MED 443
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TEAM 443
PHARMACOLOGY

Drugs used in depression
Old & New group

- Main text
- Male slide
- Female slide
- Important
- Dr, notes
- Extra info

EDITING FILE

Objectives

No Objectives

Dr.Foda

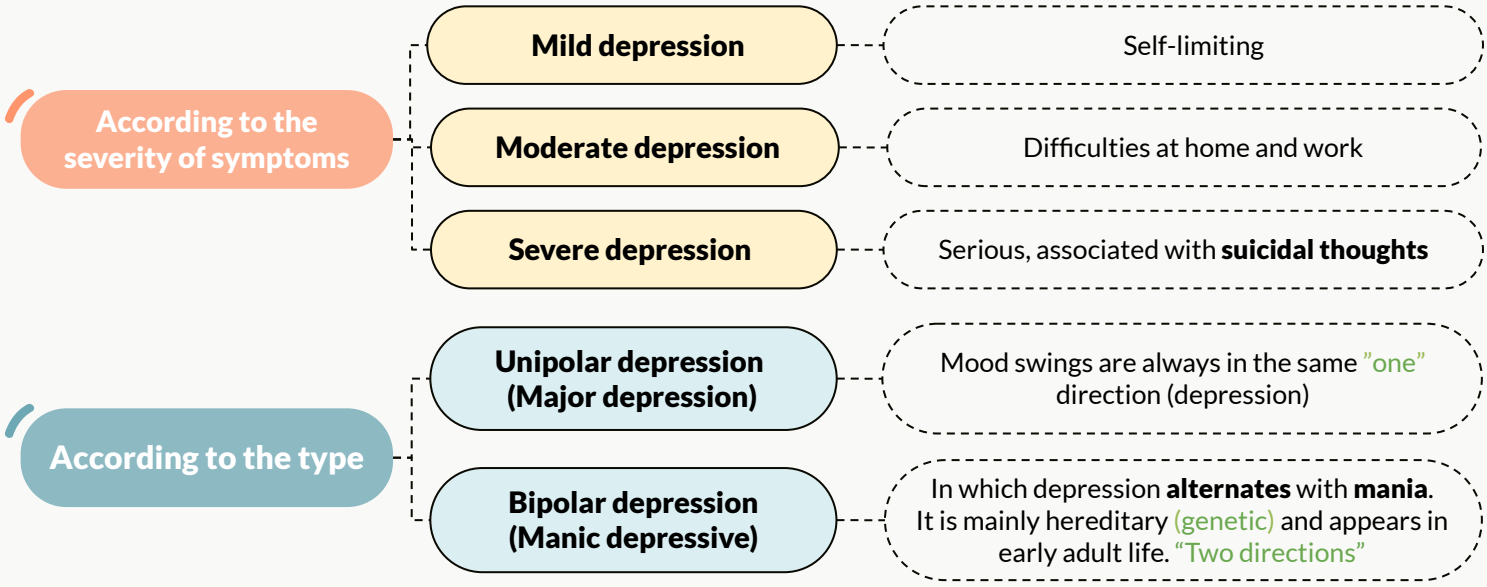
Male Dr.’s notes

- You won't be asked about Mania from these lectures
- We won't ask about the potency of drugs with its groups, just know the difference between drug families
- I won’t ask about Pharmacokinetics or the metabolism of drugs
- Focus on MOA and side effects (ADR’s)
- The questions at the end of the lecture are VERY important

Depression

1. "**Depression**" is a very common psychiatric disorder that is related to the "**mood**" (affective disorder).
2. Changes in mood are associated with **depression and/or mania** (great euphoria)
3. Disorders of mood rather than disturbance in thought or cognition. as in schizophrenia
4. • **Incidence**: Depression is a chronic and recurrent illness that can affect at least 20% of the population at some period in their lifetime.
   • **Cost**: 15-35 billions $ / year in USA only.

