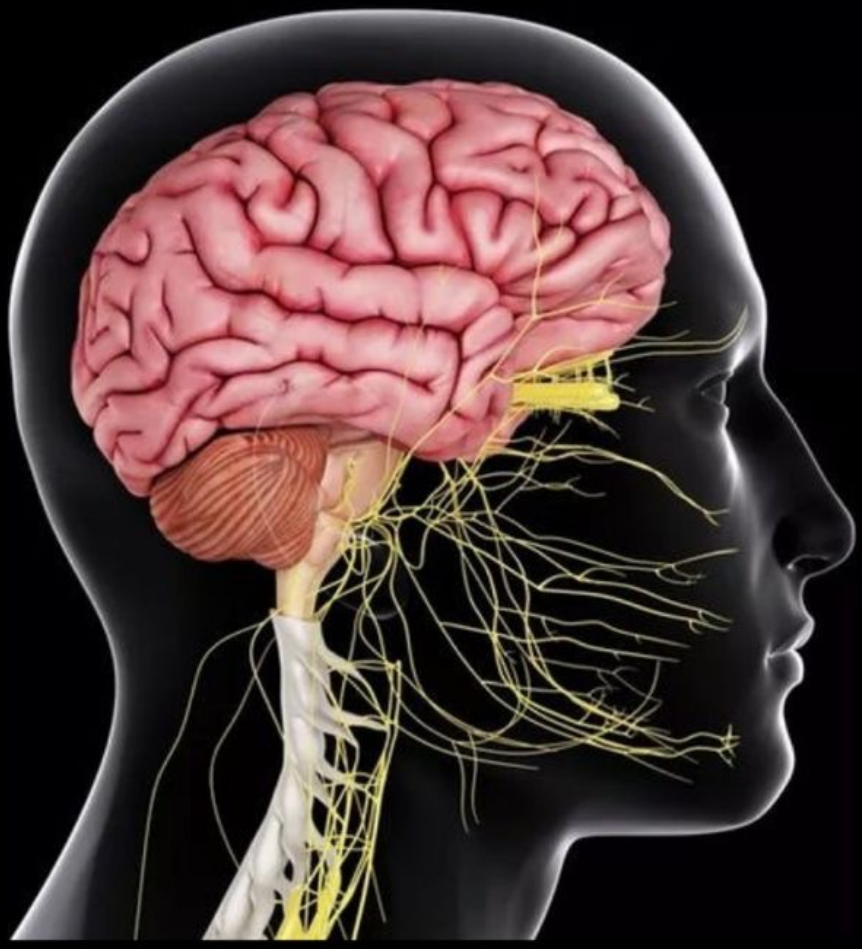
CENTRAL NERVOUS SYSTEM



SUBJECT : Pharmacology

LEC NO. : 5

DONE BY : Batool ALzubaidi

وَقُل رَّبِّ زِدْنِي عِلْمًا

قبل ما نبلش بس حبيت احكي ملاحظة عشان اطلع من خطيتكم، بحكم كنت اعمل التفاريغ الي في بعض السلايدات بتكون بالملف بس خلال المحاضرة اونلاين او وجاهي ما تكون موجودة و هو يشرح مش يفشق عنها ما كانت تبين ف انا كنت احذفهم و لما نزلت اول ٤ محاضرات راحت هاي النقطة عن بالي راح احطهم بهاد التفريغ عشان ما يكون علي اشي

محاضرة ال opioid

Definitions

Hyperalgesia: abnormally increased sensitivity to pain

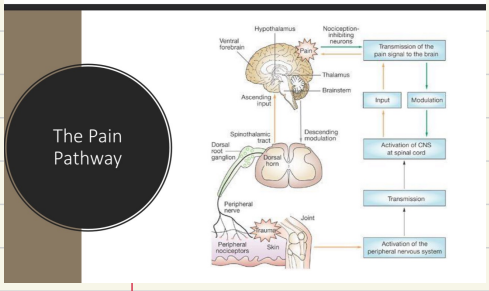
Allodynia: pain resulting from an originally non-painful stimulus

Hypoalgesia: decreased sensitivity to painful stimuli

Analgesia: reduction or relief of pain sensation without affecting other sensations

Anesthesia: local or general reduction or absence of all sensations (touch, pain, temperature, ...) with or without loss of motor function. This may be accompanied by loss of consciousness

Paresthesia: abnormal or altered sensation of the body (numbness, tingling, or burning)



Summary of Morphine's Therapeutic Uses

Therapeutic Use	Comments
Analgesia	*Morphine* is the prototype opioid agonist. Opioids are used for pain in trauma, cancer, and other types of severe pain.
Treatment of diarrhea	Opioids decrease the motility and increase the tone of intestinal circular smooth muscle. [Note: Agents commonly used include *diphenoxylate* and *loperamide* (see Chapter 31).]
Relief of cough	*Morphine* does suppress the cough reflex, but *codeine* and *dextromethorphan* are more commonly used.
Treatment of acute pulmonary edema	Intravenous *morphine* dramatically relieves dyspnea caused by pulmonary edema associated with left ventricular failure, possibly via the vaso-dilatory effect. This, in effect, decreases cardiac preload and afterload, as well as anxiety experienced by the patient.
Anesthesia	Opioids are used as pre-anesthetic medications, for systemic and spinal anesthesia, and for postoperative analgesia.

Meperidine (Pethidine)

Synthetic

- κ agonist
- Some μ agonist activity
- anticholinergic

Uses

- Used only for short-term analgesia management
- Preferred over morphine during labor

How to manage pain?

Assessment

Diagnosis

Support

Analgesics

- Paracetamol
- NSAIDs
- Opioids

High-Yield Terms to Learn

Opiate	A drug derived from alkaloids of the opium poppy
Opioid	The class of drugs that includes opiates, opiopeptins, and all synthetic and semisynthetic drugs that mimic the actions of the opiates
Opioid peptides	Endogenous peptides that act on opioid receptors
Opioid agonist	A drug that activates some or all opioid receptor subtypes and does not block any
Partial agonist	A drug that can activate an opioid receptor to effect a submaximal response
Opioid antagonist	A drug that blocks some or all opioid receptor subtypes
Mixed agonist-antagonist	A drug that activates some opioid receptor subtypes and blocks other opioid receptor subtypes

Can you tell the difference between these terms:

- Opium?
- Opioid?
- Opiate?
- Narcotic?

Opioid Partial Agonists

tial opioid agonists bind to opioid receptors but have only partial icacy relative to full opioid agonists.

prenorphine
ntazocine
lbuphine

Opioids

Phenanthrenes	Action on Opioid Receptors
Morphine	Agonist
Codeine	Agonist
Oxycodone	Agonist
Oxymorphone	Agonist
Hydromorphone	Agonist
Hydrocodone	Agonist
Buprenorphine	Partial agonist
Nalbuphine	Mixed Agonist/Antagonist
Butorphanol	Mixed Agonist/Antagonist

Benzmorphan	
Pentazocine	Mixed Agonist/Antagonist
Phenylpiperidines	
Fentanyl	Agonist
Alfentanil	Agonist
Sufentanil	Agonist
Meperidine	Agonist
Diphenylheptane	
Methadone	Agonist

Pentazocine

Partial agonist at κ
Antagonist at μ and δ

Less euphoria

Contraindicated in patients with coronary artery disease

Uses

- Analgesia (limited use because of side effects)

محاضرة ال antidepressants

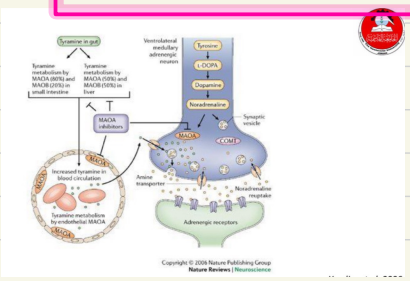
MAOIs +				
1. Tyramine - rich food ↓	**2. Cold Remedies** (sympathomimetic) ↓	**3. TCAs** (↑ CA) ↓	**4. Pethidine** ↓	**5. SSRIs** (↑ 5HT) ↓
Hypertensive crisis **(Cheese reaction)**	Hypertensive Crisis.	-Hypertension -Hyperthermia -Convulsions	-Respiratory depression -Hyperthermia -Convulsions	"**Serotonin syndrome**": -Hyperthermia -Convulsions

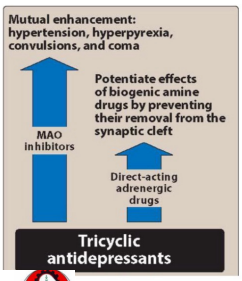
Tyramine in food is metabolized in GIT by MAO-A & MAO-B. MAOIs allow tyramine in tyramine-rich food (old cheese, chicken liver, chocolate) to escape metabolism & release ↑ ↑ ↑ amounts of catecholamines from neurons → hypertensive crisis.

