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DRUGS USED IN PARKINSON'S (LITERATURE REVIEW).

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ABSTRACT

Parkinson's is a progressive neurodegenerative disease characterized by tremor and bradykinesia and is one of the most common neurological disorders. Male sex and advancing age are independent risk factors and, as the population ages, is taking an increasing toll on productivity and medical resources. There are a number of other extrapyramidal conditions that can make the diagnosis challenging. Unlike other neurodegenerative diseases, idiopathic Parkinson's disease has effective treatments that mitigate symptoms. Medications can improve day-to-day function and, in cases where medication does not give a sustained benefit or has significant side effects, treatments like deep brain stimulation result in improved quality of life. Parkinson's disease represents a rapidly developing neurodegenerative condition; the increased worldwide prevalence, apart from an infectious cause, resembles many of the features commonly observed during pandemics. In most populations, 3-5% of Parkinson's disease is explained by genetic causes associated with certain Parkinson's disease genes, representing monogenic Parkinson's disease, while 90 genetic risk variants account for 16-36% of the inherited risk of non-monogenic Parkinson's disease. explains together. . Additional causal associations include having a relative with Parkinson's disease or tremors, constipation, and being a nonsmoker, each of which at least doubles the risk of Parkinson's disease. The diagnosis is clinically based; auxiliary test is reserved for people with atypical appearance. Current criteria define Parkinson's disease as the presence of bradykinesia with rest tremor, rigidity, or both. However, the clinical presentation is multifaceted and includes many non-motor symptoms. Prognostic advice is guided by knowledge of the subtypes of the

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disease. There is a potentially long prodromal period before clinically manifest Parkinson's disease.

Key words

Parkinson's disease; Parkinsonism; Pharmacologic treatment, Levodopa , dopa decarboxylase inhibitor, carbidopa, benserazide, COMT inhibitors, dopamine agonists, MAO-B inhibitors.

Introduction. This disease was recorded in 1817 by the English doctor Parkinson (he and his family members were ill). Men are affected twice as often as women. It is a chronic disease characterized by a sudden increase in muscle tone, tremors, and lack of movement (akinesias). The function of salivary and skin sebaceous glands increases. The etiology of the disease is unknown. In this disease, the nuclei of the extrapyramidal system are damaged, that is, they have an inhibitory effect on the neostriatum. the amount of dopamine in the basal nucleus and core substance decreases. Neostriatum, on the other hand, increases in control of orca brain function. Decreased dopaminergic inhibitory effect increases the excitatory effect of cholinergic neurons of the caudate nucleus. In addition, the stimulating effect of glutamatergic neurons prevails. Due to this, changes occur in the muscles. So. for the treatment of parkinson's disease, it is necessary to increase the dopaminergic effect in the MNS or to decrease the cholinergic effect. Dopamine cannot be used for this purpose because it does not cross the blood-brain barrier. That is why L-DOFA (levodopa) is used, because it passes through GEB with the body and is converted into dopamine under the action of the enzyme dopa recarboxylase. Dopaminergic system activity can be enhanced by stimulating dopamine receptors or by reducing dopamine neuronal uptake. Current drugs against parkinsonism. **Parkinson's disease** (**PD**), or simply **Parkinson's**, is a chronic degenerative disorder of the central nervous system that affects both the motor system and non-motor systems. The symptoms usually emerge slowly, and as the disease progresses, non-motor symptoms become more common. Early symptoms are tremor, rigidity, slowness of movement, and difficulty with walking. Problems may also arise with cognition, behaviour, sleep, and sensory systems. Parkinson's disease dementia is common in advanced stages.

The motor symptoms of the disease result from the nerve cell death in the substantia nigra, a midbrain region that supplies dopamine to the basal ganglia. The cause of this cell death is poorly understood but involves the aggregation of the protein alpha-synuclein into Lewy bodies within the neurons. Collectively, the main motor symptoms are known as parkinsonism. Contributing factors include a combination of genetic and environmental factors. Those with an affected family member are at an increased risk of getting the disease, with certain genes known to

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be inheritable risk factors. Environmental risks include exposure to pesticides and prior head injuries; a history of exposure to trichloroethylene is also suspected.

