Parkinson Disease Treat by Using Herbal Treatment

Prepaid by – Kalyankar Prerana Dnyaneshwar.
Gauided by- Dr . Megha Salve Mam.
Shivajirao pawar college of pharmacy .

ABSTRACT –
Parkinson Disease (PD) is neurodegenerative illness and has a common onset between the ages of 55 and 65 years. They are show this mov. like movement problems such as rigidity, slowness, and tremor. More than 6 million individuals worldwide have Parkinson disease.

PD could be a central anxious framework clutter caused by the passing of the substantia nigra portion of basal ganglia within the brain that can't create sufficient dopamine and some cases is heredity and in some, it is caused by a specific gene mutation. They are additionally related with non-motor indications, which may go before engine side effects by more than a decade. These non-motor symptoms become troublesome side effects within the afterward stages of PD. Objective: There is a growing trend to use natural products, especially herbal ones, for the treatment of PD. This article reviews the basic properties of medicinal plants and biological compounds that can be used to treat PD. Methods: Authentic articles selected in ISI, SID, PubMed, PubMed Central, Scopus and Web of Science databases were used. It contains a total of 12 active ingredients derived from plants and 18 herbal extracts. Currently, isolated from various plant compounds that have an effect. PD is treated by targeting the pathways involved in the pathogenesis of the disease.Product: Even herbal extracts like rough hibiscus hook. F. (Malvaceae), Ginkgo biloba L. (Ginkgoaceae), Carthamus Tinctorius L (Asteraceae) and some active ingredients such as berberine and curcumin have shown positive effects in animal models of PE, and the active ingredients and operation methods are as follows . : Investigate. Another study. Discussion and conclusion: Despite the wide range of ideas Although there are many types of plants around the world, few of them have been studied for their activity against Parkinson's disease. Therefore, this field has many prospects for future research on plants and their bioactive compounds.
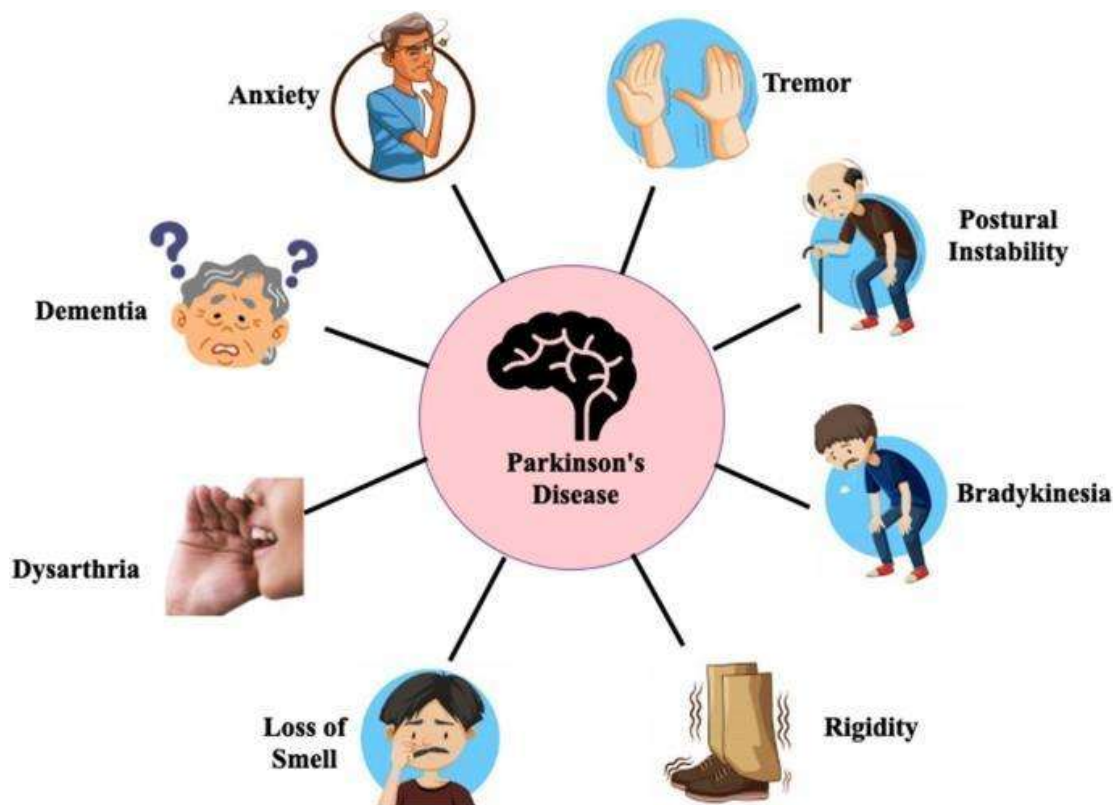
I. INTRODUCTION –

Parkinson's infection may be a neurological development disorder. Common indications of Parkinson's are tremor, gradualness of development, solid muscles, unsteady walk and balance and coordination problems. Parkinson's infection could be a anxious framework infection which influences the ability to control development of the quiet . The malady more often than not begins gradually and after that compounds over time. Parkinson's malady understanding shake, have muscle solidness, and have inconvenience strolling and keeping up adjust and coordination.As the infection compounds, the understanding may have inconvenience in talking, resting and have mental & memory issues, patients involvement behavioral changes and have other indications.