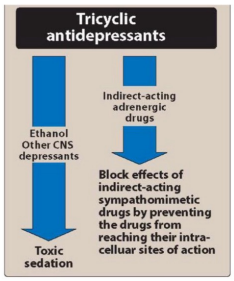
Monoamine Oxidase Inhibitors (MAOi)

Precautions with MAOi

- Patients on nonselective MAOIs should be warned against serious drug interactions and should be given a list of the foods they should avoid.
- Patients on MAOIs should not receive TCAs or SSRIs except after 2 weeks from stopping MAOIs (effect persists for 2 weeks or 6 for fluoxetine).
- Avoid in the elderly because of postural hypotension.







Summary of antidepressants mechanisms of action

Mechanisms of Increase of Biogenic Amines by Antidepressants

Amine Pump Inhibitors	**MAO Inhibitors**	**Presynaptic α_2 Blockers**
Inhibit uptake-1 of biogenic amines into neurons resulting in their accumulation in synaptic cleft, potentiating their action at post synaptic receptors.	Inhibit metabolism of biogenic amines by MAO enzyme inside nerve endings → ↑stores available for release.	↑ NA release into synaptic cleft by preventing α_2 auto-inhibition.
Members **1. TCAs** **2. TTAD** **3. SSRI** **4. NSRI** **5. Bupropion**	**Members** **Tranylcypromine** **Phenelzine** **Moclobemide**	**Members** **Mirtazapine**

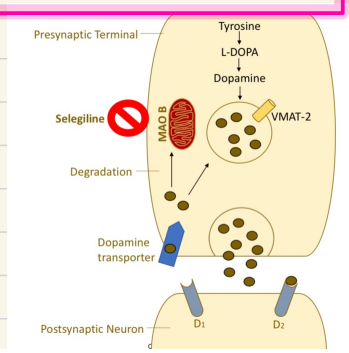
TCAs: Tricyclic antidepressants **NSRI:** Norepinephrine Serotonin Reuptake Inhibitor
TTADs: Tetracyclic antidepressants **SSRIs:** Selective Serotonin Reuptake Inhibitor.

محاضرة ال parkinson

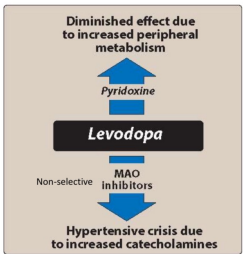
Parkinsonism: Etiology

- **Idiopathic (Parkinson's disease)**: primary or idiopathic destruction of dopaminergic neurons in the basal ganglia.
- **Secondary parkinsonism**:

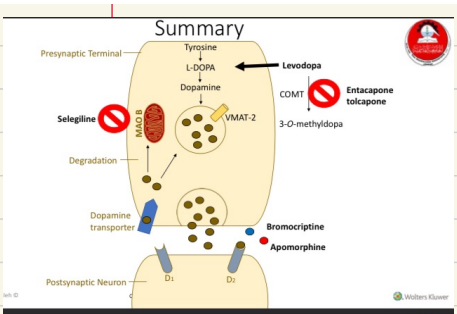
❑Viral encephalitis
❑CO or manganese poisoning.
❑Drug-Induced parkinsonism "pseudoparkinsonism" e.g., *haloperidol*



Drug-drug Interaction



Summary



Summary

Drug	Mechanism of Action	Adverse Effects
I. Bromocriptine, Pramipexole &Ropinirole (Given alone or with L-dopa). **Apomorphine**	Direct D_2 agonists. (Less fluctuation due to rapid absorption - longer $t_{1/2}$). is given **SC in emergency** (sudden freezing i.e. immobility) as it is rapid and more effective than L-dopa.	- Similar to L-dopa, with more psychosis. - Vasospasm & cardiac fibrosis **(bromocriptine)**
II. Amantadine (Given alone or with L-dopa).	- ↑ DA release (mild effect) → enhances L-dopa effect. - Blocking cholinergic receptors - Block glutamate receptor (NMDA) → ↓ glutamate excitotoxicity → ↓ neuronal degeneration. **- more effective against rigidity and bradykinesia**	- Insomnia. - Hallucination. - Livedo reticularis: purple spotting of skin 

Summary

III. Selegiline (Adjunct to L-dopa/carbidopa). **Rasagiline**	Selective inhibitor of MAO-B → delays breakdown of nigrostriatal DA → prolongs L-dopa action → ↓ fluctuation **5 times more potent**	- Insomnia (due to its metabolism to methamphetamine and amphetamine) - Hallucination. - Very low risk of cheese reaction. **No Insomnia**
IV. Entacapone (Adjunct to L-dopa/carbidopa). **Tolcapone**	COMT inhibitor → ↓ L-dopa peripheral metabolism → ↑ its bioavailability & **prolongs its action → ↓ fluctuations.** **Relatively longer duration**	- Similar to L-dopa /carbidopa. **+ Diarrhea.** **Fulminant hepatic necrosis**

Dopamine Receptor Agonists

Pharmacokinetics

Characteristic	*Pramipexole*	*Ropinirole*	*Rotigotine*
Bioavailability	>90%	55%	45%
V_d	7 L/kg	7.5 L/kg	84 L/kg
Half-life	8 hours[1]	6 hours	7 hours[3]
Metabolism	Negligible	Extensive	Extensive
Elimination	Renal	Renal[2]	Renal[2]

Amantadine

Mechanism of action:

- Antiviral used to treat influenza.
- Amantadine increases the release of dopamine, blocks cholinergic receptors and inhibit NMDA glutamate receptors.

Amantadine

Therapeutic uses:

- Amantadine is less efficacious than levodopa in the treatment of Parkinson's disease.
- Effective against rigidity and bradykinesia

Summary Of The Therapeutic Strategy

- Levodopa+carbidopa is the mainstay (first-line) therapy of Parkinson's disease (mostly in combination with a MAO B inhibitor or COMT inhibitor).
- MAO B inhibitors and COMT inhibitors are given in adjunct to levodopa+carbidopa therapy.
  - ----- MAO B inhibitors increase efficacy of levodopa and decrease fluctuation in motor response
  - ----- COMT inhibitors increase efficacy of levodopa and decrease "wearing off" mechanism.
- Dopamine agonists can be given alone in young and mild parkinsonians (to delay levodopa use) OR in combination with levodopa+carbidopa if disease is in progress.
- Antimuscarinics are used in adjunct with levodopa+carbidopa (or in cases of antipsychotics-induced parkinsonism).

Strategy of treatment

Antiparkinsonian Drugs aim to restore DA/Ach balance

I. ↓ Cholinergic Activity
Anticholinergics.
Benzhexol - Benztropine

II. ↑ Dopaminergic Activity
Dopaminergic Drugs
1. Levodopa. **(DA precursor)**
2. Bromocriptine- pramipexole- ropinirole. **(D_2 agonists)**
3. Amantadine. **(↑DA release)**
4. Entacapone. **(↓DA degredation)**
5. Selegiline **(↓DA degredation)**

Summary of the therapeutic strategy

How to decrease fluctuation in motor response to levodopa?