Diagnosis of Parkinson's disease is mainly based on symptoms, usually motor-related. PD typically occurs in people over the age of 60, of whom about one percent are affected. In those younger than 50, it is termed early-onset PD. The average post-diagnosis life expectancy is 7–15 years. No cure for PD is known, and treatment aims to mitigate symptoms. Initial treatment typically includes L-DOPA, MAO-B inhibitors, or dopamine agonists. As the disease progresses, these medications become less effective and produce a side effect marked by involuntary muscle movements. Diet and certain forms of rehabilitation have shown some effectiveness at improving symptoms. Surgery to place microelectrodes for deep brain stimulation has been used to reduce severe motor symptoms where drugs are ineffective. Evidence for treatments for the non-movement-related symptoms of PD, such as sleep disturbances and emotional problems, is less strong.

The disease is named after English doctor James Parkinson, who published the first detailed description in *An Essay on the Shaking Palsy*, in 1817. Public awareness campaigns include World Parkinson's Day and the use of a red tulip symbolizes Parkinson's awareness. People with PD who have increased the public's awareness of the condition include boxer Muhammad Ali and actor Michael J. Fox. [1,2].

The purpose of the work: to provide general knowledge about the drugs used against Parkinsonism, their use. Give a classification of drugs used against parkinsonism. To provide an understanding of the main effect of drugs used against Parkinsonism. Understanding the mechanisms of action of drugs used against Parkinsonism. Educate about the side effects of anti-parkinsonism drugs. Providing knowledge about instructions for use and contraindications of anti-parkinsonism drugs.

Analysis Of The Work divided into 3 groups:

1. Dopaminergic enhancing substances

• a source of dopamine production (levodopa)

2. substances that stimulate dopamine receptors (bromocriptii)

• monoamine oxidase V blocking substances (selegiline)

Substances that reduce glutamatergic effects (midantan)

Substances that enhance the cholinergic effect (cyclodol)

L-DOFA is a left-handed isomer of dioxyphenylala. dopamine is produced from it under the action of DOFA decarboxylase enzyme. Levodopa is considered the most effective in the treatment of Parkinson's disease, because it reduces its symptoms: it strongly relieves akinesia, but has little effect on tension and tremor. The treatment is prolonged, the effect starts after one or several weeks, and gives a