Home grown solutions, as the basic portion of conventional medication (such as in China and India), have been gradually acknowledged for utilize within the treatment of different infections around the world due to their multilevel work characteristics and momentous adequacy (in a few cases) with less antagonistic impacts [10]. For illustration, characteristic items inferred from Chinese home grown solutions, such as curcumin, epigallocatechin gallate, ginsenosides, berberine, artemisinins, emodin, ursolic corrosive, silibinin, triptolide, cucurbitacins, oridonin, tanshinone, artesunate, shikonin, β-elemene, gambogic corrosive, cepharanthine, and wogonin, have been illustrated with numerous bioactivities counting proapoptotic, antiangiogenic, and antifibrotic impacts, as well as resistance adjust, autophagy control, and chemotherapy enhancement both in vitro and in vivo [11, 12]. In antiquated China, numerous home grown medications recorded in Shennong's Classic of Materia Medica, the earliest total pharmacopeia of China, are still being practiced within the treatment of PD, such as

Radix achyranthis bidentatae, Herba asari, Fructus viticis, and Fructus xanthii [13]. In India, there has too been a long history of utilizing home grown solutions within the treatment of neurodegenerative illnesses, such as Withania somnifera, Mucuna pruriens, and Tinospora cordifolia. These lines of prove demonstrated that home grown medications may be promising candidates to get disease-modifying drugs for PD.Herbal recipes for treating PD date back 2200 years.

Causes –

Although the exact cause of Parkinson's disease is still a mystery, there are many pathogenic factors that play an important role in Parkinson's disease, such as oxidative stress, free radical production, mitochondrial disease, apoptosis, neuroinflammation9,10 and genetic susceptibility. 6-hydroxydopamine and 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine 12,13, rotenone, parquet, maneb, manganese, toluene, N-hexane, carbon monoxide, mercury, cyanide Included in the cargo some endogenous toxins. , or external. , as a load Copper, lead, and trichlorethylene10,14, some drugs, viral diseases, Alzheimer's disease, amyotrophic lateral sclerosis (ALS), Creutzfeldt-Jakob disease, Wilson's disease, and Huntington's disease .Parkinson's disease occurs when dopamine enters the brain directly, causing cell death in the nigrostriatal dopamine areas 16 and 11 of the brain and aging. 17. Symptoms The four main symptoms of Parkinson's disease are tremors. ripe; Bradykinesia and Seo.

Symptoms –

Trembling,
Rigidity,
Bradykinesia
Postural Instability,
Non-motor
features includes abdominal cramps,
Disturbed sleep,
Walk,
Talk,
Co-ordinate movements,
Shuffling gait,
Digestion,
Emotion,
Blood Pressure,
Fixed facial expression,

lack of blinking,
 Micrographia,
Autonomic dysfunction,
 Cognitive,

Psychiatric changes,
 Sensory symptoms,
Seborrhea
 Muscle atrophy.
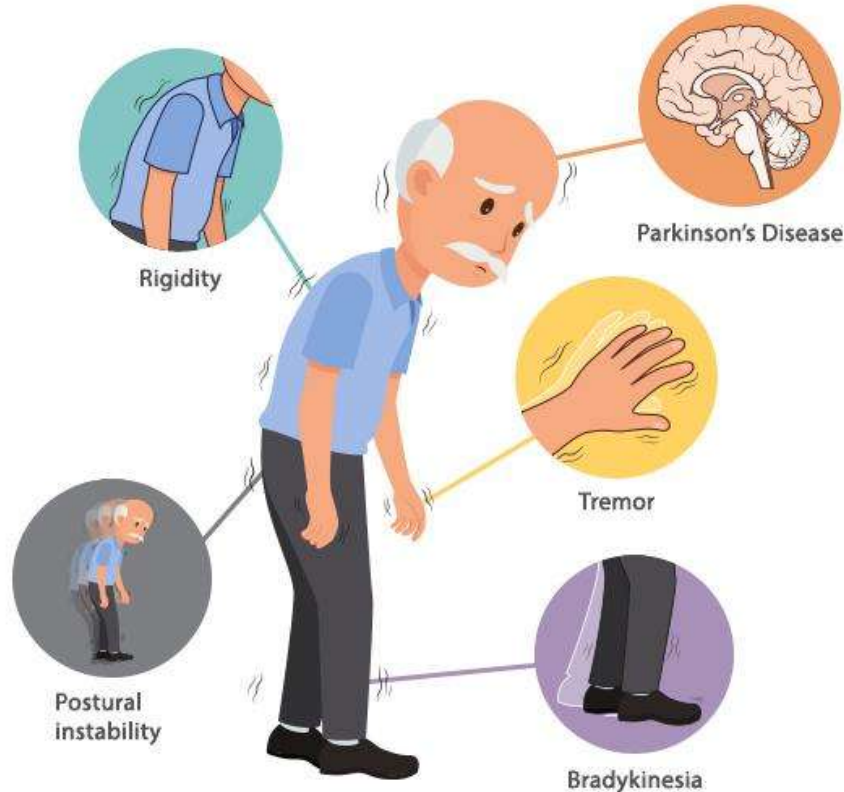
Causes -

Although the exact cause of the disease is still a mystery, there are many pathogenic factors that play an important role in Parkinson's disease, such as oxidative stress, free radical production, mitochondrial disease, apoptosis, neuroinflammation9,10 and susceptibility genetics. The precise cause of malady is still a secret, But numerous pathogenetic components such as oxidative stretch, free radical arrangement, mitochondria brokenness, apoptosis, neuroinflammation 9,10, and hereditary helplessness [11] are fundamentally included in PD. Certain endogenous or exogenous poisons such as 6-hydroxydopamine and 1-methyl-4-phenyl-1,2,3,6- tetrahydropyridine12,13, rotenone, Paraquat, Maneb, manganese, toluene, N-Hexane, carbonmonoxide, Mercury, Cyanide, Copper, Lead and Trichloroethylene10,14, certain medicines, viral contamination, Alzheimer's illness, amyotrophic horizontal sclerosis (ALS), Creutzfeldt-Jakob malady, Wilson's malady and Huntington's disease15, Organization.

