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Scope of some Indian medicinal plants in the management of a few neuro-degenetative disorders *in-silico*: a review

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Abstract

Neuro-degenetative disorders like Alzheimers, Parkinsonism, Multiple Sclerosis, Migraine and Schizophrenia are studied in this work. Interaction studies of receptors involved in the above disorders with compounds from Indian medicinal plants are studied *in-silico*. Best interacting phytocompounds are selected based on the docking score of the individual phytocompound with the receptor protein. These selected phytocompounds can be further checked for their efficacy by *in-vitro* receptor-ligand binding assay studies.

Keywords: **Alzheimers, Parkinsonism, Lewy Body Dementia, Multiple Sclerosis, Migraine, Schizophrenia**

Introduction

Modern people lead a very stressful life. Modernization has contributed restlessness & stress which directly affects Central Nervous System (CNS) leading to CNS degeneration, dysfuncions & disorders. Change of life-style with too much dependency on synthetic chemical drugs, preserved food items, pollution infested urban life have been disturbing delicate balance or different homeostasis prevailing in the human biochemical system. A new trend of neurosis with inner emptiness or a sense of meaninglessness of life is presently one of the major challenges to modern psycho-somatic problems. Even new generation chemical drugs are not sufficient to combat this alarming situation. From time immemorial Indian medicinal plants have been in use for various diseases including psychological disorders. In Charaka Samhita (chapter IV *sharira sthana*) it is surprising to note that a detailed account of the varieties of body mind relations has been given, which not only tally with the modern psychology, even it elaborates further. However Charaka strongly emphasized to keep our body & mind healthy "through self-discipline and by observing some simple rules of ethical, moral conducts and habits in relation to food, exercise, sleep, personal hygiene & cleanliness."

Drugs that act upon the CNS influence the lives of everyone, everyday – because they can produce specific physiological & psychological effects. Most of the chemical drugs (psychotropic agents) are mainly intended to sedate, stimulate or to change mood, thinking or behavior. Four major categories of CNS drugs are, (a) anti-anxiety, mostly tranquilizing and sedative agents used for the therapy of anxiety disorders and insomnia; (b) anti-depressants or mood elavating agents and to treat

obsessive compulsive disorders; (c) anti-psychotic drugs used to treat severe psychiatric illness like schizophrenia; (d) anti-manic or mood stabilizing drugs including certain anti-convalescent.

CNS-degenerative disorders are characterized by progressive and irreversible loss of neurons from specific regions on the brain, including death of neurons. Many neurodegenerative diseases including Schizophrenia, Alzheimer's, migraine and dementia with Lewy bodies etc. occur as a result of neurodegenerative processes. As research progresses, many similarities appear which relate these diseases to one another on a sub-cellular level. Discovering these similarities research offers hope for therapeutic advances that could ameliorate many diseases simultaneously. There are many parallels between different neurodegenerative disorders including atypical protein assemblies as well as induced cell death. Neurodegeneration can be found in many different levels of neuronal circuitry ranging from molecular to systemic. The word dementia comes from the Laden de meaning "apart" and mens from the genitive mentis meaning "mind". Dementia is the progressive deterioration in cognitive function - the ability to process thought (intelligence).

For the management of different diseases of CNS where chemical & synthetic drugs are not fully effective, different phyto medicinal compounds (phyto chemicals from herbal sources) are successfully utilized with minimum or without side effects even for long term therapy. These drugs are used to treat anxiety disorders, mania, depression, acute & chronic cases dementia, etc. without much altering consciousness.

In this review article selected phyto-compounds from different Indian medicinal plants have been used to understand the scope & activity on CNS using bioinformatic parameter.

Alzheimer's disease (AD), also known in medical literature as Alzheimer disease, is the most common form of dementia. Alzheimer's disease is a neurological disorder in which the death of brain cells causes memory loss and cognitive decline. A neurodegenerative type of dementia, the disease starts mild and gets progressively worse. The brain region most vulnerable to neuronal dysfunction and cell loss in AD is the medial temporal lobe, including entorhinal cortex and hippocampus. There is no cure for the disease, which worsens as it progresses, and eventually leads to death. As the disease advances, symptoms can include confusion, irritability, aggression, mood swings, trouble with language, and long-term memory loss. As the sufferer declines they often withdraw from family and society. Gradually, bodily functions are lost, ultimately leading to death. Gore M, et. al. 2010, did interactions studies of phytocompounds with alzheimer receptors [1].

Early onset of AD are associated with mutations in the β-amyloid precursor protein (APP), apolipoprotein E (ApoE), presenilin-1 and 2 (PS1 and PS2).

Usage of phytocompounds as remedy/cure for AD.

Several ayurvedic herbs are seen as used for treating AD. Some of the studied herbs for AD include shankhapushpi (*Canscora decussate*), *Nardostachys jatamansi* and Kavacha (*Mucuna pruriens*). It is seen that xanthone, the active component of *Canscora decussate*, nardal, the active component of *Nardostachys jatamansi* and ergotamine, the active

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component of *Mucuna pruriens* are seen to interact with the receptor proteins of AD namely APP, ApoE, PS1 and PS2.

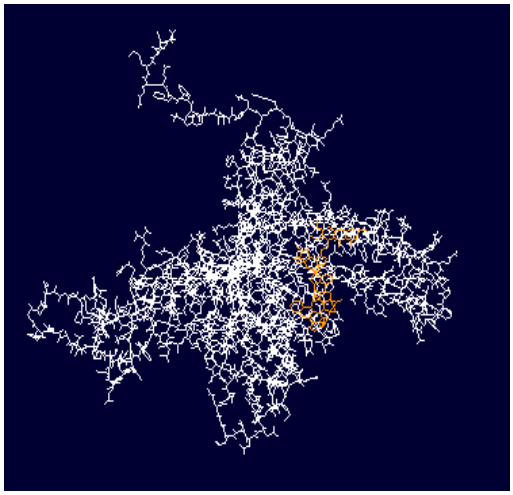

Fig. 1- Docked structure APP with nardal, ergotamine & xanthone combination (figure source [1])

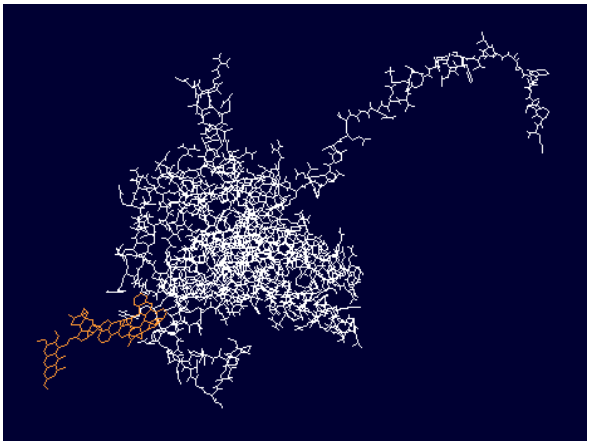

Fig. 2- Docked structure APOE with nardal, ergotamine & xanthone combination (figure source [1])

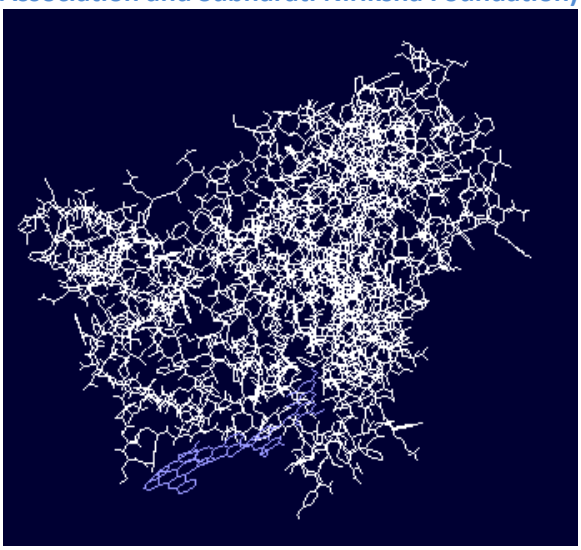

Fig 3- Docked structure PS1 with nardal, ergotamine & xanthone combination (figure source [1])