- Addition of a MAO B inhibitor or a COMT inhibitor or a dopamine agonist
- Shortening of the interval between doses of levodopa+carbidopa
- Using slow-release preparations of levodopa+carbidopa

Drugs Used in Parkinson's Disease

- Levodopa and carbidopa
- Selegiline and rasagiline
- Catechol-O-methyltransferase inhibitors (COMTis).
- Dopamine receptor agonist
- Amantadine
- Antimuscarinic agents

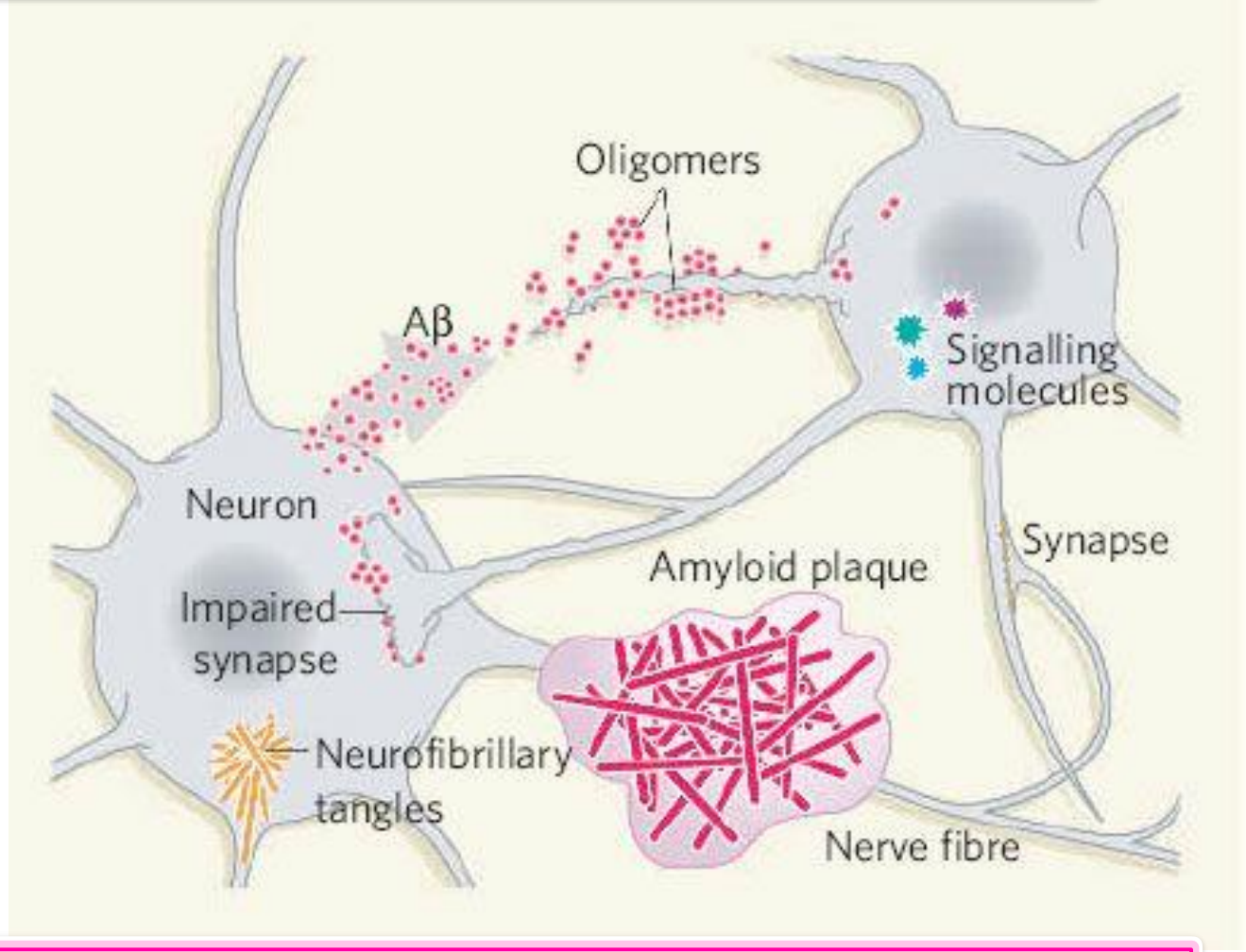
ANTI-PARKINSON DRUGS
Amantadine SYMMETREL
Apomorphine APOKYN
Benztropine COGENTIN
Biperiden AKINETON
Bromocriptine PARLODEL
Carbidopa LODOSYN
Entacapone COMTAN
Levodopa (w/*Carbidopa*) SINEMET, PARCOPA
Pramipexole MIRAPEX
Procyclidine KEMADRIN
Rasagiline AZILECT
Ropinirole REQUIP
Rotigotine NEUPRO
Selegiline (Deprenyl) ELDEPRYL, ZELAPAR
Tolcapone TASMAR
Trihexyphenidyl ARTANE

Overview: Alzheimer's Disease

One of the main features of alzheimer disease is neural destruction and degeneration caused by accumulation of amyloid plaque and neurofibrillary tangles

- is a neurodegenerative disorder characterized by impairment of memory and cognitive function together with mood and personality changes.
- is the most common cause of dementia in the elderly.

→ Severe memory loss



Pathophysiology is known, etiology unknown, aging disorder, physical damage is due to destruction due to protein byproducts several byproducts of metabolism most likely protein polymers like amyloid will start accumulating in neurons in the brain their accumulation is associated with neural damage , related to higher functions in cerebral function like memory and learning abilities and decline in IQ

Alzheimer's Disease: Pathophysiology

- Dementia of Alzheimer's disease has three distinct features:

1. Accumulation of senile plaques (β-amyloid accumulations)
2. Formation of numerous neurofibrillary tangles
3. Loss of cortical neurons (cholinergic neurons)

وحدة من المشاكل الرئيسية الي لازم نصلحها to try to restore as انه with treatment much cholinergic stimulation as possible

That's why Alzheimer's patients lose their cognitive abilities like learning and memory

Related to accumulation of aging related unprocessed metabolic byproducts like amyloid plaques and neurofibrillary tangles leading to neuronal degeneration and cell death

Alzheimer's Disease

Strategy of therapy

Improve brain cholinergic transmission

Reduce degradation and metabolism by using cholinergic antagonists

Reduce glutamate-NMDA-induced excitotoxicity

Since Alzheimer's disease is associated with continuous destruction of neurons we can try to protect them by glutamate antagonists

Most important excitatory peripheral neurotransmitter is acetylcholine, glutamate is a neurotransmitter that exerts an important function in the brain related to movement thinking recognition but it carries with it some sort of toxicity called excitotoxicity because it facilitates rapid extensive influx of calcium in the neurons neurons as it's activating them, rapid calcium influx in any cell not just neurons can trigger apoptosis, normal glutamate physiology is associated with nerve damage with aging

Drugs Used in Alzheimer's Disease

- Acetylcholinesterase inhibitors
  - Work on degradation step
  - Progression of disease cannot be stopped they only reduce symptoms
  - ❑ Donepezil
    - Cross BBB first line for mild
  - ❑ Galantamine
  - ❑ Rivastigmine
    - cross BBB
  - There's some drugs of this class don't cross blood brain barrier like neostigmine which treats peripheral cholinergic deficiency associated with diseases like myasthenia gravis

