POTENTIAL NEUROPROTECTIVE EFFECTS OF HERBAL COMPOUNDS
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ABSTRACT
Herbal bioactive compounds have been investigated to possess neuroprotective properties. They are involved in the modulation of different signaling pathways that may facilitate neuroprotection. In this brief review, some of the promising compounds and their potential neuroprotective effects have been reported. They can be potential sources of therapeutics for neurodegenerative disorders.

KEYWORDS: neuroprotective, herbal, bioactive compounds, neurodegenerative diseases

INTRODUCTION
In the past, there have been enormous efforts to prevent neurodegenerative diseases. However, there have been no available remedies for these kinds of diseases because they have delayed onset.\(^1\) Probably, the disease progression can be halted to greater degree if it can be alleviated at the initial stage.\(^1\) Such therapeutic remedies may rejuvenate neuronal activities that slow down or even stop the progression of neurodegenerative diseases. Herbal bioactive substances can be potential sources of therapeutics. They are known to have promising biological functions for the prevention of neurodegenerative disorders.

Bioactive compounds:
1. Kukoamine A from Cortex Lycii Radicis (bark of Lycium chinense or L. barbarum)\(^2\)
   Mechanism: anti-oxidative stress and this effect may be partly via blocking NMDARs in SH-SY5Y cells\(^2\)
   Effect: potential therapeutic interventions for brain injury\(^2\)
2. Ginkgetin from Ginkgo biloba L.\(^3\)
   Mechanism: decreasing the levels of intracellular reactive oxygen species (ROS) and maintaining mitochondrial membrane potential; dramatically inhibited cell apoptosis induced by MPP+ through the caspase-3 and Bcl2/Bax pathway; inhibiting the decrease of tyrosine hydroxylase expression in the substantia nigra and superoxide dismutase activity in the striatum; strongly chelates ferrous ion and thereby inhibits the increase of the intracellular labile iron pool through down-regulating L-ferritin and upregulating transferrin receptor 1\(^3\)
   Effect: improved sensorimotor coordination; may provide neuroprotective therapy for Parkinson’s disease (PD) and iron metabolism disorder related diseases\(^3\)
3. Eucommia ulmoides Oliv compounds\(^4\)
   Effect: amelioration of ubiquitin-proteasome system
   Potential treatment for Parkinson’s disease\(^4\)
4. Aqueous extract of Pimpinella anisum L. seeds\(^5\)
   Effect: habituation-related central action\(^5\)
   Caution: for patients with Alzheimer’s disease (AD)\(^5\)
5. Polygonum cuspidatum\(^6\)
   Effect: significant effects against PC12 cells injured by rotenone\(^6\)
6. Phellopterin from Citrus junos\(^7\)
   Mechanism: stimulates the phosphorylation of Extracellular signal-regulated kinase (ERK) and ERK-cAMP response element binding protein (CREB)\(^7\)
   Effect: regulation of long-term memory formation\(^7\)
7. [6]-gingerol and epigallocatechingallate from Tea⁹
   **Mechanism:** synergistically induced apoptosis and inhibits the proliferation of glioma cancer cells')(⁸
8. Furanocoumarins from stems of *Clausena lansium*²⁹
   **Effect:** Neuroprotection at a concentration of 10 μM³⁹
9. 3,7-dihydroxy-2,4,6-trimethoxy-phenanthrene from rhizomes of *Dioscorea nipponica*¹⁰
   **Mechanism:** strongly reduced NO levels with an IC₅₀ value of 19.56 μM in BV2 microglial cells. Also, it significantly increased neurite outgrowth in N2a cells.¹⁰
   **Effect:** anti-inflammatory and neuroprotective effects¹⁰
10. Extracts from *Bombycis excrementum* (BE)¹¹
    **Mechanism:** significantly ameliorated AβO-induced memory impairments and inhibited AβO-induced neuronal loss in cultured cells and the brains of mice. BE also significantly inhibited microgliosis and astrogliosis following intra-hippocampal AβO injections in mice. It significantly attenuated the release of nitric oxide from microglia and reduced AβO-induced S100-β cytokine release from activated astrocytes¹¹
    **Effect:** may be a candidate agent for the treatment of Alzheimer’s disease.¹¹
11. Bilobalide from *Ginkgo biloba* L¹²
    **Mechanism:** inhibition of pro-inflammatory mediator production and down-regulation of JNK1/2 and p38 MAPK activation¹²
    **Effect:** protection against cerebral ischemia and reperfusion injury¹²
12. Clerodanditerpenes from *Croton yanhuii*¹³
    **Mechanism:** enhanced NGF-mediated neurite outgrowth from PC12 cells¹³
    **Effect:** potentially useful for the medical treatment of Alzheimer’s disease¹³

**CONCLUSION**

The role of plant metabolites in the alleviation of neurodegeneration is a promising area of research. Many bioactive compounds from plants have already been isolated and identified to have neuro-protective attributes. Further studies are warranted for the development of these isolated compounds to develop new therapeutics to remedy initial progression of neurodegenerative diseases.

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