Stages of PD-
1.   Onset of PD: adolescence, early features and typical features-

There are different classifications of PD based on the time of onset of the disease. Juvenile PD occurs at age 20, and PD occurs as early as age 40 (some authors extend the period to age 45). After that, the onset of the disease is considered normal. For early-onset PD in adolescence or before the age of 35, a genetic diagnosis is required, even without a positive family history, related to autosomal recessive inheritance, such as: Unrelated parents are important (Sheerin et al. 2014).In many cases, patients are more likely to have earlier onset and less severe cognitive impairment. However, when motor differences are identified, interventional treatment options can be determined within the advPD concept (Hassan et al. 2015). In general, since this form of PD is rare, it is a progressive neurodegenerative disease, and most patients die in a more or less rare form. anus. so, At age 65, the incidence is 50 per 100,000, 75–150, 50 per 100,000, 85–400, high risk (de Lau et al. 2006; Pringsheim et al. 2014;).In an attempt to

understand the extent of prodromal PD, which occurs 10 years before clinical diagnosis, Berg et al. We estimate the prevalence of prodromal symptoms to be 0.5% at age 55, 1.5% at age 65, and 4% at age 75 (Berg et al. 2015).That is, about 1% at the age of 75. Although the population is diagnosed with PD, only 4% have prodromal PD and may develop typical motor symptoms within the next 10 years. These statistics show the strong impact that PD has on our aging society.

2. Global classification of clinical disease and motor scales: HY stage and UPDRS scale In an early study of PD patients between 1949 and 1964, Margaret M. Hoehn and Melvin D. Yahr classified patients into five categories (most commonly used HY categories I through V) depending on the degree of disability.Of all patients who were matched, approximately 25% developed significant disability or died within 5 years of disease onset. At 5 to 9 years of hunting, this period increases to 67 years, at 80 to 10 to 14 years. Few patients have symptoms such as: The disease progressed slowly with balance and postural stability over 10 years, with some without significant disability even after 20 years (Hoehn and Yahr 1967). In a recent study of 142 PD patients with long-term follow-up between 2000 and 2012, approximately 77% progressed 10 years after diagnosis, mainly due to the person's decline due to lack of body. Most of the causes of death are not related to Parkinson's disease, but to pneumonia, cancer, heart disease, etc. and other factors (Williams-Gray et al.2013).

3. Non-motor indications and PD subtypes-

Parkinson's illness (PD) has customarily been considered a engine framework clutter, but it is presently broadly respected as complex clutter with particular clinical highlights that too incorporate neuropsychiatric and non-motor appearances (Chaudhuri and Sauerbier 2016). The foremost pertinent non-motor highlights contain cognitive brokenness and dementia, psychosis and mental trips, disposition clutters counting discouragement, uneasiness, and apathy/abulia, rest unsettling influences, weakness, autonomic dysfunction,

Olfactory brokenness, gastrointestinal brokenness, torment and tactile unsettling influences as well as dermatologic discoveries (seborrhea). In spite of the fact that these indications are in portion included within the MDS-

UPDRS scale, more particular scales exist which only assess non-motor work such as the persistent self-questionnaire NMS-Quest (Chaudhuri et al. 2006) or the physician-assisted NMS Scale (Chaudhuri et al. 2007).These scales capture the non-motor burden of illness and empower a more all encompassing see on PD, since non-motor side effects were appeared to unequivocally impact by and large seriousness of malady in PD patients (Chaudhuri et al. 2013, 2015). Besides, later classifications of advPD are clearly alluding to non-motor side effects, e.g., symptomatic dysautonomia (counting orthostatic symptomatic hypotension), over the top daytime languor, mental trips and cognitive impedance (Luquin et al., 2017).

While an exertion to classify PD agreeing to engine indications into diverse overwhelming phenotypes such as tremor-dominant and non-tremor-dominant (postural flimsiness stride disorder/akinetic-rigid) subtypes has as of now been embraced, such a classification has as of late moved into center moreover for non-motor phenotypes. Here, the taking after non-motor subtypes are recognized: cognitive, neuropsychiatric (lack of concern, depression/anxiety), rest (REM rest behavior clutter), (central) torment, fatigue,Autonomic (gastrointestinal tract brokenness, genital-urinary clutters, symptomatic hypotension), and "Park weight" (combined with olfactory brokenness and dyskinesia) subtype (Marras and Chaudhuri 2016; Sauerbier et al. 2016b). Interests, the non-motor side effect designs reflect phenotypes which can be characterized by overwhelming inclusion of either neocortical, olfactory/limbic or brain stem zones and hence illustrate the solid interface to the basic neuropathological and biochemical

(e.g., cholinergic, serotonergic, opioidergic, adrenergic) unsettling influences (Marras and Chaudhuri 2016).

