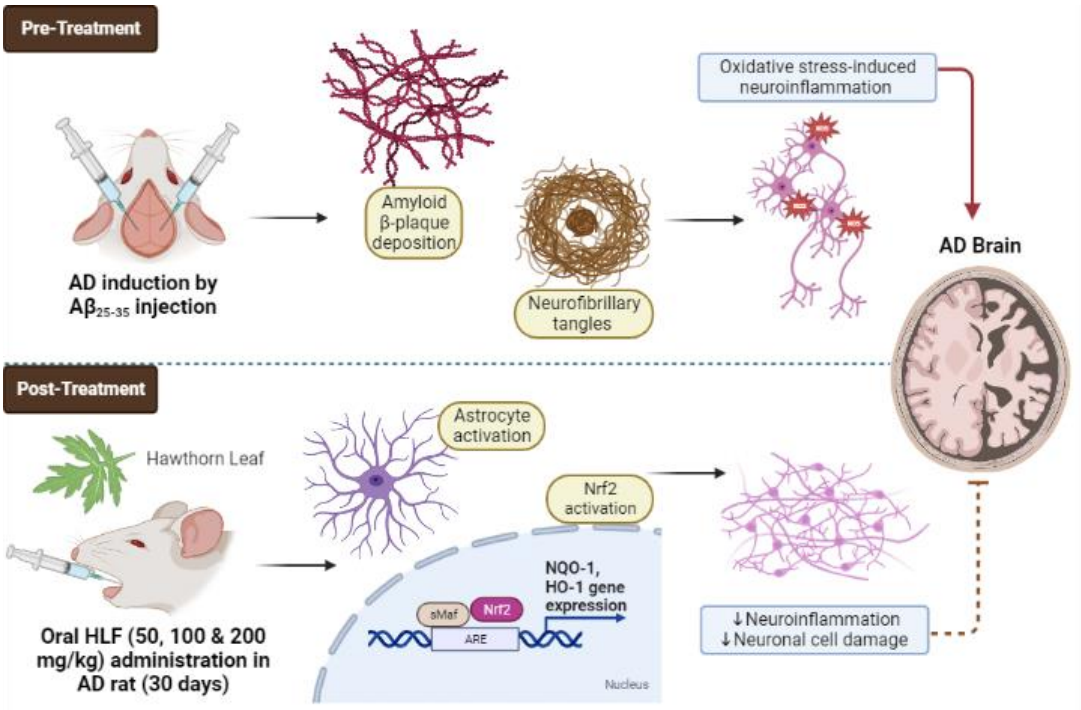
Neuroprotection in Aβ25-35 -induced Alzheimer's disease model by Hawthorn leaf flavonoids (doi.org/10.55627/ppc.002.002.0166).

It has been observed that increased accumulation of Aβ peptides in the brain triggers endogenous oxidative stress, neuroinflammation, and memory dysfunction in Alzheimer's Disease (AD). *Crataegus pinnatifida* Bge. Commonly referred to as the Hawthorn leaf is a well-known traditional Chinese medicine. The plant is used for conditions such as hyperlipidemia, heart palpitations, forgetfulness, and tinnitus in mainstream medicinal practice.