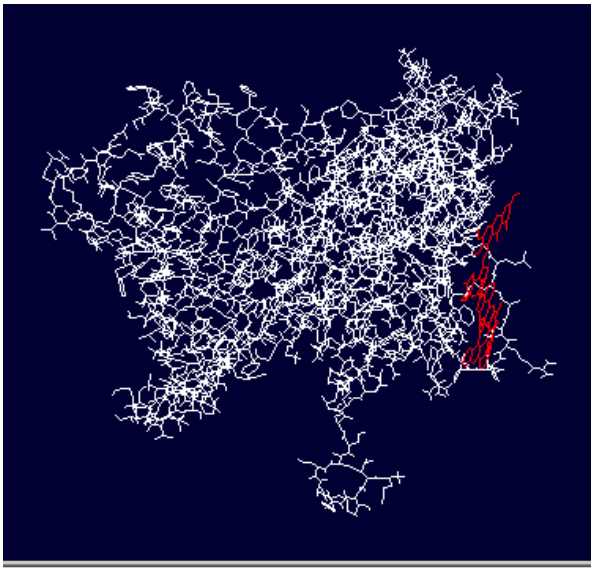

Fig. 4- Docked structure PS2 with nardal, ergotamine & xanthone combination (figure source [1])

Dementia with Lewy bodies (DLB), also known under a variety of other names including Lewy body dementia (LBD), diffuse Lewy body disease, cortical

Lewy body disease, and senile dementia of Lewy type, is a type of dementia closely associated with both Alzheimer's and Parkinson's diseases. It is characterized anatomically by the presence of Lewy bodies, clumps of alpha-synuclein and ubiquitin protein in neurons, detectable in *post mortem* brain histology.

LBD is a progressive degenerative dementia of the elderly. Its primary feature is cognitive decline, particularly of executive functioning. The patient will display an inability to plan, or a loss of analytical or abstract thinking. Persons with LBD will show markedly fluctuating cognition. Wakefulness will vary from day to day, and alertness and short term memory will rise and fall. Persistent or recurring visual hallucinations with vivid and detailed pictures are often as an early diagnostic symptom.

α-synuclein is a component seen in LBD [2].

Usage of phytocompounds as remedy/cure for LBD. Bagchi P, Hopper W, 2011 did interaction studies of α-synuclein receptor with compounds of *Valeriana jatamansi* [2].

Valeriana jatamansi is a small herbaceous species found in Himalayas. Eleven jatamanins including a new lignin, (+)-9'-isovaleroxylariciresinol are compounds found in *Valeriana jatamansi*. Screening studies show that jatamanin 11 showed good interaction with α-synuclein receptor [2].

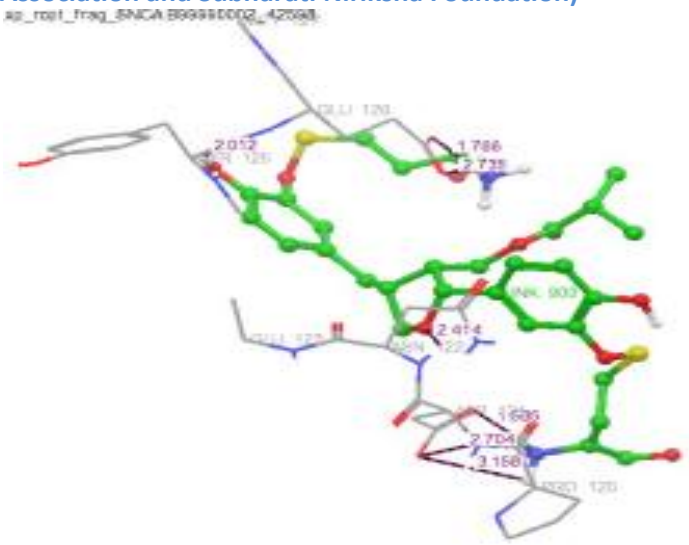

Fig. 5- Interaction of SNCA protein with jatamanin11 from *Valeriana jatamansi* (figure source [2]).

A **migraine** is a severe, painful headache that is often preceded or accompanied by sensory warning signs such as flashes of light, blind spots, tingling in the arms and legs, nausea, vomiting, and increased sensitivity to light and sound. The excruciating pain that migraines bring can last for hours or even days. Childhood migraines are linked to behavioral problems.

Most of us go through about six sleep cycles with about four stages of sleep, plus rapid eye movement (REM) sleep. The deepest stages of sleep (stages 3 and 4) are necessary for the production of sufficient serotonin and dopamine, both neurotransmitters. These neurotransmitters are the "feel good" chemical messengers in the brain, and both depend on adequate sleep; a decrease in serotonin and dopamine is associated with poor sleep or sleep problems. One reason for waking with migraines is that REM sleep is most powerful just before awakening. Sleep problems can then trigger migraines by causing

instability of serotonin and a lowering of dopamine levels [3, 4].

Mutation in mammalian serotonin hydroxytryptamine receptor 2 (HTR2) is implicated as factor in migraine. Migraine remains the most common yet least understood neurological syndrome.

Usage of phytocompounds as remedy/cure for migraine.

In-silico studies alone prove that Acoradin from *Acorus calamus* has the potential to treat migraine [3] and both *in-silico* and *in-vitro* studies proves that the colchicine from *Colchicum autumnale* (hiranyatutha, surinjan) and hypophyllanthanthin from *Phyllanthus amarus* (type Bhumi amla) has the potential to treat migraine [4].

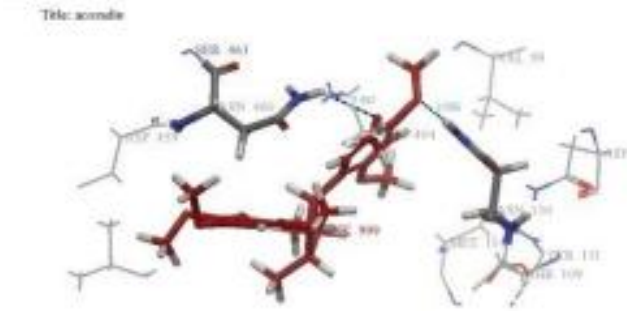

Fig. 6- HTR2A receptor with acoradin (figure source [3])

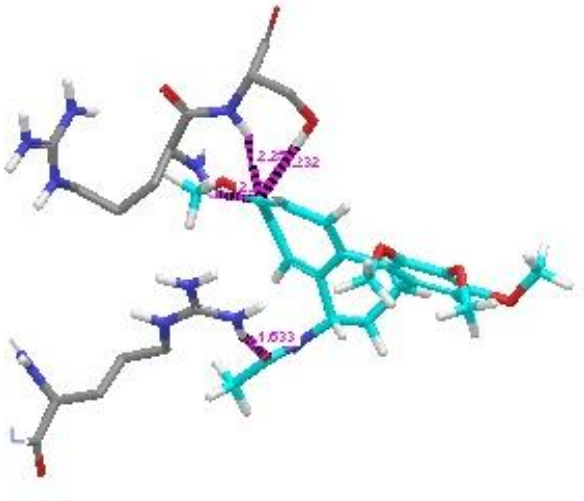

Fig. 7-5HT2 receptor with colchicines (figure source [4])