4.Neuropathological staging With the assistance of a exact portrayal of engine and non-motor phenotypes, a relationship with neuroanatomical structures and ensuing neuropathological modifications gets to be attainable. In common, idiopathic PD is respected as a gradually dynamic infection spreading inside the anxious framework, which clarifies that to begin with side effects are frequently exceptionally troublesome to pinpoint inside an person persistent. Through exceptionally point by point neuropathological investigations of autopsy fabric of PD patients, Braak et al. (2003)

and Shoreline et al. (2009) portrayed particular pathways of neuronal degeneration, Lewy body pathology and spreading of malady within the CNS (Braak et al.2003; Shoreline et al. 2009). Braak recommended that the illness prepare counting synucleinopathy with Lewy body testimony may begin in non-dopaminergic structures within the fringe and after that spread in an rising way to the olfactory bulb and lower brainstem which might clarify early autonomic unsettling influences and hyposmia (Braak stages I/II). At that point, brainstem synucleinopathy was found emigrate rostrally to the substantia nigra pars compacta and other neuronal clusters of the midbrain and basal forebrain and classic engine side effects show up (Stages III/IV). Eventually, the telencephalic cortex of the transient and frontal flaps was appeared to be included (Stages V/VI) (Braak et al.2003). Agreeing to this concept, advPD connects with the suggestion of neocortical structures inferring cognitive disability. Interests and in understanding with the concept of a pathophysiological prepare influencing dopaminergic and non-dopaminergic structures, patients with a speedier infection movement towards advPD display with prior cognitive impedance and postural flimsiness .

5. Challenges to classify illness stages at the boundary of advPD and atypical parkinsonism During infection movement and based on the transcendent engine and non-motor highlights related with advPD, the division from atypical parkinsonism (AP) may be troublesome and cover disorders like 'minimal change' numerous framework decay (MSA) or dynamic supranuclear paralysis with overwhelming parkinsonism (PSP-P) have been depicted (Petrovic et al. 2012; Respondek and Höglinger 2016). AP incorporates a heterogeneous bunch of disorders, all characterized by clinically show parkinsonism in combination with other clinical highlights and a destitute restorative reaction to dopaminergic medicine. As it were autopsy investigations can clearly separate from advPD, as their neuropathology is characteristically distinctive: in MSA, alpha-synuclein aggregation is found and characterizes an alpha-syncleinopathy as PD, but basically in glial cells as cytoplasmic incorporations (coiled bodies). In differentiate, PSP and corticobasal degeneration (CBD) are alluded to as tauopathies due to characteristic intraneuronal tau conglomeration and a few TDP-43 proteinopathies might moreover create

clinical parkinsonism (Dickson 2012; Siuda et al.2014; Stamelou et al. 2013). In all parkinsonian disorders, rectify symptomatic classification is basic for the definition of treatment choices and the precision of any guess. In any case, indeed in experienced centers, the conclusion of PD and its symptomatic separation from AP have destitute unwavering quality and are regularly erroneous, on the off chance that solely based on clinical criteria. In a number of clinical thinks about, there's an mistake rate of at slightest 10–30% in such cases.Symptomatic exactness can progress by ensuing utilize of standardized symptomatic rebellious such as the Queens-Square-Brain-Bank (QSBB)-criteria, counting its steady signs. QSBB-criteria incorporate basically engine side effects and, thus, non-motor indications are under-represented in these criteria. In any case, there's still a exceptional contrast within the demonstrative accuracy between specialists and non-experts, indeed in the event that such standardized criteria are utilized, conjointly among specialists a outstanding rate of misdiagnosis has been watched in longitudinal perceptions (Hughes et al. 1992; Postuma et al. 2015; Rizzo et al.2016).

Management –

PD is an extrapyramidal disease that affects the motor structures of the basal ganglia and is characterized by loss of dopaminergic function and subsequent reduction in motor function, leading to the clinical features of the disease. Studies in the late 1950s demonstrated depletion of striatal dopamine. The presence of non-motor characteristics associated with glutamatergic, cholinergic, In addition to the neuromodulators adenosine and enkephalin, it also includes the serotoninergic and adrenergic systems. Additional evidence suggests that PD may originate in the posterior and anterolateral motor nuclei of the vagus and glossopharyngeal nerves, suggesting a disease pattern that begins in the brain and ascends to higher cortical levels. The histopathological characteristics of PE are: The presence of pigmented dopaminergic neurons and the presence of Lewy bodies (LB). A decrease in the number of dopaminergic neurons in the substantia nigra (SNpc) that project to the striatum (nigrostriatal pathway) causes dopaminergic dysfunction in PD patients.In fact, patients experience the motor symptoms of PD only after 50-80% of their

dopaminergic neurons have been lost, indicating the involvement of compensatory mechanisms in the early stages of the disease. There are two types of dopamine receptors, D1 (excitatory type) and D2. (Inhibitory type) It affects the motor functions of the extrapyramidal system. The components of this system are the basal ganglia, which include the internal part of the globus pallidus (GPi) of the ventral striatum and the reticular part of the substantia nigra (SNpr). These units are part of a larger circuit in the thalamus and cortex. Dopamine loss in the striatum of PD patients increases GPi/SNpr circuit activity and subsequently increases gamma-aminobutyric acid. GABA deficiency inhibits the thalamus.The result is a reduced ability of the thalamus to activate the frontal cortex, which reduces the motor function characteristic of PD. Therefore, restoration of striatal dopaminergic activity through activation of D2 and D1 receptors and dopaminergic therapy may mediate clinical improvement of motor symptoms in PD. Furthermore, dopaminergic disease does not simply reduce thalamic activation; Loss of the normal inhibitory action of dopamine results in increased cholinergic activity. Research continues to support evidence that PD is associated with global network dysfunction that spans multiple levels of the nervous system.

