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Dr. Annegowda Hardur Venkatappa
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From the Editor's Desk:

Dr. Annegowda H V

It is with immense pride and heartfelt enthusiasm that we present the inaugural issue of the *Journal of Advanced Pharmacy and Drug Development (JAPDD*), a platform envisioned to bridge innovation and evidence-based research in pharmaceutical sciences. This marks the beginning of an exciting journey toward fostering scientific dialogue, innovation, and discovery within the global pharmaceutical research community.

The **September 2025** issue represents a significant milestone in our academic and research endeavours. *JAPDD* was founded with a collective vision to create a credible and inclusive platform that promotes original research, critical reviews, and discussions on emerging trends in drug development, pharmacological sciences, ethnomedicine, regulatory innovations, and personalized healthcare. Our mission is to encourage the exchange of knowledge and inspire collaborations that advance both scientific understanding and practical application in these diverse domains.

In this volume, readers will find six carefully curated articles covering a broad spectrum of contemporary topics including polyherbal interventions in Parkinson's disease, the evolving role of personalized medicine, and phytopharmacological reviews that highlight the potential of natural products in modern therapeutics. Each manuscript has undergone a rigorous peer-review process to ensure scientific quality, ethical compliance, and clarity of presentation. To enhance accessibility and citation visibility, Digital Object Identifiers (DOIs) have been assigned to every article through Crossref sponsorship. While ISSN registration is currently in progress, our commitment to academic integrity, scientific rigor, and transparency remains unwavering. We aspire for *JAPDD* to grow into a reliable, respected, and collaborative platform for researchers, academicians, clinicians, and industry professionals dedicated to advancing pharmaceutical sciences.

On behalf of the Editorial Board, I extend my sincere gratitude to all authors, reviewers, and supporters who have contributed to the success of this inaugural issue. Your dedication, expertise, and trust have been instrumental in transforming this vision into reality. We invite continued support, constructive feedback, and future submissions as we move forward in our shared pursuit of scientific excellence.

Warm regards,

Dr. Annegowda Hardur Venkatappa

Editor-in-Chief

Journal of Advanced Pharmacy and Drug Development (JAPDD)

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Review Article

Polyherbal Interventions In Parkinson's Disease: Mechanistic And Neuroprotective Benefits

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Abstract

Parkinson's disease (PD) is a progressive neurodegenerative disorder marked by motor impairment, a decrease in dopamine levels, and oxidative stress. Current therapies primarily alleviate symptoms but has limitations in preventing the progression of the disease. Polyherbal formulations, which consist of various plant extracts, have emerged as promising treatment options due to their synergistic effects in addressing the complex pathology of PD. According to research, employing Polyherbal Formulations (PHFs) that combine several plants can produce better results than using plant extracts alone or their additive effects. This review focuses on the potential mechanisms of action through which PHFs act in treating Parkinson's disease. Additionally, showcasing their therapeutic potential through preclinical and clinical data highlights their efficacy and synergistic qualities. The article explores the neuroprotective advantages emphasizing how well they may treat neuroinflammation, oxidative stress, neurotransmitter modulation, and α-synuclein aggregation mitigation. It emphasizes PHFs importance as supplementary and alternative approaches to controlling neurodegenerative illnesses and explores how they may improve patient outcomes, decrease side effects, and increase therapeutic efficacy. This thorough evaluation highlights the need for additional study to confirm efficacy, improve formulations, and elucidate safety profiles, opening the door to novel, nature-based Parkinson's disease treatment options

Keywords: Parkinson's disease, Polyherbal formulation, Synergism, Clinical trial, Herbal medicine, Challenges

Introduction

Neurodegenerative diseases (ND) involve the gradual deterioration of neuronal structure or function, frequently linked to neuronal loss. Parkinson's disease (PD), Alzheimer's disease (AD), Amyotrophic lateral sclerosis (ALS) and prion's disease (PrD) are few examples of ND [1]. PD is a long-lasting and advancing ND that impacts at least 1% of individuals by the time they reach age 70 [2]. It is marked by movement difficulties, including tremors, stiffness, and slow movements (bradykinesia), along with a range of non-motor manifestations, including sleep disturbances, autonomic dysregulation, cognitive impairment, neuropsychiatric disorders, gastrointestinal dysfunction, weight fluctuations, visual disturbances, and

fatigue [3]. Cognitive impairments, mental health issues, and the neurodegenerative effects of levodopa-induced dyskinesia are additional anomalies linked to PD [4]. Despite the fact that the signs and treatments for PD were initially referenced in ancient texts like “Indian Ayu rveda” (5000 BC) and the Chinese medical classic “Nei-Jing” (500 BC), it was James Parkinson, a British doctor, who first gave a detailed account of the condition, referring to it as “the shaking palsy” in his writings, "An Essay on the Shaking Palsy "in 1817 [5,6]. The main characteristics James Parkinson outlines are postural instability, muscle weakness, and a resting tremor that lessens with intentional movement, resulting in a distinctive forward-leaning posture and shuffling stride [7]. PD is the neurological disorder with the highest growth rate worldwide regarding mortality and disability [8], as illustrated in Figure 1.

Global Health Estimates the incidence of PD has increased twofold in the last quarter-century, impacting more than 8.5 million individuals in 2019. The rise in disability and mortality rates linked to PD is outpacing that of any other neurological condition. In 2019, Parkinson's disease was responsible for 5.8 million disability-adjusted life years, marking an 81% increase since the year 2000, and it led to 329,000 fatalities, which is more than twice the number reported in 2000 [9]. There was a notable rise in the reported cases, going from 2.5 million in 1990 to 6.1 million in 2016. It is anticipated that by 2040, the worldwide prevalence of PD will surpass 17 million cases [10].

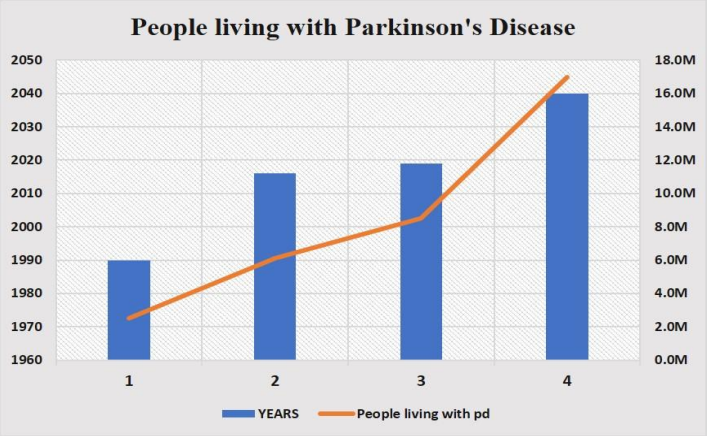

Figure 1: Illustrates the prevalence data of Parkinson's disease as reported by the World Health Organization.

Although PD was identified over 200 years ago, no disease-modifying drugs have been developed. Patients rely on symptomatic relief through conventional medications like dopamine agonists (ropinirole, rotigotine, bromocriptine, and pramipexole), COMT-inhibitors, MAO-B inhibitors, and non-pharmacological treatments like deep brain stimulation, MRI-guided focused ultrasound, rehabilitation, and exercise [11]. For decades, levodopa has been the primary monotherapy, providing consistent clinical benefits in the early stage as it raises dopamine levels and is the best way to treat PD. However, numerous individuals experience motor issues after several years of using these conventional medications [12,13]. After long-term levodopa medication, dyskinesia and the return of Parkinsonian symptoms are common [14]. Treatment with these conventional medications can cause adverse effects including queasiness, vomiting, postural low blood pressure (a drop in blood pressure when standing), drowsiness, cognitive confusion, sleep disruptions,

hallucinations, and involuntary movements (dyskinesias) [15].

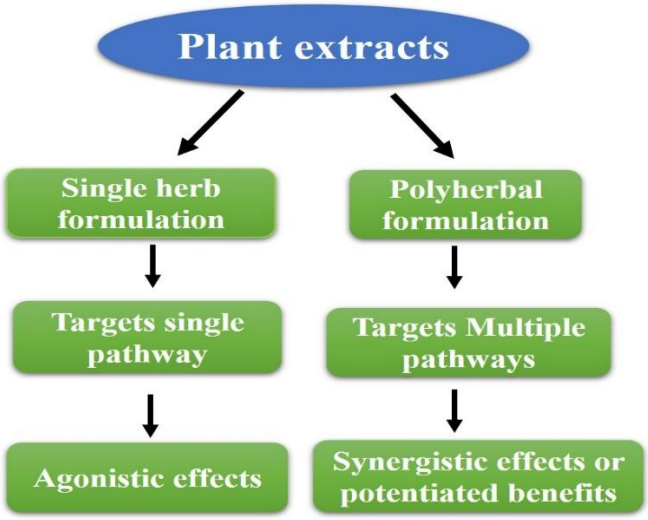

Figure 2: Single Herbal vs. Polyherbal Formulation

New treatments aimed at addressing the root causes of neuronal death are crucial. Since medicinal plants and herbs comprise a complex blend of phytocompounds with a wide range of pharmaco-biological significance, herbal therapies are explored for treating several illnesses. A variety of therapeutic methods continue to rely heavily on plants as a source of medications [16]. Natural products from medicinal plants, fruits, and vegetables have been utilized for a long time to help manage PD because of their antioxidant, anti-inflammatory, and neuroprotective benefits. These include preventing iron accumulation, protein misfolding, and supporting mitochondrial and proteasomal functions [17]. While individual plant phytochemicals are well-known, their therapeutic levels are often insufficient. However, studies show that combining plants of varying potencies can produce more effective results than using them individually [18]. This review offers detailed and up-to-date information on various polyherbal formulations used to manage PD, primarily focusing on their mechanism and therapeutic potential.

Herbs treating Parkinson's disease

The compounds obtained from plants provide a safe and natural substitute for prescription medications in addressing neuroinflammation in PD and other neurodegenerative illnesses [19]. Numerous preclinical, clinical, *in vitro*, and *in vivo* investigations have examined the potential application of herbs in treating PD over the last several decades, and the level of safety related to the utilization of herbal remedies is fairly large in addition to their efficacy [20].

Due to their multilevel function characteristics and remarkable efficacy (in certain cases) with fewer side effects, herbal medicines, which are the foundation of traditional medicine, have gradually gained acceptance for application in the management of different illnesses worldwide [21]. Many ancient herbal medicines, including those involving plants like *Withania somnifera*, *Mucuna pruriens, Tinospora, Acanthopanax, Alpinia*, and *Astragalus*, have been utilized in India to treat neurological disorders. The components or extracts of herbal remedies have been shown to have consistent and significant effects on PD models in contemporary pharmacological research [17]. The table below includes some of the plant-derived compounds employed in treating PD **(Table**

1).

Table 1: Therapeutic Effects and Dosage of Plant-Based Compounds in Parkinson's Disease Animal Models

Plant	Plant part used	Active components	Animal model	Dose	Result	Reference
Gynostemma pentaphyllum	Leaves	Gypenoside derivatives	6-OHDA-induced PD in rats	10 and 30 mg/kg	Enhanced the reduction of TH-immunopositive neurons in the rat brain brought on by 6-OHDA damage. Following the injury, it also restored the levels of norepinephrine, homovanillic acid, 3,4-dihydroxyphenylacetic acid, and dopamine.	[22]
Curcuma longa	Rhizome	Curcuminoids	MPTP-induced PD in mice	150mg/kg	Averted the loss of dopamine and tyrosine hydroxylase (TH) immune response caused by MPTP. It also restored the expression of glial fibrillary acidic protein (GFAP) and inducible nitric oxide synthase (iNOS) proteins.	[23]
		Curcumin	Rotenone-induced PD in mice	50, 100 and 200mg/kg	It markedly enhanced behavioral changes, reduced oxidative damage, and boosted the activities of mitochondrial enzyme complexes. It also decreased the elevated levels of acetylcholine esterase enzyme.	[24]
Punica granatum L	Seeds Juice	Propyl gallate Nobiletin Ellagic acid Ellagic acid Vitexin isovitexin	Paraquat-induced PD in mice	500 mg/kg 5ml	Dopamine (DA) and 3,4-Dihydroxyphenylacetic acid (DOPAC) levels increased, nuclear factor-kappa-B (NF-KB) was downregulated, the Interleukin-10 (IL-10) level increased, and the cytokine level decreased. The level of transforming growth factor (TGF-β) significantly decreased while Glial cell line-derived neurotrophic factor (GDNF) substantially increased.	[25]

Zingiber officinale	Rhizome	2-Butanone Zingiberene Zingerone	Rotenone-induced PD in mice	50 mg/kg and 100mg/kg	Inhibited the development of Lewy bodies by blocking the aggregation of α-Syn proteins in the nigrostriatal region, reducing the release of pro-oxidant and pro-inflammatory factors and the expression of COX-2 protein, enhancing glutathione enzyme activity, and slightly lessening the degeneration of dopaminergic neurons.	[26]
Bacopa monnieri	Plant	Bacosides	Rotenone-induced PD in rats	40mg/kg	Pre-treatment with *Bacopa monnieri* notably reduced ($p < 0.01$) the levels of α-synuclein when compared to the rotenone-injected subjects. Elevated levels of IL-1β and Tumour necrosis factor-α (TNF-α) during treatment mitigated the increased expression of α-synuclein. It also lowered ROS production and enhanced antioxidant activity in the striatum.	[27]
Spondias mombin L.	Leaves	Cyclogallipharaol dl-α-tocopherol Quercetin Rutin	Rotenone-induced PD in zebrafish	5mg/L, 15mg/L and 25mg/L	Decreased thiobarbituric acid reactive substances (TBARS) and total thiol levels. Increased Catalase (CAT), Superoxide dismutase (SOD), and glutathione (GSH) enzymes and decrease in Glutathione-S-Transferase (GST) activity in Zebrafish.	[28]
Trigonella foenum-graecum	Seeds	Trigonelline	6-OHDA and MPTP-induced PD in rats	10mg/kg, 30mg/kg and 100mg/kg	Showed a considerable improvement in motor impairment and a notable rise in the count of ipsilateral rotations.	[29]
Centella asiatica	Whole plant	Madecassoside Asiaticoside	Rotenone-induced PD in rats	10mg/kg, 30mg/kg and 100mg/kg	Standardized extract of *Centella asiatica* (ECa233) (30 mg/kg) provided protection against the inhibition of mitochondrial complex-I, lowered malondialdehyde (MDA) levels, and enhanced the expression of SOD and CAT.	[30]
Withania somnifera	Root	Withaferin A	MPTP-induced PD in mice	100mg/kg	A mouse that received MPTP treatment exhibited decreased levels of DA, DOPAC, homovanillic acid (HVA), GSH, and	[31]

					glutathione peroxidase (GPx) while showing increased levels of TBARS in comparison to the control group.	
Allium sativum	Bulb	Garlic derived compounds	6-OHDA-induced PD in Rats	500mg/kg	The number of TH positive cells in the groups treated with garlic extract was considerably greater ($p<0.001$) compared to the lesion group. The motor impairments showed considerable improvement in hanging, rotarod, open-field, and apomorphine-induced rotational assessments.	[32]
Smilax china	Bark	Tannins and fibres	Rotenone-induced PD in Wistar rats	100mg/kg and 200mg/kg	The body weight, mobility, coordination, and occurrence of catalepsy in animals treated with Smilax China ethanolic extract all showed improvement. Furthermore, it safeguarded the brain from oxidative stress by enhancing SOD levels in the group induced with rotenone. The degradation of α-synuclein and inflammation were diminished in the substantia nigra, basal ganglia, and vagus nerve of the treated groups.	[33]
Vitex negundo	Leaves	Vitexin	Haloperidol induced PD in rats	100mg/kg, 200mg/kg and 400mg/kg	Strong antioxidant activity and inhibition of Acetylcholinesterase (AChE) were shown in vitro. Significant pathological changes, which included elevated levels of AChE, Butyrylcholinesterase (BChE), and MDA alongside reduced levels of GSH, SOD, CAT, and DA, were notably reversed in rats administered haloperidol. Treatment with *V. negundo* at a dosage of 400 mg/kg markedly enhanced dopaminergic activity, bolstered antioxidant defenses, and reinstated cholinergic function.	[34]

Polyherbal Formulations: Synergistic Effects and Mechanisms

Although many plants include well-known active phytochemical components, the compounds are usually found in minimal concentrations and rarely sufficiently produce the desired therapeutic effects.

Because of this, scientific studies suggest that combining several plants with varying potencies could theoretically produce greater results than utilizing them separately or adding up their distinct effects [18].

Polyherbal formulations (PHFs) are highly effective in treating various diseases, offering a favorable risk-benefit ratio due to their efficacy at low doses and safety at higher doses. As natural products, PHFs are cost-effective, environmentally friendly, and widely accessible, making them increasingly popular, particularly in rural regions and developing countries where contemporary medical treatments are frequently too expensive [35,36]. Because of the synergism, PHFs provide advantages that are impossible with single herbal formulations. It lowers the chance of unfavourable side effects by enabling stronger therapeutic results with lower dosages. Additionally, PHFs improve patient comfort by removing the need to take many single herbal formulations at once [37]. Synergistic activity is important in herbal therapeutics, as numerous studies have shown that herbal extracts as whole or multiple herbs in complex formulations give superior efficacies to similar doses of individual active components or herbs when used alone [38]. Clinical and pharmacological studies have confirmed the therapeutic effects of many PHFs. PHFs are renowned for their remarkable efficacy in treating various ailments. When the right herbs are combined to make PHFs, the therapeutic benefits of herbal remedies are enhanced by the presence of many phytoconstituents [37].

Possible ways through which Polyherbal May Aid Parkinson's Management

Antioxidant pathway

Oxidative stress is widely recognized as one of the primary pathogenetic processes responsible for neuronal loss in PD [39]. It results from a breakdown in antioxidative processes involving GSH, SOD, and DJ-1(protein) in people with PD, which upsets the balance of reactive oxygen species (ROS) [40]. Elevated lipid peroxidation (LPO) and DNA damage in the Substantia Nigra (SN) are indicators of oxidative harm resulting from an abundance of ROS in PD brains [39]. A vital element of the therapeutic strategy for treating PD is using antioxidants to lower oxidative stress [41]. Research conducted both *in vitro* and *in vivo* on Parkinson's models has demonstrated that natural and endogenous antioxidants like polyphenols, coenzyme Q10, and vitamins A, C, and E offer protective benefits against neuronal death caused by oxidative stress [42]. Curcumin, baicalein, quercetin, resveratrol, kaempferol, amentoflavone, caffeine, rosmarinic acid, neoandrographolide, naringenin are some of the examples of phytochemicals originating from plants that may primarily demonstrate their biological activity through antioxidant pathways. These substances aid in reducing oxidative stress, which is a major factor in several clinical disorders [43-48]. In PD, chrysin

pretreatment preserves SOD activity and GSH levels while lowering LPO and oxidative strain . In PC12 cells, chrysin also obstructs NF-κB phosphorylation and transcriptional activity, lowers intracellular Nitric oxide (NO), and downregulates iNOS production [49,50]. In SH-SY5Y cells, vanillin possesses potent neuroprotective potential by strengthening antioxidant defences, diminishing LPO and NO levels, and reducing rotenone-induced ROS, mitochondrial dysfunction, caspase activation, and signalling molecule expression in PD [51-53].

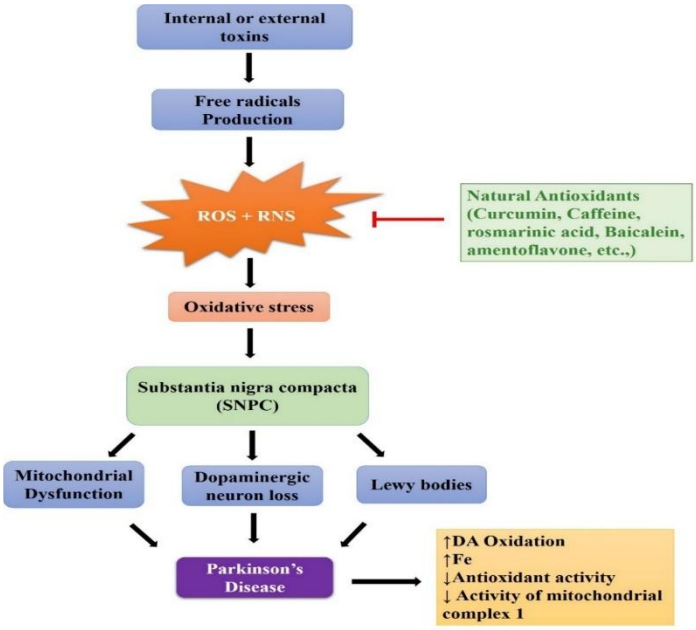

Figure 3: Phytochemicals function as neuroprotective agents against oxidative stress in PD

Anti-Neuroinflammatory pathway

Neuronal degeneration in PD is associated with persistent neuroinflammation, mainly driven by microglia, along with involvement from astrocytes and oligodendrocytes [54]. Activated microglia produce ROS that result in oxidative harm, which consequently causes dopaminergic neurons to degenerate in PD [55]. Increased concentrations of growth factors like Epidermal growth factor (EGF) and Transforming Growth Factor-β1 (TGF-β1) and cytokines such as Tumour necrosis factor-α (TNF-α), Interleukin (IL)-1β, IL-2, IL-6, and IL-4 are significant indicators of inflammation in the striatum and cerebrospinal fluid of individuals with PD [56]. Nicotine, berberine, capsaicin, and kavalactone are phytochemicals with anti-inflammatory qualities that lessen inflammation in PD by reducing oxidative stress and TNF-α levels [57]. Significant anti-inflammatory and neuroprotective benefits are demonstrated by extracts of *Ginkgo biloba, Lindera neesiana*, *Scutellaria baicalensis,* spicatoside A (*Liriope platyphylla*), quercetin, apigenin, and ginger components (6-gingerol, 6-shogaol, 6-paradol, and zingerone) [58-60]. By lowering neuroinflammation, broccoli extract containing sulforaphane also provides neuroprotection [61]. DA neurons are protected from lipopolysaccharide (LPS)-induced toxicity by 2,3,5,4′-Tetrahydroxystilbene-2-O-beta-d-glucoside (TSG), the primary bioactive compound in *Polygonum multiflorum*. TSG also increases astrocyte-derived neurotrophic support and reduces microglia-induced neuroinflammation [62]. Furthermore, diallyl sulfide and curcumin (found in *Allium sativum*) inhibit pro-inflammatory substances like TNF-α, which makes them promising treatments for neurodegenerative illnesses like PD

[63,64]. In the SN of PD rats, polyphenols extracted from *Toona sinensis* seeds protect dopaminergic neurons by decreasing the quantity of microglia, and astrocytes is decreased along with the downregulation of mRNA and protein levels of inflammatory markers TNF-α and Cyclooxygenase-2 (COX-2) [65]. **Figure 4** represents the mechanism of Phytochemicals in an anti-neuroinflammatory pathway in PD.

