ALZHEIMER'S DISEASE AND TREATMENT
Phytopharmaceuticals in Neurodegenerative Disorders

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Abstract
Millions of individuals worldwide suffer from Neurodegenerative Diseases (NDs). Two of the most common neurodegenerative disorders, Parkinson’s Disease (PD) and Alzheimer’s Disease (AD), have a significant socioeconomic impact. People desire a cure for these disorders from the natural herals since several centuries. Scientific literature has reported many medicinal plants and their secondary metabolites with their ability to relieve the symptoms. Medicinal plants represent a largely unexploited reservoir of natural medicines which have potential cure for Alzheimer’s & Parkinson’s diseases. The structural diversity of their phytocompounds make these plants a valuable source of novel lead compounds. On the basis of traditional literature and up-to-date research, several new therapeutically active compounds have been identified from herbal extracts, which may be valuable in the treatment of cognitive disorders. This chapter focuses on illnesses such as AD, PD, and trinucleotide disorders of repletion, as well as the significance of natural phytoconstituents/extracts and their possible mechanisms of neuroprotection.

Introduction
Alzheimer’s Disease (AD) produces continuous and increasing cognitive decline, whereas Neurodegenerative Disorders (NDs) affect specific regions of the body, based on neuronal cells experiencing careful deterioration.

In the neurodegeneration of neurons, an important role is played by oxidative damage by highly reactive compounds. Different types of neurological disarrays influence advanced destruction of motor, sensory neurons, which can identify exterior objects as sensory information. [1]. Particularly in brain tissue sensitive to reactive oxygen species which damage cell, build up may lead to AD by lipid peroxidation.

Acetylcholine level , which is neurotransmitter involved in memory and learning [2,3] is also affected. Most conventional dementia causes degradation of neurons apart from AD, PD, and Huntington’s Disease(HD), [4,5]. Dementia is typified by chronic progressive mental disorder while AD distinctly distresses memory, intelligent, knowledge, design, and linguistic. Abundant in older population effecting 5% of people over 65 years and 50% of people above 85 years [6]. AD broadly affects all the ageing persons in world, characterized by universal cerebral malfunction, memory damage, change in behaviour personality, and performance injuries of activities of daily activities that leads the patient bedridden, incontinent, and reliant on supervisory care [7]. This devastating disease represents 22 million people globally developing in 3 years [8]. 20% of the occurrence is of Vascular dementia. It is estimated to increase more than 0.8 billion by 2040 [9]. Dementia affects millions of individuals worldwide, with two-thirds of them living in third-world nations.

Etiology
Alzheimer’s Disease (AD)
Insulin resistance is linked to the development of Alzheimer’s disease via promoting tau phosphorylation. [10], secretion of APPsα, which decreases the Amyloid beta intracellular pool [11]. Apart genomic, environmental abnormalities also cause AD. It is not understood by modified βAPP metabolism and amyloid β accumulation in sporic cases, but include age-dependant reasons in, compromised energy metabolism, oxidative stress, and disturbed ion homeostasis in cells.

Little education, history of AD trauma, with feeding of high-fat/ high-caloric and a lethargy life improve the risk of incidence of disease [12]. Ca²⁺ dysregulation, oxidative damage, and mitochondrial impairment, in Endoplasmic Reticulum (ER), apoptosis and ultimately death of neurons can be persuaded by

beta amyloid [13,14]. AD pathogenesis of neurotoxic forms of amyloid beta from APP appear to be a critical step in the development of β and γ secretase inhibitors and blockers [15]. In the treatment for AD by drugs acetycholinesterase inhibitors (AChE), which enhances the intensity of acetylcholine in the brain can be used. Aβ seems to be an important initiator [16].

Copper and iron chelators should also help neurons deal with oxidative stress that’s why they are labeled as indirect antioxidants as accumulation of amyloid is reduced in brain [17]. Testosterone and Estrogen [18] are additional therapeutic approaches which are being examined including anti-inflammatory agents such as steroids and COX-2 inhibitors that naturally diminish with age.