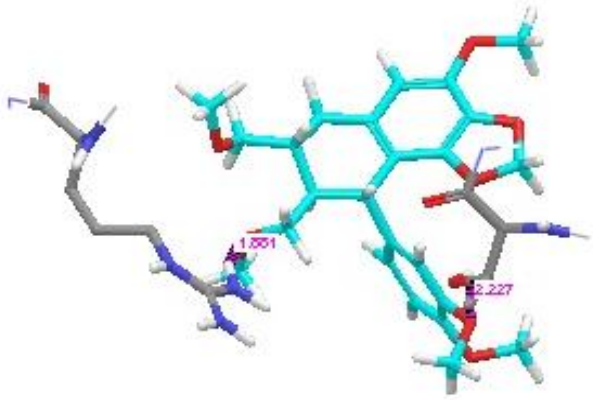

Fig. 8-5HT2 receptor with hypophyllanthin (figure source [4])

Parkinson's disease (**PD)** is a degenerative disorder of the central nervous system which affects the basal ganglia. The motor symptoms of PD result from the death of dopamine-generating cells in the substantia nigra, a region of the midbrain; the cause of this cell death is unknown. Early in the course of the disease, the most obvious symptoms are movement-related; these include shaking, rigidity, slowness of movement and difficulty with walking and gait. Later, thinking and behavioral problems may arise,

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with dementia commonly occurring in the advanced stages of the disease, whereas depression is the most common psychiatric symptom. Other symptoms include sensory, sleep and emotional problems. PD is more common in older people, with most cases occurring after the age of 50.

Parkinson's disease can cause neuropsychiatric disturbances which can range from mild to severe. This includes disorders of speech, cognition, mood, behaviour, and thought. 2013 work of Bagchi P highlighted the interaction studies of phytocompounds with receptors implicated in parkinsonism [5].

The PARK2 gene, one of the largest human genes, provides instructions for making a protein called parkin and leucine-rich repeat kinase 2 (LRRK2), also known as dardarin (from the Basque word "dardara" which means trembling), is an enzyme that in humans is encoded by the PARK8 gene are seen as components in Parkinson's disease.

- PARK2 protein best docks with Kaempferol, Curcumin, Racemosol, Calamusenone, Jatamanin1, Bacopaside I, Beta-sitosterol, Alpha and beta-santalol *and no docking with Eugenol and Levodopa*.

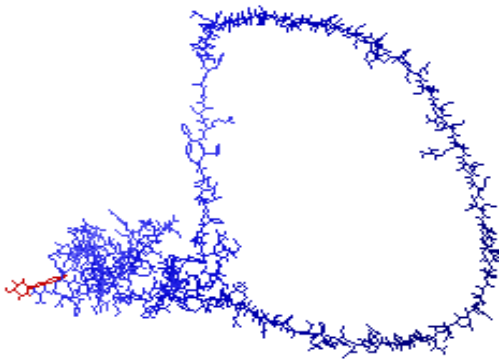

Fig. 9: PARK2 receptor with Kaempferol (figure source [5])

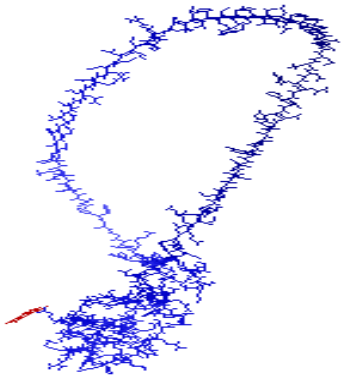

Fig. 10: PARK2 receptor with Curcumin (figure source [5])

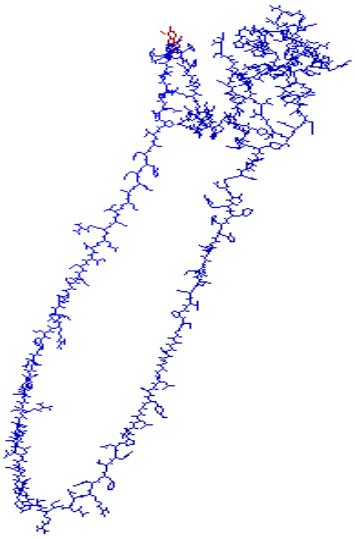

Fig. 11: PARK2 receptor with Racemosol (figure source [5])

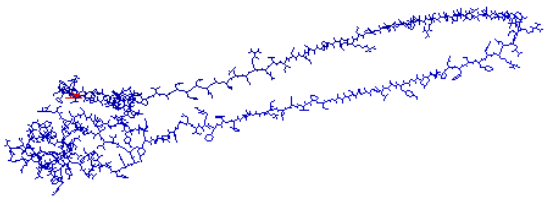

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Fig. 12: PARK2 receptor with Calamusenone (figure source [5])

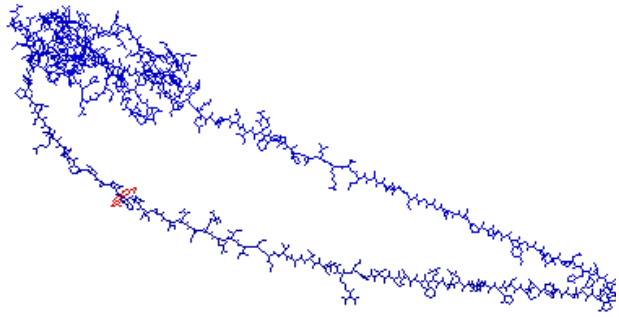

Fig. 13: PARK2 receptor with Jatamanin1(figure source [5])

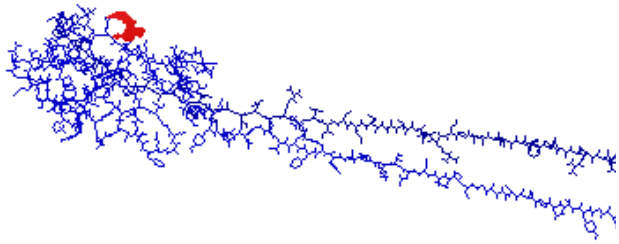

Fig. 14: PARK2 receptor with Bacopaside I (figure source [5])

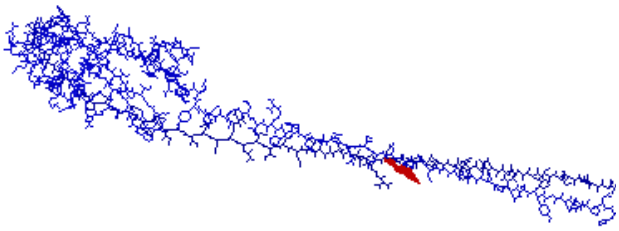

Fig. 15: PARK2 receptor with Beta-sitosterol (figure source [5])

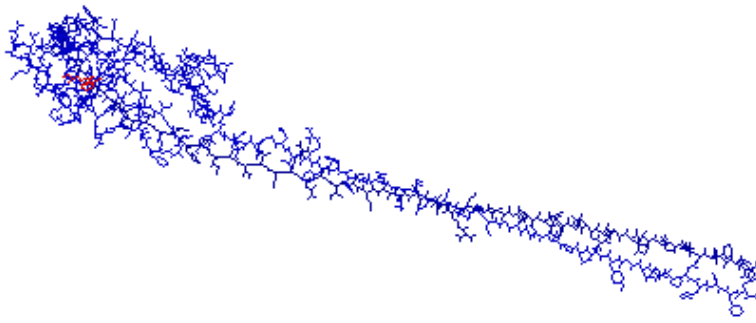

Fig. 16: PARK2 receptor with Alpha-santalol (figure source [5])

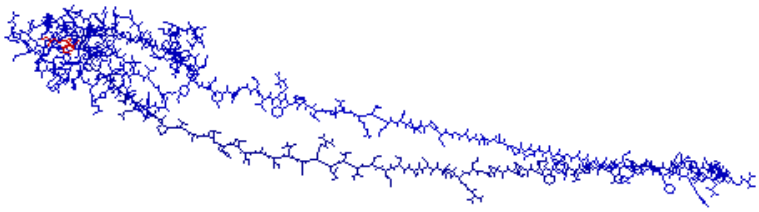

Fig. 17: PARK2 receptor with Beta-santalol (figure source [5])

- LRRK2 protein best docks with Bacopaside, Curcumin, Withanone, Bacopaside I and Cordifolioside A.