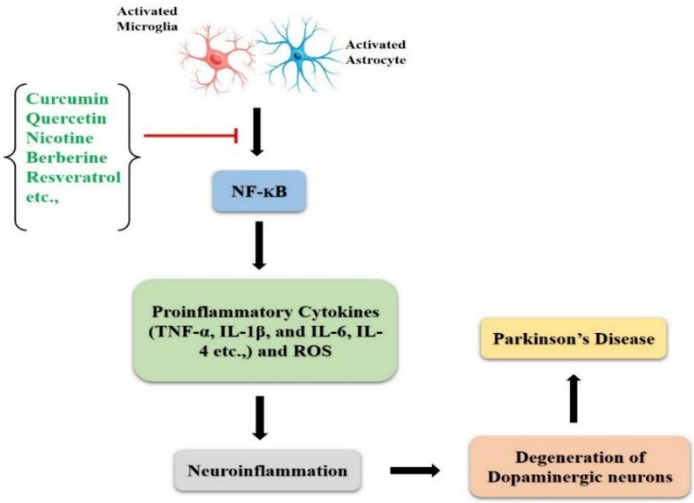

Figure 4: Role of Phytochemicals as Neuroprotective Agents Against Neuroinflammation in PD

Neurotransmitters modulation

Under pathological circumstances, an excess of glutamate in the synaptic cleft can over-activate glutamate receptors, resulting in neuronal death [66]. Elevated glutamate levels in the basal ganglia are directly linked to motor incoordination and dyskinesias experienced by PD patients [67]. The balance between dopamine and acetylcholine is necessary for the striatum's motor function, whereas glutamatergic and Gamma-Aminobutyric Acid (GABA) inputs regulate dopaminergic activity in the Substantia Nigra pars compacta (SNpc). Serotonin (5-HT) producing neurons also affect the release of dopamine in the striatum in PD [68]. The onset and progression of both motor and non-motor symptoms in PD may be due to dysregulation of the GABA system [69]. Phytochemicals like Naringenin, hesperetin, and quercetin have demonstrated the ability to improve DA uptake. By enhancing the synthesis of 5-HT, naringenin also stimulates serotonergic neurotransmission [70]. Ginsenoside Rb1 exhibits promise in PD models and offers neuroprotection against glutamate-induced excitotoxicity by altering glutamate signaling [71,72]. Several tea catechins, including epicatechin, epigallocatechin-3-gallate, catechin, and flavonol-like quercetin, are potent human catechol-O-methyltransferase (COMT) inhibitors. Tea flavonoids are a highly powerful class of neuroprotectors due to the actions of catechins and their strong antioxidant qualities [73,74].

Mitigation of α-synuclein Aggregation

A primary focus on treatment for PD is α-synuclein and a key approach to improving the condition is to prevent its aggregation, oligomerization, and fibrillation. Studies have shown that plant extracts and phytochemicals have neuroprotective effects on oligomerization and fibrillation by targeting several crucial stages of α-synuclein synthesis [75]. In PD, Curcumin prevented glial-associated inflammation,

restored GSH levels, and inhibited the production of ROS to mediate its inhibitory action on α-synuclein aggregation [76]. Treatment with dihydromyricetin (DHM), a key compound isolated from the stems and leaves of *Ampelopsis grossedentata* and salvianolic acid B (Sal B), a bioactive component found in *Salvia miltiorrhiza Bunge*, both *in vitro* and *in vivo*, successfully prevented the accumulation and aggregation of α-Syn fibrils [77]. Gallic acid inhibits the development of α-synuclein amyloid fibrils [78], and ginsenoside-Rg1, one of the active ingredients in ginseng, also lowers oligomeric, phosphorylated, and disease-related α-synuclein in the SNpc in PD [79]. **Figure 5** represents the role of phytochemicals in the mitigation of α-synuclein aggregation in PD.

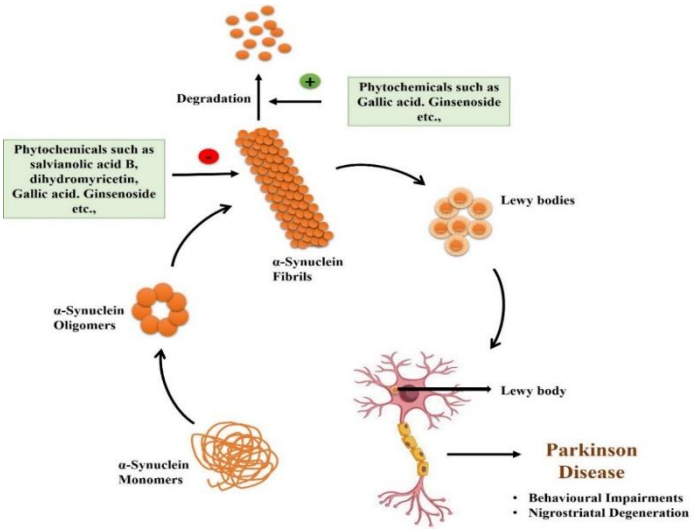

Figure 5: Role of Phytochemicals as Neuroprotective Agents Against α-synuclein aggregation in PD

Polyherbal Formulations in Parkinson's Disease

Moringa concanensis and Sesbania grandiflora

Manjusha *et al.* 2022 evaluated the neuroprotective effects of ethanolic extracts of *Sesbania grandiflora* (EESG), *Moringa concanensis nimmo* (EEMC), and their combination (MCSG-CE) which was made by employing a 1:1 ratio of their ethanolic extracts. They were tested on rats with 6-hydroxy dopamine (6-OHDA)-induced Parkinsonism. 6-OHDA+levodopa (6 mg/kg, p.o.), 6-hydroxydopamine+EEMC (100 mg/kg, p.o.), 6-OHDA+EEMC (200 mg/kg, p.o.), 6-hydroxydopamine+ EESG (100 mg/kg, p.o.), 6-hydroxydopamine+ EESG (200 mg/kg, p.o.), and 6-hydroxydopamine+ MCSG-CE (100 mg/kg, p.o.) were the eight groups each containing six rats. Each animal was subjected to its treatment for seven days following the induction. The MCSG-receiving groups' catalepsy scores notably dropped in contrast to the pre-treatment group. This implies that dopaminergic neurotransmission in the striatum, which lessens muscular rigidity and restores voluntary movements, is positively impacted by MCSG [80].

Nigella sativa, Prunus dulcis, Piper longum, Cucurbita pepo,* and *Piper nigrum

Kishore *et al.* 2022 evaluated haloperidol (1 mg/kg i.p.) and reserpine (1 mg/kg i.p.) induced PD in healthy male Swiss albino rats. PHE (Poly herbal extract) of *Piper nigrum, Nigella sativa*, *Cucurbita pepo*, *Prunus dulcis*, *Piper longum,* and *Cucurbita pepo* was given at doses of 100, 200, as well as 400 mg/kg. Animals were divided into six groups

(Normal, Disease, Standard, and PHE at 100, 200, and 400 mg/kg; six animals per group). Behavioural, along with locomotor assessments, showed that PHE significantly restored activity impaired by haloperidol and reserpine on a dose-related basis, at 400 mg/kg, yielding the best results [81].

Tongtian oral liquid

Dongjie *et al.* 2022 evaluated eleven herbal ingredients comprising the poly-herbal formulation known as Tongtian oral liquid (TTKFY) in the zebrafish treated with MPTP. The study assessed TTKFY's neuroprotective properties on the growth of DA-neurons, antioxidant properties and DA-pathway-related gene expression. Following treatment with MPTP drug (70 μM) to cause PD, the larvae of Zebrafish were subjected to varying TTKFY concentrations (0.5, 1, 2, and 4 ml/L). TTKFY shielded DA-neurons, enhanced antioxidant function, behavioural problems, dopamine pathway and mRNA gene expression in a dose-related way. TTKFY treatment at four distinct dosages in MPTP-treated zebrafish embryos resulted in a notable upregulation of TH (Tyrosine hydroxylase) mRNA expression, thereby averting the neurotoxin's harmful effects on the embryo's DA-system [82].

Itrifal Muqawwi-e-Dimagh

Siddique *et al.* 2021 estimated Itrifal Muqawwi-e-Dimagh (IMD), a Unani polyherbal preparation, for its effects on transgenic *Drosophila melanogaster* neurons that express human α-synuclein. IMD is a polyherbal mixture that contains the following: *Emblica officinalis, Terminalia bellirica, Terminalia chebula, Papaver somniferum, Malva sylvestris, Rosa damascene, Coriandrum sativum, Amygdalus communis,* cane sugar, silver, and clarified butter. IMD is recommended by the Unani medical system to cure mental illness and increase mental fortitude. The recommended dosage for humans was converted into equivalents for 20g of fly food. The PD flies were permitted to consume it for an entire day before the studies. The PD flies received L-Dopa of concentration of 10^{-3} M on an individual basis. In comparison to PD flies that were not subjected to IMD, those who were treated for it showed much-reduced levels of oxidative strain and increased levels of enzymes that act as antioxidants. Additionally, tyrosine hydroxylase activity was elevated which was dose-dependent. IMD either reduces oxidative damage in the brains of PD flies through neutralizing ROS and Preventing dopaminergic neuron damage, or it prevents Lewy bodies from developing by preventing the fibrils from aggregating [83].

***Prunus amygdalus* (PA), *Arachis hypogaea* (AH), *Citrullus lanatus* (CL)**

Nandagopal *et al.* 2020 evaluated the effects of a polyherbal mixture on PD in a rat model induced by CPZ (3 mg/kg i.p.). Rats received treatment of mixture of methanolic extract of *Prunus amygdalus, Arachis hypogaea,* and *Citrullus lanatus* (MEPAC) and a standard drug for 21 days and divided into five groups (Normal, Disease, Standard, MEPAC 200

mg/kg, and 400 mg/kg; six rats per group). MEPAC treatment significantly elevated dopamine, GSH, and SOD levels while reducing MDA levels. Histopathological analysis indicated that MEPAC-treated rats had a nearly normal cerebral cortex and hippocampus, similar to the standard drug group, with mild proliferation in the hippocampal region [84].

Terminalia chebula and Rosa damascene

Kumar *et al.* 2020 examined the antiparkinsonian properties of a polyherbal suspension containing *Terminalia chebula* and *Rosa damascena* in albino rats. Thirty animals were categorized into five groups of six: Normal, Disease (haloperidol 2 mg/kg i.p. for 11 days), Standard (levodopa 6 mg/kg p.o. for 45 days), and two Treatment groups receiving haloperidol (2 milligram/kilogram i.p. for eleven days) alongside polyherbal suspension at 100 mg/kg and 200 mg/kg per oral for 45 days. Both doses of the polyherbal treatment demonstrated neuroprotective effects, reducing oxidative stress without side effects [85].

Camellia sinensis* and *Withania somnifera

Giri M *et al.* 2020 evaluated the potential of hydroalcoholic extracts of *Camellia sinensis* (HECS), *Withania somnifera* (HEWS), and a 1:1 combination mixture to stop neuronal damage from reserpine, tacrine, and haloperidol. Intraperitoneal injections of reserpine (1 mg/kg) and tacrine (5 mg/kg) were administered to Wistar rats, whereas haloperidol (0.5 mg/kg) was administered to albino mice. HECS, HEWS, and combination were given to different groups 30 minutes prior to the administration of tacrine, reserpine, and haloperidol at different concentrations of 100 mg/kg and 30 mg/kg (p.o.). The 1:1 combination exhibits antioxidant action as well as protection against neuronal injury, where 30mg/kg exhibited more observable effects against reserpine-caused hyperlocomotion, tacrine-caused vacuous chewing movements, and orofacial burst, and haloperidol-induced catalepsy [86].

Hepad S1

Kim *et al.* 2019 assessed PD models *in vivo* and *in vitro,* The study looked into the neuroprotective properties of the herbal supplement Hepad S1, a remedy for various disease. The plants *Cnidii Rhizoma*, *Atractylodis Rhizoma, Paeonia Japonica, Glycyrrhizae Radix et Rhizoma Poria cocos Wolf* and *Zizyphi Semen*are are combined to make Hepad S1. The male Sprague-Dawley rats were given MPTP to induce PD. Hepad S1 at dose of 200, 300, 400, and 500 mg/kg/day administered orally for 4 weeks inhibited the MPP+ ability to reduce GSH and increase lipid peroxidation within cells, demonstrating anti-oxidant action. Animals treated with Hepad S1 had higher Orexin A levels in the serum and serotonin. Hepad S1 also improved dopamine levels and complex I enzyme activity in SN [87].

Gami–Chunggan

Ahn *et al.* 2019 assessed Gami-Chunggan Formula's

(GCF) benefits of neuroprotection in chronic PD models in animals. *A. gigas* root, *G. jasminoides Ellis* fruit, *P. lactiflora* root, *L. chuanxiong* root, *S. aromaticum* bud, *P. suffruticosaandrews* root bark, *B. falcatum* Linne root, and *A. rugosa O. Kuntze* were the most prevalent plants in GCF. For five weeks, MPTP and probenecid were administered to C57BL/6 mice. For 38 days, GCF (300 mg/kg, 100 mg/kg, and 200 mg/kg) was given concurrently alongside MPTP injection. GCF was also given to the animals with overexpressed A53T α-synuclein for 60 days. GCF reduced motor impairment in mice with A53T α-synuclein overexpression and those induced by MPTP. Additionally, GCF prevented the depletion of SN neurons and dopaminergic fibers within the striatum [88].

***Bacopa monnieri* (BM), *Mucuna Pruriens* (MP), *Embelica Officinalis* (EO) and *Withania somnifera* (WS)**

Srivastava *et al.* 2019 evaluated polyherbal formulations (PHFs 1–5) that were made using extracts of BM, EO, MP, and WS in ratios of 1:1:1:1, 1:2:2:2, 2:1:2:2, 2:2:1:2, and 2:2:2:1. These PHFs were refined to enhance their antioxidant capabilities by employing the DPPH assay and PHF1 showed higher activity and was selected for treatment. Mice were categorized into control, MPTP (1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine) group, and MPTP+PHF1 groups, with PHF1 administered intraperitoneally at 50 mg/kg body weight. MPTP treatment (20 mg/kg orally) was given over two weeks, with PHF1 pre-treatment. Results showed that PHF1 significantly improved dopaminergic neurons, reduced apoptosis, enhanced antioxidant activity, and reestablished dopamine levels in the brains of mice with PD induced by MPTP in comparison with the group that received only MPTP [89].

DA-9805

Eo *et al.* 2019 and Jeong *et al.* 2018 assessed DA-9805, a standardized polyherbal extract comprising *Angelica Dahuricae Radix, Moutan Cortex Radix,* and *Bupleuri Radix*, demonstrated neuroprotective effects against 6-Hydroxydopamine-caused cytotoxicity both *in vivo* and *in vitro*. Through the ERK-Nrf2 pathway, it increased PC12 cell survival, inhibited apoptosis, and triggered antioxidative enzymes. In 6-OHDA-treated animals, DA-9805 maintained locomotion, dopamine transmission, and dopaminergic neurons. In SH-SY5Y cells treated with MPP+, it decreased ROS, maintained mitochondrial activity, and restored tyrosine hydroxylase expression. DA-9805 alleviated bradykinesia, preserved striatal and SNpc neurons, increased dopamine levels, and controlled mitochondrial genes and AKT phosphorylation in the insulin pathway in MPTP-induced PD animals [90,91]. **Huh *et al.* 2022** also assessed DA-9805, which improved neurotransmitter imbalances and a motor impairment in 6-OHDA-induced PD animals. In the ipsilateral striatum and SNpc, it restored the expression of choline acetyltransferase, dopamine

transporter, and tyrosine hydroxylase, suggesting neuroprotection [92]

BR-16A **(Mentat®)**

Kumar *et al.* 2006 assessed BR-16A, an herbal psychotropic blend containing *Bacopa monnieri, Acorus calamus, Tinospora cordifolia, Centella asiatica, Withania somnifera, Embelica officinalis, Saussurea lappa, Evolvulus alsinoides, and* Triphala *(Terminalia belerica, Terminalia arjuna, Terminalia arjuna)* at dosages of 50 and 100 mg/kg (oral) for its neuroprotection in haloperidol (1 mg/kg i.p.) and reserpine (2 mg/kg i.p.) induced catalepsy in mice. The individual *Withania somnifera* and BR-16A at doses 50 and 100mg/kg showed significant protection against catalepsy, indicating they may influence both dopaminergic and serotonergic receptor-mediated neurotransmission [93].

NR-ANX-C

Nair V *et al.* 2007 assessed NR-ANX-C, a PHF containing *Ocimum sanctum, Withania somnifera*, *Camellia sinensis*, Shilajit and Triphala for anticataleptic efficacy. Catalepsy was induced by intraperitoneal administration of haloperidol (1mg/kg). The duration of the animal's-imposed posture was used to calculate its catalepsy score. All NR-ANX-C treated groups showed a significant decrease in cataleptic scores and SOD activity, with the NR-ANX-C (25 mg/kg) treated group experiencing the greatest reduction [94].

Table 2: Phase-wise Clinical Insights of Polyherbal Formulations in Parkinson's Disease Treatment

Formulation	Study Type	Trial Phase	Clinical Trial Id	Study start date	Status
Herbal Medicinal Mixture (Roucongrong, Heshouwu)	Interventional	Phase 2 Phase 3	NCT00656253	2008	Completed
Huanglian Wendan, Liu Wei Di Huang, Jin Gui Shen Qi, Bu Yang Huan Wu, Tian Ma Gou Teng	Interventional	Phase2 Phase 3	NCT05001217	2022	Completed

Composed of Roucongrong and adjuvant	Interventional	Phase2 Phase 3	NCT00629161	2008	Unknown Status
Astragalus membranaceous (Fisch.), Bunge, *Rehmannia glutinosa libosch*	Interventional	Phase 2	NCT02616120	2015	Recruiting

Challenges associated with the polyherbal formulation

As long as herbal remedies are proven safe for treating minor ailments, most countries approve them based on traditional references. Nonetheless, there are growing suggestions that polyherbal therapy could help with more severe conditions for which there are no conventional treatments. This emphasizes how regulatory frameworks, backed by scientific and clinical data, are necessary to guarantee their safety, effectiveness, and quality. Inadequate regulation, uncontrolled distribution, misuse, and subpar product quality are frequently the causes of adverse consequences in polyherbal therapy. Furthermore, there are serious worries regarding herb-drug interactions, especially for people taking several drugs from several doctors who might not coordinate their care [95]. The primary obstacle in employing PHFs for preventive or therapeutic purposes is a lack of scientific information regarding their metabolite profiles, human-equivalent dosages, unknown side effects, and potential counteragents [96]. **Figure 6** illustrates various problems associated with polyherbal formulation.

Plant combinations can produce intricate and often unexpected interactions. While enhancing therapeutic outcomes is typically the aim of these combinations, more research on herb-herb interactions is still required, and it isn't easy to forecast the effects of the numerous active ingredients in herbal preparations [97]. Because diverse medicinal plants contain a wide variety of chemical components, creating a stable polyherbal mixture is a difficult undertaking. Therefore, whether or not ingredients with specific therapeutic activity are identified, the complete herbal medication or herbal drug preparation is considered an active drug material. The chemical composition, solubility, absorption, and therapeutic response of these medications are all altered as a result of the incompatibility [98].

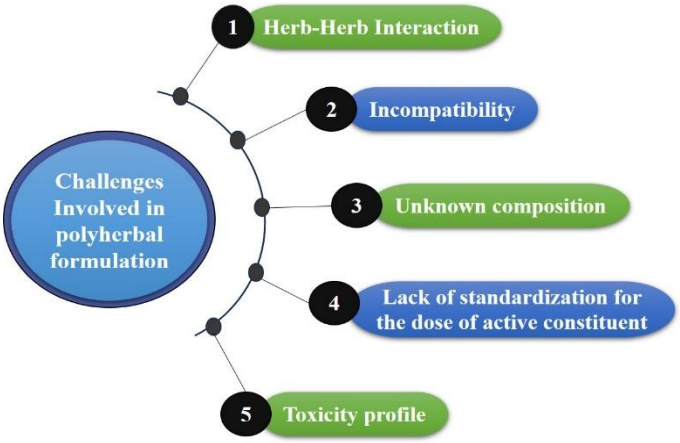

Figure 6: Various Challenges in Polyherbal Formulation

The fact that many plant-based products may interact with drugs or foods, but that data is frequently unavailable, makes quality control and drug-herb interactions serious disadvantages [99]. It is difficult to guarantee the safety and non-toxicity of polyherbal compositions since interactions between different herbs can have unanticipated consequences. To minimize potential risks, thorough toxicity testing and safety assessments are necessary [100]. Extensive safety evaluations and toxicity testing are required to reduce possible hazards [100]. It is quite challenging to standardize herbal treatments because of their inherent polypharmacy. Establishing robust quality control protocols is essential. Each herb's active ingredient must be properly identified and measured using advanced analytical techniques like HPLC [101]. The pharmaceutical industry works to develop internal standards based on the quantification of marker compounds. The task of integrating qualitative fingerprinting with other physicochemical quality measures is still ongoing, but these issues should soon be resolved [102].

Scope and Future Perspectives

PHFs hold considerable potential in addressing the complex, multifactorial pathology of PD. Through their modulation of oxidative stress, neuroinflammation, mitochondrial dysfunction, and α-synuclein aggregation, they provide multitarget effects and provide a comprehensive treatment approach. Both motor and non-motor symptoms may be relieved by PHFs and traditional herbal medicines, according to recent studies (2022–2025), especially when combined with conventional pharmaceutical drugs. The bioavailability, blood–brain barrier penetration, and sustained release of phytoconstituents have been greatly improved by advances in nanotechnology, such as liposomes, solid lipid nanoparticles, and dendrimers; green nanotechnology offers safer and more environmentally friendly substitutes. Despite these developments, clinical translation is still hampered by issues like contamination, inconsistent herbal composition, a lack of standards, and a lack of long-term safety evidence. In order to corroborate preclinical findings and develop consistent dose regimens, future initiatives should focus on large-scale, randomized clinical trials. Furthermore, combining network pharmacology, omics technology, and mechanistic research on herb-drug interactions could help elucidate the molecular

underpinnings of synergism. PHFs and cutting-edge medication delivery systems may eventually be able to supplement current therapies and develop into evidence-based, individualized approaches to the management of Parkinson's disease.