Classification of Depression



Symptoms

Depression	Mania
• **Loss of energy and interest:** ○ Diminished ability to enjoy oneself. ○ Decreased or increased "disturbed" sleeping or appetite. ○ Difficulty in concentrating; indecisiveness تردد; slowed or fuzzy thinking. ○ Exaggerated feelings of sadness, hopelessness, or anxiety.	• **Causes mood swings creating periods with the following symptoms:** ○ A high energy level with **decreased need for sleep.** ○ Unwarranted or **exaggerated belief** in one's own ability. ○ Extreme irritability. ○ Rapid, **unpredictable emotional changes.**
• **Feelings of worthlessness:** ○ Recurring thoughts about death and **suicide**. ○ If most of these symptoms last for **two weeks** or more, the person probably has Depressive illness.	

Biochemical Theory of Affective Disorders

Affective Disorders Lead to ↓ Serotonin

- ↑ NE levels → **Mania** — Rx: Drugs that ↓ NE
- ↓ NE levels → **Depression** — Rx: Drugs that ↑ NE

What is the evidence to support this theory?
Amphetamine (CNS Stimulant used in ADHD) causes mania by increasing norepinephrine and dopamine,while Reserpine (↓NE storage) & Methyldopa (↓NE synthesis) are antihypertensives that produce depression (these drugs deplete NE and Dopamine storage).

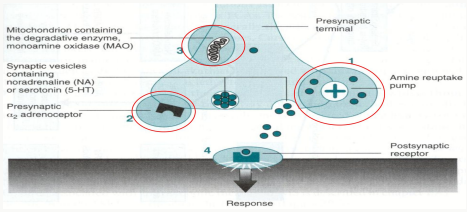
5-HT Deficiency	May cause sleep problems, irritability and anxiety associated with depression
↓ levels of NE	Disturbance in mood, alertness, arousal, appetite and reward
Dopamine	Important for pleasure, sexual function & psychomotor activity

What are the features of drugs that should be used for the treatment of depression?
Simply to increase the levels of these **mono**amines. Monoamines: NE, 5-HT, Dopamine

Important

Site of Action for Antidepressants

1. Monoamine (NE or/and 5-HT) reuptake pump inhibitors.
2. Blockade of presynaptic α2 receptors
3. Inhibition of MAO enzyme.



Classification of Antidepressants based on the site of action

Old antidepressants		New antidepressants	
Drugs that **block the reuptake of NE and 5-HT**	Drugs that **inhibit MonoAmine Oxidase (MAO)**	Drugs that **selectively block the reuptake of 5-HT (SSRIs)**	Drugs that block **presynaptic α_2** adrenoceptors
Most **Tricyclics**	MAOIs : - **Phenelzine** - **Tranylcypromine** - **Moclobemide**	- **Fluoxetine** - **Paroxetine** - **Sertraline** - **Citalopram** "Safer, no interaction with other receptors"	e.g. - **Mirtazapine** - **Mianserin**

Antidepressants available in the market (Worldwide)

Female Dr: Imp to know examples of each class

Class	Drugs
1. Tricyclics (TCAs) and Tetracyclics	1. Tricyclics: **Imipramine**, Desipramine, Clomipramine, **Amitriptyline**, **Nortriptyline** 2. Tetracyclics: Amoxapine, Maprotiline
2. MonoAmine Oxidase Inhibitors (MAOIs)	Tranylcypromine, Phenelzine, **Moclobemide**
3. Selective Serotonin Reuptake Inhibitors (SSRIs)	**Fluoxetine**, Paroxetine, Fluvoxamine, Sertraline, Citalopram, Escitalopram
4. Serotonin & Norepinephrine Reuptake Inhibitor (SNRI)	**Venlafaxine**, Duloxetine
5. Serotonin-2 Antagonist & Reuptake Inhibitors (SARIs)	**Trazodone,** Nefazodone
6. Norepinephrine & Dopamine Reuptake Inhibitor (NDRI)	**Bupropion** Male Dr.: يستخدم مع المرضى اللي بيوقفون التدخين
7. Noradrenergic and Specific Serotonergic Antidepressant (NaSSA)	**Mirtazapine**
8. Noradrenaline Reuptake Inhibitor (NRI)	Reboxetine