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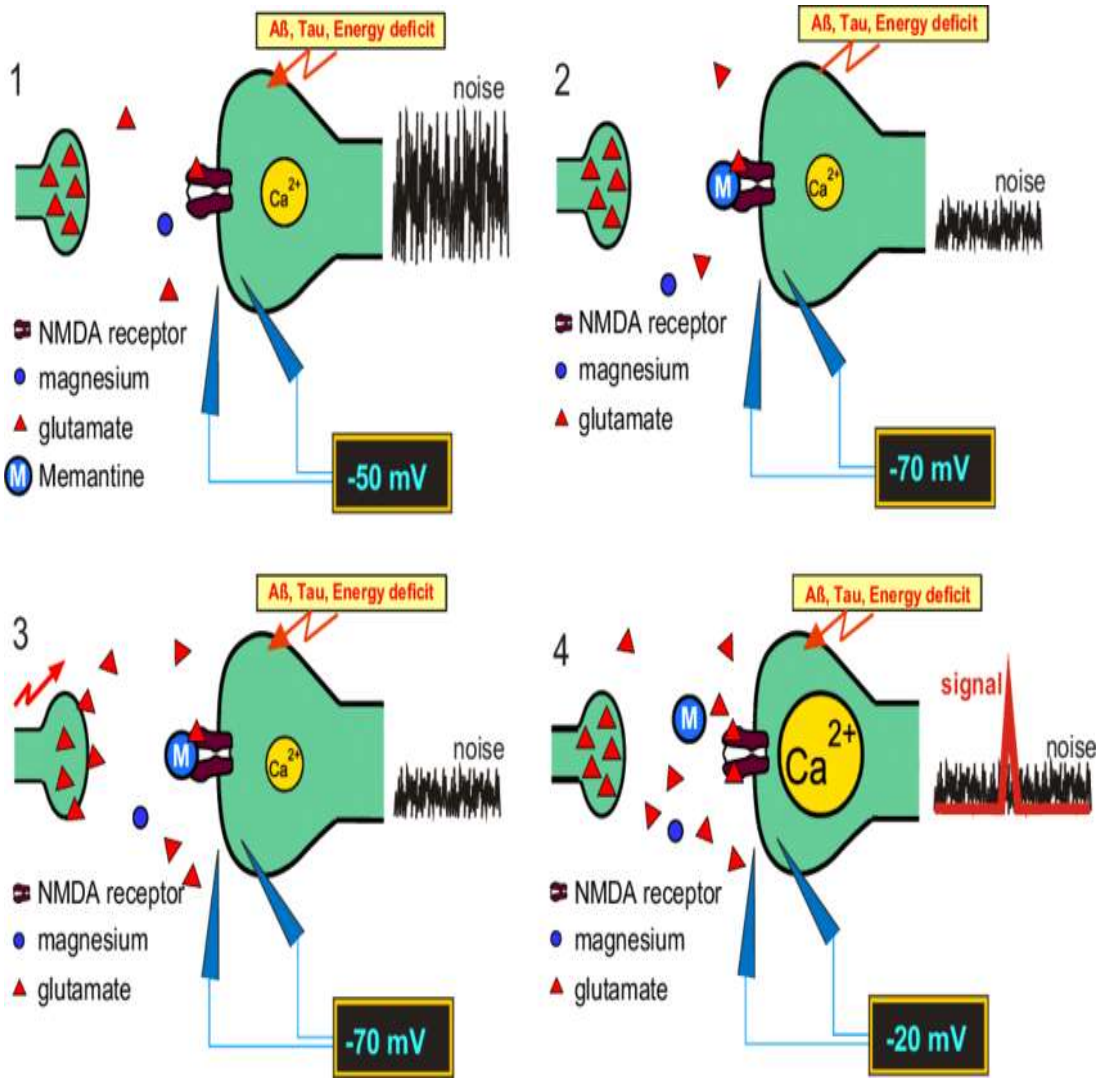
good result after 1-2 months. It is necessary to gradually increase the dose of the drug. Levodopa from MIT is well absorbed. But in the peripheral tissues, it also turns into dopamine and therefore causes many unpleasant effects on the account: appetite disturbance, dyspepsia, orthostatic hypothesis, mental disorders, cardiac arrhythmia. Levodopa can be used together with carbidopa or benserazide, an inhibitor of the DOFA-decarboxylase enzyme, in order to reduce the peripheral effect. These enzyme inhibitors do not pass through the GEB and do not affect the formation of dopamine in the brain during levodopa. They prevent the formation of dopamine in peripheral tissues. Levodopa contains 0.25 levodopa + 0.025 carbidopa. Madopar - 125-(0.1 levodopa + 0.025 benserazide) Madopar - 250 -{0.2 levodopa + 0.050 benserazide)

When using these two drugs, the amount of levodopa entering the MNS increases, and its side effects decrease. Cytamine V6 (pyridoxine) reduces the activity of levodopa. It is not possible to use levodopa in cardiovascular, liver, kidney and mental diseases.



MIDANTAN blocks glutamate receptors and reduces the stimulating effect on cortical glutamate neurons, neostratum. It has a neuroprotective effect on the