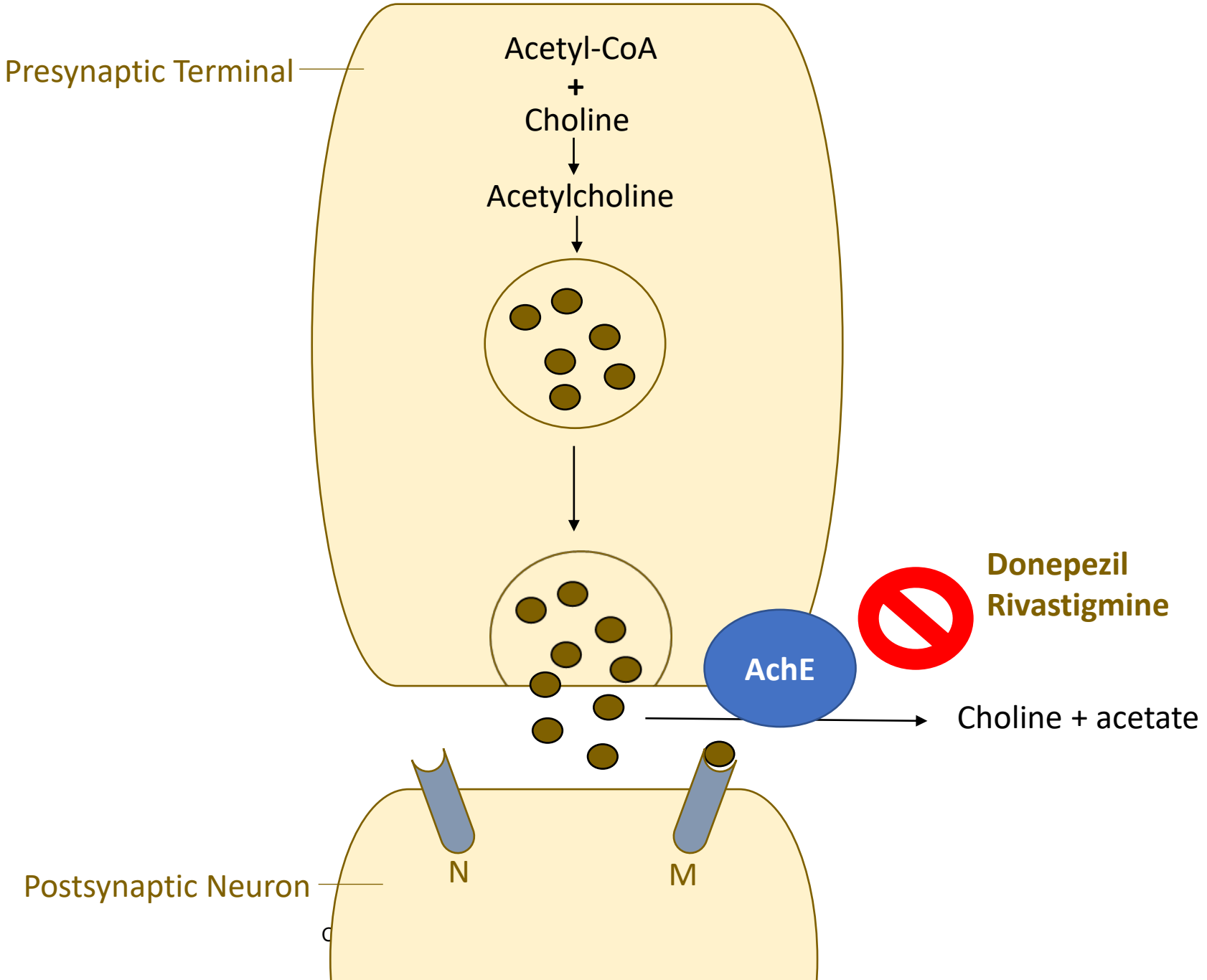
- NMDA receptor antagonists
  - ❑ Memantine

Acetylcholinesterase Inhibitors

Mechanism of action:

- hallmark of the disease: Progressive loss of cortical cholinergic transmission participates in Alzheimer's disease-associated dementia.
- Inhibition of acetylecholinesterase (AchE) → improve cholinergic transmission.

If this works it will only correct the symptoms of the disease but it has no role in slowing down the progression, acetylcholinesterase inhibitors don't reduce ongoing damage



Acetylcholinesterase Inhibitors

Therapeutic uses:

The reversible AchE inhibitors (Donepezil, galantamine and rivastigmine) are approved for the treatment of *mild* to *moderate* Alzheimer's disease.

- These drugs provide a modest reduction in rate of loss of cognitive function in Alzheimer patients.
- Rivastigmine is the ONLY agent approved for the management of dementia associated with Parkinson's disease.
- Rivastigmine is the ONLY agent available as a transdermal patch.

Acetylcholinesterase Inhibitors

Adverse effects

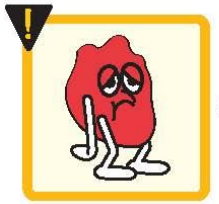
- Nausea
- Diarrhea
- Vomiting
- Anorexia
- Tremors
- Bradycardia
- Muscle cramps

Pupil dilatation

Increased cholinergic stimulation at the muscaranic receptors in the heart

Increased acetylcholine availability at the neuro-muscular junction


Tremors


Bradycardia


Nausea


Diarrhea

Anorexia


Myalgia

NMDA Receptors Antagonists

Mechanism of action:

- Overstimulation of NMDA glutamate receptors in the brain→ increases intracellular calcium → neurodegenerative or apoptotic loss of neurons (excitotoxicity)

NMDA Receptors Antagonists

Therapeutic uses

- Memantine is an NMDA receptors antagonist approved for the treatment of *moderate* to *severe* Alzheimer's disease.
- Memantine is often given in combination with an AchE inhibitor to treat Alzheimer's disease.

Treatment of Alzheimer's Disease

- Pharmacotherapy of Alzheimer's disease is symptomatic.
- The standard care includes AchE inhibitors + a NMDA antagonist.
- They both provide modest, short-term benefits but do NOT alter the underlying neurodegenerative process.

Neuronal damage will continue because it's mainly driven by accumulation of amyloid

Future alternatives for the treatment of Alzheimer's Disease

They're tested with variable results

- **Cholesterol-lowering agents**: statins
- **Insulin sensitizers**: PPAR-γ agonists (Rosiglitazone) (it sensitizes tissue to insulin and alters apolipoprotein E gene expression →↑ break-down of β-amyloid).
- **Intranasal insulin** (insulin crosses BBB from the nasal mucosa via transport from the olfactory receptor cells in the roof of the nasal cavity as patients with AD have lower insulin levels in CSF and higher plasma insulin levels)
- **NSAIDs**: low dose aspirin, celecoxib

Future alternatives for the treatment of Alzheimer's Disease

Experimental disease-modifying drugs:

- Amyloid lowering agents: Semagacestat (failed)
- Drugs interfering with amyloid-β deposition: Tramiprosate
- Drugs increasing amyloid-β clearance: anti-amyloid antibodies
- Drugs interfering with tau deposition: Li+ small dose, valproate, methylene blue

Another protein metabolism byproduct accumulates in Alzheimer's brain

Antipsychotic Drugs

=Neuroleptics, Major tranquilizers

Definitions

- **Psychosis:**
  - Mental state involving detachment from the reality
  - Madness; supernatural power
  - Diagnosis by exclusion

thought, personality, behavior, bizarre irrational thinking المشكلة بتكون بال

- **Delusion:**

Describes false bizarre fixed beliefs

بتكون قناعات عند المريض اعتقادات معينة من دون دليل او صلة بالواقع ما الها اصل حقيقي ثابتة صعب تغيرها، في انواع كتير لل delusions اشهرها persecutory delusion و هو ال delusion of safety معظمهم بكون خايفين او عندهم افكار انه they're haunted حدا بحاول يقتله او يسممه يراقب تحركاته براقبه من كاميرات و كلها اشياء ما الها بالراقع و ممكن الحدا يكون خايف منه من العيلة