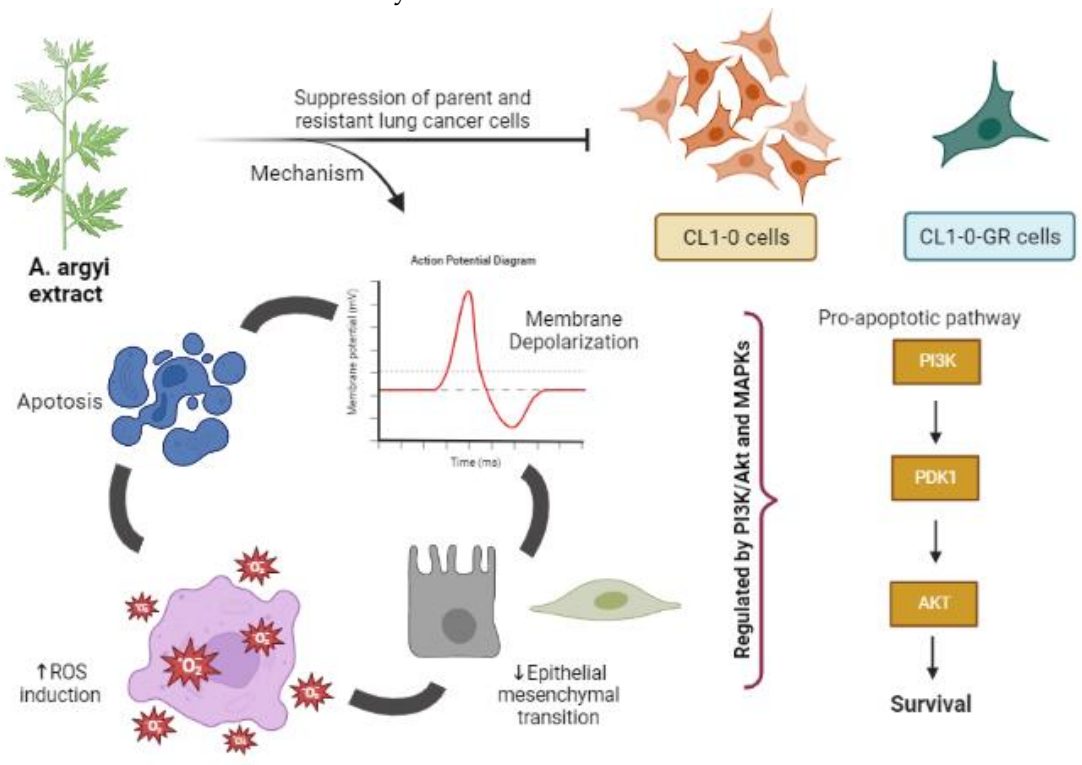


The most important bioactive components consist of Hawthorn Leaf Flavonoids (HLF). Xu and associates showed that in a rat model given Aβ25-35 (bilateral hippocampus injection), the neuroprotective effects of HLF were significant. Oral doses of 50, 100, and 200 mg/kg for a time

period of 30 days of HLF demonstrated significant attenuation of the damage to neurons alongside marked inhibition in memory deficits. The enzyme activities of superoxide dismutase and catalase and glutathione content were enhanced. In contrast, HLF decreased the malondialdehyde content in the $A\beta_{25-35}$ rat AD model and suppressed astrocytes' activation, ameliorating neuroinflammation. HLF upregulated NQO-1, Nrf-2, and HO-1 protein expressions. These results demonstrated the potential of HLF as a potential AD therapeutic. Phytother Res (2022) DOI: 10.1002/ptr.7690.

Artemisia argyi extract uses PI3K/MAPK signaling to induce apoptosis in human gemcitabine-resistant lung cancer cells (doi.org/10.55627/ppc.002.002.0169).

Artemisia argyi H. Lév. & Vaniot (Asteraceae), also called "Chinese mugwort," is frequently used as a herbal medicine in China, Japan, Korea, and eastern parts of Russia. It is termed "ai ye" and "Gaiyou" in China and Japan, respectively. It was an ancient Chinese practice that before Tomb-sweeping day, the leaves of *A. argyi* be consumed. Renal and Hepatic pathologies and conditions such as hepatitis, asthma, sinusitis, irregular menstrual cycles, and inflammatory syndromes were all treated by this plant. Although *A. argyi* extract (AAE) has demonstrated antineoplastic potential, the mechanisms by which this is achieved in lung cancer are yet to be known. Su *et al*. used CL1-0 parent and gemcitabine-resistant (CL1-0-GR) lung cancer cells to investigate the underlying mechanisms of AAE's anti-tumor effects. **In** CL1-0 and CL1-0-GR cells, a significantly decreased cell viability and induction of apoptosis were observed after AAE treatment. AAE-induced apoptosis was found to be regulated via the PI3K/AKT, and MAPK signaling pathways. AAE also prevented CL1-0 and CL1-0-GR cancer cell invasion, migration, colony formation, and spheroid formation.