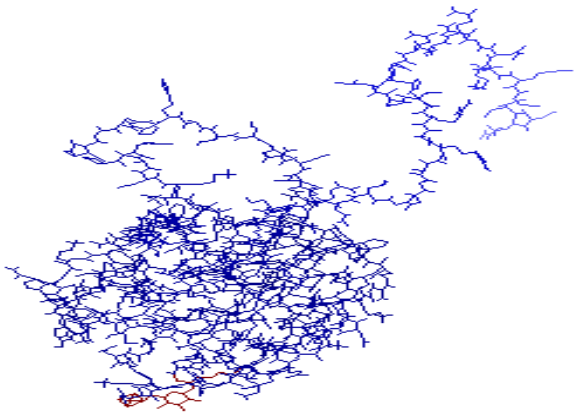

Fig. 18: LRRK2 receptor with Bacopaside (figure source [5])

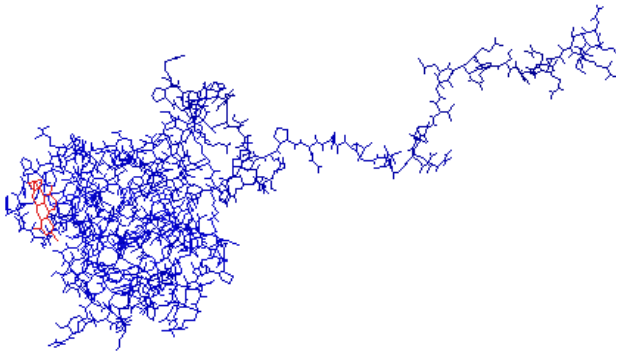

Fig. 19: LRRK2 receptor with Curcumin (figure source [5])

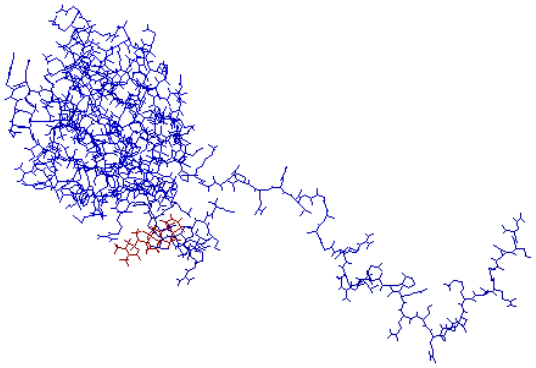

Fig. 20: LRRK2 receptor with Withanone (figure source [5])

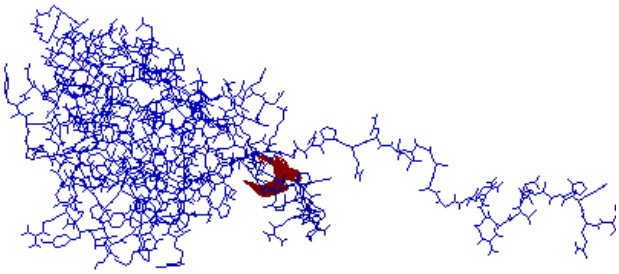

Fig. 21: LRRK2 receptor with Bacopaside I (figure source [5])

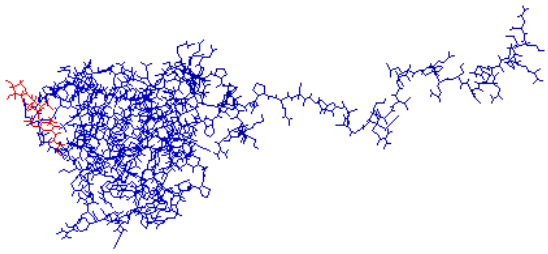

Fig. 22: LRRK2 receptor with Cordifolioside A (figure source [5])

Multiple sclerosis (abbreviated MS, also known as disseminated sclerosis or encephalomyelitis disseminate) is an important cause of long term disability in adults. MS is a disease in which the fatty myelin sheaths around the axons of the brain and spinal cord are damaged, leading to demyelination and scarring as well as a broad spectrum of signs and symptoms. Disease is twice as common in females as men. There is evidence that both genetic and environmental factors play a causative role. The peak age of onset is the fourth decade and onset before puberty or after the age of 60 years is rare. *In-silico* work by Somashekhar R *et.al.*, 2012, stated the phytocompounds interacting with receptor proteins implicated in multiple sclerosis [6]. Protein Tyrosine Phosphatase, Receptor type, C polypeptide (PTPRC), Tumor necrosis factor super family Receptor1A (TNFRSF1A), Human Leukocyte Antigen DR-1 (HLA-DR1) mutation genes along with Epstein-Barr virus is taken & suitable inhibitors are seen as components involved in multiple sclerosis [6].

Schizophrenia

Schizophrenia is a severe mental disorder characterized by two kinds of symptoms; positive psychotic symptoms -thought disorder, hallucinations, delusions, and paranoia -and negative

symptoms –impairment in emotional range, energy, and enjoyment of activities. Schizophrenia is a particular form of psychosis, a term encompassing several severe mental disorders that result in the loss of contact with reality along with major personality derangements. Presently researchers suspect a genetic cause for schizophrenia along with environmental basis. Psychosocial factors play an important role in the formation & development of this disorder. Individual as well as family counseling is necessary to help patients to lead a normal social life. Cognitive behavioral approach along with proper medication can pave a path for curing this disorder. The cause of Schizophrenia is multi-gene mutation (including environmental factors). The illness can be described as a collection of particular symptoms that usually fall into four basic categories: formal thought disorder, perception disorder, feeling/emotional disturbance, and behavior disorders. A sibling or a parent with schizophrenia increases the likelihood that a person will have the disease. Neuroscientists are facing enormous difficulties in treating schizo (& neuro-patients) since they have psychotic trend with their own world of hallucinations and delusions. The schizophrenic receptors are virtually screened with the phytocompounds and the interactions were noted [7, 8, 9, 10].