Slow Onset of Action

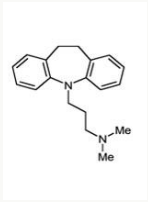
- **Antidepressants do not act immediately** (show clinical effects after 3 weeks) indicating that secondary adaptive changes must occur before the benefit is gained.
- The most consistent **adaptive change** that have to happen are seen with antidepressant drugs is the **downregulation** of β-, α_2 and 5-HT_2 adrenergic+serotonergic presynaptic receptors These receptor mediate **negative feedback** on monoamine release in the brain.
- Desensitization (down-regulation) of β- adrenoceptors (↓c-AMP) is very important and is related to clinical response.

A) Old Antidepressants

1. TriCyclic Antidepressants (TCAs)

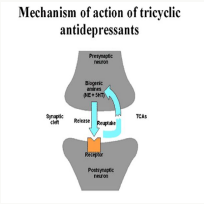
Drug

- TCAs are the **oldest** class of antidepressant drugs.
  - **Tricyclic** Antidepressants : They have characteristic **three**-ring nucleus :
    Imipramine, Desi**pramine**, Clomi**pramine**, Ami**triptyline**, Nor**triptyline.**
  - **Tetracyclic** Antidepressants (**four**-ring nucleus): Amoxapine, Maprotiline.



M.O.A

- All tricyclics **block reuptake pumps** for both **5-HT** and **NE** in nerve terminals by competing for binding site of the transport protein → so ↑ **conc. of NE & serotonin** in the synaptic cleft & at the receptor site."Simply, they **block NE and 5-HT reuptake**"
- Some have more potency for inhibition of **5-HT** uptake pump: "don't trigger mania"
  Amitriptyline, Imipramine, Clomipramine skipped by male's dr
- Others have more potency for inhibition of **NE** uptake pump: "they may trigger mania"
  Nortriptyline, Desipramine Skipped by male's dr



Actions

- Elevate mood
- Improve mental alertness
- Increase physical activity

"The antidepressant effect may develop after several weeks of continued treatment (**2-3** weeks)"

- **In non-depressed patients**: they cause sedation, confusion & motor incoordination.

P.K.

skipped by male's dr

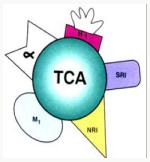
- **Peak levels:** 2-6 hours post ingestion Numbers are not imp
- TCAs are **lipophilic** in nature, therefore they are well absorbed from the GIT and readily cross the BBB to penetrate the CNS.They have a large volume of distribution
- **Elimination:** hepatic oxidation
- **Metabolism:** In the liver by demethylation (**Imipramine** to **Desipramine**, **Amitriptyline** to **Nortriptyline**) and by hydroxylation into metabolites that retain the biological activity of the parent compounds. Metabolites also active→ **Longer DOA**

Important

★ADRS

TCAs (nonselective) **block:**

1-M_1 cholinergic receptors
2- H_1 histamines receptors
3- α_1 adrenergic receptors
4- **5-HT_2** receptors



- **Anticholinergic:** Dry mouth, blurred vision, constipation & urine retention, aggravation of glaucoma. atropine like action
- **Antihistaminic:** Sedation, confusion.
- **Anti-adrenergic :** Postural hypotension, arrhythmias, conduction defects.
- Weight gain (due to **5-HT_2** block),**Sexual dysfunction** & impotence
- Lower seizure threshold Due to ↑NE which is a CNS stimulant
- **TCAs have narrow therapeutic index → toxicity can develop:** excitement, convulsions, coma, atropine-like effects, cardiac arrhythmias, sudden death

  they should be monitored, some patients attempt suicide by overdosing
- **TCAs have a large volume of distribution** therefore **hemodialysis is NOT effective** for treatment of TCAs toxicity also they are bound to plasma proteins.