2 main symptoms characterizing psychosis

- **Hallucination:**

Sensory dysfunction

the patient will create certain sensory image that's actuallyالمريض راح يحس باشياء مش موجودة بالواقع absent ممكن يكون visual or auditory ال most commonly auditory بكون بسمع اصوات مو موجودة فعليا اصوات بتكون تحكي معه شخصيات الها اسماء و بالعادة بتكون هاي الاصوات negative بتكون تحكي اشياء سيئة عنه بالعادة discouragingاذا كانت visual بكون يتخيل وجود شخصية معينة و يحكي معها و يتفاعل معها

- **Illusions?**

Misperception of stimulus

في الحقيقة ال stimulus بكون موجود بس بفهمه او بشوفه بطريقة خاطئة يعني ممكن يشوف شجرة على انها شخص بده يهجم عليه

Psychosis: Causes

Unknown, poorly understood

1% prevalence

- Genetic?
- Normal?
- Environmental
  - Stress
- Medical conditions:
  - 1. Drugs (dopa like)
  - 2. rapid withdrawal of opoid
  - 3 brain tumour
  - 4. Alzheimer and parkinson could manifest psychotic illnesses later
  - 5 Electrolytes imbalance
  - 6. Fetal infections

Can trigger psychosis in patients who are predisposed to develop psychotic illnesses

Function MRI doesn't show any abnormality

- Drugs:
  - Alcohol; Cannabis
  - Cocaine; Amphetamine; MDMA
  - K-opioid receptor agonists
  - NMDA Antagonists:

Amphetamine » increase release of dopamine in the brain

Signs and symptoms

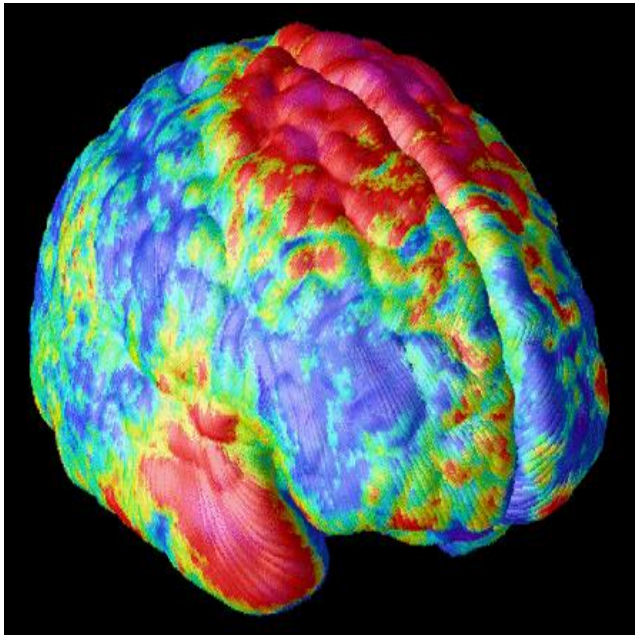
- Positive
  - Hallucination & delusions
  - Thought disorders
  - Abnormal behaviors
- Negative
  - Social contacts
  - Emotional responses
  - Decrease attention and memory
  - Anxiety and depression--→ Suicide

They withdraw from social contact

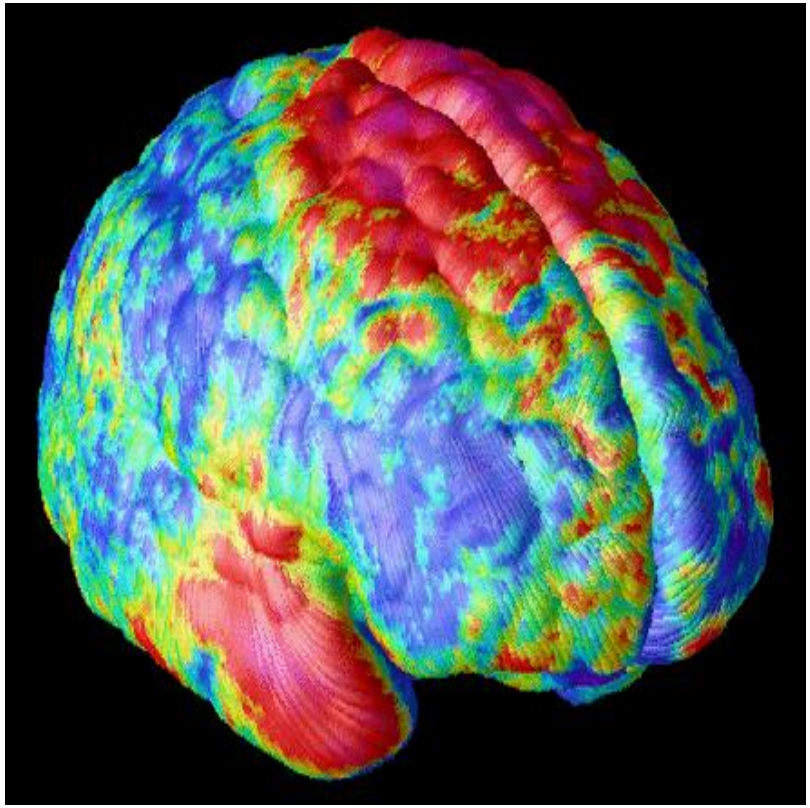
Depressed most of the time, loss of pleasure