Conclusion

In the field of neurodegenerative illnesses, PD remains a major obstacle as existing treatments mainly target symptom relief instead of stopping or reversing the disease's progression. Polyherbal formulations offer a promising alternative for holistic care, leveraging the combined effects of various bioactive compounds to provide neuroprotective benefits, counter oxidative stress, and modulate neuroinflammatory reactions. The synergistic interactions among these herbal ingredients have the potential to improve both motor and non-motor functions while minimizing the side effects often associated with conventional treatments. Emerging evidence supports the effectiveness of polyherbal combinations in enhancing motor function, mitigating non-motor symptoms, and enhancing everyone's standard of living for those affected by PD. Future research should prioritize well-structured clinical trials and thorough mechanistic studies to realize their therapeutic capabilities fully. Such initiatives are crucial for establishing a robust body of evidence to integrate polyherbal therapies into standard treatment strategies for Parkinson’s disease.

References:

1. Re F, Gregori M, Masserini M. Nanotechnology for neurodegenerative disorders. Maturitas. 2012 Sep 1;73(1):45-51.
2. Savitt JM, Dawson VL, Dawson TM. Diagnosis and treatment of Parkinson disease: molecules to medicine. The Journal of clinical investigation. 2006 Jul 3;116(7):1744-54.
3. Dovonou A, Bolduc C, Soto Linan V, Gora C, Peralta Iii MR, Lévesque M. Animal models of Parkinson’s disease: bridging the gap between disease hallmarks and research questions. Translational neurodegeneration. 2023 Jul 19;12(1):36.
4. Schapira AH, Chaudhuri KR, Jenner P. Non-motor features of Parkinson disease. Nature reviews neuroscience. 2017 Jul;18(7):435-50.
5. Davie CA. A review of Parkinson’s disease. Vol. 86, British Medical Bulletin. 2008. p. 109–27.
6. Bertram L, Tanzi RE. The genetic epidemiology of neurodegenerative disease. The Journal of clinical investigation. 2005 Jun 1;115(6):1449-57.
7. Parkinson J. An essay on the shaking palsy. The Journal of neuropsychiatry and clinical neurosciences. 2002 May;14(2):223-36.
8. Feigin VL, Nichols E, Alam T, Bannick MS, Beghi E, Blake N, Culpepper WJ, Dorsey ER, Elbaz A, Ellenbogen RG, Fisher JL. Global, regional, and national burden of neurological disorders, 1990–2016: a systematic analysis for

the Global Burden of Disease Study 2016. The Lancet Neurology. 2019 May 1;18(5):459-80.

9. World Health Organization. Launch of WHO's Parkinson disease technical brief. World Health Organization: Geneva, Switzerland. 2022.
10. Bhidayasiri R, Sringean J, Phumphid S, Anan C, Thanawattano C, Deoisres S, Panyakaew P, Phokaewvarangkul O, Maytharakcheep S, Buranasrikul V, Prasertpan T. The rise of Parkinson's disease is a global challenge, but efforts to tackle this must begin at a national level: a protocol for national digital screening and "eat, move, sleep" lifestyle interventions to prevent or slow the rise of non-communicable diseases in Thailand. Frontiers in neurology. 2024 May 13; 15:1386608.
11. Monge-Fuentes V, Biolchi Mayer A, Lima MR, Geraldes LR, Zanotto LN, Moreira KG, Martins OP, Piva HL, Felipe MS, Amaral AC, Bocca AL. Dopamine-loaded nanoparticle systems circumvent the blood–brain barrier restoring motor function in mouse model for Parkinson's Disease. Scientific reports. 2021 Jul 26;11(1):15185.
12. Casanova Y, Negro S, Slowing K, García-García L, Fernández-Carballido A, Rahmani M, Barcia E. Micro-and nano-systems developed for tolcapone in Parkinson's disease. Pharmaceutics. 2022 May 17;14(5):1080.
13. Bezard E. Experimental reappraisal of continuous dopaminergic stimulation against L-dopa-induced dyskinesia. Movement Disorders. 2013 Jul;28(8):1021-2.
14. Essa MM, Braidy N, Bridge W, Subash S, Manivasagam T, Vijayan RK, Al-Adawi S, Guillemin GJ. Review of natural products on Parkinson's disease pathology. Journal of Aging Research and Lifestyle 2014;3(1):1-8.
15. Jankovic J, Tan EK. Parkinson's disease: etiopathogenesis and treatment. Journal of Neurology, Neurosurgery & Psychiatry. 2020 Aug 1;91(8):795-808.
16. Pradeep S, Jain AS, Dharmashekara C, Prasad SK, Kollur SP, Syed A, Shivamallu C. Alzheimer's disease and herbal combination therapy: A comprehensive review. Journal of Alzheimer's disease Reports. 2020 Oct 24;4(1):417-29.
17. Rahman MM, Wang X, Islam MR, Akash S, Supti FA, Mitu MI, Harun-Or-Rashid M, Aktar MN, Khatun Kali MS, Jahan FI, Singla RK. Multifunctional role of natural products for the treatment of Parkinson's disease: at a glance. Frontiers in Pharmacology. 2022 Oct 6; 13:976385.
18. Parasuraman S, Thing GS, Dhanaraj SA. Polyherbal formulation: Concept of ayurveda. Pharmacognosy reviews. 2014 Jul;8(16):73.
19. Sharma P, Kishore A, De I, Negi S, Kumar G, Bhardwaj S, Singh M. Mitigating neuroinflammation in Parkinson's disease: exploring the role of proinflammatory cytokines

and the potential of phytochemicals as natural therapeutics. Neurochemistry international. 2023 Nov 1;170:105604.

20. Shokeen A, Dhawan B, Sarwat M, Gupta S. Advancement in herbal drugs for the treatment of Parkinson's disease. InTargeting Angiogenesis, Inflammation, and Oxidative Stress in Chronic Diseases 2024 Jan 1 (pp. 251-276). Academic Press.
21. Yin R, Xue J, Tan Y, Fang C, Hu C, Yang Q, Mei X, Qi D. The positive role and mechanism of herbal medicine in Parkinson's disease. Oxidative medicine and cellular longevity. 2021;2021(1):9923331.
22. Choi HS, Park MS, Kim SH, Hwang BY, Lee CK, Lee MK. Neuroprotective effects of herbal ethanol extracts from Gynostemma pentaphyllum in the 6-hydroxydopamine-lesioned rat model of Parkinson's disease. Molecules. 2010 Apr 16;15(4):2814-24.
23. Ojha RP, Rastogi M, Devi BP, Agrawal A, Dubey GP. Neuroprotective effect of curcuminoids against inflammation-mediated dopaminergic neurodegeneration in the MPTP model of Parkinson's disease. Journal of Neuroimmune Pharmacology. 2012 Sep;7(3):609-18.
24. Khatri D, Juvekar A. Combination of curcumin and ellagic acid mitigates rotenone induced locomotor, oxidative and mitochondrial deficits in mice. Parkinsonism & Related Disorders. 2018 Jan 1;46:e42.
25. Fathy SM, El-Dash HA, Said NI. Neuroprotective effects of pomegranate (Punica granatum L.) juice and seed extract in paraquat-induced mouse model of Parkinson's disease. BMC Complementary Medicine and Therapies. 2021 Apr 26;21(1):130.
26. Adebayo OG, Ben-Azu B, Akpofure E, Modo EU, Ndidiamaka IP, Enya JI, Udoeyop FA, Oritsemuelebi B, Chidebe EO, Chimezie J, Omeiza NA. Zingiber officinale Roscoe extract improves nigrostriatal dopaminergic activity in rotenone-induced Parkinsonian mice: Implication of COX-2/TNF-α/IL-6 and antioxidant enzyme crosstalk in the immunoinflammatory responses. Phytomedicine Plus. 2024 Nov 1;4(4):100610.
27. Singh B, Pandey S, Rumman M, Mahdi AA. Neuroprotective effects of Bacopa monnieri in Parkinson's disease model. Metabolic brain disease. 2020 Mar;35(3):517-25.
28. Dal Santo G, de Veras BO, Rico E, Dal Magro J, Agostini JF, Vieira LD, Calisto JF, Mocelin R, de Sá Fonseca V, Wanderley AG. Hexane extract from *Spondias mombin* L.(Anacardiaceae) prevents behavioral and oxidative status changes on model of Parkinson's disease in zebrafish. Comparative Biochemistry and Physiology Part C: Toxicology & Pharmacology. 2021 Mar 1;241:108953.

29. Gaur V, Bodhankar SL, Mohan V, Thakurdesai PA. Neurobehavioral assessment of hydroalcoholic extract of Trigonella foenum-graecum seeds in rodent models of Parkinson's disease. Pharmaceutical Biology. 2013 May 1;51(5):550-7.
30. Teerapattarakan N, Benya-aphikul H, Tansawat R, Wanakhachornkrai O, Tantisira MH, Rodsiri R. Neuroprotective effect of a standardized extract of Centella asiatica ECa233 in rotenone-induced parkinsonism rats. Phytomedicine. 2018 May 15;44:65–73.
31. RajaSankar S, Manivasagam T, Sankar V, Prakash S, Muthusamy R, Krishnamurti A, Surendran S. Withania somnifera root extract improves catecholamines and physiological abnormalities seen in a Parkinson's disease model mouse. Journal of Ethnopharmacology. 2009 Sep 25;125(3):369-73.
32. Bigham M, Mohammadipour A, Hosseini M, Malvandi AM, Ebrahimzadeh-Bideskan A. Neuroprotective effects of garlic extract on dopaminergic neurons of substantia nigra in a rat model of Parkinson's disease: motor and non-motor outcomes. Metabolic brain disease. 2021 Jun;36(5):927-37.
33. Sayyaed A, Saraswat N, Kulkarni A, Vyawahare N. Neuroprotective action of Smilax china ethanolic bark extract in treatment of a prominent aging disorder: Parkinson's disease induced by rotenone. Future Journal of Pharmaceutical Sciences. 2023 Sep 14;9(1):79.
34. Vannur A, Biradar PR, Patil V. Experimental validation of Vitex negundo leaves hydroalcoholic extract for neuroprotection in haloperidol induced parkinson's disease in rat. Metabolic Brain Disease. 2022 Feb;37(2):411-26.
35. Little CV. Simply because it works better: Exploring motives for the use of medical herbalism in contemporary UK health care. Complementary therapies in medicine. 2009 Oct 1;17(5-6):300-8.
36. Joshi CS, Priya ES, Venkataraman S. Acute and subacute toxicity studies on the polyherbal antidiabetic formulation Diakyur in experimental animal models. Journal of health science. 2007;53(2):245-9.
37. Kotmire S, Desai A, Chougule N. The advances in polyherbal formulation. Journal of Pharmacognosy and phytochemistry. 2024;13(1):210-21.
38. Zhou X, Seto SW, Chang D, Kiat H, Razmovski-Naumovski V, Chan K, Bensoussan A. Synergistic effects of Chinese herbal medicine: a comprehensive review of methodology and current research. Frontiers in pharmacology. 2016 Jul 12;7:201.
39. Zhao B. Natural antioxidants protect neurons in Alzheimer's disease and Parkinson's disease. Neurochemical research. 2009 Apr;34(4):630-8.

40. Dorszewska J, Kowalska M, Prendecki M, Piekut T, Kozłowska J, Kozubski W. Oxidative stress factors in Parkinson's disease. Neural regeneration research. 2021 Jul;16(7):1383-1391.
41. Percário S, da Silva Barbosa A, Varela EL, Gomes AR, Ferreira ME, de Nazaré Araújo Moreira T, Dolabela MF. Oxidative stress in Parkinson's disease: Potential benefits of antioxidant supplementation. Oxidative medicine and cellular longevity. 2020 (1):2360872.
42. Sutachan JJ, Casas Z, Albarracin SL, Stab BR, Samudio I, Gonzalez J, Morales L, Barreto GE. Cellular and molecular mechanisms of antioxidants in Parkinson's disease. Nutritional neuroscience. 2012 May 1;15(3):120-6.
43. Abrahams S, Haylett WL, Johnson G, Carr JA, Bardien S. Antioxidant effects of curcumin in models of neurodegeneration, aging, oxidative and nitrosative stress: A review. Neuroscience. 2019 May 15;406:1-21.
44. Suk K, Lee H, Kang SS, Cho GJ, Choi WS. Flavonoid baicalein attenuates activation-induced cell death of brain microglia. The Journal of pharmacology and experimental therapeutics. 2003 May 1;305(2):638-45.
45. Ay M, Luo J, Langley M, Jin H, Anantharam V, Kanthasamy A, Kanthasamy AG. Molecular mechanisms underlying protective effects of quercetin against mitochondrial dysfunction and progressive dopaminergic neurodegeneration in cell culture and MitoPark transgenic mouse models of Parkinson's Disease. Journal of neurochemistry. 2017 Jun;141(5):766-82.
46. Menegas S, Ferreira CL, Cararo JH, Gava FF, Dal-Pont GC, Gomes ML, Agostini JF, Schuck PF, Scaini G, Andersen ML, Quevedo J. Resveratrol protects the brain against oxidative damage in a dopaminergic animal model of mania. Metabolic Brain Disease. 2019 Jun 15;34(3):941-50.
47. Al-Brakati A, Albarakati AJ, Lokman MS, Theyab A, Algahtani M, Menshawi S, AlAmri OD, Al Omairi NE, Essawy EA, Kassab RB, Abdel Moneim AE. Possible role of kaempferol in reversing oxidative damage, inflammation, and apoptosis-mediated cortical injury following cadmium exposure. Neurotoxicity research. 2021 Apr;39(2):198-209.
48. Vijayakumar S, Prabhu S, Rajalakhsmi S, Manogar P. Review on potential phytocompounds in drug development for Parkinson disease: a pharmacoinformatic approach. Informatics in Medicine Unlocked. 2016 Jan 1;5:15-25.
49. Krishnamoorthy A, Sevanan M, Mani S, Balu M, Balaji S. Chrysin restores MPTP induced neuroinflammation, oxidative stress and neurotrophic factors in an acute Parkinson's disease mouse model. Neuroscience letters. 2019 Sep 14;709:134382.

50. Zhang Z, Li G, Szeto SS, Chong CM, Quan Q, Huang C, Cui W, Guo B, Wang Y, Han Y, Siu KM. Examining the neuroprotective effects of protocatechuic acid and chrysin on in vitro and in vivo models of Parkinson disease. Free Radical Biology and Medicine. 2015 Jul 1;84:331-43.
51. Dhanalakshmi C, Manivasagam T, Nataraj J, Justin Thenmozhi A, Essa MM. Neurosupportive Role of Vanillin, a Natural Phenolic Compound, on Rotenone Induced Neurotoxicity in SH-SY5Y Neuroblastoma Cells. Evidence-Based Complementary and Alternative Medicine. 2015;2015(1):626028.
52. Kim IS, Choi DK, Jung HJ. Neuroprotective effects of vanillyl alcohol in Gastrodia elata Blume through suppression of oxidative stress and anti-apoptotic activity in toxin-induced dopaminergic MN9D cells. Molecules. 2011 Jun 24;16(7):5349-61.
53. Makni M, Chtourou Y, Barkallah M, Fetoui H. Protective effect of vanillin against carbon tetrachloride (CCl4)-induced oxidative brain injury in rats. Toxicology and industrial health. 2012 Aug;28(7):655-62.
54. Blesa J, Trigo-Damas I, Quiroga-Varela A, Jackson-Lewis VR. Oxidative stress and Parkinson's disease. Frontiers in neuroanatomy. 2015 Jul 8;9:91.
55. Huang B, Hu G, Zong X, Yang S, He D, Gao X, Liu D. α-Cyperone protects dopaminergic neurons and inhibits neuroinflammation in LPS-induced Parkinson's disease rat model via activating Nrf2/HO-1 and suppressing NF-κB signaling pathway. International Immunopharmacology. 2023 Feb 1;115:109698.
56. Mogi M, Harada M, Narabayashi H, Inagaki H, Minami M, Nagatsu T. Interleukin (IL)-1β, IL-2, IL-4, IL-6 and transforming growth factor-α levels are elevated in ventricular cerebrospinal fluid in juvenile parkinsonism and Parkinson's disease. Neuroscience letters. 1996 Jun 14;211(1):13-6.
57. Subedi L, Lee SE, Madiha S, Gaire BP, Jin M, Yumnam S, Kim SY. Phytochemicals against TNF α-mediated neuroinflammatory diseases. International journal of molecular sciences. 2020 Jan 24;21(3):764.
58. Park G, Kim HG, Ju MS, Ha SK, Park Y, Kim SY, Oh MS. 6-Shogaol, an active compound of ginger, protects dopaminergic neurons in Parkinson's disease models via anti-neuroinflammation. Acta Pharmacologica Sinica. 2013 Sep;34(9):1131-9.
59. Subedi L, Gaire BP, Do MH, Lee TH, Kim SY. Anti-neuroinflammatory and neuroprotective effects of the Lindera neesiana fruit in vitro. Phytomedicine. 2016 Jul 15;23(8):872-81.
60. Jin X, Liu MY, Zhang DF, Zhong X, Du K, Qian P, Yao WF, Gao H, Wei MJ. Baicalin mitigates cognitive impairment and protects neurons from microglia-mediated neuroinflammation via

suppressing NLRP 3 inflammasomes and TLR 4/NF-κB signaling pathway. CNS Neuroscience & Therapeutics. 2019 May;25(5):575-90.

61. Subedi L, Cho K, Park YU, Choi HJ, Kim SY. Sulforaphane-enriched broccoli sprouts pretreated by pulsed electric fields reduces neuroinflammation and ameliorates scopolamine-induced amnesia in mouse brain through its antioxidant ability via Nrf2-HO-1 activation. Oxidative medicine and cellular longevity. 2019;2019(1):3549274.
62. Zhou Y, Wang G, Li D, Wang Y, Wu Q, Shi J, Zhang F. Dual modulation on glial cells by tetrahydroxystilbene glucoside protects against dopamine neuronal loss. Journal of neuroinflammation. 2018 May 25;15(1):161.
63. Aggarwal BB, Gupta SC, Sung B. Curcumin: an orally bioavailable blocker of TNF and other pro-inflammatory biomarkers. British journal of pharmacology. 2013 Aug;169(8):1672-92.
64. Ho CY, Weng CJ, Jhang JJ, Cheng YT, Huang SM, Yen GC. Diallyl sulfide as a potential dietary agent to reduce TNF-α-and histamine-induced proinflammatory responses in A 7r5 cells. Molecular nutrition & food research. 2014 May;58(5):1069-78.
65. Li X, Zhang Y, Wang Y, Xu J, Xin P, Meng Y, Wang Q, Kuang H. The mechanisms of traditional Chinese medicine underlying the prevention and treatment of Parkinson's disease. Frontiers in pharmacology. 2017 Sep 19;8:634.
66. Liu Y, Wang S, Kan J, Zhang J, Zhou L, Huang Y, Zhang Y. Chinese herbal medicine interventions in neurological disorder therapeutics by regulating glutamate signaling. Current neuropharmacology. 2020 Apr 1;18(4):260-76.
67. Sgambato-Faure V, Cenci MA. Glutamatergic mechanisms in the dyskinesias induced by pharmacological dopamine replacement and deep brain stimulation for the treatment of Parkinson's disease. Progress in neurobiology. 2012 Jan 1;96(1):69-86.
68. Barone P. Neurotransmission in Parkinson's disease: beyond dopamine. European journal of neurology. 2010 Mar;17(3):364-76.
69. Alharbi B, Al-Kuraishy HM, Al-Gareeb AI, Elekhnawy E, Alharbi H, Alexiou A, Papadakis M, Batiha GE. Role of GABA pathway in motor and non-motor symptoms in Parkinson's disease: a bidirectional circuit. European Journal of Medical Research. 2024 Mar 27;29(1):205.
70. Rebas E, Rzajew J, Radzik T, Zylinska L. Neuroprotective polyphenols: a modulatory action on neurotransmitter pathways. Current neuropharmacology. 2020 May 1;18(5):431-45.
71. Radad K, Gille G, Moldzio R, Saito H, Rausch WD. Ginsenosides Rb1 and Rg1 effects on mesencephalic dopaminergic cells stressed with glutamate. Brain research. 2004 Sep 17;1021(1):41-53.

72. Zhang YL, Liu Y, Kang XP, Dou CY, Zhuo RG, Huang SQ, Peng L, Wen L. Ginsenoside Rb1 confers neuroprotection via promotion of glutamate transporters in a mouse model of Parkinson's disease. Neuropharmacology. 2018 Mar 15;131:223-37.
73. Kang KS, Yamabe N, Wen Y, Fukui M, Zhu BT. Beneficial effects of natural phenolics on levodopa methylation and oxidative neurodegeneration. Brain research. 2013 Feb 25;1497:1-4.
74. Kang KS, Wen Y, Yamabe N, Fukui M, Bishop SC, Zhu BT. Dual beneficial effects of (-)-epigallocatechin-3-gallate on levodopa methylation and hippocampal neurodegeneration: in vitro and in vivo studies. PloS one. 2010 Aug 5;5(8):e11951.
75. Javed H, Nagoor Meeran MF, Azimullah S, Adem A, Sadek B, Ojha SK. Plant extracts and phytochemicals targeting α-synuclein aggregation in Parkinson's disease models. Frontiers in Pharmacology. 2019 Mar 19;9:1555.
76. Sharma N, Nehru B. Curcumin affords neuroprotection and inhibits α-synuclein aggregation in lipopolysaccharide-induced Parkinson's disease model. Inflammopharmacology. 2018 Apr;26(2):349-60.
77. Wu JZ, Ardah M, Haikal C, Svanbergsson A, Diepenbroek M, Vaikath NN, Li W, Wang ZY, Outeiro TF, El-Agnaf OM, Li JY. Dihydromyricetin and Salvianolic acid B inhibit alpha-synuclein aggregation and enhance chaperone-mediated autophagy. Translational neurodegeneration. 2019 Jun 15;8(1):18.
78. Liu Y, Carver JA, Calabrese AN, Pukala TL. Gallic acid interacts with α-synuclein to prevent the structural collapse necessary for its aggregation. Biochimica et Biophysica Acta (BBA)-Proteins and Proteomics. 2014 Sep 1;1844(9):1481-5.
79. Ardah MT, Paleologou KE, Lv G, Menon SA, Khair SB, Lu JH, Safieh-Garabedian B, Al-Hayani AA, Eliezer D, Li M, El-Agnaf OM. Ginsenoside Rb1 inhibits fibrillation and toxicity of alpha-synuclein and disaggregates preformed fibrils. Neurobiology of disease. 2015 Feb 1;74:89-101.
80. Manjusha V, Suresh D, Venkatachalam V. Antiparkinsonian activity of moringa concanensis and sesbania grandiflora in 6-hydroxy dopamine induced parkinsonism in rats. JMPAS. 2022 Jan;11(1):4324-7.
81. Kishore DV, Siddiqui FM, Khasim SM, Baburao s. evaluation of anti-parkinson's activity of a polyherbal mixture in experimental mice.
82. Dongjie S, Rajendran RS, Xia Q, She G, Tu P, Zhang Y, Liu K. Neuroprotective effects of Tongtian oral liquid, a Traditional Chinese Medicine in the Parkinson's disease-induced zebrafish model. Biomedicine & Pharmacotherapy. 2022 Apr 1;148:112706.