A) Old Antidepressants

1. TriCyclic Antidepressants (TCAs)

Uses

- Endogenous (Major) **Depression** "moderate to severe".
- **Imipramine** is used for treatment of ★**nocturnal enuresis** (involuntary urination) in children & geriatric patients as it constricts internal urethral sphincter (antimuscarinic effect).
- Panic attack /acute episode of anxiety.
- Generalized Anxiety Disorder (GAD).
- Obsessive Compulsive Disorder (OCD).
- Attention Deficit Hyperkinetic Disorder (ADHD)

★**Chronic neuropathic pains or unexplained body pains.**

DDI*

- **TCAs are strongly bound to plasma protein,** therefore their effect can be potentiated by drugs that compete for **their plasma protein binding site** (NSAIDs like Aspirin and Phenylbutazone) "displacement of aspirin → ↑ effect/toxicity"
- TCAs are metabolized by **liver microsomal enzymes,** therefore their effect can be
  1. Reduced by **inducers** (Barbiturates)"either we change the drug or the dosage"
  2. Potentiated by **inhibitors** of liver enzymes (Oral contraceptives, Antipsychotics, and SSRIs).
- **TCAs** (inhibitors of monoamine reuptake) should not be given with ***MAOI*** (inhibitors of monoamine degradation) ,it may lead to **serotonergic & hypertensive crisis** "they have the same effect→ not given together"
- Additive to antipsychotics & anti-parkinsonisms → ↑anticholinergic effects.

C.I

1. in Patients with **Glaucoma or enlarged prostate** because of the **Atropine like action**

2. ↑NE :

- **in manic-depressive illness because** they tend to "switch" the depressed patient to the "manic" phase, therefore, they should be combined with "lithium salts"
  "drugs selective to serotonin do not have this effect"
- **in Seizure disorders** (TCAs increase NA levels in the brain, which is excitatory)

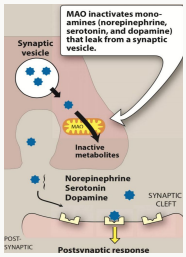
2. MonoAmine Oxidase Inhibitors (MAOIs)

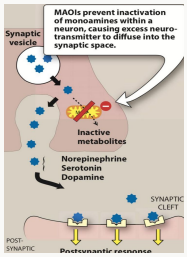
overview

-MAO is a mitochondrial enzyme found in nearly all tissues, and it exists in two forms:

MAO-A Responsible for **NE, 5-HT** catabolism. It also metabolizes **tyramine** of ingested food. "Inhibitors used in depression"

MAO-B More selective for **dopamine** metabolism ""Inhibitors used in parkinson's





MAO Inhibitors

Non Selective (MAO-A & MOA-B):
1-Phenelzine (Irreversible) long acting.
2-Tranylcypromine (irreversible)
correct: irreversible

"The effect of irreversible MAOIs persists for a period of 2-3 weeks after stopping treatment, time needed by the body to synthesize new enzyme."

Selective:
1-**Moclobemide**(**MAO-A**): (Reversible) Antidepressant action, short acting.
2-Selegiline(MAO-B): (Reversible) used in treatment of Parkinsonism.

A)Old Antidepressants

2. MonoAmine Oxidase Inhibitors (MAOIs)

Uses

- Only used for **refractory cases** and in atypical depression where phobia and anxiety are prominent symptoms.
- **Limited use because**:

1. ADRs
2. Food and drug interactions
3. Low antidepressant efficacy = Low Benefit/ risk ratio.

ADRs

- Antimuscarinic effects.
- Postural hypotension.
- Sedation,sleep disturbance.
- Weight gain.
- Specific ADRs for ***Phenelzine*** :
- Hepatotoxicity.
- Sexual Dysfunction

	Drug	Sedation	Anticholinergic effects	Hypotension
Non- selective irreversible	Isocarboxazid	+	++	+
	Phenelzine	+	++	+
	Tranylcypromine	-	+	+
Selective Reversible	Moclobemide	-	-	-

Important

Cheese reaction

This occurs when **Tyramine** rich foods are taken with **MAOIs**.