Pathogenesis –

PD is a neurodegenerative and progressive disease that is associated with various motor and debilitating disorders, including bradykinesia, muscle stiffness, resting tremor and imbalance. PD is pathologically characterized by slow and gradual degeneration of dopaminergic neurons in the SN compacta that leads to a decrease in the level of dopamine in the striatum, tailed nuclei and the putamen .The progressive loss of dopaminergic neurons in the basal complexes is the most important pathological finding In the brain of patients with PD. Destruction of these neurons results in the reduction of dopamine neurotransmitter in this area. After 50–60% of dopaminergic neurons are degraded and dopamine levels in the striatum decrease by around 80–85%, the symptoms of the disease appear. The exact molecular mechanism of the degradation of dopaminergic neurons and the incidence of PD is unclear; however, studies have shown that oxidative stress and mitochondrial dysfunction probably play a key role in the pathogenesis of PD; the loss of nigrostriatal dopaminergic neurons and the presence of intracellular cytoplasmic proteins,

i.e., Lewy bodies, are also involved. The cells are located in the nigrostriatal neurons in the SN pars compacta (SNpc) and are sent to putamen. The absence of these neurons, which typically contain small amounts of melanin, leads to depigmentation of SNpc .

Abnormal mitochondrial function and oxidative stress

Accumulating evidence suggests that in PD, the function of mitochondrial complex I partially decreases. Approximately 100% of molecular oxygen is consumed by the mitochondria during cellular respiration, and powerful oxidants, including hydrogen peroxide and superoxide radicals, are produced as a by-product. Reactive oxygen species (ROS) production increases by inhibiting mitochondrial complex I, which can produce toxic hydroxyl radicals or react with nitric oxide and produce peroxynitrites. These molecules can damage the nucleic acids, proteins and lipids by reacting with nucleic acids. One of these injuries can occur in the electron transport chain, which can lead to mitochondrial damage and the formation of ROS that, in turn, can increase the inappropriate folding of proteins .

Much research has also suggested that ROS plays a role in the degeneration of dopaminergic neurons in the brain tissues of PD patients . High levels of lipid peroxidation, glutathione depletion and increase in protein oxidation are observed in the brain tissues of PD patients. Oxidation of dopamine leads to the formation of dopamine quinone that can directly alter proteins. In healthy people, there are mechanisms that prevent the cells from inappropriate folding and accumulation. For example, these proteins are harvested by lysosomal and ubiquitin-proteasome system, as well as certain chaperones can correct these foldings. It has been suggested that defective mitochondrial function causes the death of dopaminergic neurons at advanced ages. Oxidation of RNA and DNA of membrane proteins and lipids is one of the important factors for defective mitochondrial function.

Diagnosis -

Right now, there isn't a particular test to analyze Parkinson's infection. A conclusion is made by a specialist prepared in apprehensive framework conditions, known as a neurologist. A determination of Parkinson's is based on your therapeutic history, a survey of your indications,

and a neurological and physical exam. A part of your wellbeing care group may recommend a particular single-photon outflow computerized tomography (SPECT) check called a dopamine transporter (DAT) check. In spite of the fact that this will help back the doubt that you simply have Parkinson's illness, it is your indications and comes about of a neurological exam that eventually decide the right determination.Most individuals don't require a DAT scan. Your care group may arrange lab tests, such as blood tests, to run the show out other conditions that will be causing your symptoms. Imaging tests — such as an MRI, ultrasound of the brain and PET filters — too may be utilized to assist run the show out other clutters. Imaging tests aren't especially accommodating for diagnosing Parkinson's disease. In expansion to looking at you, a part of your wellbeing care group may provide you carbidopa-levodopa (Rytary, Sinemet, others), a Parkinson's illness pharmaceutical.You must be given a adequate measurements to appear the advantage, as getting moo measurements for a day or two isn't dependable. Noteworthy change with this pharmaceutical will frequently affirm your determination of Parkinson's disease. Sometimes it takes time to analyze Parkinson's malady. Wellbeing care experts may prescribe standard follow-up arrangements with neurologists prepared in development clutters to assess your condition and side effects over time and analyze Parkinson's disease. However, a modern test may be on the skyline.Analysts are considering a Parkinson's test that can distinguish the illness some time recently indications start. The test is called an alpha-synuclein seed intensification measure. In a 2023 consider, analysts tried the spinal liquid of more than 1,000 individuals to search for clumps of the protein alpha-synuclein. Alpha-synuclein is found in Lewy bodies. It shapes clumps that the body can't break down.The clumps spread and harm brain cells. Alpha-synuclein clumps are a trademark sign of Parkinson's illness. The test precisely recognized individuals with Parkinson's illness 87.7% of the time. The test moreover was profoundly touchy for identifying individuals at chance of Parkinson's infection.

Parkinson's disease is the most common clinical disease and clinical manifestations include normal aging, essential tremor, drug-induced parkinsonism, Parkinson's disease and Parkinson's disease, parkinsonism, and normal pressure hydrocephalus19. New entities include parkinsonism, dopa-responsive dystopia 20 , juvenile-onset Huntington's disease, and globus pallidus degeneration 21 . Neuroimaging and laboratory tests are necessary in atypical cases. MRI, EEG, PET, CT and EXERCISES 22,23,24. Laboratory tests may include blood tests such as a complete blood count (CBC), chemistry readings, urinalysis, and blood sugar tests. An EKG is done to help evaluate the heart.