83. Siddique YH, Naz F, Rashid M, Mian S. Effect of Itrifal Muqawwi-e-Dimagh (a polyherbal drug) on the transgenic Drosophila model of Parkinson's Disease. Phytomedicine Plus. 2021 Nov 1;1(4):100131.
84. Nandagopal A, Ali Khan MA. Neuroprotective effect of polyherbal formulation in'Parkinson's animal model. Asian J Pharm Clin Res. 2020;13(3):121-5.
85. Kumar A, Mishra K. PHARMACOLOGICAL EVALUATION OF POLYHERBAL SUSPENSION FOR ANTIPARKINSONS ACTIVITY IN ALBINO RAT. International Journal of Pharmaceutical Sciences and Research. 2020;11(8):3848.
86. Giri MA, Bhalke RD, Prakash KV, Kasture SB. Evaluation of Camellia sinensis, Withania somnifera and their Combination for Antioxidant and Antiparkinsonian Effect. Journal of Pharmaceutical Sciences and Research. 2020 Aug 1;12(8):1093-9.
87. Kim DH, Choi JJ, Park BJ. Herbal medicine (Hepad) prevents dopaminergic neuronal death in the rat MPTP model of Parkinson's disease. Integrative Medicine Research. 2019 Sep 1;8(3):202-8.
88. Ahn S, Liu QF, Jang JH, Park J, Jeong HJ, Kim Y, Kim DH, Jeong G, Oh ST, Park SU, Cho SY. Gami–Chunggan formula prevents motor dysfunction in MPTP/p-induced and A53T α-synuclein overexpressed Parkinson's disease mouse model though DJ-1 and BDNF expression. Frontiers in Aging Neuroscience. 2019 Aug 28;11:230.
89. Srivastava AK, Naseer A, Gupta A. Evaluation of Neurobehavioral and Biochemical Parameters of Poly-Herbal Formulation on MPTP-Intoxicated Mice for the Treatment of PD. Current Bioactive Compounds. 2020 Dec 1;16(9):1290-8.
90. Eo H, Kwon Y, Huh E, Sim Y, Choi JG, Jeong JS, Du XF, Soh HY, Hong SP, Pak YK, Oh MS. Protective effects of DA-9805 on dopaminergic neurons against 6-hydroxydopamine-induced neurotoxicity in the models of Parkinson's disease. Biomedicine & Pharmacotherapy. 2019 Sep 1;117:109184.
91. Jeong JS, Piao Y, Kang S, Son M, Kang YC, Du XF, Ryu J, Cho YW, Jiang HH, Oh MS, Hong SP. Triple herbal extract DA-9805 exerts a neuroprotective effect via amelioration of mitochondrial damage in experimental models of Parkinson's disease. Scientific reports. 2018 Oct 29;8(1):15953.
92. Huh E, Kwon Y, Choi JG, Lim MG, Jeong JS, Park AY, Kim JH, Pak YK, Hong SP, Oh MS. DA-9805, a herbal mixture, restores motor manifestations in 6-Hydroxydopamine-induced Parkinson's disease mouse model by regulating striatal dopamine and acetylcholine levels. Frontiers in Pharmacology. 2022 Jun 15;13:903664.

93. Kumar A, Kulkarni SK. Effect of BR-16A (Mentat), a polyherbal formulation on drug-induced catalepsy in mice. Indian J Exp Biol. 2006 Jan 1;44(1):45-8.
94. Nair V, Arjuman A, Dorababu P, Gopalakrishna HN, Rao UC, Mohan L. Effect of NR-ANX-C (a polyherbal formulation) on haloperidol induced catalepsy in albino mice. Indian Journal of Medical Research. 2007 Nov 1;126(5):480-4.
95. Gupta D, Sudan P, Imran M, Wal A. The Importance of Pharmacovigilance in the Polyherbal Sector: Challenges and Future Potential.
96. Nachimuthu S, Kandasamy R, Ponnusamy R, Dhanasekaran M, Thilagar S. Neurotoxicity of polyherbal formulations: challenges and potential solutions. InMedicinal Herbs and Fungi: Neurotoxicity vs. Neuroprotection 2021 Jan 22 (pp. 187-201). Singapore: Springer Singapore.
97. Che CT, Wang ZJ, Chow MS, Lam CW. Herb-herb combination for therapeutic enhancement and advancement: theory, practice and future perspectives. Molecules. 2013 May 3;18(5):5125-41.
98. Bhope SG, Nagore DH, Kuber VV, Gupta PK, Patil MJ. Design and development of a stable polyherbal formulation based on the results of compatibility studies. Pharmacognosy research. 2011 Apr;3(2):122.
99. Sen S, Chakraborty R. Toward the integration and advancement of herbal medicine: a focus on traditional Indian medicine. Botanics: Targets and Therapy. 2015 Feb 13:33-44.
100. Kanoujia D, Lata C. Exploring the potential of medicinal plants and herbal combinations for supplementation of vitamin D deficiency: a critical review. International Journal of Advances in Engineering Sciences and Applied Mathematics. 2025 Aug 26:1-8.
101. Naveen P, Lingaraju HB, Prasad KS. Rapid development and validation of improved reversed-phase high-performance liquid chromatography method for the quantification of mangiferin, a polyphenol xanthone glycoside in Mangifera indica. Pharmacognosy Research. 2017 Apr;9(2):215.
102. Wani MS, Parakh SR, Dehghan MH, Polshettiwar S, Chopade V, Chepurwar S. Herbal medicine and its standardization. Pharmaceutical Reviews. 2007;5(6):411038-49

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Review Article

Personalized Medicine: Revolutionizing Treatment Approaches For Individualized Patient Care

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Abstract

Traditional medical protocols have relied on standardized approaches that often overlook individual variability in disease presentation and drug response, resulting in suboptimal outcomes and adverse effects. The advent of personalized medicine, driven by advances in genomic sequencing, molecular profiling, and biomarker analysis, enables the design of individualized treatment plans based on each patient's unique genetic and molecular characteristics. Genetically modified organisms and recombinant technologies now support the development of targeted drug therapies and biologics, allowing precise intervention at the molecular level. Further, the integration of digital health technologies and advanced analytics including wearable devices, remote monitoring, and machine-learning platforms has elevated diagnostics and disease management through continuous data collection and real-time adaptation. Global market trends indicate substantial investment in precision medicine as healthcare systems prioritize genomics-based diagnostics, biomarker-driven therapies, and data-centric models of care. As a result, the convergence of genomic science, biotechnology, and digital health is ushering in a new era of highly effective, patient-centric medicine characterized by predictive diagnostics, preventative strategies, and tailored treatments, ultimately transforming the landscape of healthcare and improving patient outcomes worldwide.

Keywords: Personalized medicine, genomic sequencing, targeted therapy, molecular diagnostics, digital health, market trends

Introduction

Personalized medicine is the process of identifying a patient's unique characteristics like genomic, biochemical, behavioral, etc., which may provide insight into how they will react to an intervention and then they can be treated. The term “individualized medicine” is also known as “Personalized medicine” [1]. Because, it makes use of the technology to meticulously compile and assess a patient's individual medical information for that patient alone [2]. It is relatively a new field of medicine where specific therapeutic techniques are prescribed for an individual. This approach has been developed based on

pharmacogenetic and pharmacogenomics data and information [3].

The phrase "personalized medicine" was first used in published works in 1999, while the fundamental ideas behind the area were established in the early 1960s [4]. The increasing trend of giving patients customized care gave rise to personalized medicine [5].

The goal of personalized medicine is to minimize drug toxicity and maximize therapy efficacy for each patient and it exemplifies appropriate patient selection, the best medication and dosage, and timely administration [6].

Early studies in personalized medicine focused on genetic differences that predict response to treatment, and this led to the expansion of clinical practice guidelines informed by the genome.[7] Personalized treatments have been connected to instances of genetically-mediated medication pharmacokinetics. This was partly caused by the scientific community in the biomedical sciences' understanding of the role of drug-metabolizing enzymes in the body's response to drugs [1]. According to the FDA, by concentrating more intently on prevention and treatment, PM seeks to enhance patient outcomes and reduce hazards. PM attempts to classify people according to how differently they respond to various therapeutic agents for their specific ailments, as opposed to developing novel drugs for patients. For example, Herceptin is a highly beneficial drug for approximately 20–30% of breast cancer patients [8]

Genomic and proteomic information, along with patient characteristics, are used by the field of personalized medicine to tailor care. The patient's genetic peculiarities are taken into account when selecting medications or treatment programs, which lowers the possibility of side effects and ensures successful outcomes. Complicated inflammatory periodontal diseases are complex and damage both the soft tissue and bone of teeth. Genetics plays a significant role in periodontitis. Individualized care is provided based on each person's unique genetic profile through the use of molecular tests such AmpliChip CYP450 test, fluorescence in-situ hybridization (FISH), microarray testing, gene mapping, DNA profiling, and receptor gene amplification [9].

Indeed, as noted by Swanskin et al., personalized medicine is receiving more and more attention. In order to treat patient diseases or predispositions, this signifies a significant shift away from "one size fits all" treatments and towards new strategies that can produce the best results: customized therapies [10]. The enormous increase in data in the health sector in recent years is one startling issue. According to certain reports, the healthcare industry is producing 48% more data each year [11]. According to Lopes-Junior, the present challenge is to provide more predictive diagnoses, treatments, and individualized care to targeted individuals and communities in order to transform the increasing medical data acquired in the health sector into clinical benefits for patients. [10]

It appears that everyone has a similar understanding of what personalized medicine is. Upon closer inspection, there are significant differences between the definitions

of personalized medicine that are already in use.

1. A medical approach that suggests customizing treatment, with choices and practices being adapted to the individual patient by use of genetic or other information, is called personalized medicine, for instance.
2. The customization of medical care to the unique needs of every individual patient." It does not actually refer to the process of developing medications or medical equipment that is specific to each patient. Instead, it entails the capacity to categorize people into subgroups that are differently or uniquely vulnerable to a given illness or responsive to a given course of therapy.
3. A type of treatment that prevents, detects, and treats disease by utilising data about an individual's genes, proteins, and surroundings.

These three definitions are enough to show how widely definitions of personalized medicine differ, even though there are many more out there.[12]

The FDA claims that by more precisely focusing on prevention and treatment, PM aims to increase patient benefits and lower hazards. PM aims to classify people into subpopulations that differ in how they react to a treatment agent for their particular ailment, rather than to develop novel medications for patients. For instance, about 20–30% of patients with breast cancer who have increased HER2 expression find Herceptin to be an incredibly helpful medication. However, due to HER2 gene alterations, certain patients with high HER2 are naturally resistant to Herceptin. Thus, stratifying patients based on their molecular characteristics—both genetic and epigenetic—enables the best possible use of Herceptin in treating breast cancer. [8]

Advantages:

- Personalized medicine has the potential to enhance clinical practice efficiency and reduce healthcare costs by administering the appropriate medication at the precise dose and time [13].
- The efficacy of personalised medicine strategies surpasses that of traditional medicine, primarily due to the availability of several customised or personalised therapeutics, such as mutation-specific medications like ivacaftor to treat cystic fibrosis and autologous CAR-T cell transplant therapies for specific cancer types [1].
- Personalized medicine can be utilized not only for disease treatment but also for early disease detection and prevention [1].
- Personalized medicine gives the genetic differences amongst people to help with illness diagnosis, prophylaxis, and treatment [14].
- Improved diagnostic evaluation that enables earlier and more successful therapeutic interventions [14].
- Less medication side effects and greater pharmacological efficacy [14].

- It helps in detection of genetic predisposition and application of preventive measures [14].

Disadvantages:

One of the many challenges that come with personalized pharmaceuticals is getting regulatory bodies to authorize their regular use. In addition, there are several challenges to the industry's widespread adoption of personalized drugs by various healthcare stakeholders, including physicians, business executives, insurance providers, and patients [1].

- Personalized medicine cannot be produced without further study, particularly genetic investigations [14].
- The underlying biology of disease is not well understood.
- It has Restricted Resources.
- It requires more usage of recent diagnostic techniques [15].

Another difficulty for PM is dealing with social, legal, and moral issues [16].

Benefits

The benefits of personalized medicine are vast and transformative. Personalized medicine improves diagnostic accuracy and provides tailored treatment recommendations based on patient-specific attributes, leading to targeted therapy that enhances treatment efficacy, reduces adverse effects, supports better disease prognosis, and encourages greater patient involvement. It also lowers healthcare costs and fosters research and innovation [10]. By offering superior medical care, it significantly benefits both society and healthcare systems. Furthermore, PM enhances the productivity of novel pharmaceutical development [6] and positively impacts the biopharmaceutical sector by reducing the time and cost associated with drug development [16]. PM enables better medication selection, reduces adverse effects, increases patient compliance, and shifts the focus of medicine from reactive treatment to proactive prevention. This not only improves cost-effectiveness but also builds patient confidence post-marketing through approval of novel therapeutic strategies and changing medical paradigms [8]. The approach avoids trial-and-error prescriptions, thereby preventing treatment delays and improving the quality of life, while also making care more affordable [17]. Although the net monetary benefit (NMB) of PM therapies ranges from zero to negative in some cases, particularly in the 'neoplasm' group, gene therapies were found to have more positive health impacts compared to other PM interventions despite their higher costs. Pricing policies might be necessary to reduce expenses in such interventions. The lack of statistically significant factors explaining the variance in cost-effectiveness suggests that PM, as a term, might be too broad; categorizing it further could help identify profitable investment areas [18].

In terms of applications, personalized medicine facilitates early disease detection through surveillance, supports improved treatment strategies, and offers better health outcomes compared to non-personalized approaches. It optimizes treatment by using the right drug in the right dosage, especially accounting for

genetic variations that influence metabolism [6]. PM also promotes adherence to preventive measures in at-risk patients, utilizes pharmacogenomics in cancer therapy, and supports the development of companion diagnostics [19]. It aims to eliminate side effects, minimize pharmacological risks, and ensures treatment is genetically tailored, including precise dosage administration and conceptual drug efficacy validation [9]. Ultimately, personalized medicine integrates an inclusive therapeutic approach based on genetic information [20], and its central philosophy is to design treatments that are as unique as the patient's symptoms—leveraging targeted strategies for better disease control. It leads to improved diagnostic techniques, timely detection, advanced drug development methods, and more precise treatments tailored to the patient's genetic profile [21]

Market Trend

The global personalized medicine market was estimated at USD 529.28 billion in 2023, and it is expected to increase at an 8.20% CAGR from 2024 to 2030. The personalized medicine market is primarily driven by the increasing demand for novel drug discoveries to tackle the global rise in cancer and other disorders. Furthermore, various collaborations between academics and market companies are expected to boost the personalized medicine market growth (Table 1).

Market Concentration & Characteristics

The market grows at a medium rate, and the pace is increasing. Due to ground-breaking innovations, the market for personalized medicine is expanding rapidly. Tailored medicines, diagnostics, and preventive measures are driven by cutting edge technologies such as proteomics, genomics, and artificial intelligence. This changing environment, which is influencing healthcare through previously unheard-of levels of effectiveness and precision, presents an abundance of opportunities for experts and investors.

U.S. Personalized Medicine Market Trends

It is predicted that the U.S. personalized medicine market would expand between 2024 and 2030 due to the existence of major players like Abbott Laboratories, GE Healthcare, Illumina, Inc., ASURAGEN, Inc., Danaher, and 23andMe, Inc. Important businesses are joining forces more frequently to increase access to personalized medicine through alliances, collaborations, and agreements.

Europe Personalized Medicine Market Trends

Europe's Market Trends for Customized Medicine The market for customized medication in Europe is anticipated to expand significantly between 2024 and 2030 at a compound annual growth rate (CAGR of substantial). Moreover, during the course of the projected period, the existing states of development in Germany, the UK, and France should present profitable prospects for market expansion.

The UK's personalized medicine market is expected to grow in concert with the European market as a result of the increasing advancement of companion diagnostics (CDxs) and the subsequent launch of molecular diagnostics by a major player in the industry.

The personalized medicine market in China

The government's encouragement and participation in cross-border international partnerships for preventative and personalized medicine are responsible for the lucrative growth of the personalized medicine sector in China. The increased acceptance of Western medicine and anticipated changes to Chinese insurance laws are also anticipated to fuel industry expansion.

The Japan personalized medicine market

From 2024 to2030, the personalized medicine market in Japan is predicted to expand at a significant CAGR. This share is the result of multiple market players actively pursuing an expansion strategy to fortify their positions in the Asia Pacific area. For example, in October 2023, NTT Corporation (Japan) signed a contract to create the Japan Precision Medicine Platform (JPP) with BC Platforms AG, PRIME-R, and Bioxcellerator [25]

Table 1 – Attributes and Details

Attribute	Details
Global Personalized medicine market (2023)	US$ 327.7 Billion
Global Personalized medicine market (2033)	US$ 690.9 Billion
Global personalized medicine market CAGR (2023 to 2033)	7.8%
USA personalized medicine market CAGR (2023 to 2033)	6.9%
Key companies profile	• GE Healthcare • Illumina, Inc. • ASURAGEN, INC. • Abbott Dako A/S • Exact Science Corporation • Cepheid, Inc. • Decode Genetics, Inc. • QIAGEN • Exagen Inc. • Precision Biologics • Celera Diagnostics LLC • Biogen

- Genelex
- Genentech, Inc.
- 23andMe, Inc.

Market Segments Covered in Personalized Medicine Market Analysis

1) Personalized Medical Care
    * Genetic Testing for Diagnostics
    * Diagnostics DTC
    * Occult Lab Services
    * Mysterious Laboratory Tests
2) Personalized Medical therapeutic
    * Pharmaceuticals
    * Genomic Medicine in Pharma
    * Medical Equipment
3) Personalized Nutrition Plans and Health
    * Complementary and
    * Alternative Medicine in Retail Nutrition (26)

Future Aspects

Personalized medicine is rapidly advancing, promising significant improvements in patient care and outcomes. This approach tailor's medical treatment to the individual characteristics of each patient, such as their genetic profile, lifestyle, and environmental factors. One of the most notable future aspects of personalized medicine is the integration of advanced genomics and biotechnology, which enable more precise and predictive diagnostics. For instance, genomic sequencing can identify genetic predispositions to various diseases, allowing for early intervention and customized treatment plans [27]

Moreover, the development of personalized therapies, including targeted drugs and gene editing technologies like CRISPR, is transforming treatment paradigms. These innovations aim to address the underlying causes of diseases at a molecular level rather than just managing symptoms (28). Additionally, the incorporation of artificial intelligence (AI) and machine learning into personalized medicine is enhancing the ability to analyze complex data sets, leading to more accurate predictions and individualized treatment strategies [29]

However, these advancements also present challenges such as ethical considerations, data privacy concerns, and the need for equitable access to personalized treatments. Addressing these issues will be crucial to realizing the full potential of personalized medicine in improving health outcomes for diverse population [30]

In the long run, patients, doctors, biopharmaceutical companies, and society at large are the stakeholders most likely to profit. Medical patients are significantly impacted by PM.

In the rapidly developing field of genomic and molecular medicine, it makes sense for practitioners to gravitate towards specialization in clinical

practice. In order to ensure higher quality, pharmaceutical medicines must be more effective and have fewer side effects, which largely determine patient satisfaction with illness care.

Therapy for those affected rises in tandem with the usage of customized medication. When personalized treatment is being used, more tests ought to be done to diagnose the ailment. Utilizing customized medication will be advantageous as it will enable the user to take action [23]

Enabling doctors to create more individualized treatment plans for individual patients based on their DNA profiles will lead to better health results. A significant amount of genetic patient data will soon become accessible thanks to the data generated by this new PM approach for patient care. We will be able to direct best practices for different therapeutic goals and keep highlighting positive patient results if we have a thorough grasp of preventative medicine [5].

Conclusion

The use of application of personalized technology for the diagnosis of cancer has contributed towards a paradigm shift in the treatment of cancer. Medical experts have the freedom of choosing from a wide range of medications by using state of the art techniques which involve genomic analysis, bioinformatics and advanced imaging techniques. By incorporating the recent developments and adoption of advanced methodologies personalized medicine has achieved a greater potential of mitigating the chances of cancer and enhanced the safety and efficiency of the treatment.

The developments in the area of targeted medicines and the approval for the usage of precision medicines created the opportunity for the betterment of treatments offered to patients also making healthcare safe, efficient and convenient. The application of the modern advancements enables the discovery of underlying conditions which lead to the development of a disorder. Through proper diagnosis and early detection of the symptoms and factors responsible for the disorder can be arrested in a primitive stage to prevent the development into a stage wherein the treatment becomes difficult.

The addition of technology and acquired knowledge has led to a shift in the use of medicine with the passage of time. The introduction of precision medicines has not only changed the traditional medical diagnosis technique but also has a significant economic impact through the development of cost effective and trustworthy diagnostic methods.

The techniques which have enabled the development in the areas of cell and gene therapy which are a result of the higher investments, good clinical trial methods and an effort to target the number of diseases to introduce personalized medicines.

Personalized medicine has been developed into a technique of medicine administration which has the potential to cater to the requirement of patients who

expect outcomes such as cost effectiveness, time savings and safety. The revolution achieved will not only enable us to achieve entrepreneurial benefits but also promotes awareness among the patients about the efficacy and performance of personalized drug. The improvement in the field of personalized medicines has a positive effect and has enabled a paradigm shift in the healthcare system.

Despite having tremendous improvements in the field of medicines and healthcare, few of the concerns exist in the minds of patients. The concerns have raised due to the incorporation of genetically modified organisms (GMO). Since there is not much awareness and information exists in the minds of people there is a hindrance to follow the practice of personalized medicine. However, with proper knowledge and communication of the techniques and advancements in the field of personalized medicine can lead to a better trust in the minds of the people.