Parkinson’s Disease (PD)

PD is most widespread, after Alzheimer’s that often impairs speech, motor skills, and other functions and detected pathologically by the loss of neurons in the nucleus basalis of Meynert additionally the septal forebrain areas [19] in connotation with the occurrence of inquinated protein in cytoplasm of neurons [20]. According to epidemiological research, the incidence of Parkinson’s disease is around 1% among those aged 65-69, rising to 3% among people aged 80 and up around one million people, or 1% of those over the age of fifty-five, are affected by PD [21,22].

L-hydroxy phenylalanine (L-DOPA) enhancement of dopamine levels is enhanced by this monoamine oxidase inhibitors, which is precursor. Cholinesterase inhibitors enhance the cholinergic activity [23]. Alzheimer’s, muscular rigidity, resting tremor, and an impairment of postural balance leading to disturbances of gait and falling is a clinical syndrome consisting of four cardinal features [24].

Causes of the Disease

It gets difficult for the cells to get the nutrients as brain and tangles in the nerve cells make it difficult for the cells to get the nutrients. Sixty percent to seventy percent of cases of dementia, Alzheimer’s accounts for chronic neuro-degenerative disease which normally begins gradually and worsens with time.

Down’s syndrome

Higherrisk of developing AD is because of a genetic flaw that results in Down’s syndrome can also lead to AD in some people because amyloid plaques builds up in the brain over time.

Genes

Genes that relate to causing Alzheimer’s due to mutation in them are- Amyloid Protein Precursor (APP), PSEN1 (Presenilin 1) & PSEN 2 (Presenilin 2). Additional genes associated are- CLU, SORL1, PICALM, CR1 etc. Most of autosomal dominant family diseases like Alzheimer’s attributable to mutations in either one of three genes: Those encoding amyloid precursor protein (APP) and presenilin’s 1 and 2. At 65 years of age. Familial variants of autosomal dominant inheritance account for around 0.1 percent of cases before the age of 65.

APOE apolipoprotein E

Apolipoprotein C1 and C2 genes is present in chromosome 19. Mutations develop in type III hyper lipoproteinemia enhances blood plasma cholesterol and triglycerides in consequence of impaired clearance of chylomicron and VLDL remnants. Even though mutations reduce the overall quantity of Aβ generated, they can induce sickness and indicate to additional functions of presenilin or changes in the function of APP. Senile plaques are main components which increases Aβ42 production is increased by mutations in the presenilin genes and APP. The brain cells die in neurofibrillar tangles formed by the tau protein particles, and the signs and symptoms of AD.

Sodium nitrite mediated neurodegeneration

D-galactose and NaNO₂ in combination has been stated in medical conditions of Alzheimer’s. Sodium Nitrite (NaNO₂) having capability to change haemoglobin into methaemoglobin, which lowers the blood’s oxygen carrying ability [25,26].

Vascular dementia and Alzheimer’s disease -Difference in symptoms

Clinical condition characterized by a steady decline in brain intellectual function, with the individual affected unable to carry out daily tasks adequately [27].

The extent of the lacunae dementia was shown to be more linked with the degree of hippocampal and cortical atrophy [28,29]. Most common in elderly persons have multi-infract dementia [30] where the brain has been damaged by frequent small strokes. In sum, Japan accounts for 50 percent of all dementias, 20-40 percent in Europe, and 15% in Latin America. Men than in women rate is found to be high and it increases with age [31]. High blood pressure, abnormal heart rhythms, and disorders that damage the arteries in the brain are all causes of vascular dementia. In patients with a mild degree of vascular dementia, memory impairment may be limited or non-existent [32]. Microvascular pathology, such as pallor, neuronal loss, and gliosis, has been discovered to play a causal role in dementia, with cholinergic impairments being seen in individuals in later stages.