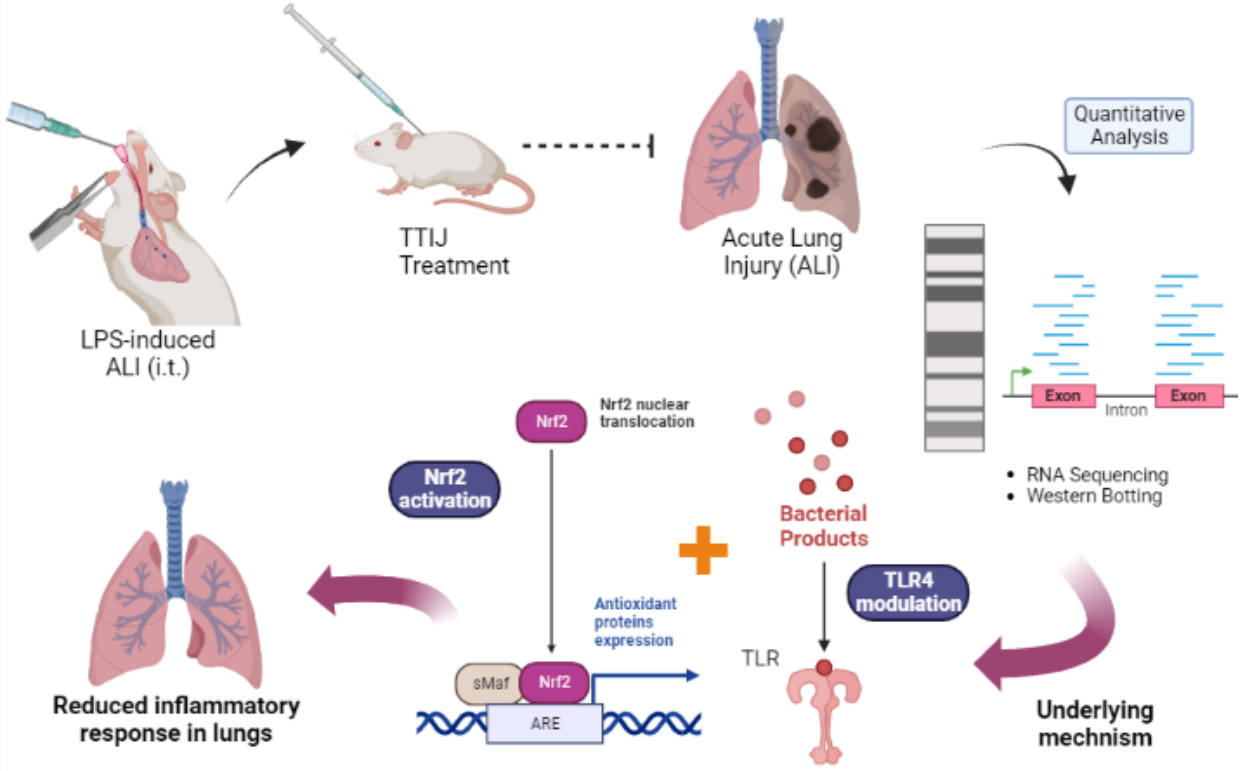


In addition, AAE acted cooperatively with commercial chemotherapy drugs to enhance tumor spheroid shrinkage. Their study provides the first evidence that *A. argyi* treatment suppresses both parent and gemcitabine-resistant lung cancer cells by inducing ROS, mitochondrial

membrane depolarization, and apoptosis. Their findings provide insights into the anti-cancer activity of *A. argyi* and suggest that the plant may serve as a chemotherapy adjuvant that potentiates the efficacy of chemotherapeutic agents. J Ethnopharmacol (2022) DOI: 10.1016/j.jep.2022.115658.