References:

1. Goetz LH, Schork NJ. Personalized medicine: motivation, challenges, and progress. Fertility and Sterility. 2018;109(6):952–63. doi:10.1016/j.fertnstert.2018.05.006. PMID: 29935653; PMCID: PMC6366451.
2. Ho D, Quake SR, McCabe ERB, Chng WJ, Chow EK, Ding X, et al. Enabling technologies for personalized and precision medicine. Trends in Biotechnology. 2020;38(5):497–518. doi:10.1016/j.tibtech.2019.12.021. PMID: 31980301; PMCID: PMC7924935.
3. Rogers K. Personalized medicine [Internet]. Encyclopaedia Britannica. 2024 Jan 29 [cited 2024 Jan 30]. Available from: https://www.britannica.com/science/personalized-medicine
4. Arjmand B, Goodarzi P, Mohamadi-Jahani F, Falahzadeh K, Larijani B. Personalized regenerative medicine. Acta Medica Iranica. 2017;55(3):144–9.
5. Erden A. Personalized medicine. Yale Journal of Biology and Medicine. 2015;88(4):349. PMID: 26930723; PMCID: PMC4654182.
6. Gajare SR, Deshmukh AS, Shinde CK. Review on personalized medicine. International Journal of Pharmaceutical Sciences Review and Research. 2021;69(1):23. doi:10.47583/ijpsrr.2021.v69i01.023.
7. Abul-Husn NS, Kenny EE. Personalized medicine and the power of electronic health records. Cell. 2019;177(1):58–69. doi:10.1016/j.cell.2019.02.039. PMID: 30901549; PMCID: PMC6921466.
8. Mathur S, Mathur S. Personalized medicine could transform healthcare: a review. Biomedical Reports. 2017;7:3–5. doi:10.3892/br.2017.922.
9. Gomathi GD, Gopalakrishnan S, Sudhakar U. Personalized medicine – a recent revolution in periodontics. International Journal of

Periodontology and Implantology. 2019;4(3):69–72.

10. Stefanicka-Wojtas D, Kurpas D. Personalised medicine: implementation to the healthcare system in Europe (focus group discussions). Journal of Personalized Medicine. 2023;13(3):380. doi:10.3390/jpm13030380. PMID: 36983562; PMCID: PMC10058568.
11. Abul-Husn NS, Kenny EE. Personalized medicine and the power of electronic health records. Cell. 2019;177(1):58–69. doi:10.1016/j.cell.2019.02.039. PMID: 30901549; PMCID: PMC6921466.
12. Redekop WK, Mladsi D. The faces of personalized medicine: a framework for understanding its meaning and scope. Value in Health. 2013;16(6):S4–9. doi:10.1016/j.jval.2013.06.005.
13. Iriart JAB. Precision medicine/personalized medicine: a critical analysis of movements in the transformation of biomedicine in the early 21st century. Cadernos de Saúde Pública. 2019;35(3):e00153118. doi:10.1590/0102-311X00153118. PMID: 30916181.
14. Salari P, Larijani B. Ethical issues surrounding personalized medicine: a literature review. Acta Medica Iranica. 2017;55(3):209–17.
15. Research and Markets. Personalized medicine market size and share analysis: global forecast 2025–2033 [Internet]. 2025 [cited 2025 Jan 10]. Available from: https://www.researchandmarkets.com/reports/5952081/personalized-medicine-market-size-share
16. Agyeman AA, Ofori-Asenso R. Perspective: does personalized medicine hold the future for medicine? Journal of Pharmacy and Bioallied Sciences. 2015;7(3):239–44. doi:10.4103/0975-7406.160040. PMID: 26229361; PMCID: PMC4517329.
17. Yousif T, Bizanti K, Elnazir B. Uses of personalized medicine in current pediatrics. International Journal of Clinical Pediatrics. 2016;5(1):1–5.
18. Thokala P, Duenas A, Sculpher M. Value in precision medicine: selected approaches, challenges, and opportunities. Value in Health. 2022;25(2):189–96. doi:10.1016/j.jval.2022.01.006.
19. Payne K, Annemans L. Reflections on market access for personalized medicine: recommendations for Europe. Value in Health. 2013;16(6):S32–8.
20. Boomika S, Ezhilarasi S, Gokulnath S, et al. Review on personalised medicine for cancer treatment. International Journal of Drug Development and Research. 2022;14(9):976.
21. Badhe P, Otari K, Badhe A. Personalized medicine: the future of modern medicine. Authorea. 2021 Dec 13.
22. Allied Market Research. Personalized medicine market size and growth insights 2031 [Internet]. 2022 [cited 2024 Jan 2]. Available from:

https://www.alliedmarketresearch.com/personalized-medicine-market-A13388

23. Di Sanzo M, Cipolloni L, Borro M, La Russa R, Santurro A, Scopetti M, et al. Clinical applications of personalized medicine: a new paradigm and challenge. Current Pharmaceutical Biotechnology. 2017;18(3):194–203. doi:10.2174/1389201018666170224105600.
24. Parmar P, et al. Personalized medicine: time for one person, one medicine. International Journal of Pharmaceutics and Drug Analysis. 2021;9(2):86–92. doi:10.47957/ijpda.v9i2.462.
25. Grand View Research. Personalized medicine market size, share and trends analysis report [Internet]. 2025 [cited 2025 Jan 5]. Available from: https://www.grandviewresearch.com/industry-analysis/personalized-medicine-market
26. Future Market Insights. Personalized medicine market outlook from 2025 to 2035 [Internet]. 2025 [cited 2025 Jan 8]. Available from: https://www.futuremarketinsights.com/reports/personalized-medicine-market
27. Collins FS, Varmus H. A new initiative on precision medicine. New England Journal of Medicine. 2015;372(9):793–5. doi:10.1056/NEJMp1500523.
28. Doudna JA, Charpentier E. The new frontier of genome engineering with CRISPR-Cas9. Science. 2014;346(6213):1258096.
29. Esteva A, Kuprel B, Novoa RA, et al. Dermatologist-level classification of skin cancer with deep neural networks. Nature. 2017;542(7639):115–8.
30. Williams J. Ethical and social issues in personalized medicine. Journal of Medical Ethics. 2019;45(4):248–53.

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Review Article

Mimosa pudica Linn: A Comprehensive Review on Phytochemical, Pharmacology, and Therapeutic Potential

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Abstract

Mimosa pudica is recognized as a sensitive plant. It is derived from the Latin word "pudica," which means shy or shrinking. Commonly known as the touch-me-not, it is a creeping annual and perennial herb. This species originates from South America and Central America. Mimosa is classified within the taxonomic group Magnoliopsida and is a member of the family *Mimosoideae.* The plant exhibits a responsive behavior by folding its leaves upon contact, subsequently reopening them. The characteristics of *Mimosa pudica* roots include bitterness, astringency, acridity, and a cooling effect. These substances are utilized in addressing conditions such as ulcers, inflammation, asthma, diarrhea, urinary issues, and fistulas. The plant is characterized by the presence of the alkaloid mimosine, while its leaf extract is noted for containing a substance similar to adrenaline. *Mimosa pudica* has a historical application in addressing urogenital disorders, dysentery, sinus issues, and in the treatment of wounds. Juices derived from fresh leaves may be utilized for both internal and external applications in the treatment of cuts and wounds. It is additionally utilized for external application on fissures, skin wounds, and ulcers. The hemostatic properties of *Mimosa pudica* contribute to the management of bleeding associated with piles. This research sought to investigate and gather various Pharmacognostic characteristics of *M. pudica.* It also represents a significant and contentious species derived from natural sources, holding great potential for future research, necessitating further investigation.

Keywords: *Mimosa pudica,* Phytochemistry, Pharmacology, Therapeutic potential, Traditional medicine, antimicrobial activity, Antioxidant activity, Wound healing.

Introduction

Mimosa pudica is known by numerous names, including sensitive plants, touch me not, humble plants, and shame plants. It belongs to the family *Mimosoideae* and is known by different names in different parts of the world [1]. *Mimosa pudica* is a creeping annual or perennial herb. It was first formally introduced by Carl Linnaeus in Species Plantarum in 1753. The word '*Mimosa*" is obtained by a Greek word means mimic and "*pudica*" is a Latin word which has a meaning, shy.

Folding movement of leaves observed by this plant, which undergoes changes in leaf orientation at night, is called nyctinasty movement and is controlled by a biological clock. The plant has 500 species and is approximately 50-70 cm high [2]. *Mimosa pudica* has compound leaves and small globular pink or mauve flower puffs. It is commonly grown as curiosity in greenhouses [3]. Phytochemical studies of It have revealed the presence of alkaloids, non-protein amino acids (mimosine), flavonoid C-glycosides, sterols, terpenoids, tannins, and fatty acids. *Mimosa pudica* (Lajwanti) or Chui Mui is a creeper belonging to the Fabaceae family. Ayurveda describes Lajwanti as tikta (bitter), kashaya (astringent), and sheetha (cold)[4]. Epidemiological studies have shown that it contains metabolites such as phenols and flavonoid compounds, which possess pharmacological properties such as antidiabetic, antimicrobial, antiulcer, antidepressants, and anti-inflammatory. Ecological studies have shown that *M. pudica* grows in all types of soil and can survive in soils with low nutrient concentrations. This usually requires disturbed soil for establishment. It is commonly seen in the wastelands and along roadsides, and is an ethnomedical plant that may be used to manage various types of diseases.[5] It is used in traditional medicine, such as Ayurveda and Unani, to treat a range of conditions, including digestive issues such as diarrhea and dysentery, skin problems such as ulcers and infections, respiratory ailments such as asthma and bronchitis, sleep disorders, epilepsy, weakness, and female reproductive health issues such as vaginal and uterine infections. The plant contains beneficial chemicals, such as alkaloids, flavonoids, tannins, and mimosine, which provide these healing qualities. However, it is a troublesome invasive species in tropical regions. It frequently overruns crops such as corn, soybeans, rice, cotton, bananas, sugarcane, coffee, and rubber, forming dense mats that block other plants, have thorny stems that complicate manual removal, and can even increase the risk of wildfire when dry. Overall, *Mimosa pudica* is both a valuable medicinal herb and persistent agricultural pest.[6] In folklore medicine, the entire plant parts, such as the roots, leaves, and flowers, are used in the treatment of several diseases, including dysentery, leprosy, pile, skin diseases, leukoderma, fever, cough, cholera, tuberculosis, biliousness, burning sensation, uterine problems, cancer, rheumatism, edema, elephantiasis, syphilis, and jaundice [10-15]. It can also be used as an antidote for snakebites and scorpion stings. This plant can be used as an herbal treatment. M. pudica is the most important controversial and effective natural origin and has a tremendous future for research [7].

Taxonomy: [8]

Kingdom: Plantae

Division: Mangoliophyta

Phylum: Tracheophyta

Class: Magnoliopsida

Clade: Tracheophytes (vascular plants)

Clade: Angiosperms (flowering plants)

Order: Fabales

Family: *Fabaceae (Leguminosae)*

Subfamily: *Mimosoideae*

Species: *Mimosa*

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Genus: *Mimosa pudica*

Sub-species: *Mimosa pudica*

VERNACULAR NAME:[2]

Kannada – Lajja, Nachika and Mudugu-davar

English – Sensitive plant

Hindi – Laajvanti and Chhui-mui

Telugu – Attapatti and Peddanidrakanni

Tamil – Tottaaladi and Thottalchnungi

Botanical Description:[9]

Mimosa pudica, also known as the sensitive or touch-me-not plant, is a low-growing, prickly subshrub native to South and Central America, but is now pantropical. It typically reaches approximately 30–50 cm tall and spreads similarly, with slender, branching stems covered in spines. Its fern-like, bipinnate leaves consist of 15–25 pairs of small, bristly leaflets that rapidly fold inward when touched, shaken, or even in darkness, a defense mechanism driven by changes in turgor pressure in specialized joints called pulvini. The plant produces clusters of pale pink to lilac globe-like flowers in the leaf axils, followed by curved, prickly pods measuring 1.5–2.5 cm long. In India, it flowers between August and October. This species was first introduced by Carl Linnaeus in 1753 to Plantarum.

Table 1 Botanical description of Mimosa pudica

Characters	*Mimosa pudica*
Plant	Short prickly branches, hairs glandular
Leaves	Bipinnate, sensitive to touch
Flowers	Axillary, globose head, lilac pink in colour
Stem	Erect, slender, prickly and well branched
Calyxes	Campanulate
Petals	Petals crenate towards base
Pods	1.5–2.5 cm long, closely prickly on sutures and falcate
Flowering and Fruiting time	August to October in India

Distribution And Habitat:[3]

Mimosa pudica is found in many parts of Asia, including Singapore, Bangladesh, Thailand, India, Nepal, Indonesia, Taiwan, Malaysia, Vietnam, Cambodia, Laos, Japan, Sri Lanka, and Philippines. It has also reached other places, such as Uganda, Ghana, Nigeria, Seychelles, Mauritius, and East Asia.

Philippines *Mimosa pudica* grows almost everywhere in the Philippines. It can be observed on roadsides, empty lots, farms, and grassy fields. In Angat and Bulacan, the plant is common in open spaces and areas where the soil has been disturbed. It spreads easily because of its seeds and its ability to grow in many types of soil. It loves sunlight and warm weather, which is why it grows well in tropical areas, such as the Philippines.

Although *Mimosa pudica* is common, it is not considered a major problem in the Philippines. However, in other countries, it is known to be an invasive species. This means that it spreads too much and can take over land where native plants are expected

to grow. In countries like Tanzania and many Pacific islands, *Mimosa pudica* is a threat to the environment in countries such as Tanzania and many Pacific islands. In Australia, it has even been declared a weed in some parts, such as the Northern Territory and Western Australia. In Queensland, people are advised to control growth.

In the United States, *Mimosa pudica* grows in states such as Louisiana, Florida, Hawaii, Texas, and others. It is also found in places such as Puerto Rico, Guam, and Virgin Islands.

In Angat, Bulacan, *Mimosa pudica* is a part of the local environment. It does not cause major problems, but can spread quickly if not controlled. Farmers and gardeners may remove it if they take up too much space or compete with the crops.

Mimosa pudica is known for its movement. It also has small pink or purple flowers and tiny thorns on its stem. Some people have used it in traditional medicine. It is believed to help treat minor wounds or skin problems, but people should always be careful and ask experts before using plants as medicines.

Mimosa pudica is a plant that grows in Angat, Bulacan, and many other parts of the Philippines. Although not native to the country, it has become common. It is interesting and fun to observe how they move. While it is a problem in some countries, in the Philippines it is mostly just a part of the native country.

Cultivation And Collection:[10]

This plant grows naturally in warm tropical areas, but also spreads to subtropical regions. It can grow well even in hilly places up to 1,300 meters above sea level. The best temperature for growth is between 22°C and 28°C, it can survive at temperatures as low as 10°C and as high as 32°C. However, they cannot survive in cold or frosty weather.

The plant prefers places where the rainfall is between 1,000 to 2,000 millimeters per year, but it can still grow in areas where the rainfall is as low as 900 mm or as high as 3,000 mm. It grows best in sunny places, but can also survive in shaded areas, although it may not grow as well. They can live in many types of soil, even if the soil is shallow or does not have many nutrients. The ideal soil pH is between 6 and 7, but it can tolerate a pH range of 5 to 7.5. It is also effective at surviving in humid and windy areas.

This plant has spread widely and has become common in many tropics and subtropics. It often grows in forests, farmlands, orchards, and pastures. Occasionally, it becomes a problem and is considered a weed, especially in dry farmlands, rice fields, and plantations. When the plant dries up, it can easily catch fire, which makes it dangerous during dry seasons.

Pharmacological activities of *Mimosa pudica*:

Wound healing activity:[11]

A 2% w/w methanolic extract of *Mimosa pudica* (Linn.) promoted faster wound healing. Compared with the control group, wounds treated with this extract showed quicker epithelialization and a higher rate of wound contraction on days 8, 12, and 16. The aqueous extract of 2% w/w *Mimosa pudica* also proved effective when applied topically to wounds, helping increase cell growth and collagen production at the injury site, which was confirmed by improved wound strength.

Additionally, there was a higher level of hydroxyproline in the wound scabs, supporting the healing process. Overall, while both methanolic and aqueous root extracts aided wound healing, the 2% w/w methanolic extract showed the best results. This suggests that the roots of *Mimosa pudica* are potentially useful in treating wounds.

Anti-Diabetic activity:[12]

In the present study, the antidiabetic potential of the leaves of *Mimosa pudica* Linn., a member of the family Mimosaceae, was investigated. The ethanolic and petroleum ether extracts of *Mimosa pudica* were tested and compared with metformin, a standard antidiabetic drug. Diabetes was induced in Wistar rats of both sexes by using alloxan. Plasma glucose levels were measured using the Glucose Oxidase-Peroxidase method. The ethanolic extract significantly reduced blood glucose levels.

Hepatoprotective Activity:[13]

Mimosa pudica plant, also known as the "Touch-me-not" plant, has been studied for its ability to protect the liver. In this study, the methanolic extract of *M. pudica* was tested in rats whose livers were damaged by a harmful chemical called carbon tetrachloride (CCl_4). This chemical is known to cause liver injury and is often used in scientific studies to test liver-protective medicines. The rats were orally administered 200 mg of *Mimosa pudica* extract per kilogram of body weight. After the treatment, blood samples were collected and tested for signs of liver damage. The results showed that the extract exerted a significant protective effect on the liver. Several harmful markers in the blood, such as SGOT, SGPT, ALP, total bilirubin, and cholesterol were lower in rats that received the extract. These substances usually increase in concentration when the liver is injured.

Anti-Microbial Activity:[14]

The methanolic extract of *Mimosa pudica* leaves demonstrated notable antimicrobial activity when tested at various concentrations (50, 100, and 200 µg/ml) against several microorganisms, including Aspergillus fumigatus, Citrobacter divergens, and Klebsiella pneumoniae. The extract exhibited excellent antimicrobial effects that were attributed to the presence of several bioactive compounds. Phytochemical analysis revealed that terpenoids, flavonoids, glycosides, alkaloids, quinones, phenols, tannins, saponins, and coumarins were present in the extract, and these constituents may play a significant role in their antimicrobial potential. Antimicrobial mechanisms may involve the disruption of microbial cell membranes, inhibition of enzymes, or interference with genetic material. Flavonoids and alkaloids are known for their broad-spectrum antimicrobial properties. In a related study, Gandhiraja *et al.* (2009) further confirmed the antifungal activity of both methanolic and aqueous extracts of Mimosa pudica, specifically against fungal pathogens such as Aspergillus fumigatus, using the well diffusion assay method. Their findings support earlier observations and highlight the potential use of plants in treating fungal infections. Overall, these studies suggest that *Mimosa pudica* could be a promising natural source of antimicrobial agents and potentially useful in the development of herbal medicines for bacterial and

fungal diseases.

Anti-Oxidant Activity:[15]

The ethanolic extract of *Mimosa pudica* was tested for antioxidant activity using various standard methods such as DPPH, Nitric Oxide, ABTS, and Hydrogen Peroxide free radical models. The extract exhibited a strong ability to protect against free radical damage. It was especially effective in reducing Nitric Oxide and DPPH free radicals, with IC50 values of 78.1±1.75 µg/ml and 35.00±1.15 µg/ml, respectively.

Anti-Hypolipidemic Activity:[16]

For hypolipidemic activity, the 80% ethanol extract of the whole plant of *Mimosa pudica* was tested for its hypolipidemic (fat-lowering) effects in diabetic male albino Wistar rats. In diabetic rats, the plant extract and the standard drug glibenclamide significantly reduced the levels of total cholesterol (TC), triglycerides (TG), and low-density lipoprotein (LDL). At a dose of 500 mg/kg body weight, the plant extract also increased the levels of high-density lipoprotein (HDL), which is the "good" cholesterol. These results suggest that M. pudica helps to lower blood fat levels and may reduce the risk of heart disease in diabetic rats.

Another study by Purkayastha et al. showed that an ethanol extract of M. pudica leaves had hypolipidemic effects in Wistar albino rats with liver damage caused by carbon tetrachloride (CCl_4). The extract, at a dose of 400 mg/kg, significantly reduced levels of triglycerides (96.8 mg/dL), total cholesterol (98.7 mg/dL), very low-density lipoprotein (VLDL) (26.9 mg/dL), and LDL (37.4 mg/dL), while HDL was found to be 34.3 mg/dL.

Diuretic Activity:[17]

The ethanolic extract of *Mimosa pudica* showed strong diuretic effects and increased urine output at doses of 100 mg/kg and 200 mg/kg. Similarly, the leaf decoction of the plant showed diuretic activity in dogs and rats at doses of 200, 500, 1000, and 2000 mg/kg, with a noticeable decrease in chloride (Cl-) and sodium (Na^+) levels, but did not affect potassium (K^+) excretion. In another study of Wistar albino rats administered aqueous leaf extract at doses of 100, 200, and 400 mg/kg, an increase in Na^+, K^+, and Cl^- was observed at a dose of 100 mg/kg. Both ethanolic and aqueous extracts of *Mimosa pudica* were tested for diuretic effects using furosemide (20 mg/kg].

Anti-Ulcer Activity:[11]

The anti-ulcer potential of *Mimosa pudica* extracts was investigated in albino rats using various solvents: 90% ethanol, methanol, chloroform, and diethyl ether. Multiple ulcer models were employed to evaluate efficacy, including aspirin-induced, alcohol-induced, and pylorus ligation-induced ulcers. Key parameters measured included the ulcer index, gastric protection, volume of gastric juice, and both free and total acidity of gastric secretions. The extracts were administered orally at doses of 100 and 200 mg/kg, with ranitidine (20 mg/kg) as the standard reference drug. Toxicity studies indicated that the extracts were safe up to a dose of 2000 mg/kg. Among the tested doses, 100 mg/kg extract showed particularly significant anti-ulcer activity.

Anti- Estrogenic Activity:[17]

The anti-estrogenic activity of *Mimosa pudica* root powder was evaluated using immature female rats as an

experimental model. This study focused on determining the estrogenic and anti-estrogenic properties of the plant material. To assess estrogenic activity, researchers have employed the uterotrophic assay, which involves measuring changes in uterine weight. These findings indicated that administration of M. pudica root powder alone did not lead to a significant increase in uterine weight, suggesting an absence of intrinsic estrogenic activity. To evaluate anti-estrogenic effects, estradiol monobenzoate, a known estrogen, was administered to induce uterine growth. In animals treated with both estradiol monobenzoate and *M. pudica* root powder, the expected increase in uterine weight was significantly reduced, indicating that root powder effectively counteracted the estrogenic action of estradiol monobenzoate. These results demonstrate that, while M. pudica root powder does not exhibit estrogenic properties on its own, it possesses notable anti-estrogenic activity by inhibiting the uterotrophic effects induced by exogenous estrogen.

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Conflict of Interest

The authors declare no conflict of interest.

Conclusion:

This overview provides a comprehensive review of the traditional use of *Mimosa pudica* in treating various diseases, along with the extensive biological activities that have been well-documented. Its broad pharmacological effects, such as treatment of ulcers, inflammation, asthma, diarrhea, urinary complaints, and fistula, have also been used traditionally in the treatment of urogenital disorders, dysentery, sinus, and wounds. Juices prepared from fresh leaves can be used both internally and externally in piles to treat cuts and wounds. This study is an attempt to explore and compile different Pharmacognostic aspects of the plant M. pudica, which requires further investigation.