The active form of Vitamin D, 1,25 dihydroxyvitamin D₃, governs neutrophil expression, including nerve growth factor, neurotrophin 3, and glial-derived neurotrophic factor, as well as neural cell survival, development, and function [33,34]. Reducing the cost of randomized controlled trials and improving the design, Vitamin D pills are being studied to see if they can help elderly people delay or prevent dementia and AD [35]. Vitamin D stimulates macrophages, which promotes phagocytic clearance of amyloid plaques under in vitro conditions [36,37]. All forms of dementia and Alzheimer’s is associated with lower concentration of vitamin D [38,39].

Neuroprotection - possible ways

As revealed in literature there are numerous scientific studies and confirmations of AD, traditional literature claims herbal research has demonstrated its efficacy to prevent the neurodegeneration.

Herbal Neuroprotectives

Oxygen delivery to the brain is the main focus of all modalities. None of these approaches has shown to be optimal thereby demonstrating the demand of alternate medicine especially those mentioned in the various AD.

Conventional crude form to cure has appeared in the form of standardized herbal extract, its formulations, and composite preparations [40,41]. (Table 1) summarizes the potential phytochemicals and their studies for the management of AD, PD and HD used. Medicines give a safe alternative to traditional treat-
ment and well borne remedies for a persistent sickness with fewer adverse effects [42]. Few case studies of herbal extracts are discussed below:

Effect of *Bacopa monniera* on lipofuscin genesis and D-Galactose fluorescent product in the brain induced elderly mice was studied. D-Galactose, react with amino groups and protein AEGs which are ironic source of free radicals in AD causing chemical oxidation of AEGs causes oxidative stress. AGE receptor binding and activation of signalling pathways. *Bacopa monniera* is a tropical Ayurvedic herb used to treat many central nervous system disorders [43]. At physiological pH, free radicals cause increased oxidative stress and, as a result, damage to micromolecules, particularly mitochondria [44].

*Withania somnifera* therapy causes an increase in cAMP through superoxide dismutase, lipid peroxidation activity, and a decrease in total sulphahydral levels, making it an antioxidant and antistress agent. *Withania somnifera* is used for several clinical condition like development of memory and cognition augmentation [45]. *Withania somnifera* root extract in mice shows, nootropic effects, and AchE is reversed. Indirect cholinergic spread facilitation, which could be very useful in NDS with cholinergic deficits [46,47]. indicated reduction of long term hypoperfusion caused anxiety and restlessness and memory shortage [48]. Moderate attenuation of histological abnormalities is seen in long-term reperfusion injury, such as partial reperfusion induced inflammatory cell penetration, lympholytic infiltration and cell proliferation. Aptogenic properties of *Withania somnifera* is studied for neuroprotection [47].

The antioxidant mechanism of *Centella asiatica* was investigated by boosting endogenous antioxidant enzymes in the brain. When tested utilising a range of AD paradigms, aqueous extract of *Centella asiatica* at doses of 200mg/kg and 300 mg/kg showed improvement in memory and learning, with significant reductions in malondialdehyde and concurrently significant levels of glutathione [49].

When tested utilising a conditioned avoidance response paradigm, an extract of *Ocimum sanctum* in ethanol alleviated scopolamine (0.4mg/kg). Amelioration indicated possible mechanism of its action by cholinergic modulation and AD and age-related dementia benefit from its use.

*Semecarpus anacardium* documented its potential utility along with Methanolic extract of *Semecarpus anacardium* which demonstrated inhibition of AchE with *Nardostachys jatamansi*. *Semecarpus anacardium* prevents stress induced neurodegeneration.

AcE has close association and hence *Semecarpus anacardium* therapy could be of dual benefits. The effect of *Citrinia ternatea* roots in alcohol on scopolamine-persuaded memory impairment was seen by Vyawahare.

*Caryocar brasiliense* (Camb), a Caryocaraceae family member popularly known as “pique”. A decoction of the leaves and petals is used as an energetic, tonic, aphrodisiac, and treatment for liver disorders, while the fruit pulp is used as a stomachic and flu treatment. Flavonoids found in *C. brasiliense* have been shown to have leishmanicidal, antifungal, antioxidant, and vasorelaxant effects [50]. Antioxidant substances such as gallic acid, quinic acid, quercetin, and quercetin 3-o-arabinose were found in *C. brasiliense* leaves in studies [51,52]. Anticholinesterase actions and protection from *C. brasiliense* extract consumption are currently restricted [53].