Treatment –
1. Acanthopanax senticosus Damage; (Family: Alariaceae) Takahiko Fujikawa et al found that administration of 250 mg/kg p.o. 100% ethanol, 50% ethanol and hot water extracts of Acanthophyllum trunk bark were effective in preventing the behavioral symptoms of parkinsonism such as bradykinesia, catalepsy and depression. Dopamine levels increase significantly We demonstrate cytoprotection of the SN and VTA during prolonged exposure to neurotoxins by significantly inhibiting DA cell degeneration in those areas through specific activation of the nigrostriatal DAergic system. The extract is administered orally for 2 weeks before IP administration of MPTP and 2 weeks together with MPTP .

2. Withania somnifera; (Family: Solanaceae); Without: Physalis somnifera Sankar Surendran et al. The effect of Withania somnifera root extract on Parkinson's disease was studied. Animals were treated with MPTP for 4 days, followed by root extracts for 7 and 28 days. A dose of 100 mg/kg has been shown to improve motor neuron activity, catecholamines, antioxidant capacity and prevent lipid peroxidation.Increased levels of TBARS28 are reduced.

3.   Uncaria rhynchophylla; (Family: Madderidae) By Oh Myeong-sook et al. Scientific evidence supports the traditional use of Uncaria rhynchophylla for Parkinson's disease. Uncaria rhynchophylla has neuroprotective activity against 6-OHDA toxicity in PC12 cells. In PC12 cells in vitro, URE significantly reduced neuronal death and increased GSH levels $(74.55 \pm 1.57\%)$, attenuated ROS and inhibited 6-OHDA-induced caspase-3 activation in a dose-dependent manner. In vivo, low-dose extraction reduced the number of induced APOs. Protection of DA neurons by reducing hypersensitivity mediated by irreversible MAO-B inhibition of URE in the striatum

4)Nardostachys jatamansi; (Family: Valirenaceae); Syn: jatamansi Muzamil Ahmad et al studied the neuroprotective effect of the ethanolic extract of Nardostachys Jatamansi in the 6-OHDA model of Parkinson's disease. The dose of the extract prevents significant increases in drug-induced reflexes and deficiencies in locomotor activity and muscular coordination, which are reliable indicators of nigrostriatal dopamine depletion. Increased depletion of D2 receptors in the striatum;

In 6-OHDA-lesioned rats, SOD, CAT and GSH activities were significantly restored by Jatamansi pretreatment due to the antioxidant or GSH-enhancing effects, and the pretreatment-induced increase in TH-IR fiber density clearly shows a dose increase. dependency. increasing the number of living neurons and Nardostachys To determine the antiparkinsonian effect of the ethanol extract of Jatamansi.

5) Chrysanthemum morifolium Ramat; (Family: Asteraceae) Auga used by Choi Dong-guk et al. Chrysanthemum morifolium Ramat extract in SH-SY5Y cell culture induced by MPP+ in an in vitro

parkinsonism model. SH-SY5Y cell cultures were evaluated for measurement of cell viability, monitoring and analysis of total RNA expression,

immunoblot analysis, flow cytometry of apoptotic cells and measurement of intracellular reactivity.

Oxidizing species (ROS) and elimination of free radicals. Chrysanthemum water extract at various concentrations inhibits the mitochondrial apoptosis pathway and significantly enhances the increase of Bax/Bcl-2 in SH-SY5Y cells, inhibits the accumulation of ROS, and weakens the cells SH-SY5Y. that In a dose-dependent manner, PARP cleavage results in inhibition of caspase-3 expression and downstream apoptosis. A signaling pathway that interrupts the activation of PARP protein degradation. And it shows strong antioxidant activity and radical scavenging activity against DPPH, superoxide, hydroxyl and alkyl radicals. Cassia seed; (Series: Legumes) According to Myung-sook et al, daily oral administration of 85% ethanol extract of Cassia seed (moon tree seeds) for 15 days significantly inhibited motor dysfunction and DA neuron loss at a dose of 50 mg. / kg. Contains ~ Various concentrations (0.1-50 lg/ml) prevented cell death against 6-OHDA-induced DA neurotoxicity through antioxidant and antimitochondrial apoptosis mechanisms in PC12 cells, and also in 6-OHDA and primary midbrain cultures. MPP+ induced neurotoxicity

6) Anemopegma mirandu; (Family: Bigoniaceae); Syn: Catuaba Lisandro Diego Giraldez et al investigated the neuroprotective activity of the following extracts: Anemopaegma mirandu in rotenone-induced apoptosis in human neuroblastoma SH-SY5Y cells using an in vitro Parkinson's disease model. At concentrations ranging from 0.0097 mg/mL to 1.250 mg/mL, the extract showed the effect of increasing cell survival by 22.3±3.6%, 22.0±2.1% and 15.8±0.7%, recovery of cell morphology and nucleus to levels indistinguishable from those of active cell management and protection of cytoplasmic membranes and mitochondria. In human neuroblastoma SH-SY5Y cells.