- **Tyramine** in food is normally degraded in the gut by **MAO-A**.
- **Tyramine** rich foods include: Old cheese , Concentrated yeast products, Pickled or smoked fish, Red beans,Red Wine, Chicken liver, Sausages.
- Since the enzyme is **inhibited** by **MAOIs**, **tyramine** from ingested food is **absorbed**, and then taken up into adrenergic neurons where it is converted into **octopamine** (a false transmitter) which causes massive release of **NE** and may result in **hypertensive crisis** ; severe hypertension vasoconstriction"peripherally", severe headache and fatal intracranial haemorrhage.
- **The special advantage** claimed for ★**Moclobemide** is that, **No cheese reaction** occurs with its use.

Drug-inter action

1. **Pethidine**: **MAOIs interact with the opioid receptor agonist (*pethidine*)** which may cause: severe **hyperpyrexia, restlessness, coma, hypotension.**
   The mechanism is still unclear, but it is likely that an abnormal pethidine metabolite is produced because of inhibition of normal demethylation pathway.

2. **Levodopa:** Precursor of dopamine can interact with MAOIs leading to **mania** and **hypertensive crisis**

3. ***Amphetamine & Ephedrine:*** Indirectly acting **sympathomimetics** can interact with MAOIs causing the liberation of accumulated monoamines in neuronal terminals leading to **hypertensive crisis**.
   "Due to ↑NE levels"

4. **TCAs** :inhibitors of monoamine reuptake can interact with MAOIs leading to **Serotonergic & hypertensive crisis** "they have the same effect→ not given together"

5. **SSRIs**: Serotonin syndrome **Give 1-2 weeks gap before initiating SSRIs.**

A 54 year old patient came to the psychiatry clinic, not controlled with any other medication, he was prescribed Moclobemide and felt relieved after a couple of months , what is the rationale for Moclobemide to be effective in depression?
Moclobemide is a drug that inhibits MAO-A, an enzyme that degrades NE and Serotonin, so when inhibited these neurotransmitters will increase.

B) New Antidepressants

- **SSRI :Selective Serotonin Reuptake Inhibitors**
  Increase serotonin on all receptors, ADRs related to overstimulation of 5HT2A(sexual dysfunction) & 5HT3(weight loss, nausea, vomiting ,diarrhea)
- **SARI : Serotonin-2A Antagonist & Reuptake Inhibitors**
  1- Inhibit serotonin reuptake selectively 2- 5-HT2A antagonists: no ADRs related to 2A overstimulation like sexual dysfunction
- **NaSSA: Noradrenergic and specific Serotonergic Antidepressant**
  1- Inhibit NE reuptake 2- Inhibit Serotonin reuptake selectively: Antagonists of 5HT2A no sexual dysfunction /5HT2C weight gain / 5HT3 antiemetic
- **NDRI: Norepinephrine and Dopamine Reuptake Inhibitors**
  No ADRs related to increased serotonin, only due to high NE
- **SNRI: Serotonin and Noradrenaline Reuptake Inhibitors**
  Basically glorified TCAs with no ADRs
- **NRI: Norepinephrine selective Reuptake Inhibitors**