References:

1. Adurosakin OE, Iweala EJ, Otike JO, Dike ED, Uche ME, Owanta JI, Ugbogu OC, Chinedu SN, Ugbogu EA. Ethnomedicinal uses, phytochemistry, pharmacological activities and toxicological effects of *Mimosa pudica*-A review. Pharmacological Research-Modern Chinese Medicine. 2023 Jun 1;7:100241.
2. Ahmad H, Sehgal S, Mishra A, Gupta R. *Mimosa pudica* L.(Laajvanti): an overview. Pharmacognosy reviews. 2012 Jul;6(12):115.
3. Hagihara T, Toyota M. Mechanical signaling in the sensitive plant *Mimosa pudica* L. Plants. 2020 May 4;9(5):587.
4. Jagetia GC, Vanlalhruaii F. Anticancer potential of *Mimosa pudica* Linn. Lajwanti in cultured Dalton's ascites lymphoma cells. International Journal of Complementary and Alternative Medicine. 2020;13(3):91-4.
5. Hassan NA, Karunakaran RO, Abdulmumin SU. A review on the pharmacological and traditional

properties of *Mimosa pudica*. Int J Pharm Pharm Sci. 2019;11(3):12-6.

6. Gunawardhana CB, Ranasinghe SJ, Waisundara VY. *Mimosa pudica* Linn.: the garden weed with therapeutic properties. Israel Journal of Plant Sciences. 2015 May 18;62(4):234-41.
7. Shaikh Z, Roy SP, Patel P, Gohil K. Medicinal value of *Mimosa pudica* as an anxiolytic and antidepressant: A comprehensive review. World journal of pharmacy and pharmaceutical sciences. 2016 Jan 13;5(3):420-32.
8. Muhammad G, Hussain MA, Jantan I, Bukhari SN. *Mimosa pudica* L., a high-value medicinal plant as a source of bioactives for pharmaceuticals. Comprehensive Reviews in Food Science and Food Safety. 2016 Mar;15(2):303-15.
9. Joseph B, George J, Mohan J. Pharmacology and traditional uses of Mimosa pudica. International journal of pharmaceutical sciences and drug research. 2013 Apr 1;5(2):41-4.
10. Tripathi AK, Soni R, Verma S. A review on ethnopharmacological applications, pharmacological activities, and bioactive compounds of *Mimosa pudica* (linn.). Research Journal of Pharmacy and Technology. 2022;15(9):4293-9.
11. Sankari S. Antioxidant and Antidiabetic Efficacy of Mimosa Pudica: A Phytochemical Perspective.
12. Zaware BB, Chaudhari SR, Shinde MT. An overview of *Mimosa pudica* Linn.: chemistry and pharmacological profile.
13. Rawat D, Kumar H, Kumar A, Kumar V, Kumar S. Ethnomedicinal and pharmacological aspects of *Mimosa pudica* plant.
14. Azmi L, Singh MK, Akhtar AK. Pharmacological and biological overview on *Mimosa pudica* Linn. International journal of pharmacy & life sciences. 2011 Nov 1;2(11).
15. Majeed I, Rizwan K, Ashar A, Rasheed T, Amarowicz R, Kausar H, Zia-Ul-Haq M, Marceanu LG. A comprehensive review of the ethnotraditional uses and biological and pharmacological potential of the genus mimosa. International Journal of Molecular Sciences. 2021 Jul 12;22(14):7463.
16. Wagh C, Bangar S, Gawade M, Shirke H. ItL (*Mimosaceae*) herb with impressive health benefits
17. Ughade M, Chaudhary PH, Burange PJ, Baig A, Pawar D, Warhekar T, Deshmukh V, Darokar P. A comprehensive review On *Mimosa pudica* Linn.

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Review Article

Herbal Remedies For Skin Aging: Insights Into Anti-Aging Cream Formulations

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Abstract

Aging represents a complex biological phenomenon characterized by the progressive deterioration of physiological functions, resulting in heightened susceptibility to chronic diseases and diminished quality of life. Key mechanisms underlying this process include oxidative stress, chronic inflammation, telomere shortening, mitochondrial dysfunction, and declining collagen biosynthesis. This review examines the current landscape of anti-aging formulations that incorporate natural bioactive compounds with established antioxidant, anti-inflammatory, and collagen-enhancing properties. We analyze the therapeutic potential of various phytoconstituents, including polyphenols, flavonoids, vitamins C and E, retinoids, peptides, and botanical extracts, which function synergistically to neutralize reactive oxygen species, protect dermal cells, facilitate cellular repair mechanisms, and promote extracellular matrix regeneration. The review also discusses the incorporation of adaptogenic herbs and nutraceuticals that restore cellular homeostasis and counteract age-related metabolic decline. Through comprehensive analysis of existing literature, we demonstrate that multi-targeted anti-aging approaches offer synergistic benefits by reducing oxidative damage, preventing dermal aging, improving skin elasticity, and supporting systemic longevity pathways. This review concludes that evidence-based, holistic anti-aging formulations may provide safe, effective, and sustainable interventions for mitigating premature aging and promoting healthy longevity

Keywords: Anti-ageing, Herbal, Moisturizing, Skin Brightening, Anti-wrinkle.

Introduction:

The skin, comprising approximately 20 square feet in total area, is the largest organ of the human body. It serves essential protective functions against pathogens and environmental hazards, assists in body temperature regulation, and mediates sensory perceptions such as touch, heat, and cold [1,2].

Physiology of the skin:

Epidermis: The epidermis is the outermost layer of the skin, consisting of stratified keratinized squamous epithelium. Its thickness varies across different regions, being most pronounced on the palms of the hands and soles of the feet. While the epidermis lacks blood vessels and nerve endings, its deeper layers are sustained by interstitial fluids from the dermis, which provide essential oxygen and nutrients and facilitate lymphatic drainage.

Dermis: Located beneath the epidermis, the dermis

is a resilient and flexible layer formed from connective tissue that contains a matrix of collagen and elastic fibers. Overstretching the skin may rupture elastic fibers, resulting in permanent stretch marks often observed in conditions such as pregnancy and obesity. Collagen fibers, the predominant cellular component, play a crucial role in binding water and conferring tensile strength to the skin; as the capacity of these fibers declines with age, wrinkles develop. The deepest region of the dermis is underlain by areolar tissue and variable amounts of adipose tissue. Fibroblasts, macrophages, and mast cells are among the principal cells within this layer.

Sebaceous (subcutaneous) glands: These glands are composed of secretory epithelial cells originating from the same tissues as hair follicles. They release sebum, an oily substance, into the hair follicles, and are distributed throughout the skin except on the palms of the hands and soles of the feet [3].

Functions of the Skin [4-8]:

Protection: The skin acts as a physical barrier against pathogens and environmental insults, defending the body from both chemical and mechanical injury. Langerhans cells, present within the skin, play an essential role in the adaptive immune response.

Sensation: Embedded sensory nerve endings enable the skin to detect a range of stimuli, including heat, cold, touch, pressure, vibration, and tissue damage, thereby contributing to the body's sensory perception system.

Thermoregulation: The skin possesses a vascular network that exceeds its metabolic needs, enabling regulation of heat loss via mechanisms such as radiation, convection, and conduction. Vasodilation increases blood flow and heat dissipation, whereas vasoconstriction decreases perfusion to conserve body heat.

Prevention of Evaporation: The skin's surface acts as a dry, semipermeable barrier, significantly minimizing fluid loss. When this barrier is compromised, as seen in severe burns, excessive fluid loss can occur.

Structural Organization: The outermost layer is composed of skin cells, pigments, and structural proteins. The mid-layer houses sweat glands, hair follicles, blood vessels, and adipose tissue, and supplies nutrients to the epidermis. The innermost layer contains additional skin cells, nerves, hair follicles, blood vessels, and sebaceous glands. Each skin layer incorporates connective tissue—collagen fibers provide support, while elastin fibers supply flexibility and strength.

Anti-aging Cosmeceuticals and Skin Physiology [9]:

Anti-aging creams represent moisturizer-based cosmeceutical formulations designed to diminish visible signs of skin aging and enhance youthful appearance. Skin aging results from continuous cellular degradation processes affecting DNA and protein integrity. This aging phenomenon is categorized into two distinct types: intrinsic (chronological) aging and photoaging, each exhibiting unique histological and clinical

characteristics.

Intrinsic aging represents a universal, time-dependent process characterized by alterations in skin's physiological functions. As aging progresses, keratinocytes lose their capacity to maintain a functional stratum corneum, while neutral lipid synthesis rates decline, resulting in dry, pallid skin with visible wrinkles. Conversely, photoaging occurs due to excessive ultraviolet radiation exposure, manifesting as dry, sallow skin with deep furrows and fine wrinkles caused by disruption of dermal and epidermal structures associated with solar elastosis and helodermatid [10].

Plant-based therapeutics have demonstrated significant potential as complementary therapeutic interventions. Cosmetic formulations serve dual purposes: enhancing aesthetic appearance and providing protection against endogenous and environmental hazards. Regular cosmetic use promotes external beauty while supporting long-term skin health by reducing the incidence of dermatological conditions. Skincare products contain natural or synthetic components that enhance skin health, texture, and structural integrity through mechanisms including hydration maintenance, collagen preservation, and photoprotection.

The cosmeceutical market, particularly herbal-based products, is experiencing rapid expansion. These formulations effectively address various skin concerns including hyperpigmentation, wrinkles, aging, and textural irregularities. Olive oil, rich in vitamin A, functions as a potent antioxidant that decelerates aging processes. Vitamin C plays a crucial role in collagen biosynthesis, a protein essential for maintaining skin elasticity and preventing wrinkle formation. Research demonstrates that antioxidant compounds operate synergistically as a "protective network," wherein multiple antioxidant species collaborate to neutralize free radicals and reactive oxygen species while protecting each other from oxidative damage [11].

Polyherbal cosmetic formulations have gained widespread recognition across diverse populations for their efficacy in managing various skin characteristics. The crude extracts of selected botanical ingredients investigated in this study have demonstrated therapeutic benefits for numerous dermatological conditions.

The pursuit of anti-aging interventions has persisted throughout human history, evolving significantly through scientific advancement. Contemporary understanding of underlying mechanisms, particularly inflammatory processes, has led to innovative treatments incorporating ingredients such as retinoids and bioactive eggshell membrane components. Traditional civilizations have long recognized the therapeutic potential of botanical compounds, establishing herbal medicine as a cornerstone of anti-aging practices. This historical foundation has facilitated the development of modern anti-aging formulations [12].

Benefits of Anti-Aging Formulations:

- Enhanced skin hydration and firmness
- Improved skin radiance and luminosity

- Increased self-confidence and psychological well-being
- Positive impact on overall health and wellness
- Prevention of age spots and hyperpigmentation
- Cost-effective alternative to invasive dermatological procedures

Advantages of Anti-Aging Products:

- Youthful appearance restoration
- Wrinkle reduction and prevention
- Enhanced self-esteem
- Protection against skin flaking and dryness
- Promotion of holistic health benefits

Limitations:

Multiple daily applications may be required to achieve optimal anti-aging and skin-brightening effects. However, discontinuation of product use typically results in gradual return to baseline skin appearance. Potential adverse effects include cutaneous irritation, erythema, burning sensations, or allergic dermatitis. Dermatological professionals recommend initiating anti-aging regimens after age 24, as this represents the appropriate developmental stage for implementing comprehensive skincare protocols [13].

Guidelines:

Anti-aging formulations are designed to moisturize, brighten, firm, and lift sagging skin, with particular efficacy in the periorbital and cervical regions. Quality formulations are typically formulated without artificial colorants, fragrances, or parabens. The moisturizing properties of these preparations provide beneficial effects for both male and female skin types [14].

Various Herbs Used in Herbal Anti-Aging Creams [15-20]:

Papaya

Carica papaya is highly valued in skincare for its enzyme papain, which is known for properties such as skin lightening, reducing unwanted hair, exfoliating dead skin, and aiding in the repair of aging skin.

Biological Source: "*Carica papaya*"

Family: *Caricaceae*

Chemical Constituents: Papain, pantothenic acid, folate, magnesium, potassium; also rich in vitamins C, A, and E

Uses: Possesses antioxidant activity, helps prevent and treat acne, diminishes dark spots and wrinkles, improves skin texture, reduces pigmentation, and removes tanning.

Amla

Known as amla or amalaki, *Phyllanthus emblica Linn* is recognized for its abundant antioxidants and high vitamin C content. It promotes skin health by toning, reducing signs of aging, and imparting a natural glow.

Biological Source: "*Phyllanthus emblica Linn*"

Family: *Phyllanthaceae*

Chemical Constituents: Ellagic acid, gallic acid, emblicanin A and B, phyllembein, quercetin, ascorbic acid

Uses: Collagen-boosting effects improve skin firmness and smoothness, while regular consumption of amla juice elevates vitamin C and enhances

collagen production, giving skin a youthful appearance.

Olive Oil

Olive oil, sourced from Olea europaea, is rich in vitamins, healthy fats, and antioxidants, all contributing to improved skin health.

Biological Source: "*Olea europaea*"

Family: *Oleaceae*

Chemical Constituents: Monounsaturated fatty acids, polyphenols, vitamins

Uses: Provides antioxidant, anti-inflammatory, and skin-nourishing benefits; moisturizes and softens skin, protects against UV-induced damage, reduces inflammation, and supports wound healing.

Tulsi (Holy Basil)

Ocimum sanctum is celebrated for preventing signs of aging and maintaining skin health.

Biological Source: "*Ocimum sanctum*"

Family: *Lamiaceae*

Chemical Constituents: Methyl cinnamate, linalool, β-elemene, camphor

Uses: Combines antibacterial and anti-inflammatory effects; cleanses pores, removes excess oil and impurities, and soothes irritations associated with acne [16–17].

Ashwagandha

Withania somnifera is renowned for its anti-aging potential, supporting skin elasticity while cleansing, moisturizing, and calming the skin.

Biological Source: "*Withania somnifera*"

Family: *Solanaceae*

Chemical Constituents: Withanolides, saponins, alkaloids

Uses: Displays antioxidant, anti-inflammatory, immunomodulatory, and neuroprotective effects; enhances skin regeneration and wound healing [18–19].

Ginger

Zingiber officinale offers potent antioxidants beneficial for combatting skin aging.

Biological Source: "*Zingiber officinale"*

Family: *Zingiberaceae*

Chemical Constituents: Gingerols, shogaols, zingiberene, zingiberol, camphene

Uses: Antioxidant, anti-inflammatory, antimicrobial, analgesic, anti-aging, and supportive of skin health.

Cinnamon

Cinnamomum zeylanicum helps protect skin structure by inhibiting collagen degradation and loss of elasticity.

Biological Source: "*Cinnamomum zeylanicum*"

Family: *Lauraceae*

Chemical Constituents: Cinnamaldehyde, cinnamate, cinnamic acid, essential oils

Uses: Improves skin plumpness, addresses eczema, alleviates acne, delays aging symptoms, soothes dry skin, and enhances complexion.

Turmeric

Curcuma longa provides antibacterial and anti-inflammatory properties, reducing facial wrinkles and fine lines.

Biological Source: "*Curcuma longa*"

Family: *Zingiberaceae*

Chemical Constituents: Volatile oils, curcuminoids, curcumin

Uses: Employed for skin brightening, glowing complexion, acne treatment, lightening dark circles, and preventing early aging signs.

Green Tea

Camellia sinensis is a popular ingredient in anti-aging formulations owing to its high antioxidant capacity.

Biological Source: "*Camellia sinensis*"

Family: *Theaceae*

Chemical Constituents: Vitamins (B, C & E), enzymes, peptides, sugars, caffeine, theophylline, chlorophyll, carotenoids

Uses: Catechins provide anti-aging benefits and help prevent skin redness.

Aloe Vera

Aloe barbadensis is valued for its moisturizing properties and its ability to stimulate collagen and elastin synthesis for firmer, more elastic skin.

Biological Source: *"Aloe barbadensis"*

Family: *Liliaceae*

Chemical Constituents: Vitamins A, C, E, B1, B2, B6, B12, aloin, anthranol, emodin

Uses: Displays anti-aging, antifungal, antioxidant, wound healing, anti-inflammatory, and moisturizing effects.

Evaluation of Anti-Aging Cream:

Organoleptic Evaluation:

The cream's physical characteristics including colour, odour, appearance, and homogeneity are assessed visually to determine organoleptic properties after storage.

Homogeneity:

Homogeneity can be evaluated by visually inspecting the cream after formulation. A small quantity will be pressed between the thumb and index finger to assess consistency and uniformity.

pH Measurement:

A calibrated pH meter can be used for this test. Approximately 1 g of cream dissolved in 50 ml of distilled water, and the suspension's pH should be measured at ideal temperature [22].

Spreadability:

To determine Spreadability, the cream should be sandwiched between two standard glass slides (area spanning 7.5 cm). A 100 g weight can be placed on top to form an even layer, then remove the excess cream it is scraped off. The setup can be arranged so that only the upper slide is free to move when a 20 g weight is attached. The time required for the upper slide to travel 7.5 cm is recorded, and the experiment can be repeated three times to obtain the mean value. Spreadability (S) is calculated using:

$$S = M \times L/T$$

Where S = Spreadability, M = mass tied to upper slide (20 g), L = length moved (7.5 cm), and T = time taken (seconds) [23].

Irritancy Test:

A 1 cm^2 area on the dorsal surface is marked, and cream is applied. Any irritation, redness, or swelling is monitored and recorded at regular intervals over 24 hours [20].

Viscosity:

Viscosity is measured using a Brookfield viscometer with spindle no. 7 at 100 rpm [21].

Dilution Test:

This assesses emulsion type: an oil-in-water (O/W) emulsion mixes with water but separates with oil, while a water-in-oil (W/O) emulsion mixes with oil but separates with water. This assesses emulsion type: an oil-in-water (O/W) emulsion mixes with water but separates with oil, whereas a water-in-oil (W/O) emulsion mixes with oil but separates with water [21].

Stability Studies:

Stability testing follows ICH guidelines. Cream samples are stored in bottles under controlled conditions: 30 ± 2 °C/65 ± 5% RH and 40 ± 2 °C/75 ± 5% RH for two months. Physical properties and viscosity are analyzed at the conclusion of the study [24].

Evaluation Parameters for Herbal Anti-aging Cream [25]

Parameter	Method	Significance
pH Measurement	Digital pH meter; cream mixed with distilled water; average of three readings	Ensures compatibility with skin pH; determines stability
Irritancy Test	Applied to a marked skin area; monitored for redness/swelling over 24h	Assesses safety; identifies potential skin irritancy
Spreadability	Cream between slides; pressed with weight; time to slide apart measured	Evaluates ease of application and distribution
Viscosity	Measured with Brookfield viscometer at set speed (e.g., 100 rpm)	Assesses flow properties and consistency for application
Dilution Test	Cream mixed with water/oil; observed for mixing or separation	Identifies emulsion type; determines stability and compatibility

Conclusion:

Continuous exposure of human skin to ultraviolet radiation leads to a range of pathobiological alterations, including irregular pigmentation, increased wrinkle formation, diminished elasticity, dryness, and surface roughness. Herbal formulations have emerged as effective interventions to mitigate these visible signs of aging and promote skin health. This comprehensive review focused on select herbs of notable therapeutic efficacy, emphasizing their active chemical constituents and mechanisms of action against cutaneous aging. Despite the vast diversity of botanical resources, the scope was limited to those demonstrating the most significant benefits for skin aging. Future research may uncover additional herbs exhibiting secondary anti-aging effects and expand the repertoire of botanical agents with potential dermatological and medical

applications.

References:

1. Gavarkar P, Thorat S, Adnaik R, Mohite S, Magdum C. Characterization and formulation of skin cream from seed oil extracted from Cucumis Melo. Pharm Lett2016;8:90-3.
2. Mohammad Ali* 1, Pooja V. 2, Shalini B. V. 2, Sahana L. 2 and Sindhu S. 2, review on: natural facial scrub, European journal of pharmaceutical and medical research ejpmr, 2024, 11(12), 252-257.
3. Kaur IP, Kapila M, Agarwal R. Role of novel delivery systems in developing topical antioxidants as therapeutics to combat photo aging, Int J of Pharm Sci, 2007; 6:271 288.
4. Watson, Ogden S, Cotterell LF, Bowden JJ, Bastrilles JY, Long SP et al. A cosmetic 'anti-ageing' product improves photo aged skin, a double-blind, randomized controlled trial British. J Dermatol, 2009; 161:419-426.
5. Saraf S, Kaur CD. Phytoconstituents as photo protective novel cosmetic formulations, Pharmacogn. Rev., 2010; 4(7):1-11.
6. Hema Sharma Datta, Rangesh Paramesh, Trends in aging and skin care: Ayurvedic concepts, Journal of Ayurveda and Integrative Medicine, 2010; 1(2):110-113.
7. Geesin JC, Darr D, Kaufmann R, Murad S, Pinnel SR. Ascorbic acid especially increases type I and type III procollagen messenger RNA levels in human skin fibroblast. J. Invest. Dermatol, 1998; 90(4):420-444.
8. Mishra AK, Mishra A, Chattopadhyay P. Herbal Cosmeceuticals for Photoprotection from Ultraviolet B Radiation, A Review, T.J.P.R., 2011; 10(3):351-360.
9. Eichler O, Sies H, Stahl W. Divergent optimum levels of lycopene, beta-carotene and lutein protecting against UVB irradiation in human fibroblasts. Photochem. Photobiol, 2002; 75:503-506.
10. Wrona M, Korytowski W, Róanowska M, Sarna T, Truscott TG. Cooperation of antioxidants in protection against photosensitized oxidation. Free Radic. Biol. Med, 2003; 25(10):1319-1329.
11. Retrieved from Alfaro Danilo, what are herbs;2022. http://www.thesproceeat.com. (Accessed Date; 02/03/2024).\
12. Gupta R, Singh NP, Mukhopadhyay S. A review article on anti-aging herbal cream. International journal of health sciences.;6(S6):3631-41.
13. Kala SL, Palaparthi S. Formulation and invitro evaluation of poly herbal anti aging face cream. World Journal of Pharamaceutical Research. 2017 Sep 9;6(13):713.
14. Suryasa, I. W., Rodríguez-Gámez, M., & Koldoris, T. (2022). Post-pandemic health and its sustainability: Educational situation. International Journal of Health Sciences, 6(1), i-v. https://doi.org/10.53730/ijhs.v6n1.5949.
15. Rodrigues F, Pimentel FB, Oliveira MBPP. Olive by-products: challenge application in the cosmetic industry. Industrial Crops and Products.

2015;70:116-124. doi:10.1016/j.indcrop.2015.03.027.

