

# Pharmacology of Parkinson's and Alzheimer's Diseases

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لا تنسوا أهلنا في غزة من دعواتكم

# Introduction

- Parkinson's and Alzheimer's diseases are neurodegenerative disorders
- They are chronic diseases we try to improve the lifestyle of the patient
- Monitoring the disease to:
  - control the symptoms
  - slow the deterioration
  - get a better prognosis
- Understanding their treatment is crucial for effective management.



# Pathophysiology of Parkinson's Disease

In simple words, this disease is dopamine deficiency

- Progressive loss of dopaminergic neurons in the substantia nigra Why we don't give dopamine supplements? Because of its metabolism and it can't cross BBB, so normally dopamine is synthesised in the brain
- Decreased dopamine levels in the basal ganglia
- Accumulation of Lewy bodies ( $\alpha$ -synuclein aggregates)
- Motor symptoms: Bradykinesia, rigidity, tremor, postural instability
- Non-motor symptoms: Depression, cognitive impairment, autonomic dysfunction



# Pharmacology of Parkinson's Disease

As we said we can't give the pure dopamine, but we can give its precursor which is L-DOPA  
REMEMBER >> L-DOPA came from tyrosine

- Levodopa (L-DOPA) + Carbidopa: Dopamine precursor with peripheral dopa decarboxylase

inhibition So the dopamine is not consumed periphery

- Dopamine Agonists: Pramipexole, Ropinirole

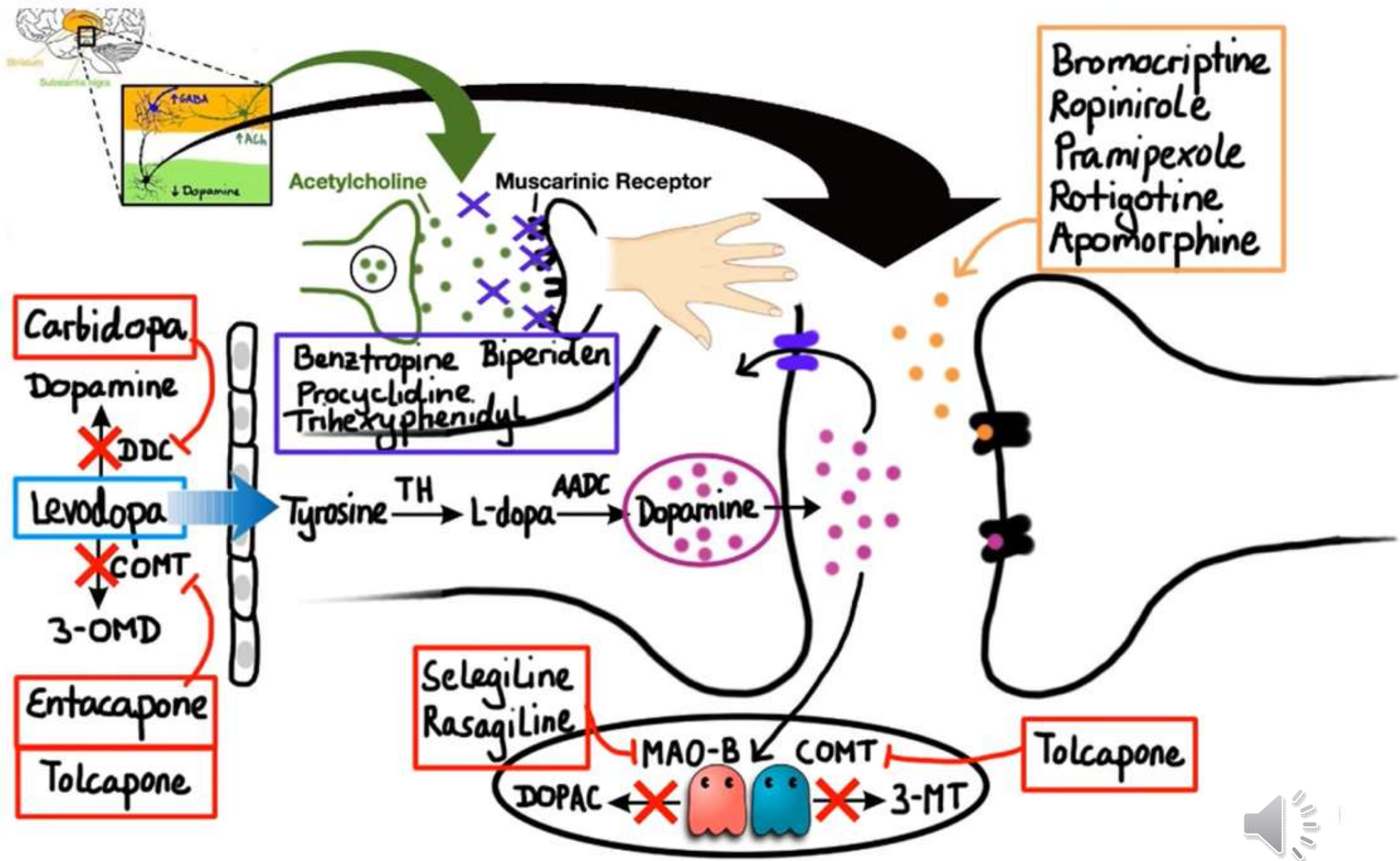
- MAO-B Inhibitors: Selegiline, Rasagiline