### Details of Phyto compounds

![Image](https://via.placeholder.com/150)

### Table 1: Promising phytochemical and their studies for the management of AD, PD, HD.

<table>
<thead>
<tr>
<th>Phytochemicals</th>
<th>Studied materials</th>
<th>Dose</th>
<th>Physiological effects</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chrysin</td>
<td>C57BL/6mice (Male)</td>
<td>10 mg/kg</td>
<td>↑Behavioral functions↑TH-positive cells in the SN and ST↑DA, DOPAC, and HVA levels</td>
<td>[54]</td>
</tr>
<tr>
<td></td>
<td></td>
<td>50 and 100 mg/kg</td>
<td>↑DA and its metabolites↑AKT/GSK3β/MEF2D pathway↓MAO-B activity</td>
<td>[55]</td>
</tr>
<tr>
<td></td>
<td></td>
<td>50, 100, and 200 mg/kg</td>
<td>↑BDNF and GDNF protein expression↓IL-10, IL-6, TNF-α, and NF-κB protein expression</td>
<td>[56]</td>
</tr>
<tr>
<td>Vanillin</td>
<td>C57BL/6mice (Male)</td>
<td>5, 10, and 20 mg/kg</td>
<td>↓iNOS, COX-2, IL-1β, and IL-6 protein expression</td>
<td>[56]</td>
</tr>
<tr>
<td></td>
<td></td>
<td>5, 10, and20 mg/kg</td>
<td>↓ERK1/2, p38, and NF-κB signaling↓Microglia activation↑Striatal DA and its metabolite levels↑Behavioral function</td>
<td>[57]</td>
</tr>
<tr>
<td></td>
<td></td>
<td>20, 40, and80 mg/kg</td>
<td>↑Striatal DA levels↑Striatal TH, TLR4, BDNF, and GFAP proteinexpression↓α-Synuclein and lowered AIF proteinexpression</td>
<td>[58]</td>
</tr>
<tr>
<td>Asiatic acid</td>
<td>C57BL/6mice (Male)</td>
<td>100 mg/kg</td>
<td>↑Motor functions↑P3K, Akt, GSK-3β, and mTOR phosphorylation↑TrKB protein expression↓NLRP3 inflammasome expression immicroglia cells</td>
<td>[59]</td>
</tr>
<tr>
<td></td>
<td></td>
<td>100 mg/kg</td>
<td>↓Mitochondrial Drp1 expression↑PGC1αgene and protein expression↑Mfn2 and mitochondrial dynamics</td>
<td>[60]</td>
</tr>
<tr>
<td>Phytochemicals</td>
<td>Experimental Model</td>
<td>Dose</td>
<td>Physiological effects</td>
<td></td>
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<tr>
<td>Ferulic acid</td>
<td>C57BL/6 mice (Male)</td>
<td>100 mg/kg</td>
<td>↑HSP-70 protein expression</td>
<td></td>
</tr>
<tr>
<td></td>
<td>C57BL/6 mice (Male)</td>
<td>20 mg/kg and muscle exercise</td>
<td>↑Motor behavior, ↑CAT, SOD, GPx, and GSH activity, ↓TBARS activity</td>
<td></td>
</tr>
<tr>
<td></td>
<td>C57BL/6 mice (Male)</td>
<td>40 mg/kg</td>
<td>↓iNOS, COX-2, IL-1β, and IL-6 protein expression</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Wistar albino rats (Male)</td>
<td>7.5 and 15 mg/kg</td>
<td>↑Parkin, Drp1, TH-positive cells in the SN and ST, ↑DA, DOPAC, and HVA levels</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Wistar albino rats (Male)</td>
<td>5 and 10 mg/kg</td>
<td>↑Behavioral functions, ↑DA level in the SN, ↓MDA level</td>
<td></td>
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<tr>
<td></td>
<td>Wistar albino rats (Male)</td>
<td>50 mg/kg</td>
<td>↑Motor function and electrophysiological performance, ↑CAT, SOD, GPx, and GSH cerebral activity</td>