Receptor genes involved:

Regulator of G protein signaling 4 (RGS4)

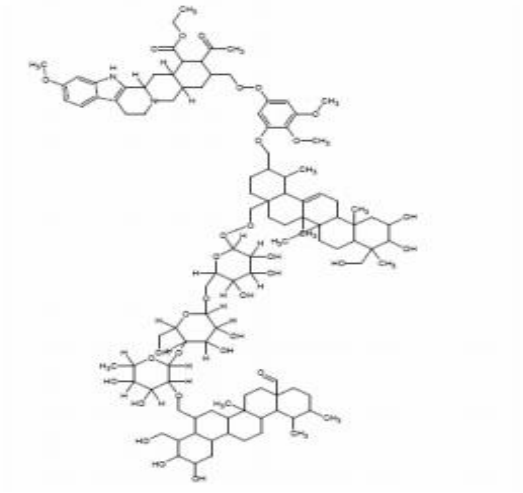

Fig. 24: Combined Structure of reserpine, asiaticoside & withanolides (figure source [7])

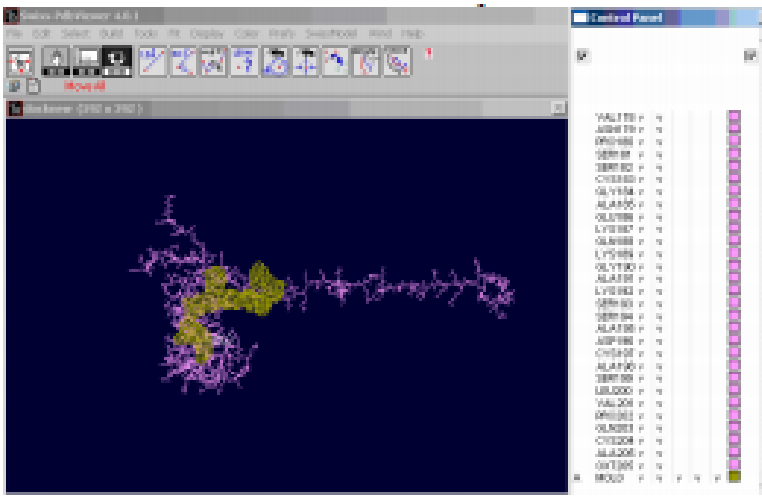

Fig. 25: Docked structure of RGS4 with combined Structure of reserpine, asiaticoside & withanolides (figure source [7])

Mutant protein hSKCa3 responsible for Schizophrenia. The structure of Withanolide is determined and docked with conotoxin protein; this combination is docked with hSKCa3 protein, hence establishing a remedy [8].

Catechol-O-methyltransferase (COMT)

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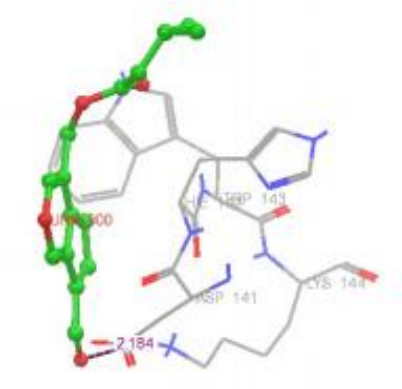

Fig. 26: Docked structure of COMT protein with baldrinal (figure source [9])

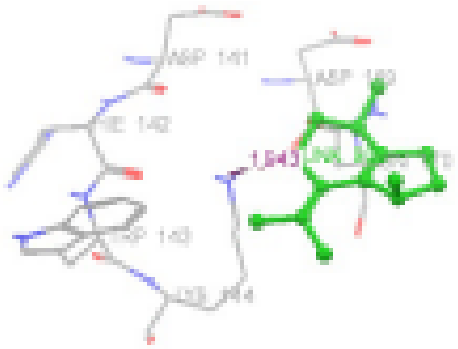

Fig. 27: Docked structure of COMT receptor with calamusenone (figure source [9])

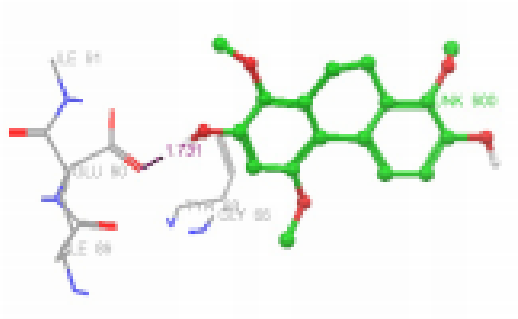

Fig. 28: Docked structure of COMT receptor with racemosol (figure source [9])

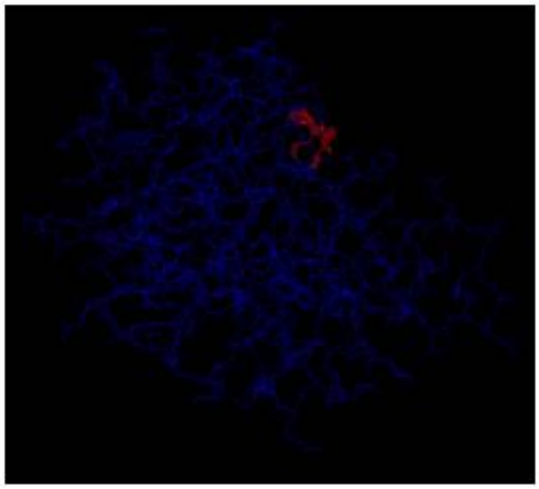

Fig. 29: Docked structure of COMT receptor with β santalol (figure source [10])

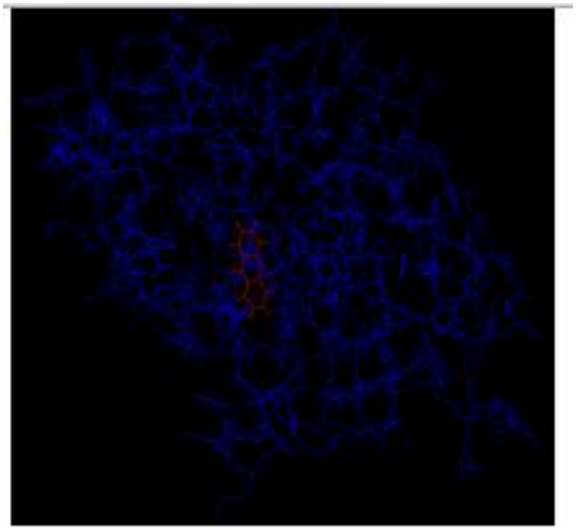

Fig. 30: Docked structure of COMT receptor with xanthone (figure source [10])

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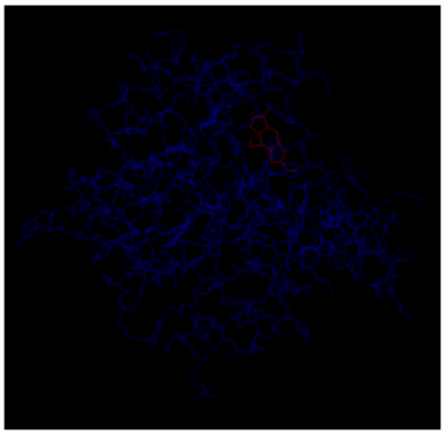

Fig. 31: Docked structure of COMT receptor with nardal (figure source [10])

GRM3: Glutamate

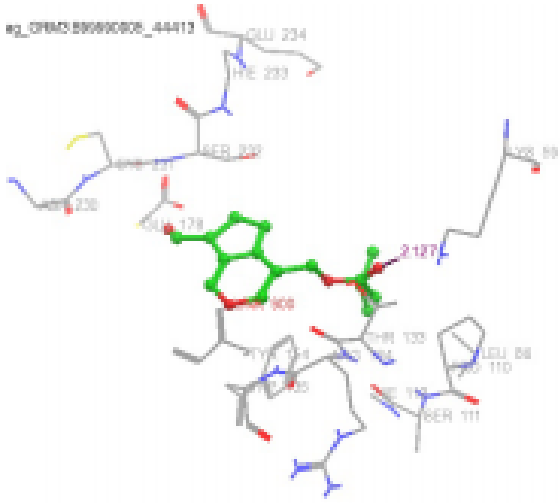

Fig. 32: Interaction of GRM3 receptor with homobaldrine (figure source [9])