B)New Antidepressants

1. Selective Serotonin Reuptake Inhibitors (SSRIs)

	Fluoxetine *(Prozac)*	Paroxetine	Sertraline	Citalopram	Escitalopram	Fluvoxamine
Overview	• The most widely utilized class of antidepressants in clinical practice. • They act within the brain to **increase** the level of **serotonin** (5-HT) in the synaptic gap by inhibiting its re-uptake. • Described as 'selective' because they affect only the reuptake pumps responsible for Serotonin.					
M.O.A	• Block 5-HT transport → increase 5-HT levels in synapse "**Inhibits 5-HT reuptake**" • They have No effect on **NET** (norepinephrine transporter). • They don't block **mAch, H, or α_1 Adrenoceptor** unlike TCAs and MAOIs ➔ so no antimuscarinic nor sedative effect, Except **Paroxetine** • They are nearly of comparable efficacy but of preferential response in each individual					
Advantages	• The most commonly prescribed antidepressants. • **Lacks cardiovascular & anticholinergic side effects compared to TCAs.** • In contrast to MAOI, **they do not cause 'cheese' reaction** so there's no food restrictions. • **Safer** (low risk of overdose). • Acute toxicity is less than that of MAOIs or TCAs.					

B)New Antidepressants

1. Selective Serotonin Reuptake Inhibitors (SSRIs) cont.

	Long acting (3-11 days)	Moderate length (~24hr)
	Fluoxetine	**Paroxetine, Sertraline, Citalopram**

P.k

- Metabolized by P450 then conjugation.
  → They are **enzyme inhibitors.** you only need to know that they are inhibitors
  → **Weak** inhibitors → Sertraline, Citalopram → ↓ interaction
  → **Strong** inhibitors → Fluoxetine, Paroxetine → ↓ metabolism of TCAs, neuroleptics, some antiarrhythmics, β-blockers
- **Fluoxetine** differs from other members of this class in:
  1. It has a **longer $T_{1/2}$** (50 hrs).
  2. Available as sustained release preparations → once weekly.
  3. Its metabolite **norfluoxetine** = **potent** as parent drug $T_{1/2}$(10 days).

Uses

Same as for TCA, in addition effective in the following conditions:
- **Anxiety** disorders.
- Eating disorders: Bulimia nervosa **(Fluoxetine)**, Anorexia nervosa "restricting eating".
- Post traumatic stress disorder (**PTSD**).
- Attention Deficit Hyperkinetic Disorder (**ADHD**).
- Treatment of **Premature Ejaculation** (via stimulation of **$5HT_{2A}$**).

ADRs

Important

Adverse effects of SSRIs:
SSRIs are selective to serotonin, but they increases the activity on all 5HT receptors, that's why they have more ADRs compared to more specific NASSA & SARI

- **$5\text{-}HT_3$** stimulation:
  1.GIT symptoms: ★**Nausea vomiting** & diarrhea. 2.Changes in appetite
- **$5\text{-}HT_{2A}$** stimulation: **Sexual dysfunction**: Loss of libido, **delayed ejaculation**
- Sleep disturbances: Drowsiness with **Fluvoxamine**, **Paroxetine**
- Anxiety & Tremors misuse

Drug	Cardiotoxicity	Nausea	Anticholinergic effects	Sedation
Fluoxetine	-	++	-	-
Paroxetine	-	++	+	+
Sertraline	-	++	-	-
Fluvoxamine	-	+++	-	+

Discontinuation syndrome:
- Symptoms are headache, malaise & flu-like symptoms, agitation , irritability & nervousness. To avoid discontinuation syndrome : decrease the dose instead of stopping it immediately

Drug-interaction

- **SSRIs** are potent **inhibitors** of liver microsomal enzymes. Therefore they should not be used in combination with **TCAs** because they can inhibit their metabolism increasing their toxicity.
- **SSRIs** should not be used in combination with **MAOIs** because of the risk of life threatening **Serotonin syndrome: tremors, hyperthermia, cardiovascular collapse and death**

Both drugs require a "washout" period of 6 weeks before the administration of the other.