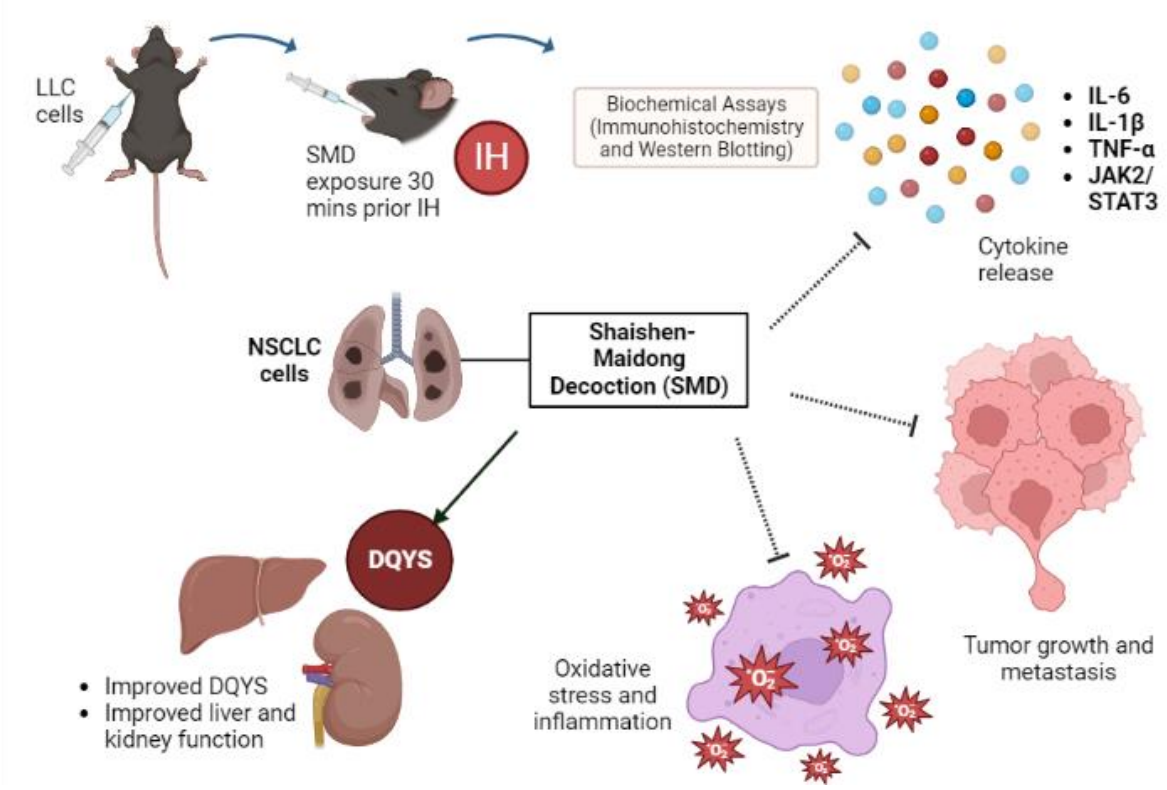
Inula japonica alleviates inflammation and oxidative stress in LPS-induced acute lung injury (doi.org/10.55627/ppc.002.002.0167).