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<tr>
<td></td>
<td>Wistar albino rats (Male)</td>
<td>50 mg/kg</td>
<td>↓MAO-B activity, ↑ERβ/Nrf2/NO-1 signaling cascade</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Drosophila melanogaster</td>
<td>0.5, 1, and 2 mg/g</td>
<td>↑CAT, SOD, GPx, and GSH cerebral activity, ↑TBARS activity</td>
<td></td>
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<tr>
<td></td>
<td>A53T transgenic mice</td>
<td>5 mg/kg</td>
<td>A53T α-synuclein, ↑Bcl-2-mediated autophagy pathway, ↑Behavioral functions</td>
<td></td>
</tr>
<tr>
<td></td>
<td>C57BL/6 mice (Male)</td>
<td>0.5, 1, and 2 g/kg</td>
<td>↑DA synthesis, ↑TH-positive cells, ↑BDNF and GDNF protein expression, maintained loss, ↓IL-1β, IL-6, TNF-α, iNOS, and COX-2 expression, ↓GFAP protein expression</td>
<td></td>
</tr>
<tr>
<td></td>
<td>C57BL/6 mice (Male)</td>
<td>50 mg/kg</td>
<td>↑Iron-export protein ferroportin in SN, ↑CAT, SOD, GPx, and GSH cerebral activity, ↑TBARS activity, ↑DA synthesis</td>
<td></td>
</tr>
<tr>
<td></td>
<td>C57BL/6U mice (Male)</td>
<td>25 mg/kg</td>
<td>↑Movement behavior, ↑TH-positive cells in the SN region, ↑CD3'CD4' to CD3'CD8' T-cell lymphocyte ratio in the peripheral blood, ↓TNF-α and IL-6 cytokine expression in serum</td>
<td></td>
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<tr>
<td></td>
<td>Postmortem PD tissue</td>
<td>100 nM</td>
<td>↓α-Synuclein aggregates</td>
<td></td>
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<tr>
<td></td>
<td>C57BL/6 mice (Male)</td>
<td>10 mg/kg</td>
<td>↑HVA, DOPAC, 5-HIAA levels, ↑TH-positive cells in the ST region, ↓JNK and p-JNK expression, ↑Bcl-2 protein expression</td>
<td></td>
</tr>
<tr>
<td></td>
<td>C57BL/6 mice (Male)</td>
<td>10 mg/kg</td>
<td>↑DAT and VMAT-2 expression, ↑Behavioral functions, ↑TH-positive cells in the ST region, ↑Behavioral characterization</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Sprague Dawley rats</td>
<td>15 mg/kg</td>
<td>↑CAT, SOD, GPx, and GSH cerebral activity, ↓TBARS activity, ↑PERK/CHOP/Bcl-2/Bcl-2 pathway, ↓GRP78 levels</td>
<td></td>
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<tr>
<td></td>
<td>C57BL/6 mice (Male)</td>
<td>10 mg/kg</td>
<td>↑OAT and VMAT-2 expression, ↑Behavioral functions, ↑CAT, SOD, GPx, and GSH cerebral activity, ↑TBARS activity</td>
<td></td>
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<tr>
<td></td>
<td>C57BL/6 mice (Male)</td>
<td>10 mg/kg</td>
<td>↑Behavioral characterization, ↑TH-positive cells in the ST region</td>
<td></td>
</tr>
<tr>
<td></td>
<td>C57BL/6 mice (Male)</td>
<td>10 mg/kg</td>
<td>↓Caspase-3, caspase-8, and caspase-9 activity, ↓Bax expression, ↑Bcl-2 protein expressions, ↑Behavioral characterization, ↓IL-4 and IL-10 protein expressions</td>
<td></td>
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Promising phytochemicals and their physiological effects for the management of Alzheimer’s disease (AD)