B)New Antidepressants

2. Serotonin-2A Antagonist and Reuptake Inhibitors (SARI)

	Trazodone	Nefazodone
MOA	• **Blocks 5-HT uptake selectively** but in a less potent manner than tricyclics. This reduces depression. • However, they are powerful **5-HT$_{2A}$ antagonists**: 1. Blockade of 5-HT$_{2A}$ receptors **stimulates 5-HT$_{1A}$ receptors**, which may help reduce depression. **2.** **5-HT$_{2A}$** antagonism also reduces the risk of anxiety, sedation **or sexual dysfunction** which is normally associated with SSRIs. • **Nefazodone:** structurally related to *Trazodone* but has **less sedative** effect, however; like most SSRI it **inhibits P450** 3A4 isoenzyme.	

3. Noradrenergic and specific serotonergic Antidepressants (NaSSA)

	★Mirtazapine
MOA	• **★α_2 receptors antagonist** special feature for mirtazapine • Increases NE and 5-HT levels. • **Blocks 5-HT$_{2A}$, 5-HT$_3$ reducing** side effects of anxiety & **Sexual dysfunction** (5-**HT$_{2A}$**) + relieves nausea & vomiting(5-HT$_3$)
ADRs	Blocking **5-HT$_{2C}$**, and **H$_1$** receptors cause side effects: • **Sedation** due to **H$_1$** blocking effect • **Weight gain**
Uses (Important)	**★★Preferred in cancer patients because:** 1. **Improves appetite**. weight gain 2. **↓ Nausea & vomiting** (Blocks 5-HT$_3$). 3. **↑ Body weight.** 4. Sedation (potent **Anti-histaminic**). MirtaZZZapine H$_1$ blocking 5. Less sexual dysfunction (5-HT$_{2a}$ blocking). 6. Has no antimuscarinic effect.

B)New Antidepressants

4. Serotonin and Noradrenaline Reuptake Inhibitors (SNRIs)

	Venlafaxine (Effexor) فين الفاكسين!
MOA	• Venlafaxine is the first and most commonly used SNRI. • **Selective 5-HT and NE uptake blockers** combines the action of SSRI and NRI, but **without α1, M1 cholinergic or H receptor blocking properties.** Similar to TCA mechanism but without the undesired blockade of multiple receptors "ADRs". • **Des**venlafaxine is a metabolite of Venlafaxine **Q:** Can SNRI's be combined w/ SSRI's? **A:** No, because they both have the same mechanism/synergistic effect → Toxicity
Uses	• Depression • Generalized anxiety disorder • Social anxiety disorder in adults. ★ يستخدم للناس اللي عندهم اصابه او اعاقه مع الانتباه للمشاكل اللي ممكن يسببها على القلب

5. Norepinephrine & Dopamine Reuptake inhibitors (NDRIs)

	Bupropion
MOA	Is unique in possessing significant potency as **NE (Norepinephrine)** and **DA (Dopamine) reuptake inhibitor**, with no direct action on 5-HT. Can be given with SSRIs
Uses	• Treatment of major depression and bipolar depression. • Used for **smoking cessation** → As it reduces the severity of nicotine craving & withdrawal symptoms.
Advantages ★ **Important**	1. **No sexual dysfunction** → given in young (Combination with SSRIs to avoid sexual dysfunction). 2. **No weight gain** (No 5-HT effect). 3. **No orthostatic hypotension.**
★**ADRS** **Important**	• **Seizures**; it ↓**threshold of neuronal firing** Contraindicated in epilepsy

6. Norepinephrine selective reuptake inhibitors (NRIs)

	Reboxetine
MOA	• **Blocks only NET** (Norepinephrine transporter) • No affinity for 5-HT, DA, ADR (Adrenergic receptor), H, mAch receptors. So, has positive effects on the concentration and motivation in particular.
Advantages	• Safe to combine with SSRIs.
ADRS	• Minimal side effects only related to activation of **ADR system** as tremor, tachycardia, and urinary hesitancy.