- COMT Inhibitors: Entacapone, Tolcapone

- Anticholinergics: Benztropine, Trihexyphenidyl

حتي يبقى  
وقت اطول  
بالدماغ

The dopamine makes a negative feedback on the synthesis of the Ach, however in the Parkinson's disease we don't have enough dopamine, so the level of the Ach will increase, and Ach will flow in the blood and bind to the muscarinic receptors (responsible for skeletal muscles movement), as a result the extra pyramidal symptoms will occur (tremor, rigidity)



# Pathophysiology of Alzheimer's Disease

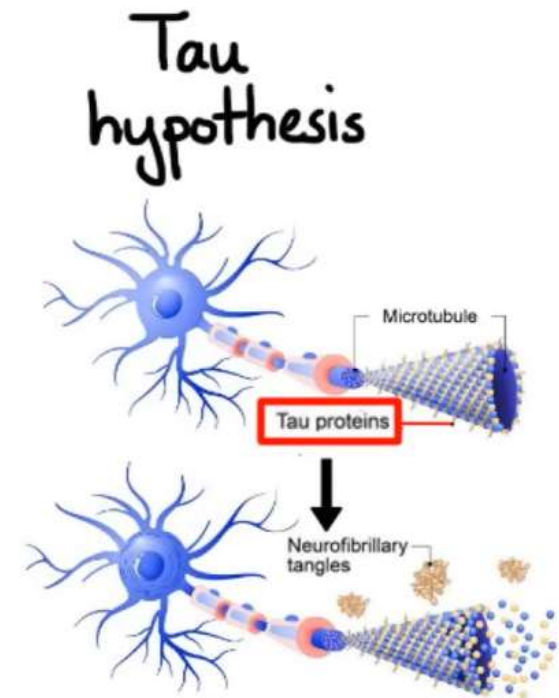
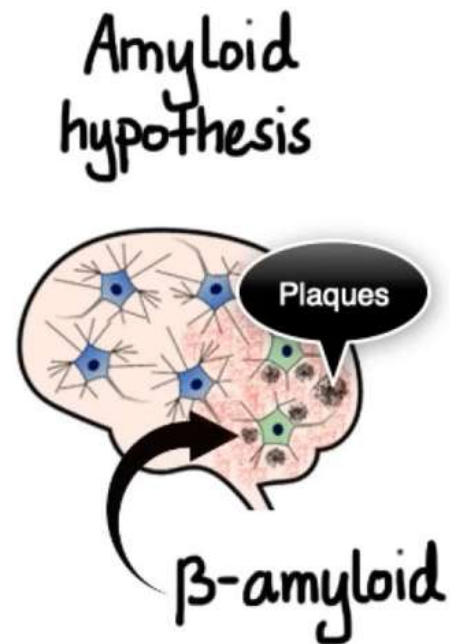
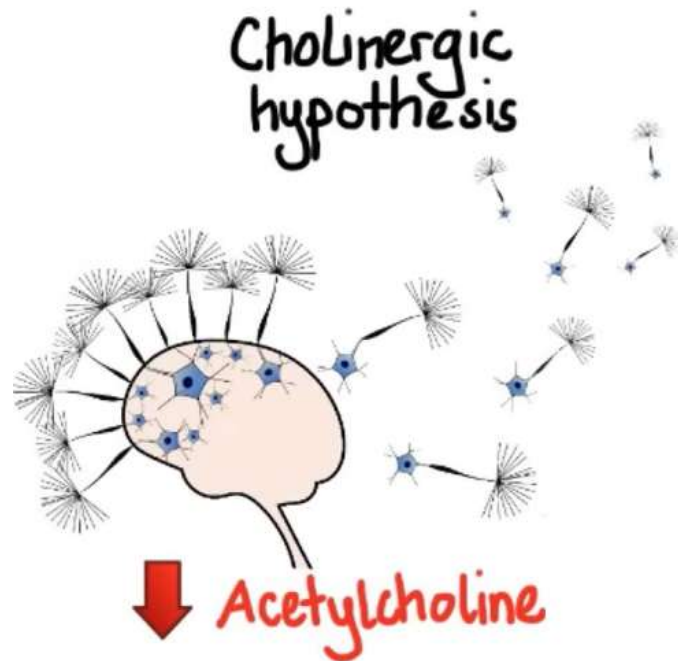
In simple words, it's Ach deficiency, and here we can't give pure Ach for the same reasons mentioned above,