<table>
<thead>
<tr>
<th>Phytochemicals</th>
<th>Experimental Model</th>
<th>Dose</th>
<th>Physiological effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apigenin</td>
<td>In vitro induced neurogenesis in vivo mouse model of AD</td>
<td>-</td>
<td>↓inflammatory cytokines, ↓cortical hyperexcitation, ↓Aβ burden, ↓oxidative stress, ↑ERK/CREB/BDNF pathway, ↓β-amyloid neurotoxicity, ↑mitochondrion protection</td>
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</tbody>
</table>

References:
[63, 64, 65, 66, 67, 68, 69, 70, 71, 72, 73, 74, 75, 76, 77, 78, 79, 80, 81-83]
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<th>Dose</th>
<th>Physiological effects</th>
<th>References</th>
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</thead>
<tbody>
<tr>
<td>α-Mangostin</td>
<td>3-NP-induced CGNs</td>
<td>Cell death inhibition</td>
<td>Mitochondrial reductant capacity</td>
<td>[100]</td>
</tr>
<tr>
<td>Astragalan</td>
<td>Ischemic male Wistar rats</td>
<td>Hemeoxygenase-1 expression</td>
<td></td>
<td>[101]</td>
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<tr>
<td>Berberine</td>
<td>Ischemic middle cerebral artery occlusion model, oxygen-glucose-deprived PC12 cell line</td>
<td>Blocking of the mitochondrial apoptotic pathway</td>
<td></td>
<td>[102]</td>
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<tr>
<td>Celastrol</td>
<td>G93A SOD1 transgenic mouse model</td>
<td>HSP70 immunoreactivity</td>
<td></td>
<td>[103]</td>
</tr>
<tr>
<td>Sesamol</td>
<td>3-NP-induced rats</td>
<td>Oxidative defense</td>
<td></td>
<td>[104]</td>
</tr>
<tr>
<td>(−) Schisandrin B</td>
<td>3-NP-treated neuronal PC12 cells</td>
<td>Pyruvate dehydrogenase activation</td>
<td></td>
<td>[105]</td>
</tr>
<tr>
<td>Quercetin</td>
<td>3-NP-treated rats</td>
<td>Neuromodulation</td>
<td></td>
<td>[106]</td>
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<tr>
<td>Naringin</td>
<td>3-NP-treated rats</td>
<td>Neuromodulation</td>
<td></td>
<td>[107]</td>
</tr>
<tr>
<td>Lycopenes</td>
<td>3-NP-treated rats</td>
<td>Oxidative defense</td>
<td></td>
<td>[108]</td>
</tr>
<tr>
<td>Kaempferol</td>
<td>3-NP-induced rats</td>
<td>Oxidative damage reversal</td>
<td></td>
<td>[109]</td>
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<tr>
<td>Ginsenosides</td>
<td>Glutamate-induced YAC128 medium spiny neurons</td>
<td>Ca (2+) response inhibition</td>
<td></td>
<td>[110]</td>
</tr>
<tr>
<td>Curcumin</td>
<td>3-NP-induced rats</td>
<td>Nrf2 and HO-1 expression</td>
<td></td>
<td>[111]</td>
</tr>
</tbody>
</table>

**Promising phytochemicals and their physiological effects for the management of Huntington’s disease (HD)**

**Diet Management**

Nutritional support can help patients with AD decrease the progression of dementia and possibly enhance their quality of life not affecting their survival chances. Folic acid increases the concentration of omega-3 PUFAs such as Eicosapentaenoic Acid (EPA) and Docosahexaenoic Acid (DHA), which is beneficial in the impediment and treatment of AD and dementia.

Fruits, fish, nuts, vegetables and even Indian spices have been shown to reduce AD risk by leading to 45 percent. Fructose for example should be consumed at less than 25 g/day.

Antioxidants such as beta-carotene, vitamin A, vitamins E and C, and others are found in small amounts and can be increased to normal levels to cure disease [112,113].