7) Hypericum perforatum; (Family: Hpericaceae) J. Benedı et al before rotenone treatment of rats with 4 mg/kg standard extract of Hypericum perforatum for 45 days, decreased MnSOD activity, mRNA levels, SOD and CAT activities, and change the redox index. protects cells from the harmful effects of hydrogen peroxide and exhibits neuroprotective activity . M. Sabesan et al. The combination of bromocriptine and ethanol extract of Hypericum perforatum prevented behavioral deficits and biological changes, such as improvement of dopamine, DOPAC levels, antioxidant status and significant reduction of lipid peroxidation.

8) Gastrodia elata Blume; (Family: Orchidaceae) Dong Kug Choi et al found that pre-treatment with ethanolic extricate of Gastrodia elata Blume at different concentrations (10, 100, 200_g/mL)

enhance the MPP+-induced Bax/Bcl-2 proportion rise in SH-SY5Y cells, weakened capase-3 enactment and PARP cleavage in a dose-dependent way, appears anti-oxidant impact with critical radical rummaging movement for DPPH, and alkyl radicals, stifled the collection of ROS and hinder the both intracellular ROS generation and downstream apoptotic signaling pathways.

9))Centella asiatica; (Family: Umbelliferae); Syn: Hydrocotyl asiatica Kumar ponnusamy et al examined that watery extricate of Centella asiatica at a measurements of 300mg/kg for 14 days is viable against MPTP actuated parkinsonism. It acts by showing its antioxidant movement in hippocampus and corpus striatum locale of brain. Extricate diminishes lipid peroxidation, protein carbonyls substance and increments Super oxide dimutase, Glutathione peroxidase, Catalase, Total antioxitants, Xanthine oxidase .

10)Thuja orientalis; (Family: Cupressaceae); Syn: Biota orientalis Myung Sook Goodness et al detailed the defensive impacts of standardized ethanolic extricate of Thuja orientalis take off in

SH-SY5Y cells. Pretreatment with dosages of 0.1–100 lg/ml in 6-OHDA actuated neurotoxicity curbed the neuronal cell passing, repressed overabundance ROS and NO generation and tall radical rummaging movement, blocked the cytochrome c discharge, and caspase-3 enactment, stifled the expanded level of ERK phosphorylation and anti-mitochondrial-mediated apoptosis.

11) Mucuna pruriens; (Family: leguminosae) ; Syn: Velvet bean A. Pinna et al found that of Mucuna pruriens extricate at a measurements 16 mg/kg (containing 2 mg/kg of L-DOPA) and 48mg/kg (containing 6 mg/kg of L-DOPA) reliably antagonized the shortage in inactivity of step start, MP extricate intensely initiated a altogether higher contralateral turning, at dosage of 48 mg/kg (containing 6 mg/kg of L-DOPA), suggested a critical opposing movement on both engine and sensory-motor shortages .

12) Ginkgo biloba; (Family: Ginkgoaceae) ; Syn: Pterophyllus salisburiensis Muzamil Ahmad et al detailed useful impacts of Standard unrefined Extricate of Ginkgo biloba in Parkinsonian rats. The pre-treatment with EGb (50, 100, and 150 mg/kg body weight) for 3 weeks obviously deliver diminish in sedate initiated revolution and a critical reclamation of striatal DA and its metabolites, it could be a strong inhibitor of MAO which anticipate the debasement of DA and increment its availability, The locomotor shortfalls were reestablished, causes increment within the substance of GSH and diminish within the degree of lipid peroxidation. Ginkgo biloba shows up to act by means of antioxidant, free radical rummaging, MAO-B-inhibiting and DA-enhancing components that protect the compromised cells inside the dopaminergic injuries .

13) Plumbago scandens (Family: Plumbaginaceae); Syn: Jasmim azulQ L.C.S.L.Morais et al found that Unrefined ethanolic extricate (CEE) and add up to acetic acid derivation division (TAF) of Plumbago scandens (1000 mg/kg, i.p.) Diminish locomotor action, the nearness of catalepsy and palpebral ptosis, in this way acts against parkinsonism .

14)Bacopa monniera; (Family: Scrophulariaceae) ; Syn: Brahmi Deepak Sharma et al found that Ethanolic extricate of entire plant of Bacopa monniera appears the restorative impact in treatment of parkinsonism actuated by aluminium neurotoxicity. It acts by lessening Turf movement altogether, avoids the increment in TBARS, lipofuscin aggregation and ultrastructural changes . Muralidharan et al inspected the neuroprotective properties of standardize extricate of Bacopa monniera against rotenone initiated oxidative harm and neurotoxicity. At concentrations of 0.05 and 0.1% for 7 days within the count calories it exhibited critical lessening within the levels of endogenous oxidative markers viz., malondialdehyde, hydroperoxide and protein carbonyl substance.Encourage, BM advertised total assurance against rotenone (500 mM) actuated oxidative stretch and particularly hindered dopamine consumption (head locale, 33%; body locale, 44%) conjointly conferred critical resistance (43–54% assurance) in a paraquat oxidative stretch bioassay in Drosophila melanogaster .

16) Pueraria thomsonii ;(Family:Fabaceae) Mei-Hsien Lee et al explored the Neurocytoprotective impacts of Pueraria thomsonii bioactive constituents ie daidzein and genistein in 6-OHDA actuated aptosis in separated PC12 cells. daidzein and genistein at concentrations of 50 μM and 100μM hindered caspase-8 and mostly repressed caspase-3 actuation, giving a defensive instrument against 6-OHDA-induced cytotoxicity in NGF-differentiated PC12 cells.Welcome to Gboard clipboard, any text you copy will be saved here.Welcome to Gboard clipboard, any text you copy will be saved here.Tap on a clip to paste it in the text box.Tap on a clip to paste it in the text box.Tap on a clip to paste it in the text box.