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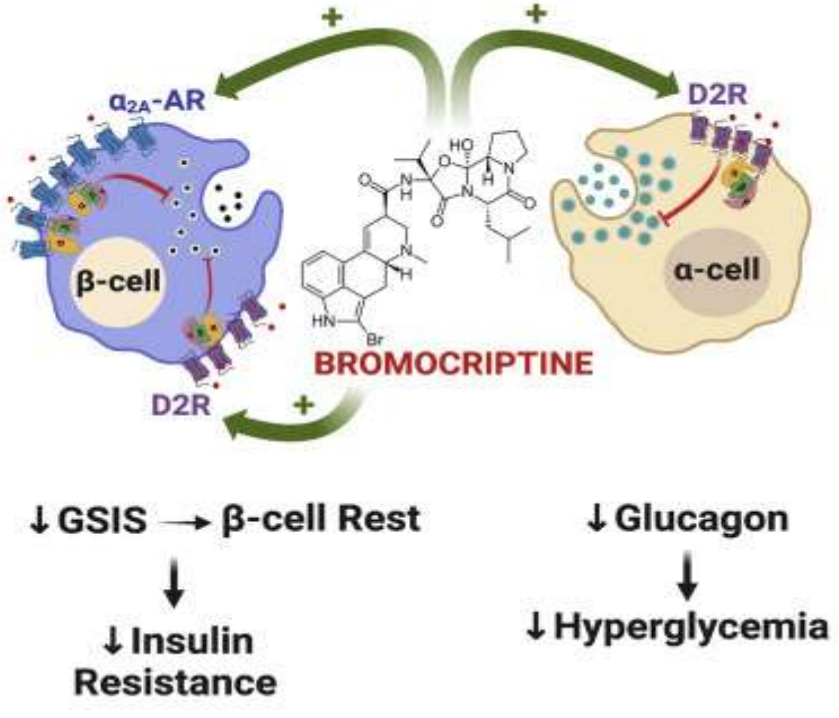
neurons of the substantia nigra, as it reduces the entry of calcium ions into the cells (resists destruction). In addition, it weakens the generation of impulses in the motor neurons of the MNS. An antiviral drug is recommended before midan. Midantan reduces excessive akinesia, reduces muscle tension, does not affect tremors at all. but the effect of the drug begins much earlier than that of levodopa (after 1-2 days). After a few days, the peak of development is reached. That is, mila is considered a fast-acting drug. It can be used together with other drugs used in Parkinson's disease (cholinelytic, L-DOFA). Midantanin is well tolerated by patients. Sometimes a headache. orthostatic hypotension, hallucinations, insomnia, general weakness. dyspepsia, dizziness are observed.

Cannot be used in acute and chronic liver diseases, kidney failure. in pregnancy, thyrotoxicosis, epilepsy, mental illness.

BROMOCRIPTIN is a semi-synthetic product of ergocriptine, a kora mig alkaloid, and is a specific agonist (enhancer) of O2dopamine receptors (lophaminmimetnk). Actively affects the metabolism of dopamine and NA in the MNS, like apomorphine. reduces serotonin release, Bromcriptine induces vomiting, lowers body temperature, reduces akinesia caused by phenothiazine products and reserpine. It reduces the amount of catecholamines in the blood. It relaxes the smooth muscles of the veins. It lowers blood pressure. It relaxes the muscles of the uterus. The anterior part of the pituitary gland reduces the secretion of prolactin and motility. Bromcriptine is due to the stimulating effect of L-receptors. Parused in the treatment of kinsonism. Patients accept these drugs well. Well absorbed from MIT. It is mainly excreted in feces. Urine contains very little 3-6 hours.