- Progressive loss of cholinergic neurons in the cortex and hippocampus
- Amyloid-beta plaque accumulation
- Neurofibrillary tangles (tau protein hyperphosphorylation)
- Neuroinflammation and oxidative stress
- Symptoms: Memory loss, cognitive decline, behavioral changes



يُقال انه الكركم يقوي من هذا المرض، عشان هيك الهنود مش منتشر عندهم

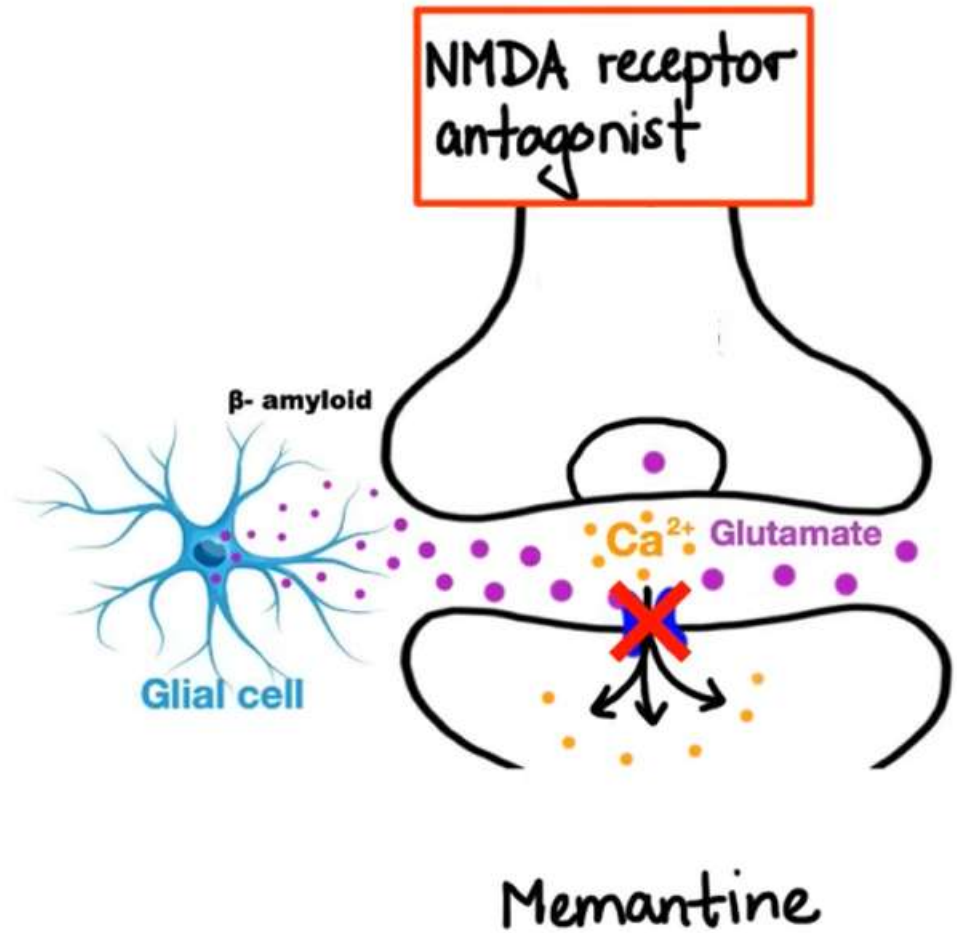
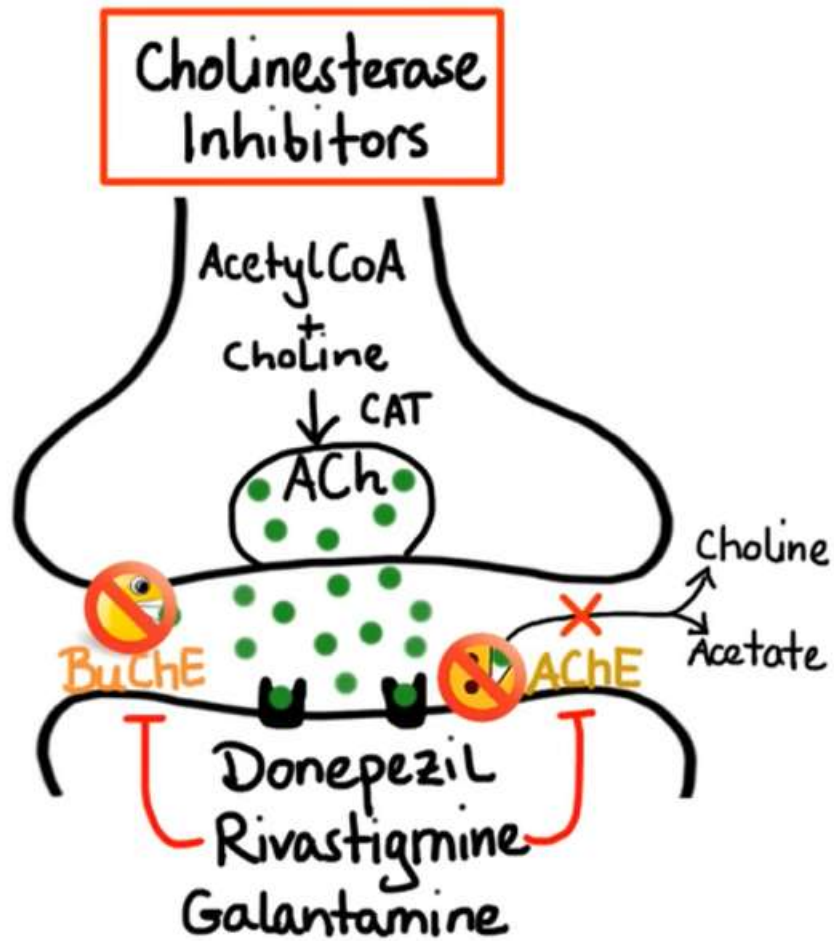
## Hypothesis according Alzheimer's disease occurrence



# Pharmacology of Alzheimer's Disease

- Cholinesterase Inhibitors: Donepezil, Rivastigmine, Galantamine
- NMDA Receptor Antagonist: Memantine
- Emerging Therapies: Anti-amyloid monoclonal antibodies (Aducanumab, Lecanemab)
- Symptomatic Management: Antidepressants, antipsychotics





# Clinical Applications and Guidelines

- Parkinson's: Start treatment based on symptom severity, consider age and side effects
- Alzheimer's: Initiate cholinesterase inhibitors early, memantine for moderate-severe cases
- Monitor for drug interactions and side effects
- Non-pharmacological interventions remain crucial



# Adverse Effects and Drug Interactions

لما يوقف بنزل ضغطه

- Levodopa: Dyskinesia, orthostatic hypotension, hallucinations
- Dopamine Agonists: Impulse control disorders, sedation
- Cholinesterase Inhibitors: GI distress, bradycardia
- NMDA Antagonists: Dizziness, confusion
- Drug Interactions: Antipsychotics, antihypertensives, anticholinergics