II. CONCLUSION –

We have selected 17 herbs for Parkinson's disease and calculated the appropriate daily dose. In addition, this study applied statistical methods to traditional medicine literature and presented a new method of preselection of herbs that are effective for specific diseases. Parkinson's disease is a rare disease that progresses rapidly. It seems to be moving fast. Treatment is medical treatment. Method (prescribed with levodopa preparations) or no other drug) and non-pharmacological approaches. (eg exercise, exercise, speech therapy, etc.) circle). Methods such as deep brain stimulation and therapy The use of levodopa-carbidopa intestinal suspension may benefit individuals. If you have drug-resistant epilepsy, your symptoms may get worse if: When the drug wears off, movement problems .

REFERENCE –

1. https://drjitendrashukla.com/parkinson/?gad_source=1&gclid=Cj0KCQiApOyqBhDlARIsAGfnyMq2mz3_9UV3ZZAsPGcHkepux-9HQ_3xnf9A7f78P33zR5HrcD9D0p0aApHiEALw_wcB
2. https://www.parkinsonseurope.org/living-well/therapies/complementary-therapies/ayurveda/
3. https://www.sciencedirect.com/science/article/abs/pii/S0367326X1200336X
4. https://www.researchgate.net/publication/216302150_Herbal_Treatment_of_Parkinsonism_A_Review

5. https://www.mountsinai.org/health-library/condition/parkinson-disease
6. https://www.frontiersin.org/articles/10.3389/fnins.2018.00612/full
7. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8046395/#:~:text=pruriens%20seeds%20extract%20(400%20mg,anti%2DParkinson%20effect%20in%20rats.&text=Treatment%20with%20M.,OHDA%2Dinduced%20PD%20rat%20model
8. https://academic.oup.com/bmb/article/86/1/109/379503
9. https://www.hindawi.com/journals/omcl/2021/9923331/
10. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3352906/.
11. https://www.tandfonline.com/doi/full/10.1080/13880209.2019.1618344.
12. https://www.jkom.org/journal/view.php?number=5060
13. https://www.e-jmd.org/journal/view.php?doi=10.14802/jmd.14009
14. https://www.mayoclinic.org/diseases-conditions/parkinsons-disease/diagnosis-treatment/drc-20376062
15. https://www.jarlife.net/614-review-of-natural-products-on-parkinsons-disease-pathology.html
16. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4517533/
17. https://dementech.com/2022/11/10/what-are-the-5-stages-of-parkinsons-disease/stages
18. https://www.researchgate.net/publication/216302150_Herbal_Treatment_of_Parkinsonism_A_Review
19. https://jamanetwork.com/journals/jama/article-abstract/2760741#:~:text=Importance%20Parkinson%20disease%20is%20the,individuals%20worldwide%20have%20Parkinson%20disease
20. https://www.oaepublish.com/articles/2347-8659.2020.58
21. https://www.neurologyindia.com/article.asp?issn=0028-3886;year=2018;volume=66;issue=7;spage=26;epage=35;aulast=RadhakrishnanAB
22. https://pubmed.ncbi.nlm.nih.gov/31141426/
23. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5514193/
24. https://images.app.goo.gl/xp2FG4eD2u8Qcy9e8
25. https://www.google.com/imgres?imgurl=https%3A%2F%2Fneurologysleepcentre.com%2Fblog%2Fwp-content%2Fuploads%2F2021%2F12%2Fparkinson2.jpg&tbnid=VdTjaxohoEYN2M&vet=1&imgrefurl=https%3A%2F%2Fneurologysleepcentre.com%2Fblog%2Fwhat-is-parkinsons-disease&docid=LWbi52AmvekTTM&w=560&h=500&hl=en-US&source=sh%2Fx%2Fim%2Fm1%2F4.
26. https://images.app.goo.gl/dGUrLQMpunNnRcid7.
27. https://images.app.goo.gl/ZR7R3fTLZzoGMd4X8.
28. https://images.app.goo.gl/NnmYcLwMLyjVjJZv8.
29. https://images.app.goo.gl/Nif3C6eSj3JwV2ep8.
30. https://www.google.com/imgres?imgurl=https%3A%2F%2Fd2seqvvyy3b8p2.cloudfront.net%2F6c6e3a165d7ed0010568bc217825dfcf.jpg&tbnid=Y0oCqNVXNjmDjM&vet=1&imgrefurl=https%3A%2F%2Fpowo.science.kew.org%2Ftaxon%2Furn%3Alsid%3Aipni.org%3Anames%3A859418-1&docid=87aphyBH4baawM&w=1600&h=1071&hl=en-US&source=sh%2Fx%2Fim%2Fm1%2F4.
31. https://images.app.goo.gl/fsf33STGpPbz65v27.
32. https://images.app.goo.gl/Fmyb8t3LuJZvwJrXA.
33. https://images.app.goo.gl/daLHcQ99si45DQWZ7
34. https://images.app.goo.gl/djk4KbjEGbrkhmp36.
35. https://images.app.goo.gl/WzGHZavxdV9vcfHQ8
36. https://images.app.goo.gl/5VetHfTxpjct8G5W8
37. https://images.app.goo.gl/uaUWbKAKcLKUTtJf8
38. https://images.app.goo.gl/tt7SXvrKvVSTN2Pd8