Clinical use of antidepressants

Disorder	Drugs		
Endogenous Depression	SSRI	New generation	Tricyclics
Panic Disorders		Imipramine	
Obsessive Compulsive Disorders (OCD)		Clomipramine	
Anorexia nervosa* & Bulimia Eating disorders (common in young pts)			
Premature ejaculation			
Schizo-Affective Disorders	SSRI +Haloperidol	Amoxapine	
Chronic pain	Amitriptyline		
Anxiety disorders			
Migraine & Anxiety & irritable bowel syndrome			
Nocturnal Enuresis in children	Imipramine		

MCQ

1. A 45 year old woman suffering from feelings of worthlessness and diminished ability to concentrate for over three weeks after her husband passed away. Upon visiting the doctor she was diagnosed with major depressive disorder and was prescribed Antidepressants, how long until the medication starts showing clinical effects?			
A. Immediately	B. After 3 weeks	C. After 1 week	D. After 3 days
2. Which of the following drugs block the reuptake of both 5-HT and NE?			
A. Fluoxetine	B. Escitalopram	C.Imipramine	D. Nefazodone
3. A 7 year old child suffering from nocturnal enuresis was Prescribed Ipramine to constrict internal urethral sphincter, which of the following drugs are contraindicated in this patient?			
A. Amitriptyline	B. Desipramine	C. Moclobemide	D. None of the above
4. A 67 old Woman with diabetes and Glaucoma was diagnosed with OCD, which of the following drugs are contraindicated in this case?			
A. TCAs	B. Selective serotonin reuptake inhibitors	C. MonoAmine Oxidase Inhibitors	D. None of the above
5. A 44 year old man was celebrating his promotion with his friends and Spent the night Eating old cheese, sausages, smoked fish and red beans and other foods which rich in Tyramine. After he got home he took MAOIs previously prescribed from is doctor and suffered a “cheese reaction” which of the following events could occur in cheese reaction?			
A. Depression	B. Hypotension	C. Vomiting	D. Hypertensive crisis

1:B ,2:C ,3:C ,4:A ,5:D

MCQ

6. Which of the following could be a side effect of taking fluoxetine (prozac).			
A. Hypertensive crisis	B. cheese reaction	C. Antimuscarinic	D. vomiting
7. What receptors does Mirtazapine (NaSSA) affect?			
A. a2	B. M1	C. a1	D. Adrenoreceptors
8. A 32 year old man diagnosed with cancer and suffering from constant emesis from his chemotherapy medications in addition to a loss of appetite. Which of the following antidepressants was prescribed in this case?			
A. Fluoxetine	B. Mirtazapine	C. Sertraline	D. None of the above
9. A 29 year old man presents to the clinic after having a difficult time dealing with nicotine cravings and withdrawal symptoms after quitting smoking which of the following drugs would help decrease withdrawal symptoms of smoking?			
A. Amoxapine	B. imipramine	C. Bupropion	D. Serotonin and noradrenaline reuptake inhibitors
10. Which of the following drugs are contraindicated In a patient suffering from epileptic epilepsy?			
A. Bupropion	B. Reboxetine	C. Flouxitine	D. None of the above

6:D ,7:A ,8:B ,9:C ,10:A

SAQ

01

7 year old boy suffering from nocturnal enuresis was given TCAs. Explain the MOA,
Mention 3 of the receptors affected, and what family of drugs is contraindicated and why?

1.block reuptake pumps for both 5-HT and NE
2- M1, H1, a1, 5-HT2
3- MOAIs because it could lead to serotonergic and hypertensive crisis if taken together

02

26 year old male was Diagnosed with major depressive disorder and was prescribed SSRIs by his doctor. What is the most commonly given SSRI? and mention its MOA

1-Fluoxetine
2-Enzyme inhibitors

03

54 year old smoker was was trying to quit smoking but could not deal with the nicotine cravings and withdrawal symptoms. What drug could be given to decrease the severity of withdrawal? and it what situation is this drug Contraindicated?

1- Bupropion
2-epilepsy

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