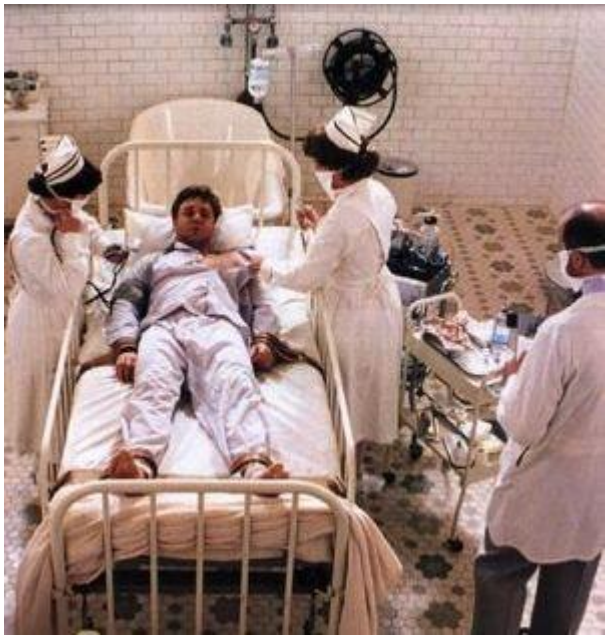
مایست

Psychosis: pathogenesis

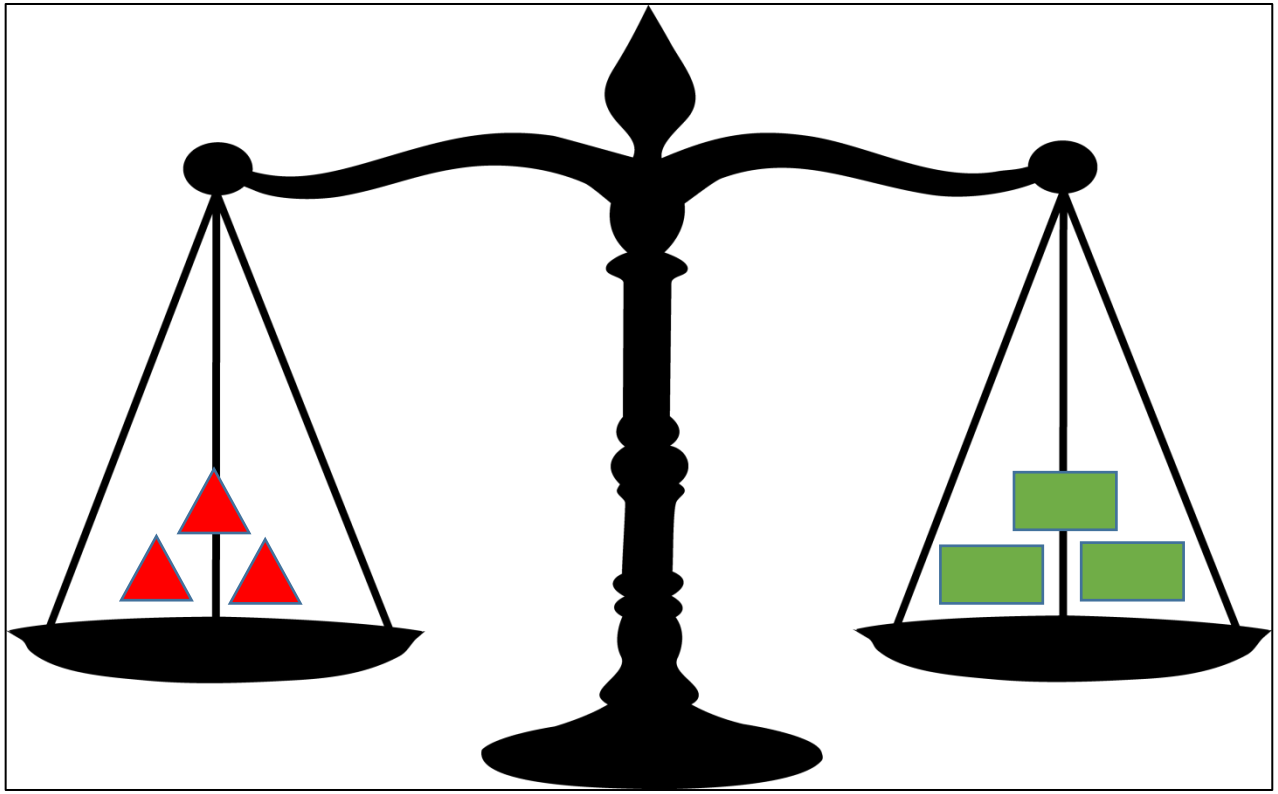


Psychosis: pathogenesis





Pathophysiology: Dopamine Hypothesis



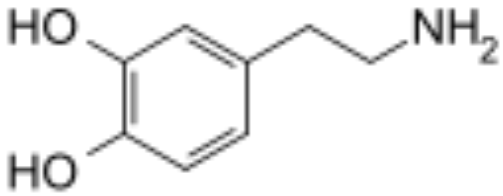
ال theory بتحكي هل ال balance between glutamate and dopamine هو السبب ؟؟

Glutamate (Glu)

Dopamine (DA)

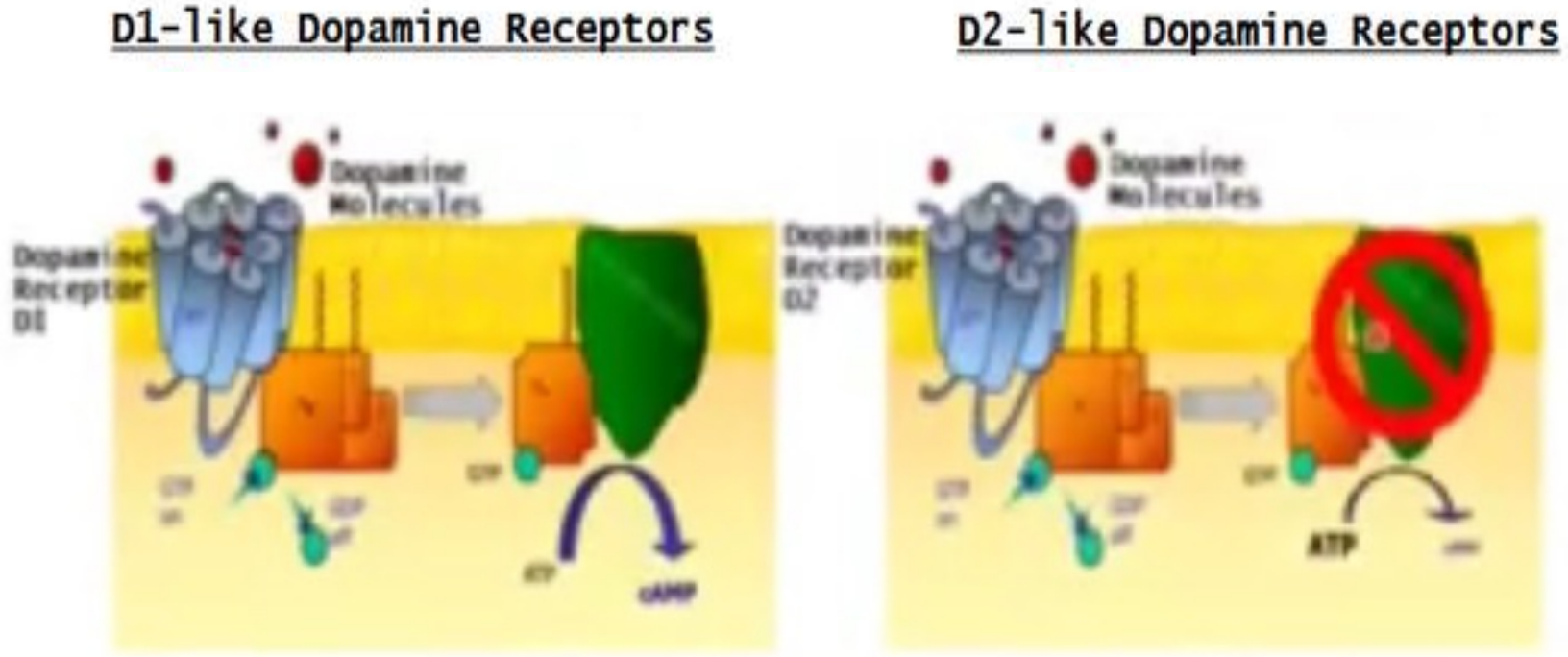
Dopamine

- A catecholamine neurotransmitter
- Projections:
  - Substantia Nigra:
    - Basal Ganglia: Movement
  - Ventral tegmental area:
    - Mesolimbic pathway: Reward and cognition
  - Arcuate nucleus:
    - Pituitary gland: Inhibit prolactin secretion
- Receptors:
  - D 1,5 (Excitatory; Metabotropic; GPRC (Gs))
  - D 2, 3, 4 (Inhibitory; Metabotropic; GPCR (Gi))