16. Puja Saha, Supriyo D. Anupam Saha Tulsi as an Active Ingredient with Antimicrobial Activity; 2021.
17. Daniells Stephen,Ashwagandha Root Extracts Show Antiaging Effects; 2016.
18. Narendra S, Mohit B, de Prashant J. An Overview on Ashwagandha,2011.
19. Mehtap O, Necla B, Işıl Takan. Vasileiou Christina,Ginger for Healthy Ageing 2022.
20. J. Varani, R. L. Warner, M. Gharaee-Kermani et al., "Vitamin A antagonizes decreased cell growth and elevated collagen- degrading matrix metalloproteinases and stimulates collagen accumulation in naturally aged human skin," Journal of Investigative Dermatology, vol. 114, no. 3, pp. 480 486, 2000.
21. Singh G, Maurya S, deLampasona MP, Catalan CAN. A comparison of chemical, antioxidant and antimicrobial studies of cinnamon leaf and bark volatile oils, oleoresins and their constituents. Food and Chemical Toxicology. 2007;45(9):1650– 1661.
22. Ines S., Masnec, Mirna Š."Skin Aging", Acta Clin Croat, Vol. 49, No. 4, 2010 18- Last reviewed by a Cleveland Clinic medical professional on 10/10/2019. https://www.dermaessentia.com/blogs/skin care/anti-aging-cream-benefits.
23. S. Sahin, M Bilgin," Olive tree (Olea europaea L.) leaf as a waste by-product of table olive and olive oil industry: a review", J Sci Food Agric (2017).
24. Sharma ma, sharma v, sharma m, mehta k. Development and evaluation anti aging multi herbal cream. Ijrrr; 2020: 13(4); 39-49.
25. Nayana PV, Navyashree PS, Nithyashree HP, Pavan NK, Preethi NS, Rakshitha C. A Review on the Formulation and Evaluation of Herbal Cold Creams Incorporating Natural Oils. Journal of Pharma Insights and Research. 2024 Aug 4;2(4):207-11.

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Review Article

An Updated Review on the Pharmacological Potential of *Clitoria ternatea* L.

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Abstract

Clitoria ternatea, a perennial twining herb indigenous to tropical Asia, flourishes optimally in well-drained soil and is adaptable to conditions ranging from full sunlight to partial shade. Various components of *Clitoria ternatea* have been utilized as active constituents in numerous Ayurvedic formulations for the treatment of diverse disorders. Several traditional Ayurvedic ' Medha ' (cognitive-enhancing) preparations incorporate *C. ternatea* in conjunction with other medicinal plants. The plant has a long-standing history of use in traditional medicine, particularly for its memory-enhancing and anxiolytic properties. Different plant parts contain a variety of bioactive compounds, including tannins, resins, starch, taraxerol, taraxerone, alkaloids, flavonoids, saponins, proteins, anthocyanins, and carbohydrates. Traditionally, *Clitoria ternatea* has been used to treat a wide range of ailments, including jaundice, migraines, throat and eye infections, skin diseases, asthma, joint inflammation, earaches, eruptions, fever, urinary tract infections, constipation, snake bites, headaches, indigestion, leprosy, and central nervous system disorders. It has also been utilized in the management of gonorrhea, stress, and infertility, and is commonly used as a natural food colorant. In Ayurveda, this plant is highly esteemed for its therapeutic benefits. Pharmacological studies have corroborated several of its medicinal properties, including anti-inflammatory, analgesic, antimicrobial, and anxiolytic activities.

Keywords: *Clitoria ternatea,* Pharmacological potential, Ayurvedic medicine – Phytochemistry, Antidiabetic activity.

Introduction

Clitoria ternatea, commonly referred to as Asian pigeonwings, bluebell, blue pea, butterfly pea, cordofan pea, and Darwin's pea, is a species of the Fabaceae family. It is also known by the synonym, Clitoria principissae. This plant exhibits two flower color variations: blue and white [1]. Indigenous to tropical and subtropical regions, the visually striking blue-flowered *Clitoria ternatea* has historically attracted the attention of traditional healers and herbalists because of its purported medicinal properties. With an increasing scientific interest in plant-based remedies, this botanical is now at the forefront of research, receiving renewed attention for its potential therapeutic benefits [2]. In southern India, the plant is referred to as 'Shankhapuspi' Although Aparajita is prevalent in Maharashtra, its application as a Medhya (nootropic) drug remains

insufficiently explored [3]. The plant can attain a height of up to 3 m and is characterized by its pinnate leaves, each consisting of 5–7 elliptical leaflets. The flowers are typically solitary and axillary, displaying a vibrant blue hue, although white variants are also available. Its fruit is a flat, dehiscent pod that generally contains six to ten seeds. This species thrives in well-drained soil and flourishes under conditions ranging from full sun to partial shade [4]. Various parts of *Clitoria ternatea* have been utilized as active components in numerous Ayurvedic formulations for the treatment of a broad spectrum of disorders. Several traditional Ayurvedic 'Medha' (cognitive-enhancing) preparations incorporate *C. ternatea*, in conjunction with other medicinal plants. Scientifically, this plant has been investigated for its diverse pharmacological properties, including antihistaminic, anthelmintic, hypoglycemic, antidepressant, and sedative effects. *Clitoria ternatea*, known as Aparajita in Bengali, is a prominent plant used in Ayurvedic medicine. All parts of the herb are used for therapeutic purposes. It has been traditionally used for centuries in Ayurveda for its extensive range of benefits, including memory enhancement, nootropic effects, stress relief, anxiety reduction, depression treatment, seizure prevention, and as a natural sedative and tranquilizer [5].

Taxonomy: [6].

Subkingdom	Viridaeplanta
Infrakingdom	Streptophyta
Division	Tracheophyte
Subdivision	Spermatophytina
Infrodivision	angiosperms
Class	Magnoliopsida
Main order	Rosanae
Order	Fabale
Family	*Fabiaceae*
Genus	*Clitoria L.*
Species	*Clitoria ternatea*

Vernacular names:
English: Conch flower, Winged leaved Clitoria, Butterfly pea flower
Guajarati: Garani, Koyal ni vel
Hindi: Khagin, Kalizer, Khajina, Koyal
Kannada: Koyala, Koyila, Girikarnike
Marathi: Gokarni, Gokarnika
Tamil: Karuvilai, Kakkanam

Morphology: **Macroscopic characters**:
The leaves are arranged alternately and are imparipinnately compound, with each leaflet positioned oppositely. The total length from the base of the petiole to the leaflet apex ranged from 10.4 to 12.5 cm. The terminal (apical) leaflet measured between 4.3 and 5.0 cm in length and 2.6 to 3.4 cm in width. The lower leaflets range from 4.2 to 4.8 cm in length and 2.3 to 2.5 cm in width, respectively. Each leaf is supported by a petiole, and each leaflet is attached via a petiolule, with stipules at its base. The leaflets possess an ovate lamina with a symmetrical base, entire margin, and mucronate apex. The venation was unicostate and reticulate. The lamina surface was smooth and hairy. The upper surface was dark green, whereas the lower surface was light green. The leaves emit a characteristic Odor and possess a bitter taste [7].

Microscopic characters:
In the transverse section, the leaf revealed a dorsiventral structure. The epidermis is differentiated into an upper and lower layer, both of which are unilayered and covered with a thick cuticle. The cuticle is striated, imparting a lobed appearance to the cut section, particularly at the midrib. The mesophyll is differentiated into palisade and spongy tissue. In the midrib region, the vascular bundle is situated within the cortex. The stele is of the haplostele type, with xylem elements encircled by the phloem. Stomata are present on both surfaces of the leaf and are of the

anomocytic and anisocytic types, with surrounding epidermal cells exhibiting wavy walls. The petiole exhibited a triangular outline, with its upper two angles extending into multicellular lobe-like structures. The stem has a circular outline. The epidermis consists of small, compactly arranged cells with 6–8 angles, each projecting into a multicellular, lobular structure. The petiole shows a circular outline, with the outermost phloem composed of 12 to 25 rows of thin-walled, longitudinally elongated cells, some of which are compressed and a few exfoliating [8].

Phytochemistry:

The plant *Clitoria ternatea* comprises a diverse array of compounds, including proteins, alkaloids, anthraquinones, anthocyanins, cardiac glycosides, phenols, tannins, phlobatannins, carbohydrates, saponins, triterpenoids, flavonoids, flavonol glycosides, volatile oils, and steroids. The seeds of *Clitoria ternatea* contain fatty acids, such as palmitic, stearic, oleic, linoleic, and linolenic acids. Additionally, the seeds are characterized by the presence of water-soluble mucilage, delphinidin 3, 3', 5'-triglucoside, beta-sitosterol, anthoxanthin glucoside, and a small basic protein known as finotin. Furthermore, *Clitoria ternatea* is rich in phytochemical constituents, including pentacyclic triterpenoids such as taraxerol and taraxerone. Phytochemical analysis of its roots has revealed the presence of terpenes, alkaloids, flavonoids, saponins, tannins, carbohydrates, proteins, resins, starch, taraxerol, and taraxerone [9].

Pharmacological Activity:

Antidiabetic activity:

The diuretic properties of the dried whole roots and powdered ethanol extract were assessed. A single intravenous (I.V.) administration of the extract significantly increased the urinary excretion of sodium (Na^+) and potassium (K^+) and decreased chloride (Cl^-) levels without affecting urine volume. A comparable effect was observed with oral administration [10]. The hypoglycemic effect of a methanolic extract of *Clitoria ternatea* leaves at doses of 200 and 400 mg/kg was examined in alloxan-induced diabetic rats. The extract significantly reduced blood glucose levels ($P < 0.001$) in diabetic rats 12 hours post-administration [11]. Oral administration of *Clitoria ternatea* (CT) leaves and flowers at a dose of 400 mg/kg body weight for 84 d led to significant improvements in various biochemical parameters in diabetic rats. Treatment with both CT leaves and flowers resulted in reductions in serum glucose, glycosylated hemoglobin (HbA1c), total cholesterol, triglycerides, urea, creatinine, and the activity of the gluconeogenic enzyme glucose-6-phosphatase. Conversely, serum insulin, HDL cholesterol, total protein, liver and skeletal muscle glycogen content, and the activity of the glycolytic enzyme glucokinase were increased. Among the two, CT leaf treatment exhibited slightly superior effects compared to CT flower treatment. These findings support the potential of *Clitoria ternatea* as an antidiabetic agent [12-14]. Diabetes is a chronic metabolic disorder characterized by persistent hyperglycemia and impaired metabolism of carbohydrates and lipids [15].

Antimicrobial Activity

The antimicrobial properties of *Clitoria ternatea* have been extensively studied against various pathogenic microorganisms. Extracts derived from different plant parts, including flowers, leaves, and roots, have demonstrated significant inhibitory effects against both bacterial and fungal strains. These findings suggest that *Clitoria ternatea* possesses potential as a natural antimicrobial agent, with possible applications in the pharmaceutical, food preservation, and cosmetic industries [16].

Antidepressant Activity

Oral administration of methanolic extracts of *Clitoria*

ternatea at dosages of 100 and 400 mg/kg produced significant antidepressant effects in mice, as indicated by a reduction in immobility time during the tail suspension test. Notably, the 400 mg/kg dosage exhibited greater efficacy than fluoxetine [17]. Furthermore, ethanolic extracts of *C. ternatea* roots at dosages of 150 and 300 mg/kg also demonstrated significant antidepressant activity. Previous research has identified two bioactive compounds in the roots—(Z)-9,17-octadecadienal and n-hexadecanoic acid—which are potential selective monoamine oxidase A (MAO-A) inhibitors. These findings suggest that *C. ternatea* may provide promising herbal alternatives for the management of psychiatric disorders such as depression and anxiety [18].

Neuropharmacological Activity

Clitoria ternatea possesses neuroprotective properties, which are attributed to its antioxidant and anti-inflammatory activities. This plant has demonstrated potential in preventing neurodegenerative disorders and enhancing cognitive function. Traditionally, it is considered a brain tonic, particularly for improving mental clarity and overall mental health. Experimental studies have shown that intraperitoneal administration of alcoholic extracts from the stem, flower, leaves, and fruit of *C. ternatea* induces sedation and reduces alertness in animal models, such as rats and mice. Furthermore, root extracts administered at doses of 300–500 mg/kg enhanced memory by mitigating electroshock-induced amnesia. These effects are associated with increased acetylcholine levels and modulation of acetylcholinesterase activity in various brain regions, including the cerebral cortex, midbrain, medulla oblongata, and cerebellum [19].

Antioxidant activity

Antioxidants are essential for neutralizing free radicals, inhibiting lipid peroxidation, and mitigating oxidative stress, thereby preventing various diseases. *Clitoria ternatea* is rich in phenolic compounds, including tannins, coumarins, xanthones, and procyanidins, all of which exhibit dose-dependent free radical scavenging activity. These bioactive constituents are linked to a range of health benefits, including cardiovascular protection, cancer prevention, and a reduction in oxidative damage to lipids and low-density lipoprotein cholesterol [20].

Anticonvulsant Activity

Seizures result from an imbalance between excitatory and inhibitory neurotransmissions in the brain. Agents that elevate gamma-aminobutyric acid levels frequently exhibit anticonvulsant activity. The maximal electroshock test is a widely recognized experimental model used to evaluate the efficacy of antiepileptic drugs, particularly in the context of generalized tonic-clonic seizures. Methanolic extracts derived from the aerial parts of *Clitoria ternatea*, when administered orally at a dosage of 100 mg/kg, demonstrated anticonvulsant activity in mice. The extract significantly delayed the onset of seizures and reduced tonic hind limb extension in both MES- and pentylenetetrazol-induced (PTZ)-induced seizure models. However, the same extract did not confer protective effects against MES- and PTZ-induced seizures in rats, indicating species-specific variability in response [21].

Anti-inflammatory, Antipyretic, and Analgesic Effects

Clitoria ternatea exhibits significant anti-inflammatory, antipyretic, and analgesic properties in various experimental models. The ethanol extract of the root, when administered intraperitoneally at doses of 100, 125, and 150 mg/kg in mice, demonstrated substantial anti-histaminic activity. Notably, both chlorpheniramine maleate and ECTR significantly inhibited clonidine-induced catalepsy but had no effect on haloperidol-induced catalepsy. The methanol

extract of the root of the blue-flowered variety was assessed for antipyretic activity in albino rats. Administered orally at doses of 200, 300, and 400 mg/kg, the extract significantly reduced both normal and yeast-induced elevated body temperatures in a dose-dependent manner, with effects persisting for up to 5 h post-administration. Its efficacy was comparable to that of paracetamol.

In anti-inflammatory models, methanolic root extract inhibited carrageenan-induced paw edema and acetic acid-induced vascular permeability in rats. It also reduced yeast-induced pyrexia and significantly decreased writhing in mice subjected to the acetic acid-induced writhing test. Petroleum ether (60–80°C) extracts of *C. ternatea* flowers demonstrated significant anti-inflammatory and analgesic activity in carrageenan-induced paw edema and hot plate models in rats and mice. Similarly, the methanolic leaf extract exhibited strong analgesic effects in mice using the acetic acid-induced writhing test, with inhibition rates of 82.67% and 87.87%, respectively, which were comparable to that of diclofenac sodium. Central nervous system depressant activity was also observed through decreased locomotor activity in the open-field and hole cross tests. Further mechanistic studies using hot plate, tail-flick, and formalin-induced pain models, along with naloxone (an opioid receptor antagonist), indicated that both root and leaf extracts possessed central and peripheral antinociceptive properties. Root extracts acted at both spinal and supraspinal levels, whereas leaf extracts primarily acted at the supraspinal level. These effects suggest the involvement of the opioid system in mediating the analgesic effects of *C. ternatea* [22].

Wound Healing Activity

The wound healing efficacy of *Clitoria ternatea* seed and root extracts was assessed using excision, incision, and dead-space wound models in rats. Both oral administration and topical application of the extracts significantly enhanced wound healing in all models. The observed effects were comparable to those produced by the standard cotrimoxazole ointment. The study further indicated that *Clitoria ternatea* positively influenced all key phases of the wound healing process—namely, the inflammatory, proliferative, and remodeling phases [23]

The wound healing potential of the standardized *Clitoria ternatea* leaf extract was evaluated using various enzymatic models pertinent to skin repair. The inhibitory activity of the methanolic extract and its fractions against hyaluronidase, elastase, and matrix metalloproteinase-1 (MMP-1) was assessed using oleanolic acid as a reference standard. Reverse-phase high-performance liquid chromatography (RP-HPLC) was employed to standardize the extract and its fractions based on the isolated biomarker taraxerol, which was present at a yield of 5.27% w/w.

The methanolic extract demonstrated significant inhibitory activity against hyaluronidase (IC_{50}: 18.08 ± 0.46 µg/mL, $P < 0.001$) and MMP-1 ($P < 0.05$), although the inhibition of elastase was not significant (IC_{50}: 42.68 ± 0.46 µg/mL). Among the tested fractions, the ethyl acetate fraction exhibited strong inhibition of hyaluronidase (IC_{50}: 28.01 ± 0.48 µg/mL, $P < 0.001$) and MMP-1 ($P < 0.01$) activities. HPLC analysis confirmed enrichment of taraxerol in both the crude methanolic extract (5.32% w/w) and the ethyl acetate fraction (4.55% w/w), supporting their bioactivity in wound healing [24].

Anti-asthmatic Activity

The ethanol extract of *Clitoria ternatea* root (ECTR) was assessed for its anti-asthmatic properties using various experimental models. Administered intraperitoneally at doses of 100–150 mg/kg, ECTR significantly mitigated milk-induced leukocytosis

and eosinophilia in mice. Additionally, it effectively prevented egg albumin-induced mast cell degranulation and reduced blue dye leakage in a passive cutaneous anaphylaxis model in rats. These results suggest that *Clitoria ternatea* root extract demonstrates significant antiallergic and anti-asthmatic activity by stabilizing mast cells and suppressing allergic inflammatory responses [25]. The anti-asthmatic efficacy of the ethanol extract of *Clitoria ternatea* roots was further evaluated using a histamine aerosol-induced bronchospasm model in Wistar rats. Oral administration of the extract at a dose of 400 mg/kg provided 47.45% protection against histamine induced bronchoconstriction in mice. The findings indicate that the aqueous extract of *C. ternatea* not only exhibits bronchodilatory effects but also reduces bronchial hyperreactivity in vivo. This effect is attributed to decreased infiltration of inflammatory cells in the airways and the stabilization of mast cells, thereby inhibiting the release of histamine and other inflammatory mediators [26].

Conclusion

This review highlights *Clitoria ternatea* as a promising medicinal plant with a wide range of pharmacological activities. Its demonstrated efficacy and safety profiles indicate its significant potential for various therapeutic applications. The plant exhibits numerous pharmacological effects, including antioxidant, antidepressant, neuropharmacological, anticonvulsant, wound healing, anti-asthmatic, anti-inflammatory, analgesic, antipyretic, antidiabetic, and antimicrobial activities. Overall, *Clitoria ternatea* is a valuable plant with a rich pharmacological profile, making it an important component of natural healing systems and a potential subject for further scientific research to explore its benefits.

References:

1. Raut S, Belekar M, Jeurkar DN, Ingole S, Kakde V, Warhate V, Gajbhiye S. Pharmacognostical and pharmacological account on Clitoria ternatea: a review.
2. Mohapatra R, Induar S, Parida S. Blossoming wellness: exploring the therapeutic potential of edible flowers. European Chemical Bulletin. 2023;12:5411–9.
3. Nangare N, Menon S, Pawar V, Kurulkar M. A critical review of Aparajita with special reference to Ayurveda classical texts. International Journal of Research and Analytical Reviews. 2019;6(2):187–92.
4. Pandey AK, et al. Botanical and phytochemical aspects of Clitoria ternatea: a review. Journal of Medicinal Plants Studies. 2014;2(3):23–30.
5. Al-Snafi AE. Pharmacological importance of Clitoria ternatea: a review. IOSR Journal of Pharmacy. 2016;6(3):68–83.
6. Shahnas N, Akhila S. Phytochemical, in vitro, and in silico evaluation of *Clitoria ternatea* for Alzheimer's disease. Pharma Tutor. 2014;2(9):135–49.
7. Joseph A, Prasad DX, Prasobh G, Surabhi G. A review on ethnopharmacological study of Clitoria ternatea. i-Manager's Journal on Chemical Science. 2023;3(3).
8. Sethiya NK, Nahata A, Mishra H, Dixit VK. An update on Shankhpushpi, a cognition-boosting Ayurvedic medicine. Journal of Chinese Integrative Medicine. 2009;7(11):1001–22.
9. Rampalli S, Gopalkrishnan B. Pharmacognostical investigation of *Clitoria ternatea* L. leaves. Horizon. 2022;9(4):814–9.
10. Mukherjee PK, Kumar V, Kumar NS, Heinrich M. The Ayurvedic medicine Clitoria ternatea—from traditional use to scientific assessments. Journal of Ethnopharmacology. 2008;120:291–301. doi:10.1016/j.jep.2008.09.009.