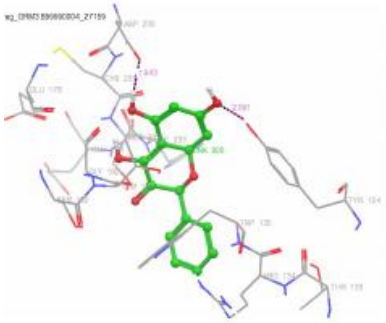

Fig. 33: Interaction of GRM3 receptor with ganglin (figure source [9])

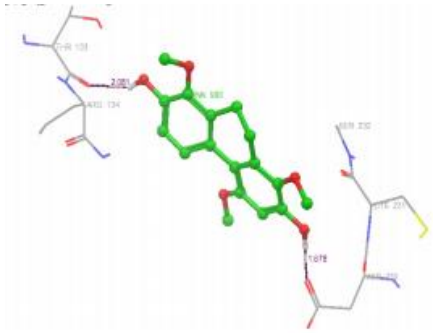

Fig. 34: Interaction of GRM3 receptor with Racemosol (figure source [9])

N-Methyl-D-Aspartate (NMDA) receptor is a sub-type of the glutamate receptor, whose function is to mediate fast excitory synaptic transmissions in the central nervous system

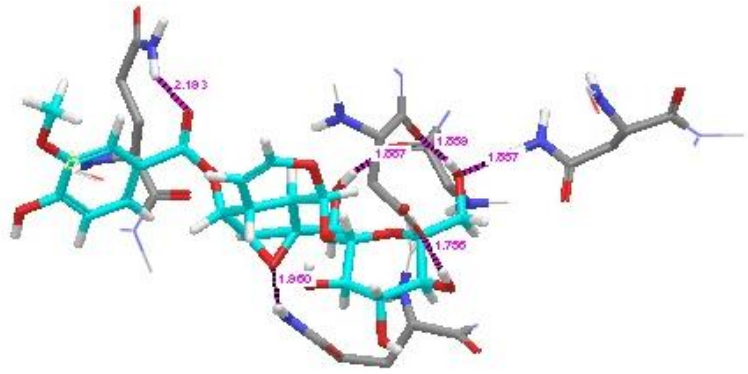

Fig. 35: Docking of NMDA receptor with picroside II (figure source [10])

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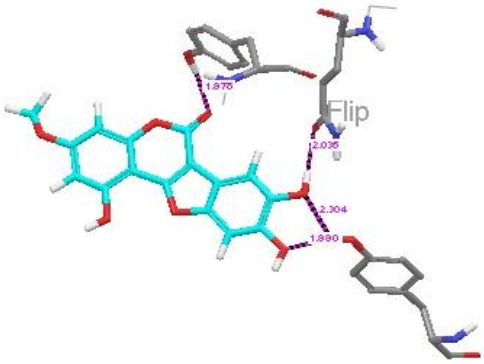

Fig. 36: Docking of NMDA receptor with wedelolactone (figure source [10])

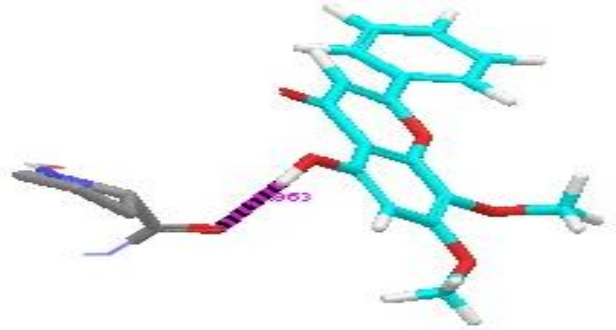

Fig. 37: Docking of NMDA receptor with 7-o-methylwogonin (figure source [10])

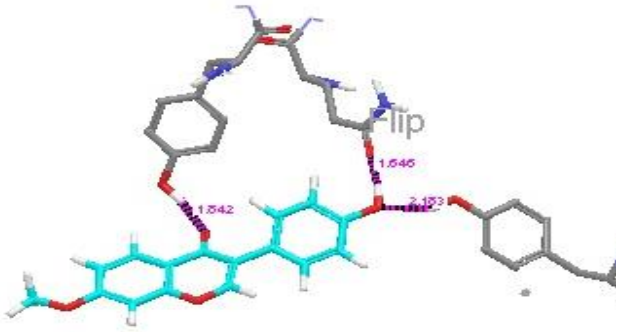

Fig. 38: Docking of NMDA receptor with isoformonocetin (figure source [10])

Conclusion

Currently available therapies for neurodegenerative disorders alleviate the disease symptoms but do not alter the underline neuro-degenerative process. The goal of recent research is to develop treatment that can prevent, retard, or reverse neuronal disorders leading to cell death. Aging related neurological disorders are associated with progressive impairment in the capacity of neurons for oxidative metabolism, perhaps in part, because of a progressive accumulation of mutations in the mitochondrial genome. A consequence of impaired oxidative capacities is the production of reactive compounds such as hydrogen peroxide and oxygen free-radicals. Unchecked, these reactive species can lead to DNA damage, peroxidation of membrane lipids and ultimately neuronal death [11]. Many genes probably contribute to aging, with those that determine durability and maintenance of somatic cell lines are particularly important. However, genetic factors only account for around 25% of variance in human life span; nutritional and environmental factors determine the rest. Further adverse drug reactions and the effects of drug interactions specially resulting from the long term use of conventional chemical psychotropic drugs which may also induce neurodegenerative diseases like parkinsonism [12]. A major contribution to random molecular neuronal damage is made by reactive oxidative species. Fresh green vegetables, fruits, natural herbs, contain a lot of anti-oxidents and free radical scavenging properties. Recent work on anti-oxidant activities of some medicinal plants show that *Nardostachys jatamanasi (Valeriana wallichii)*, used in present review, has got potent anti-oxidant properties [13].

From very ancient days different medicinal plants are part & parcel of major populations of India, China & other Asian countries. Technical committee of World Health Organization (WHO),

while studying various research needs for health for all – observed "traditional systems dealing with health care & its problem have been practiced for generations in nearly all the countries in the south east Asian region. This would clearly indicate that optimal use needs to be made of this heritage available for better delivery of health care." [14]. Hence it can be inferred that medicinal herbs have got tremendous scope and potential for long term health care especially to combat neurological disorders and maintenance of different homeostasis prevailing in the human biochemical system. As regards scope and utility of ayurvedic psychotropic medicinal plants (reviewed in this paper) – these may be used either singly or in combinations and even the specific phytochemicals may be used as adjunct to the existing therapy with chemical drugs in minimum dosage to minimize side effects.