Presenting as atelectasis and pulmonary edema, acute lung injury (ALI) is a life-threatening condition. Inula japonica Thunb is a Chinese herbal medicine traditionally used for treating lung diseases. Nonetheless, the underlying mechanisms of the terpenoids present in the plant remain to be elucidated. In mouse models with lipopolysaccharide (LPS) induced ALI, the protective effects of total terpenoids of Inula japonica (TTIJ) were studied. ALI was developed in the mice by injecting LPS into the trachea. The underlying mechanism of TTIJ was determined using bioinformatic and RNA-seq technology. For oxidative stress and inflammation, the effects of TTIJ were verified using real-time qPCR and western blotting. In the lungs, LPS-caused histopathological changes were markedly inhibited by TTIJ. Two major pathways were identified, nuclear factor-erythroid 2-related factor 2 (Nrf2) and Toll-like receptor 4 (TLR4), primarily responsible for the TTIJ protective effects. A drastically reduced inflammation and oxidative stress in LPS-induced ALI was seen through the modulation of anti-oxidative cytokines, glutathione (GSH) and superoxide dismutase (SOD), as well as tumor necrosis factor-α (TNF-α), interleukin-6 (IL-6), upon pretreatment with TTIJ. The inactivation of the MAPK/NF-κB signaling pathway reduced the phosphorylation of p65, p38, JNK, and ERK. TTIJ successfully suppressed cyclooxygenase-2 (COX-2) expression. As confirmed by the luciferase assay, several integral components of Nrf2 signaling pathways were elevated via Nrf2 receptor activation. These authors showed that TTIJ could serve as a potential therapeutic agent for ALI due to the anti-oxidant and anti-inflammatory properties. Phytomedicine (2022) DOI: 10.1016/j.phymed.2022.154377.



A Chinese decoction arrests lung cancer (doi.org/10.55627/ppc.002.002.0165).

A leading cause of cancer-associated deaths worldwide is lung cancer. During cancer growth, intermittent hypoxia (IH) is most commonly caused due to abnormal microcirculation and serves to promote the division and migration of cancer cells. Previous studies have established that exposure to IH is linked to Qi and Yin Syndrome (DQYS), where individuals suffering from advanced lung cancer have demonstrated deficiencies. It has been reported that an herbal remedy referred to as the Shashen-Maidong Decoction (SMD) can treat lung cancer by nourishing the body's "zheng qi" and resisting "xie qi". Currently, the same preparation is being used to address DQYS in clinical practice. Still, regarding IH, the benefits of the SMD are yet to be fully explained. In an attempt to address this gap, the underlying mechanism of action and subsequent effects of SMD were investigated by Zheng and associates in non-small cell lung cancer (NSCLC). Lewis Lung Cancer (LLC) cells were administered into the right axilla of C57 mice and afterward subjected to conditions of IH for 21 days (21%-5% O 2, 5 min/cycle, eight h/day). Thirty minutes before IH exposure, the mice were given SMD orally in three concentrations: (2.6, 5.2, or 10.4 g/kg/day). Hematoxylin-Eosin (HE) staining and Immunohistochemistry (IHC) techniques were used to gauge the proliferation and migration of tumor cells. Oxidative stress was measured through dihydroethidium (DHE) staining along with malondialdehyde (MDA) and superoxide dismutase (SOD) quantitation. Inflammatory markers IL-6, IL-1β, and TNF-α levels via IHC staining were determined, and western blotting was employed to detect the involvement of the IL-6/JAK2/STAT3 signaling pathway. LLC-injected mice given SMD exhibited markedly lowered tumor growth and hepatic metastasis. Furthermore, decreased VEGF, Ki67, MMP-2, and CD31 were seen.