11. Piala JJ, Madissoo H, Rubin B. Diuretic activity of roots of *Clitoria ternatea* L. in dogs. Cellular and Molecular Life Sciences. 1962;18(2):89.
12. Abhishek S, Pankaj M, Vikas S. Hypoglycemic effects of *Clitoria ternatea* leaves (Linn) extract. Journal of Pharmacology and Toxicological Studies. 2013;1(1):4–7.
13. Terahara N. Five new anthocyanins, ternatins A3, B4, B3, B2 and D2 from *Clitoria ternatea* flowers. Journal of Natural Products. 1996;59(2):139–44.
14. Saito N, Abe K, Honda T, Timberlake CF, Bridle P. Acrylates, delphinidin glucosides and flavonols from Clitoria ternatea. Phytochemistry. 1985;24(7):1583–6.
15. Selvaraj J, Pitchai D, Nithya P, Valli G, Ponnulakshmi R, Ramajayam G. Antidiabetic and antioxidant activity of novel dihydroxygymnemic triacetate (DGT) in the liver of high-fat diet and fructose-induced type-2 diabetic adult male rats. International Journal of Pharmacy and Pharmaceutical Sciences. 2015.
16. Mishra SS, Moharana SK, Dash MR. Review on Cleome gynandra. International Journal of Research in Pharmacy and Chemistry. 2011;1(3):681–9.
17. Jeyaraj R, Ramasamy K. Antimicrobial activity of *Clitoria ternatea* Linn. Asian Journal of Pharmaceutical and Clinical Research. 2012;5(4):109–11.
18. Gollen B, Mehla J, Gupta P. *Clitoria ternatea* Linn: a herb with potential pharmacological activities and future prospects as therapeutic herbal medicine. Journal of Natural Remedies. 2018.
19. Kosai P, Sirisidthi K, Jiraungkoorskul K, Jiraungkoorskul W. Review on ethnomedicinal uses of memory-boosting herb, butterfly pea (*Clitoria ternatea*). Journal of Natural Remedies. 2015.
20. Zingare ML, Zingare PL, Dubey A, Ansari A. *Clitoria ternatea* (Aparajita): a review of the antioxidant, antidiabetic and hepatoprotective potentials. Journal of Natural Remedies. 2013.
21. Gollen B, Mehla J, Gupta P. *Clitoria ternatea* Linn: a herb with potential pharmacological activities and future prospects as therapeutic herbal medicine. Journal of Natural Remedies. 2018.
22. Al-Snafi AE. Pharmacological importance of Clitoria ternatea: a review. IOSR Journal of Pharmacy. 2016;6(3):68–83.
23. Solanki YB, Jain SM. Wound healing activity of *Clitoria ternatea* L. in experimental animal model. Pharmacologia. 2012;3(6):160–8.
24. Maity N, Nema NK, Sarkar BK, Mukherjee PK. Standardized *Clitoria ternatea* leaf extract as hyaluronidase, elastase and matrix metalloproteinase-1 inhibitor. Indian Journal of Pharmacology. 2012;44(5):584–7.
25. Taur DJ, Patil RY. Evaluation of antiasthmatic activity of *Clitoria ternatea* L. roots. Journal of Ethnopharmacology. 2011;136(2):374–6.
26. Chauhan N, Rajvaidhya S, Dubey BK. Antihistaminic effect of roots of *Clitoria ternatea* Linn. International Journal of Pharmaceutical Sciences and Research. 2012;3(4):1076–9

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Research Article

Formulation and Evaluation of Herbal Anti-Aging Cream Containing Natural Bioactive Ingredients

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Abstract

The present study aimed to formulate and evaluate an herbal anti-aging cream using natural ingredients such as papaya leaves, tulsi powder, olive oil, ginger, rice water, ashwagandha, aloevera, vitamin E, beeswax, glycerin, and sandalwood oil, prepared by the emulsification method in three variations (F1, F2, and F3). The formulations were assessed for organoleptic properties, pH, spread ability, homogeneity, irritancy, dilution, sensitivity, and wash ability. All creams were pale green, semi-solid, smooth in texture, and had a pleasant sandalwood fragrance, with pH values ranging from 5.4 to 5.9, within the ideal range for skin compatibility. They exhibited excellent spread ability, homogeneity, and stability, were easily washable, and showed no signs of irritation or sensitivity, confirming dermatological safety. The creams were identified as water-in-oil emulsions, offering enhanced moisturization and prolonged hydration. Among the three, F1 demonstrated superior overall performance in terms of texture, stability, and user acceptability, indicating its potential as an effective, safe, and natural alternative to synthetic anti-aging formulations.

Keywords: Papaya leaves, anti-ageing, anti-wrinkle, skin care, moisturising, skin nourishing.

Introduction

The skin is the largest organ of the body with a total area of about 20 square feet. The skin protects us from microbes and the elements, helps regulate body temperature, and permits the sensations of touch, heat, and cold [1].

Physiology of the skin:

The skin is composed of multiple layers, each with distinct structures and functions. The outermost layer, the epidermis, is made up of stratified keratinized squamous epithelium, with varying thickness depending on the body part—it is thickest on the palms of the hands and soles of the feet. This layer lacks blood vessels and nerve endings, but its deeper cells are nourished by interstitial fluid from the underlying dermis, which provides oxygen and

nutrients and also facilitates lymphatic drainage. Beneath the epidermis lies the dermis, a tough and elastic layer composed of connective tissue with a matrix rich in collagen and interwoven elastic fibres. When the skin is overstretched, as in pregnancy or obesity, elastic fibres may rupture, resulting in permanent stretch marks. Collagen fibres in the dermis bind water and give the skin its tensile strength, though this ability declines with age, contributing to the formation of wrinkles. The dermis also contains cells such as fibroblasts, macrophages, and mast cells, and its deepest layer rests on areolar tissue with varying amounts of adipose tissue. Embedded in the skin are sebaceous glands, which consist of secretory epithelial cells originating from the same tissue as hair follicles. These glands secrete an oily substance called sebum into hair follicles and are found throughout the skin except on the palms of the hands and soles of the feet.

Functions of skin:

The skin serves several vital functions essential to maintaining overall health and homeostasis. One of its primary roles is protection, acting as an anatomical barrier that defends the body against pathogens and physical damage. Langerhans cells within the skin contribute to this defence as part of the adaptive immune system. The skin is also crucial for sensation, containing a wide array of nerve endings that respond to stimuli such as heat, cold, touch, pressure, vibration, and injury, making it an integral part of the sensory system. In terms of heat regulation, the skin has a rich blood supply that exceeds its basic needs, allowing it to regulate body temperature through mechanisms like radiation, convection, and conduction. Blood vessels dilate to increase blood flow and heat loss or constrict to reduce blood flow and conserve heat. The skin also plays a role in the control of evaporation, serving as a semi-permeable barrier that minimizes fluid loss; when this barrier is compromised, as in the case of burns, significant fluid loss can occur. Structurally, the skin is organized into layers: the outer layer contains skin cells, pigment, and proteins; the middle layer houses sweat glands, hair follicles, blood vessels, and fat; and the inner layer includes skin cells, nerves, blood vessels, hair follicles, and oil glands. The dermis supports the epidermis by supplying nutrients and is composed of connective tissue with collagen fibres for strength and elastin fibres for flexibility and resilience [2].

Anti-ageing cream:

Anti-aging creams are moisturizer-primarily based cosmeceutical skincare merchandise. With the promise of creating the patron to look more youthful by way of reducing the signs of pores and skin aging [3].

Damage to cellular DNA and proteins causes a continuous degradation process that leads to skin aging. Sequential skin aging and photo aging are the two different categories into which the aging process is divided. Each category has unique historical and clinical characteristics. The universal and predictable

process of sequential skin aging is defined by changes in the way the skin functions physiologically. The aging process causes dry, pale skin with wrinkles because keratinocytes can no longer generate a viable stratum corneum and the pace at which neutral lipids are formed slows down [4]. On the other hand, excessive exposure to UV radiation from sunshine results in photo aging. It is distinguished by dry, sallow, and pale skin that exhibits deep furrows and fine wrinkles brought on by the disarray of dermal and epidermal components linked to heliodermatis and elastosis. Plants and herbs have previously shown promise as a supplemental medical tool [5]. Cosmetics are used to improve skin's appearance and protect against both endogenous and external hazardous substances [6]. Using cosmetics helps people look better on the outside and maintain excellent health for longer by lowering the prevalence of skin conditions [7]. The natural or artificial components included in skin care products that promote skin health, texture, and integrity, as well as hydrating, preserving skin elasticity through the decrease of type I collagen, and providing photo protection, among other benefits. This characteristic of cosmetics results from inclusion of components in skin care products, since they aid in lowering the skin's generation of free radicals and maintaining its characteristics over time [8]. The greatest option for reducing skin conditions including hyperpigmentation, wrinkles, aging, rough skin texture, etc., is cosmetic items. The market for herbal cosmetics is growing quickly. Vitamin A can be found in large quantities in olive oil.

It slows down the aging process by acting as an excellent antioxidant. The body uses vitamin C to generate collagen, a protein that is necessary for our skin to be elastic and to avoid wrinkles [9]. According to research, the anti-oxidant compounds in that living organism always function as a "protective chain," meaning that various anti-oxidant compounds work in concert to shield one another from direct harm during the processes that neutralize free radicals and other reactive species [10]. The effects of the polyherbal cosmetic formulation are well acknowledged in the communities of various nations, and it has long been advised for the management of skin characteristics. Numerous skin conditions have been treated with the crude extract of the chosen herbal extract that is the subject of this inquiry [11].

History:

The fight against skin aging has existed since antiquity and has developed as a result of scientific advancements. Improvements like the usage of eggshell membranes and retinol are the result of a better understanding of the underlying causes, such as inflammation. The use of plants' therapeutic qualities by civilizations has made traditional herbal treatments essential. Modern anti-aging compositions have been made possible by this historical progression [12]

Some Incredible benefits of using anti-aging creams:

Skin tightening and proper hydration provide a range of benefits for both your appearance and overall health. They help improve the skin's natural glow, making it look more radiant and vibrant, which in turn can boost your self-esteem. These practices also support healthy skin function and contribute positively to your general well-being. By keeping the skin firm and well-moisturized, they can reduce the likelihood of developing age spots and uneven skin tone. Furthermore, taking care of your skin in this way may lessen the need for costly cosmetic treatments, offering a more affordable and natural solution for maintaining youthful skin.

Advantages of anti-aging cream:

Anti-aging creams offer several advantages, making them a valuable addition to a skincare routine. They help you look younger by reducing the appearance of wrinkles and fine lines, while also protecting the skin from dryness and flaking. By improving skin texture and radiance, they boost self-confidence and enhance overall appearance. In addition, regular use of anti-aging creams promotes better skin health, which in turn supports overall well-being and positively influences other aspects of life.

Disadvantages of anti-aging cream:

You possibly need to use the wrinkle cream a couple of times the afternoon for decreasing the getting old and brighten the pores and skin but when you discontinue the use of the product your skin is possible to return to its original 3635 look. There are some side effects also in anti-aging cream, a few merchandises may also reason pores and skin inflammation, rashes, burning, or redness on the face Anti-aging cream is dangerous for children- As in step with Dermatologists, they advise the use of anti-aging cream after 24 due to the fact it's miles the proper time to start the usage of anti-aging products or as a minimum have a proper skincare routine.

Usage of Anti-aging cream:

Use anti-getting old creams to moisturize, brighten, tighten, and lift up your sagging skin, specifically the skin around your eyes and neck. Those potions do not have artificial colourings, scents, and parabens. The moisturizing impact of those lotions will work wonders on males and females' skin [13].

Methodology:

Procedure:

The required amount of white bee's wax, glycerine, and olive oil were taken as the oil phase in a porcelain dish and melted at 70°C. Simultaneously, aloevera, vitamin E capsule, ginger, papaya leaf powder, tulsi powder, ashwagandha powder, and rice water were taken as the aqueous phase in another porcelain dish and heated to 70°C. The aqueous phase was then added gradually to the oil phase with continuous stirring, with additional rice water added in small amounts if necessary. Stirring was continued until a cream of the desired consistency was obtained, after which sandalwood oil was added as a perfume just before transferring the finished product into a suitable container. By varying the proportions of ingredients, three different formulations, namely

F1, F2, and F3, were prepared and subsequently evaluated through various tests [14].

Table 1: Ingredients Profile

Ingredient	Role of ingredients
Papaya leaves	Skin whitening; reduces unwanted hair; exfoliates dead skin; repairs ageing skin
Tulsi powder	Antioxidant; anti-inflammatory; antimicrobial; analgesic
Olive oil	Antioxidant; anti-inflammatory; antibacterial; anti-wrinkle; reduces skin inflammation; protects against sun damage; prevents acne-causing bacteria; moisturizes and hydrates skin
Ginger	Antioxidant; anti-inflammatory; antimicrobial; analgesic
Rice water	Reduces pigmentation; lightens dark spots; soothes sunburn; anti-ageing
Ashwagandha	Antioxidant; reduces wrinkles and fine lines; anti-inflammatory
Aloevera	Anti-inflammatory; anti-ageing; wound healing; soothes sunburn
Vitamin e capsule	Acts as an antioxidant
Bee's wax	Helps emulsify and stabilize the cream
Glycerine	Humectant that attracts moisture; prevents dryness; maintains skin's natural moisture balance
Sandalwood oil	Improves skin tone; hydrates and nourishes skin; treats acne; reduces signs of ageing; antioxidant and anti-inflammatory; reduces fine lines and wrinkles

Table 2: Formulation table

Sl no.	Ingredients	F1	F2	F3
1.	Papaya leaves	2.5gm	3.5gm	3gm
2.	Tulsi powder	2.5gm	3.5gm	3gm
3.	Olive oil	2ml	2.5ml	1.5ml
4.	Ginger	1.5gm	1.5gm	1.5gm
5.	Rice water	6ml	5ml	7ml
6.	Ashwagandha	2.5gm	2.5gm	2.5gm
7.	Aloevera	1.5gm	1.5gm	1.5gm
8.	Vitamin E capsule	2ml	2ml	2ml
9.	Bee's wax	2gm	2gm	2gm
10.	Glycerine	1.5ml	1.5ml	1.5ml
11.	Sandalwood oil	Q. S	Q. S	Q. S

Evaluation of Anti-Aging Cream:

1. Organoleptic evaluation: The sensory characteristics, including colour, smell, and appearance, were noted.

2. Homogeneity: All developed creams were tested for homogeneity by visual inspection after the creams have been set in the container. They were tested for their appearance and presence of any aggregates [15]

3. pH of the cream: The pH meter was calibrated using standard buffer solution. About 1 g of the cream was weighted & dissolved in 50 ml of distilled water. The pH of the suspension was determined at 27^0C [16].

4. Spreadability: Spreadability is determined by measuring the time, in seconds, it takes for two glass slides to separate from the cream; a shorter time indicates better Spread ability. To perform the measurement, 3 g of herbal cream was placed between two slides and pressed to create a uniform thin layer. A weight of 1000 g was then applied for 5 minutes. Following this, an additional 10 g was added using a pan, and the upper slide was connected to a string and hook for pulling. The time taken for the upper slide to move 10 cm over the lower slide was recorded, and Spreadability was calculated using the designated formula.

$$S = \frac{M \times L}{T}$$

Where,

S – Spreadability,

M – Weight tied to the upper slide (20g)

L - Length of the glass (7.5 cm)

T-Time taken in seconds [17].

5. Irritancy test: An area of one square centimetre is outlined on the left dorsal side. The cream is applied to the marked area, and the starting time is noted. Any signs of irritation, redness, or swelling are monitored and documented at regular intervals over a 24-hour period [18]

6. Dilution Test: The dilution test identifies the type of emulsion by mixing it with either water or oil. An O/W emulsion will mix completely with water but separate when mixed with oil, while a W/O emulsion will blend with oil but separate when mixed with water [19]

7. Sensitivity test: The cream that was prepared was applied to the skin of the hand and exposed to sunlight for 4 to 5 minutes.my research [20]

8. Washability Test: Removal of the applied cream from the skin was conducted by gentle washing under tap water, ensuring minimal force to effectively cleanse the skin [21-22].

Results and Discussion:

1. Organoleptic Evaluation

The formulated herbal anti-aging creams (F1, F2, and F3) were evaluated for their color, odor, and appearance. All formulations exhibited a pale-green color with a characteristic sandalwood odor and a semi-solid appearance, as shown in Table 3. These properties indicate uniformity and acceptable aesthetic characteristics, which enhance consumer

appeal and acceptability.

Table 3: Organoleptic Property

Sl. No	Properties	F1	F2/F3
1	Colour	Pale green	Pale green
2	Odour	Sandalwood	Sandalwood
3	Appearance	Semi-solid	Semi-solid

2. Homogeneity

All formulations exhibited good homogeneity, with uniform distribution of herbal extracts throughout the cream base. This was confirmed by visual inspection and by touch. The smooth texture without any lumps or phase separation confirmed proper emulsification and stability of the formulations.

3. pH of the Cream

The pH of all formulations was found to be in the range of 5.4 to 5.9, which is suitable for topical application and compatible with the natural skin pH (around 5.5–6.8). Maintaining the pH within this range minimizes the risk of irritation or allergic reactions. Among the formulations, F2 showed the lowest pH (5.4), while F3 had the highest (5.9).

Table 4: pH of the Formulations

Sl. No	Formulation	pH
1	F1	5.8
2	F2	5.4
3	F3	5.9

4. Spreadability

The spreadability of the formulations was determined to evaluate the ease of application on the skin. All formulations exhibited good spreadability, indicating smooth and uniform application without excessive drag. Among the three, F1 showed slightly higher spreadability (23.52 gm·cm/sec), followed by F3 (22.2 gm·cm/sec) and F2 (21.05 gm·cm/sec). This indicates that F1 provides a more desirable consistency for topical use.

Table 5: Spreadability of the formulations

Formulation	Mass (gm)	Radius (cm)	Time (sec)	Spreadability (gm·cm/sec)	Observation
F1	100	2.0	8.5	23.52	Spreadable
F2	100	2.0	9.5	21.05	Spreadable
F3	100	2.0	9.8	22.20	Spreadable

5. Irritancy Test

All three formulations were tested for skin irritancy and were found to be non-irritant. This confirms the safety of the herbal ingredients used and indicates that the formulations are suitable for regular topical use.

6. Dilution Test

The dilution test revealed that all formulations were immiscible with water but miscible with oil, confirming that the prepared creams are water-in-oil (W/O) type emulsions. Such emulsions are beneficial for dry or aged skin as they provide enhanced moisturizing effects and longer retention on the skin surface.

Table 6: Dilution test

Sl. No	Formulation	Water	Oil
1	F1	Immiscible	Miscible
2	F2	Immiscible	Miscible
3	F3	Immiscible	Miscible

7. Sensitivity Test

The sensitivity test indicated no sensitivity reactions for any of the formulations (F1, F2, or F3). This demonstrates that the herbal extracts and excipients used in the formulation are safe and non-allergenic for topical application.

Table 7: **Sensitivity test**

Sl. No	Formulation	Sensitivity
1	F1	No
2	F2	No
3	F3	No

8. Washability Test

All formulations were found to be easily washable with water, which enhances user convenience and improves consumer acceptability. This property also indicates the proper emulsification and physical stability of the formulations.

Table 8: Washability test

Sl. No	Formulation	Washability
1	F1	Easily washable
2	F2	Easily washable
3	F3	Easily washable

Overall Discussion

The prepared herbal anti-aging cream formulations (F1, F2, and F3) showed satisfactory physicochemical properties, including appropriate color, odor, texture, pH, and spreadability. All formulations were homogeneous, stable, non-irritant, and easily washable. The W/O type emulsion nature ensures better moisturization and longer retention on the skin, which is beneficial for anti-aging applications. Among the three, F1 exhibited slightly superior spreadability and balanced pH, indicating its potential as an optimized formulation for further studies and commercialization.

Conclusion:

The formulated herbal anti-ageing cream containing papaya leaves, tulsi, ginger, ashwagandha, olive oil, rice water, aloe vera, vitamin E, beeswax, glycerine, and sandalwood oil was successfully developed and evaluated. The formulation showed excellent organoleptic characteristics, good homogeneity, skin-friendly pH, smooth spreadability, and no signs of irritation or sensitivity. It also passed washability and dilution tests effectively. All evaluation parameters indicated that the cream is safe, stable, and suitable for regular use, making it a promising herbal formulation for anti-ageing skincare.

Reference:

1. Gavarkar P, Thorat S, Adnaik R, Mohite S, Magdum C. Characterization and formulation of skin cream from seed oil extracted from Cucumis melo. Pharma Letter. 2016;8:90–3.
2. Ali M, Pooja V, Shalini BV, Sahana L, Sindhu S. Review on natural facial scrub. European Journal of Pharmaceutical and Medical Research. 2024;11(12):252–7.
3. Kaur IP, Kapila M, Agarwal R. Role of novel delivery systems in developing topical antioxidants as therapeutics to combat photoaging.

International Journal of Pharmaceutical Sciences. 2007;6:271–88.

4. Watson S, Ogden S, Cotterell LF, Bowden JJ, Bastrilles JY, Long SP, et al. A cosmetic 'anti-ageing' product improves photoaged skin: a double-blind, randomized controlled trial. British Journal of Dermatology. 2009;161:419–26.
5. Saraf S, Kaur CD. Phytoconstituents as photoprotective novel cosmetic formulations. Pharmacognosy Reviews. 2010;4(7):1–11.
6. Datta HS, Paramesh R. Trends in aging and skin care: Ayurvedic concepts. Journal of Ayurveda and Integrative Medicine. 2010;1(2):110–3.
7. Geesin JC, Darr D, Kaufmann R, Murad S, Pinnel SR. Ascorbic acid increases type I and type III procollagen messenger RNA levels in human skin fibroblasts. Journal of Investigative Dermatology. 1998;90(4):420–4.
8. Mishra AK, Mishra A, Chattopadhyay P. Herbal cosmeceuticals for photoprotection from ultraviolet B radiation: a review. Tropical Journal of Pharmaceutical Research. 2011;10(3):351–60.
9. Eichler O, Sies H, Stahl W. Divergent optimum levels of lycopene, beta-carotene and lutein protecting against UVB irradiation in human fibroblasts. Photochemistry and Photobiology. 2002;75:503–6.
10. Wrona M, Korytowski W, Rózanowska M, Sarna T, Truscott TG. Cooperation of antioxidants in protection against photosensitized oxidation. Free Radical Biology and Medicine. 2003;25(10):1319–29.
11. Alfaro D. What are herbs [Internet]. 2022 [cited 2024 Mar 2]. Available from: http://www.thesproceeat.com
12. Gupta R, Singh NP, Mukhopadhyay S. A review article on anti-aging herbal cream. International Journal of Health Sciences. 2022;6(S6):3631–41.
13. Kala SL, Palaparthi S. Formulation and in vitro evaluation of polyherbal anti-aging face cream. World Journal of Pharmaceutical Research. 2017;6(13):713–21.
14. Rodrigues F, Pimentel FB, Oliveira MBPP. Olive by-products: challenge application in the cosmetic industry. Industrial Crops and Products. 2015;70:116–24. doi:10.1016/j.indcrop.2015.03.027.
15. Kumar R, Saha P, Lokare P, Datta K, Selvakumar P, Chourasia A. A systemic review of Ocimum sanctum (Tulsi): morphological characteristics, phytoconstituents and therapeutic applications. International Journal for Research in Applied Sciences and Biotechnology. 2022;9(2):221–6.
16. Nayana PV, Navyashree PS, Nithyashree HP, Pavan NK, Preethi NS, Rakshitha C. A review on the formulation and evaluation of herbal cold creams incorporating natural oils. Journal of Pharma Insights and Research. 2024;2(4):207–11.
17. Ozkur M, Benlier N, Takan I, Vasileiou C, Georgakilas AG, Pavlopoulou A, Cetin Z, Saygili EI. Ginger for healthy ageing: a systematic review

on current evidence of its antioxidant, anti-inflammatory, and anticancer properties. Oxidative medicine and cellular longevity. 2022 Jan 1;2022(1):4748447.

18. Gaurav H, Yadav D, Maurya A, Yadav H, Yadav R, Shukla AC, Sharma M, Gupta VK, Palazon J. Biodiversity, biochemical profiling, and pharmaco-commercial applications of *Withania somnifera*: A review. Molecules. 2023 Jan 26;28(3):1208.
19. Singh S. Magical ayurvedic spices and herbs that can boost our immunity. MOJ Food Process Technols. 2020;8(3):99-102.
20. Nayana PV, Navyashree PS, Nithyashree HP, Pavan NK, Preethi NS, Rakshitha C. Formulation and evaluation of cold cream using different natural oils. World Journal of Pharmaceutical Research 2024 13, 19, 1288-1299.
21. Varghese A, Devassy AM, Aswathy V, Bhagyasree S, Mahesh A. Formulation and evaluation of anti-aging cream from herbal extract World Journal of Pharmaceutical Research 2024 13,15 943-956.

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