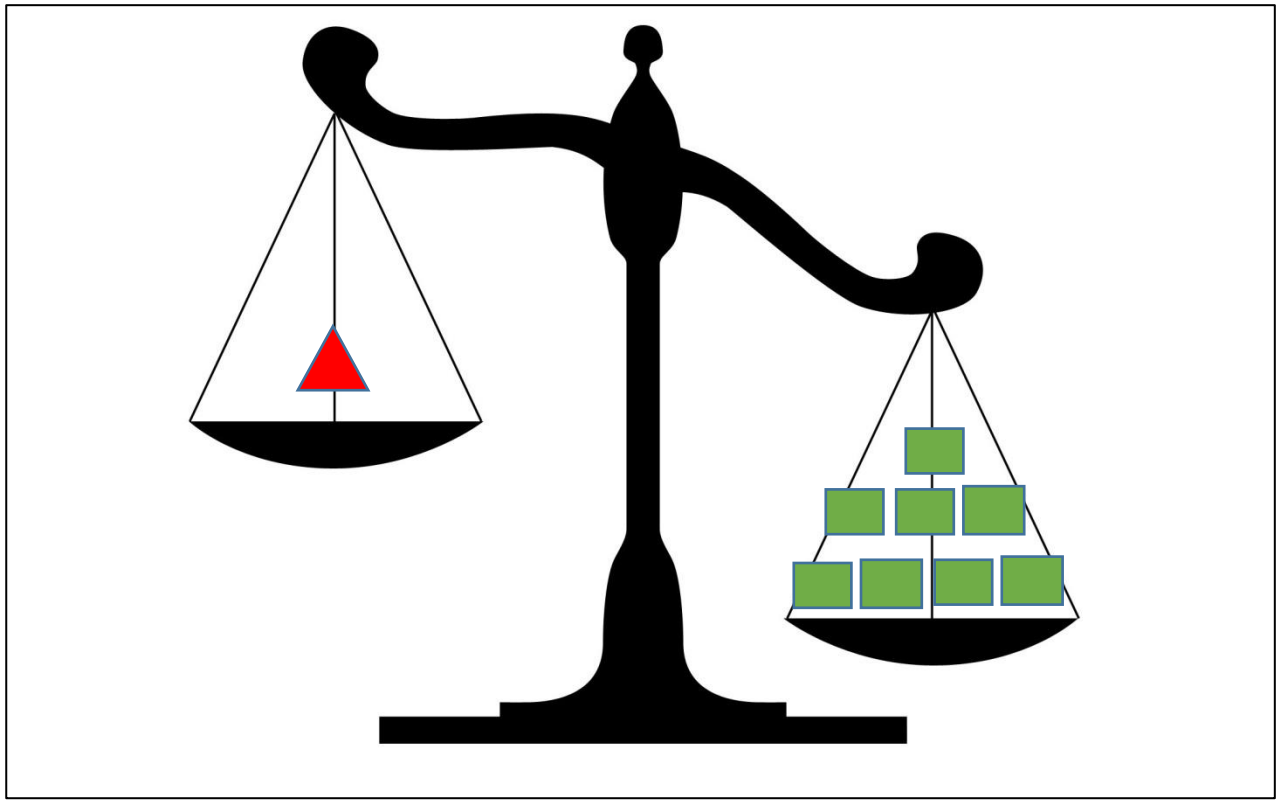
Difference in ability of dopamine to exert different brain functions because it's a mixed excitatory and inhibitory neurotransmitter

D1 vs. D2


D1-like Dopamine Receptors
Dopamine Molecules
Dopamine Receptor D1
ATP
cAMP
D2-like Dopamine Receptors
Dopamine Molecules
Dopamine Receptor D2
ATP

Most anti-psychotics aim to decrease dopamine levels

Pathophysiology: Dopamine Hypothesis



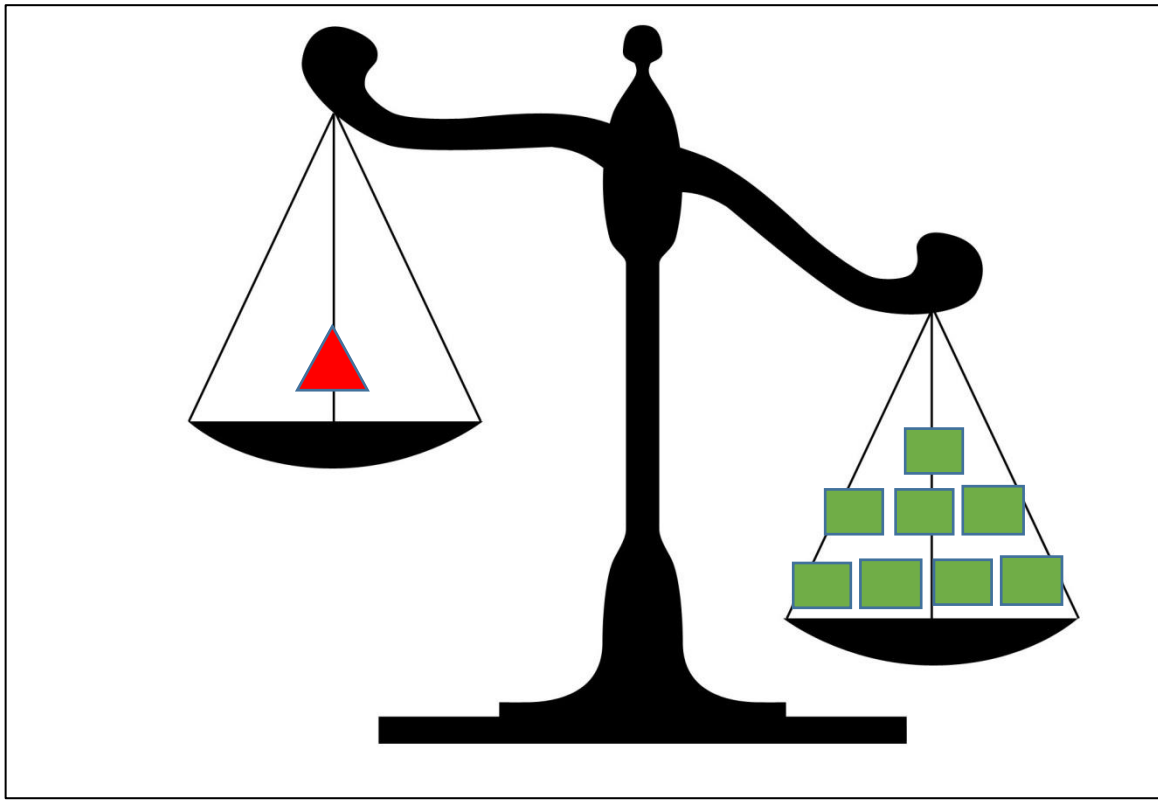
Glutamate (Glu)

Dopamine (DA)

Pathophysiology: Dopamine Hypothesis

Pros:

- D2 agonists and dopamine releasers produce psychotic-like effects.
  - Amphetamine, bromocriptine
- Drugs block NMDA receptors produce psychotic-like effect.
  - Ketamine, Phencyclidine
- Antipsychotics are dopamine (D2) antagonists.
  - Clozapine, Olanzapine



Cons:

- Antipsychotics are not always effective.
- Therapeutic effect is generally delayed.
- Newer antipsychotics have 5-HT antagonism (Pimavanserin)

Newer agents are histamine antagonists

Glutamate (Glu)

Dopamine

Antipsychotic Drugs: Principles

- The exact Pathophysiology is not well understood.
- Many people do not respond fully to medications or respond partially
- Therapeutic effect may be delayed several weeks
- Significant side effects
- More effective against positive-symptoms
- Use the minimum effective dose.

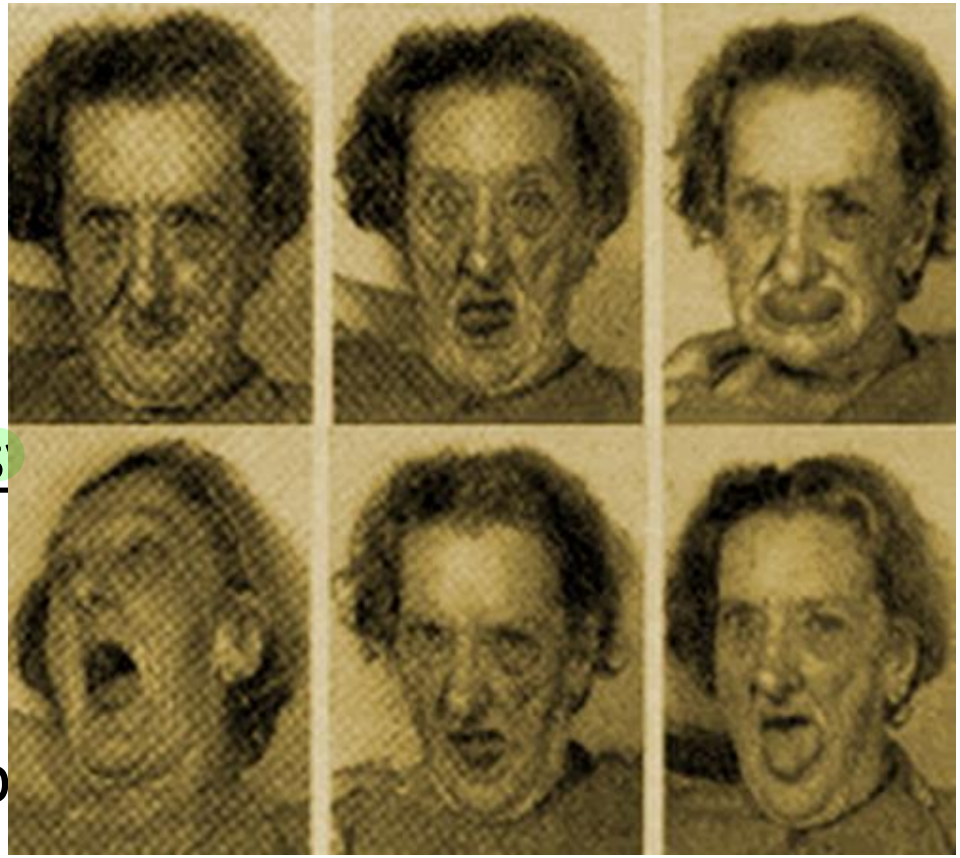
Main reason for incompliance

Negative symptoms are only treated by psychological therapy

Antipsychotic Drugs: Major S/E

Life threatening condition characterised by circulatory shock, high fever, seizures