**Astrocytes transplantation**

The hippocampus of AD mice was implanted with astrocytes. The role of astrocytes as active A clearance cells in the brain, which could have crucial ramifications for the future development of AD therapy was studied [114].

**Stem-cells transplantation**

BDNF-mediated hippocampus synaptic density increases significantly after stem cell transplantation. It improves spatial learning and memory in Alzheimer’s patients without affecting Aβ deposits [115-117].
Sternly affected areas in AD brain are with the temporal lobes, especially in hippocampus. Cognitive deficits and pathogenesis of Alzheimer’s is caused by degeneration of BFCN’s suggesting, BF CNs may be an ideal form of donor cell for treating Alzheimer’s-related cognitive impairments.

Exogenous cholinergic neurons displayed cholinergic projection in the basal forebrain, and migratory patterns, as well as morphological and functional integration into the endogenous projection system. The feasibility of developing stem cell treatment utilizing ESC-derived cells was in a behaviour test was performed to demonstrate HuCNS-SC cells which are human neural stem cells that have been produced according to cGMP standards under controlled conditions. HuCNS-SC cells can engraft, migrate, and develop into neurons, astrocytes, and oligodendrocytes over time with no evidence of tumour formation or AD side effects.

Induced pluripotent stem cells

Pluripotent stem cells are found in the early stages of development and serve as the basis for all cell types in the body. Because induced pluripotent stem cells can develop all types of cells in the body, they could provide cells that would otherwise be difficult to obtain, such as brain neurons.

Technology that allows scientists to create neurons in the lab that exhibit some of the hallmarks of AD. The development of a method for generating neurons from these IPS cells in a dish lab, where the beta amyloid protein that forms plaques in patients’ brains is released.

Future aspects

AD, for example, necessitates early detection to receive successful treatment. The number of AD patients is increasing at an alarming rate, making it critical to employ modern Alzheimer’s technology to treat the condition. Much research has been conducted and still going on the biomarkers, proteomics, and genomics level. There are numerous challenges that must be conquered. Technology alone will not be enough to treat the disease; standardization of processes and techniques is critical for preserving consistency and achieving an elevated level of reliability.

Intracellular neurofibrillary tangles, extracellular amyloid plaques, synaptic deterioration and neuronal death are all implicated in the metabolic processes that lead to AD as a neurodegenerative disorder, according to numerous research. Genetics accounts for about 70% of the risk of AD at any given age. The epsilon 4 allele of the apolipoprotein E gene is a genetic risk factor for AD (ApoE). Apart from the genetic and biochemical aspects of AD, a diet deficient in vitamin D modulates nerve growth factor in its active form, appears to be another culprit. In conclusion biomarkers and stem cell therapy may be promising strategies for early diagnosis and treatment of AD and other NDs.

Conclusion

Neuroprotective agents which are synthetically manufactured and are used in various neurodegenerative diseases like PD, AD and HD have own sluggishness, drowsiness, dry mouth, weariness, tension or apprehension, difficulties with balance, and other side effects which have been reported during treatment. Plant based natural phytomedicines are therefore being explored showing cognitive functions in humans. Numerous classes of plant based natural medicines have been mentioned in literature which stimulate cell stress reaction pathways and thus contribute to imparting neuroprotective excellence. Medicinal plants are having numerous phytochemicals with different secondary metabolites like poly phenols (phenolic acids, anthocyanins, proanthocyanidins, flavonols, tannins), isoprenoids (sesquiterpenes, diterpenes, triterpenes, steroids, saponins), alkaloids (indole alkaloids, tryptogenic acid diethylamide, tropane alkaloids, ergot group) and fatty acid they show their effect by inhibiting or even scavenging the excess free radicals produced by oxidative and neurotoxin-induced stressors in brain nerve cells. However more exploratory research is needed to demonstrate the exact molecular processes involved in neuroprotection by these phytochemicals which can lead to effective formulation development.

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Abbreviations

AD: Alzheimer’s disease; PD: Parkinson’s disease; HD: Huntington’s disease; ND: Neurodegenerative disease.

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