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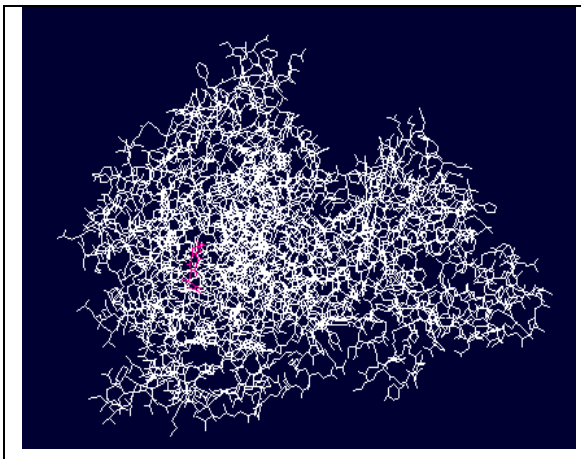
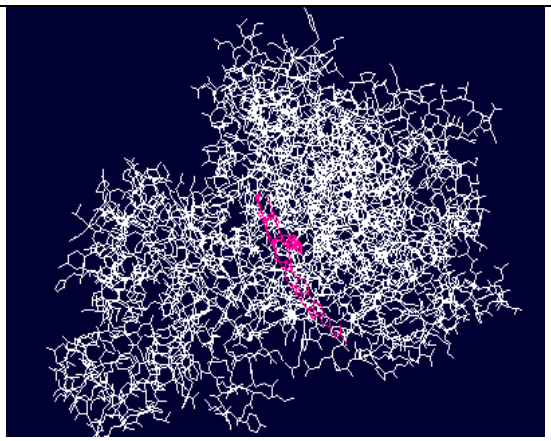
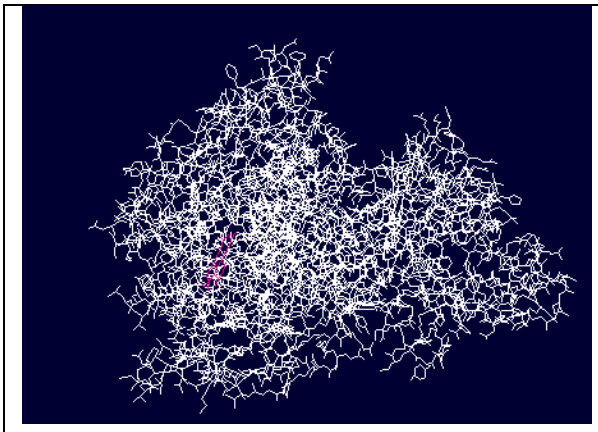
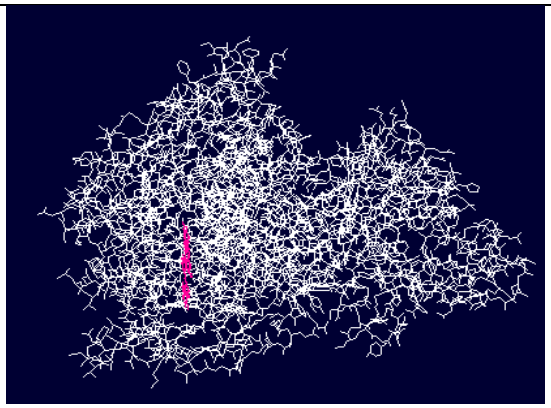
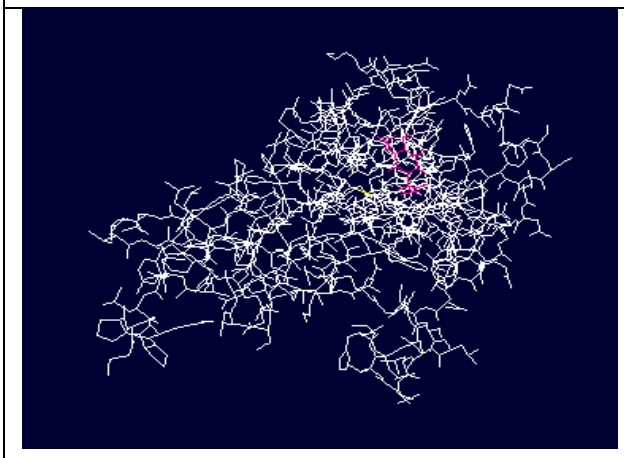
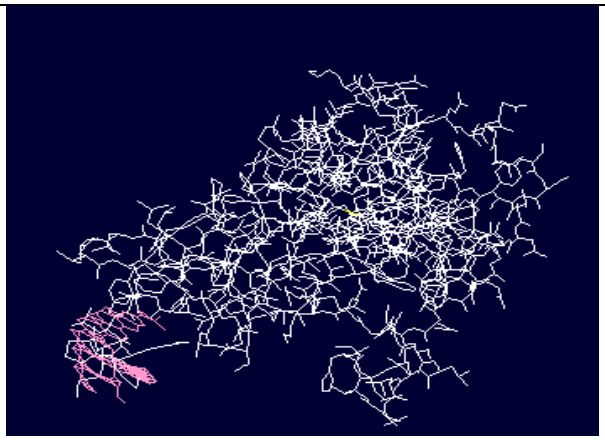
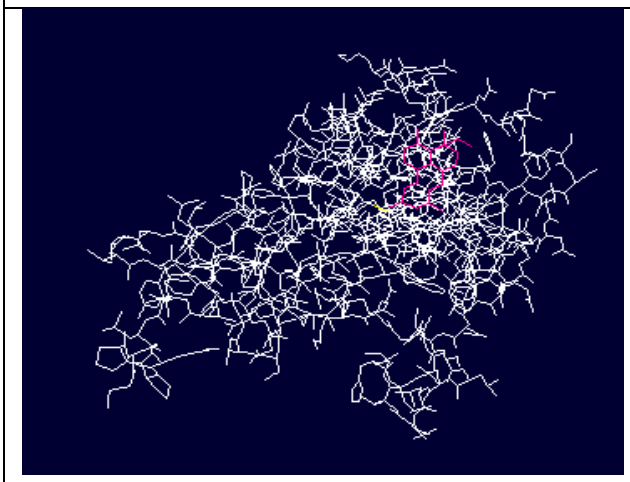
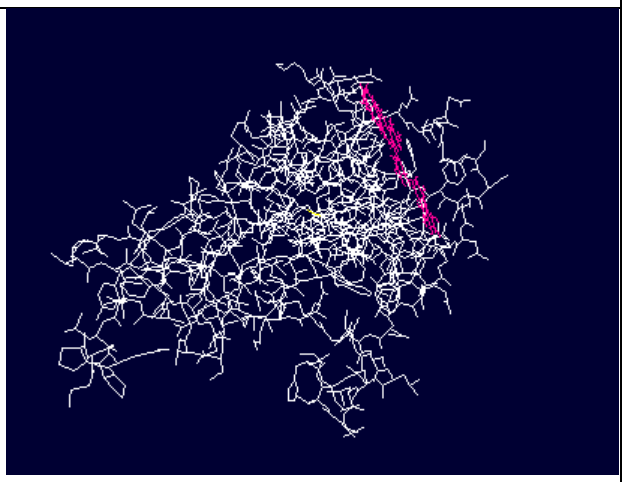
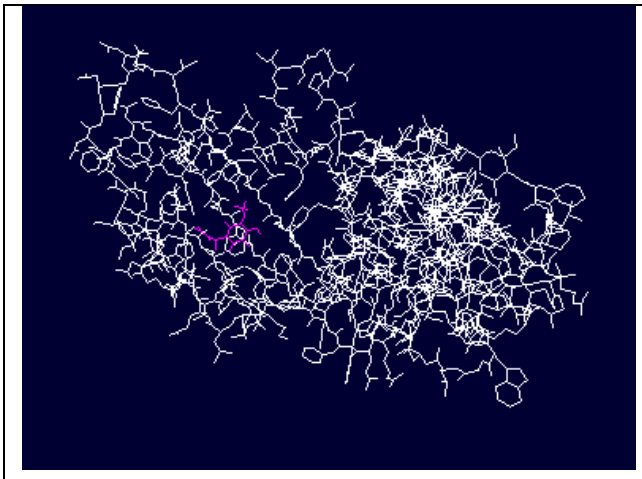
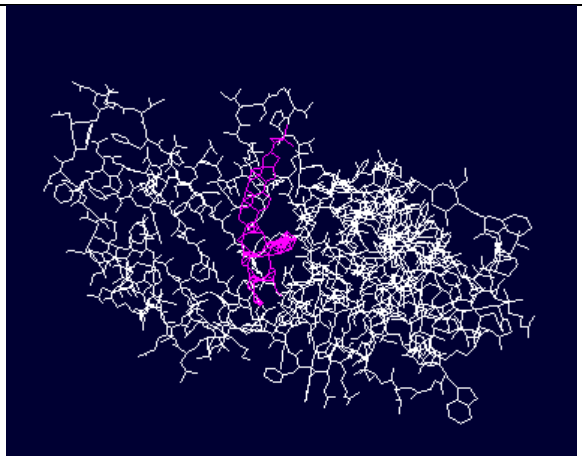
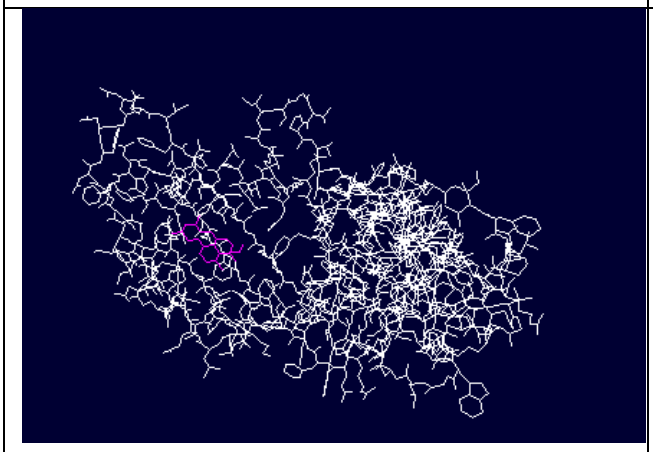
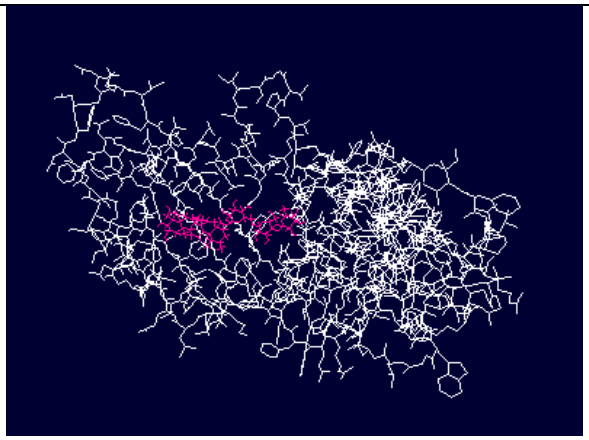
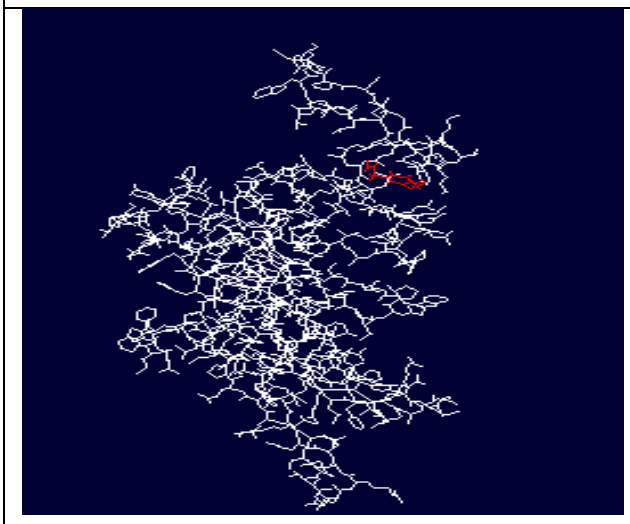
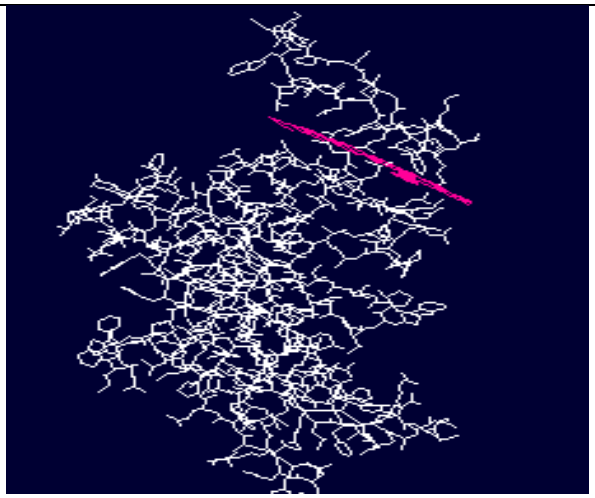
	