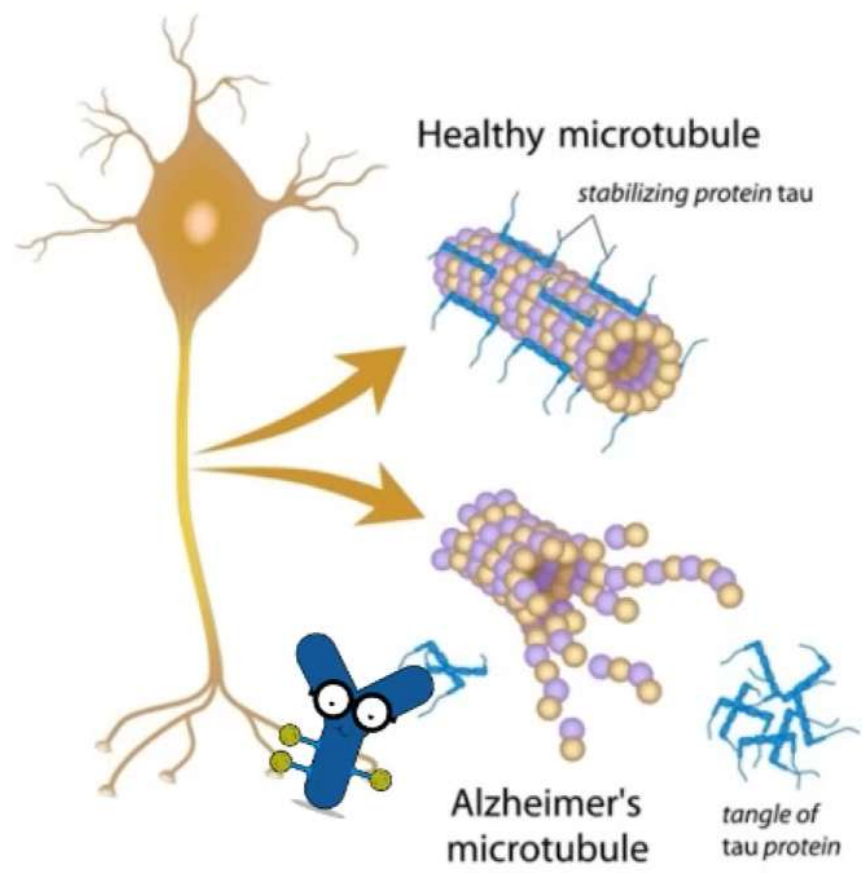
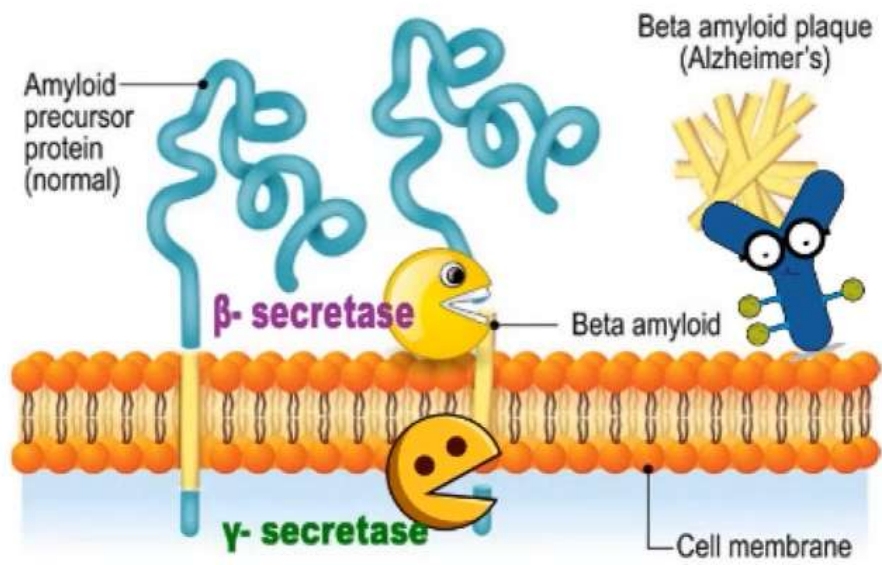
# Latest Research and Future Directions

مثل تعلم اكثر من لغة وحل مسائل الرياضيات تعتبر وقاية

- Gene therapy and neuroprotective strategies
- Stem cell-based treatments
- Novel monoclonal antibodies for Alzheimer's
- Personalized medicine approaches
- Potential disease-modifying therapies



المحاضرة سهلة، ادرسوا محاضرة الباحث الأولى قبلها، ولا تنسونا من دعواتكم.



**THANK YOU**