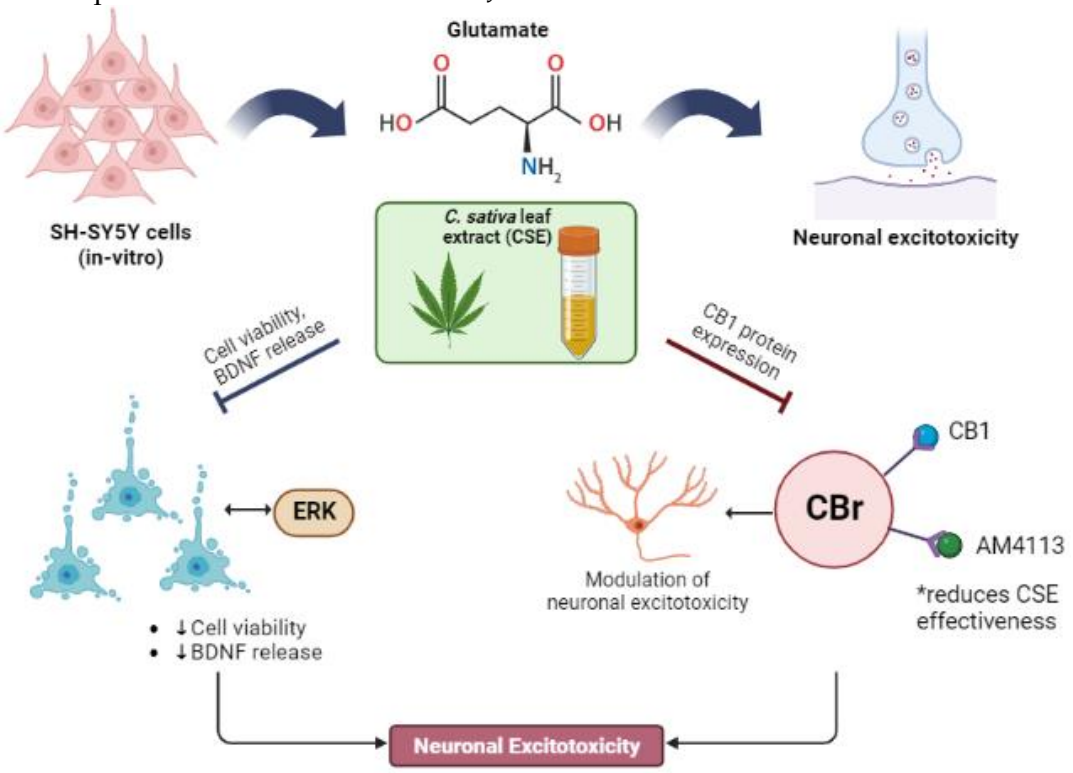


Moreover, elevated expression in tumor tissue of E-cadherin was observed. Reduction in NOX-2

expression, MDA levels, enhanced expression of SOD-2, and increased levels of SOD are evidence of the anti-oxidant potential of SMD. Diminished IL-6, IL-1β, and TNF-α levels, decreased IL-6 expression, and reduced JAK2 and STAT3 phosphorylation were also seen with SMD. Additionally, SMD treatment improved DQYS and liver and kidney function in LLC-bearing mice under IH conditions. Therefore, their investigation reasonably demonstrates that SMD treatment bears tumor-inhibiting potential under IH conditions in LLC-bearing mice. Inactivation of the IL-6/JAK2/STAT3 signaling pathway under IH conditions may be the underlying reason for the anti-oxidant and anti-inflammatory and the resultant antineoplastic effects of SMD. J Ethnopharmacol (2022) DOI: 10.1016/j.jep.2022.115654.

Cannabis sativa L. extract improves neuroprotection in a model of excitotoxicity (doi.org/10.55627/ppc.002.002.0168).

Borgonetti and colleagues investigated whether *Cannabis sativa* L. extract (CSE) and its main chemical components, β-caryophyllene, and cannabidiol, bear neuroprotective effects. An in vitro model of glutamate-induced neuronal excitotoxicity using SH-SY5Y cells was optimized. ERK levels, brain-derived neurotrophic factor release, glutamate-impaired cell viability, CB1 protein expression, and how CSE influenced them were studied.



The role of CB1 modulation was confirmed using AM4113, a CB1 antagonist. CSE, through ERK modulation, counteracted the changes in brain-derived neurotrophic factor level and significantly protected SH-SY5Y from glutamate-impaired cell viability. CSE entirely reversed CB1 receptor expression, and the use of AM4113 reduced the efficacy of CSE, suggesting the involvement of CB1 in neuro-excitotoxicity modulation. The investigation, thus, showed that CSE harbors neuroprotective effects. As a result, it can be proposed that the whole cannabis Phytocomplex could be a more effective strategy than its primary constituents alone, and suggested further investigations using more complex cellular and animal models. Fitoterapia (2022) DOI: 10.1016/j.fitote.2022.105315

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