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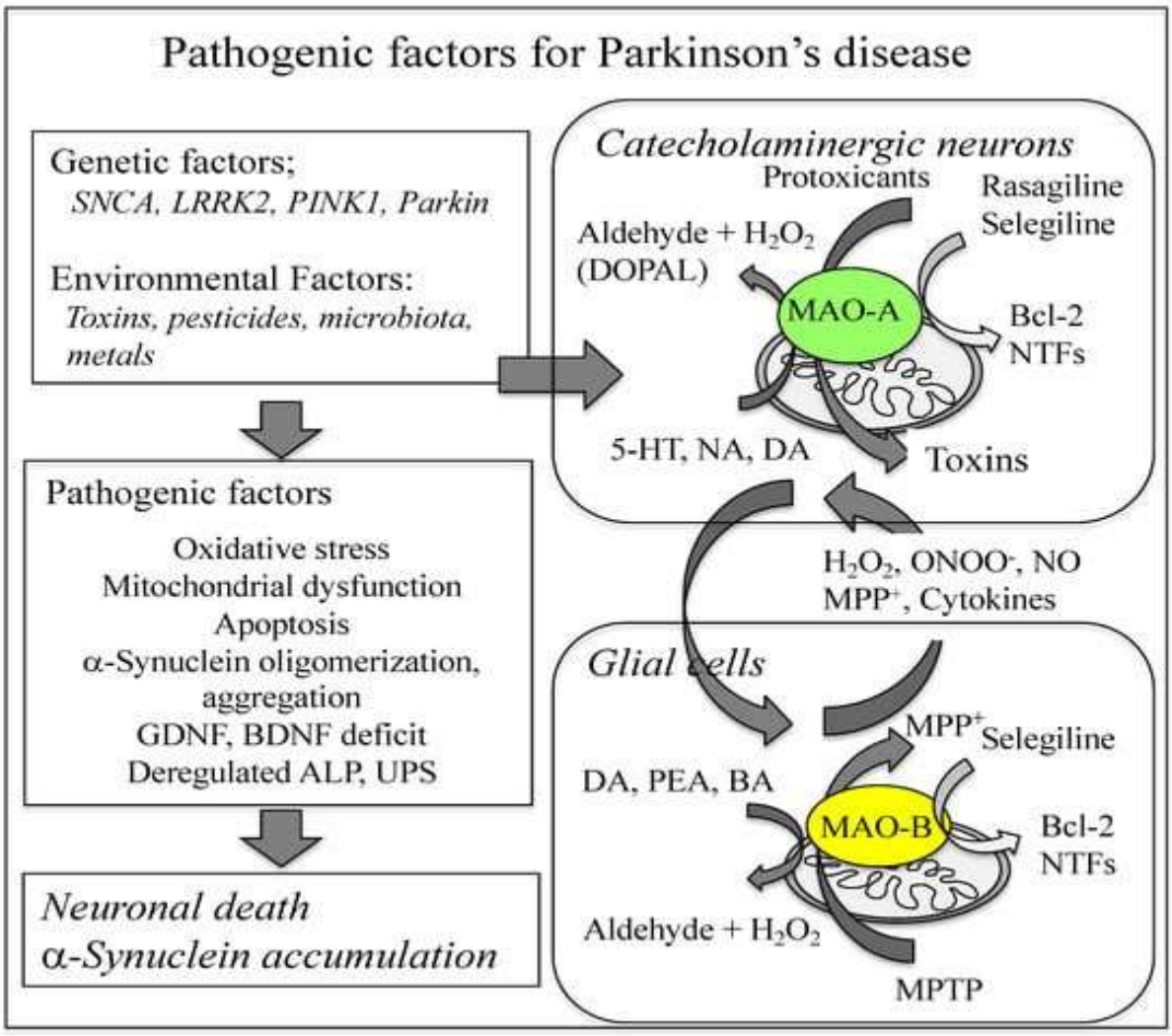
Adverse effects: nausea, vomiting, constipation, decreased blood pressure, swelling of mucous membranes, due to the sedative effect, it cannot be given to drivers and operators. Contraceptives, MAO inhibitors, MNS depressants should not be used in patients taking bromocriptine.

Selegiline has the property of inhibiting the activity of facal V-type MAO. Similar in chemical structure to ephedrine.

The mechanism of action: due to the large amount of V-type MAO in the brain, its activity decreases, the amount of dopamine in the brain, including



dopamine, increases in the body. When selegiline is used together with levodopa, the amount of dopamine increases, especially in extrapyramidal system neurons. Selegiline accelerates the onset of action of levodopa and prolongs its duration of action. Therefore, these 2 preparations are additionally used together. It also improves indicators of fat metabolism:

Adverse effects: similar to levodopa.

Siklodol.- strongly inhibits M- and N-cholenoreceptors of the peripheral and MNS, and therefore reduces the cholinergic stimulating effect on the basal ganglia,

reduces tremors, has little effect on muscle tension and akinesia [1,2,3, 4,5,6,7,8,9,10].

CONCLUSION: Parkinson's patients who don't get the right medication while in the hospital (often because they're in the hospital for unrelated illnesses) sometimes can't talk or walk. Many have suffered from poor medication management while in hospital. Parkinson's UK believes that the NHS could save up to £10m a year and improve care for Parkinson's patients if mandatory training was introduced for all hospital staff. "Almost two-thirds of people with Parkinson's do not receive their medication on time in the hospital." "More than three-quarters of Parkinson's patients we asked said their health had worsened as a result of medication mismanagement in hospital." "Only 21% of respondents told us they took their medication on time without reminding hospital staff

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