- Hyperprolactinemia.
  - Glactorrhoea
  - Gynecomastia
- Sexual dysfunction:
- Neuroleptic malignant s
  Syndrome
- Tardive dyskinesia.
- Sedation??
- Hyperglycemia and diab
- Weight gain??



Abnormal repitative movements of facial muscles » irreversible effects

مانیست

Antipsychotic Drugs: Major S/E

- Hyperprolactinemia.
  - Glactorrhoea
  - Gynecomastia
- Sexual dysfunction: loss of libido.
- Neuroleptic malignant syndrome
- Tardive dyskinesia.
- Sedation??
- Hyperglycemia and diabetes.
- Weight gain??

Typical

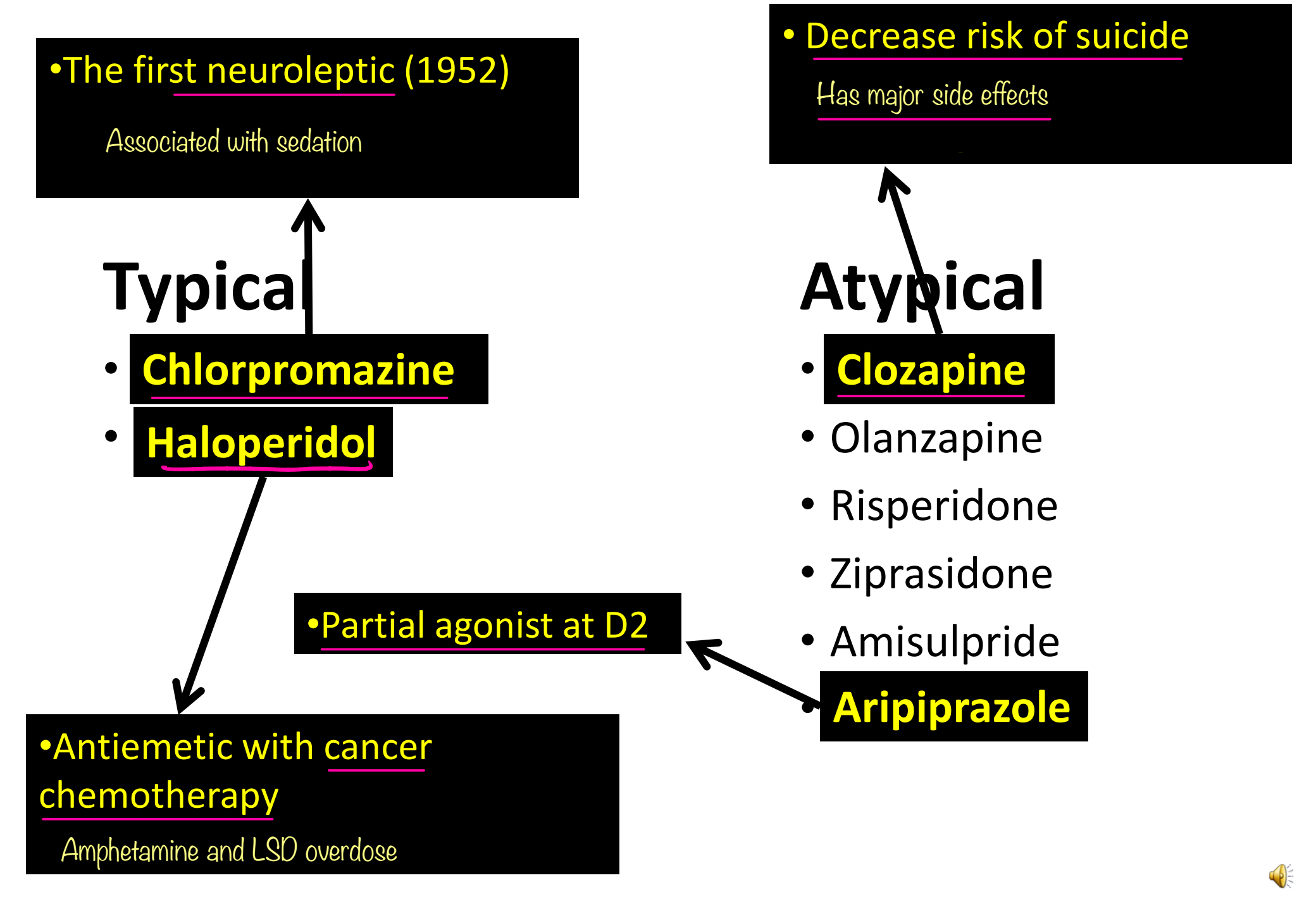
Atypical

موکس مهم

Antipsychotic Drugs

Lees adverse effects

	Typical (First Generation)	Atypical (Second Generation)
Discovery	Older	Newer
Effectiveness	Partially	Better?
MOA	D2: Antagonist	D2: antagonist 5-HT: antagonist (5-HT3c)
Extrapyramidal S/E	+++++++	++


•The first neuroleptic (1952)
Associated with sedation
Typical
• Chlorpromazine
• Haloperidol
•Antiemetic with cancer chemotherapy
Amphetamine and LSD overdose
•Partial agonist at D2
• Decrease risk of suicide
Has major side effects
Atypical
• Clozapine
• Olanzapine
• Risperidone
• Ziprasidone
• Amisulpride
• Aripiprazole

Other Antipsychotics

- Pimavanserin:
  - Approved in 2016.
  - No dopamine action.
  - 5-HT inverse agonist (i.e. antagonist)
  - $$$$$$ → Very expensive
  - Parkinson's disease psychosis.

Antipsychotics: Other uses

- Anxiety Disorders: OCD
- Huntington Disease
- Autism
- Antiemetic

Antipsychotic Drugs: Clinical notes

- Most are effective against positive symptoms.
- Smoking increase metabolism of Clozapine
- Change the medication

اذا ما كان في response او صار response و بعدها بفترة صار relapse

- Injectable forms:
  - Non-Compliance of the patient
  - Acute psychotic agitation or mania
- Safe during pregnancy

Thank you

Question 1

- **Antipsychotics can produce all of the following except:**

A. Hyperglycemia
B. sedation
C. Weight loss
D. Sexual dysfunction
E. It can produce all of the above

Question 2

- **Examples of typical antipsychotics:**

A. Olanzepine
B. Haloperidol
C. Clozapine
D. Ziprazidone
E. All of the above

Question 3

- **Dopamine (D1) receptors are:**

A. Ionotropic excitatory
B. Metabotropic excitatory
C. Ionotropic Inhibitory
D. Metabotropic inhibitory
E. Voltage-gated receptors

Question 4

- **A patient look at a tea cup, and he claims it is a bomb. This is an example of:**

A. Dillusion
B. Illusion
C. Hallucination
D. Schizophrenia
E. Sounds like normal to me!