PTPRC protein docked with EUGENOL	PTPRC protein docked with SHATAVARIN

	
PTPRC protein docked with CURCUMIN	PTPRC protein docked with ASIATICOSIDE
	
TNFRSF1A protein docked with EUGENOL	TNFRSF1A protein docked with SHATAVARIN
	
TNFRSF1A protein docked with CURCUMIN	TNFRSF1A protein docked with ASIATICOSIDE

	
HLADR1 protein docked with EUGENOL	HLADR1 protein docked with SHATAVARIN
	
HLADR protein docked with CURCUMIN	HLADR1 protein docked with ASIATICOSIDE
	
EB protein docked with EUGENOL	EB protein docked with SHATAVARIN

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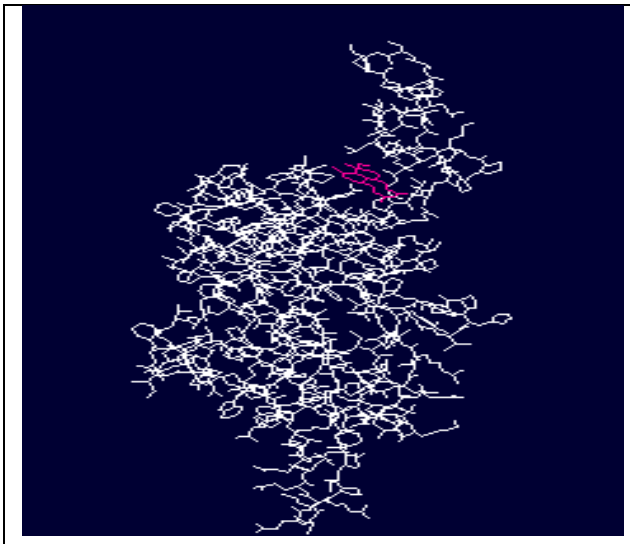	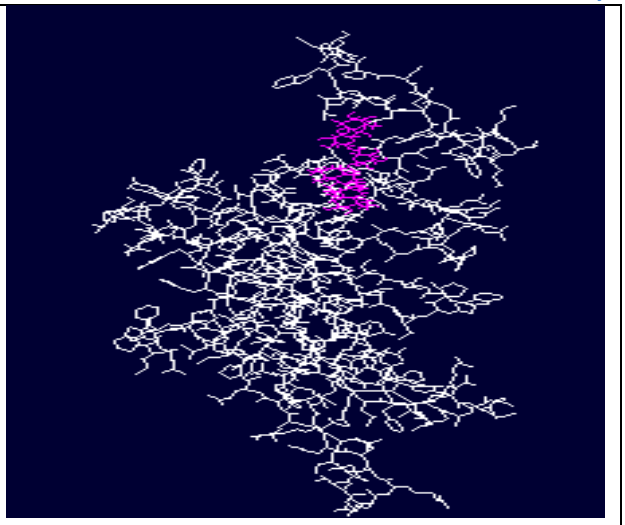
EB protein docked with CURCUMIN	EB protein docked with ASIATICOSIDE

Fig. 23: Docking of MS receptors (figure source [6])