بسم الله الرحمن الرحيم

DOPAMINE SCIENTIFIC TEAM

Antipsychotics

FINAL | Lecture 4

﴿إِنِّي تَوَكَّلْتُ عَلَى اللَّهِ رَبِّي وَرَبِّكُمْ ۚ مَا مِن دَابَّةٍ إِلَّا هُوَ آخِذٌ بِنَاصِيَتِهَا ۚ إِنَّ رَبِّي عَلَىٰ صِرَاطٍ مُّسْتَقِيمٍ﴾

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PHARMACOLOGY

رحلة اليقين مع سورة يس

بِسْمِ اللَّهِ الرَّحْمَٰنِ الرَّحِيمِ

إِن كَانَتْ إِلَّا صَيْحَةً وَاحِدَةً فَإِذَا هُمْ جَمِيعٌ لَّدَيْنَا مُحْضَرُونَ (٥٣) فَالْيَوْمَ لَا تُظْلَمُ نَفْسٌ شَيْئًا وَلَا تُجْزَوْنَ إِلَّا مَا كُنتُمْ تَعْمَلُونَ (٥٤)

{ إِنْ كَانَتْ } البعثة من القبور { إِلَّا صَيْحَةً وَاحِدَةً } ينفخ فيها إسرافيل في الصور، فتحيا الأجساد، { فَإِذَا هُمْ جَمِيعٌ لَدَيْنَا مُحْضَرُونَ } الأولون والآخرون، والإنس والجن، ليحاسبوا على أعمالهم.

{ فَالْيَوْمَ لَا تُظْلَمُ نَفْسٌ شَيْئًا } لا ينقص من حسناتها، ولا يزاد في سيئاتها، { وَلَا تُجْزَوْنَ إِلَّا مَا كُنْتُمْ تَعْمَلُونَ } من خير أو شر، فمن وجد خيرا فليحمد الله على ذلك، ومن وجد غير ذلك فلا يلومن إلا نفسه.

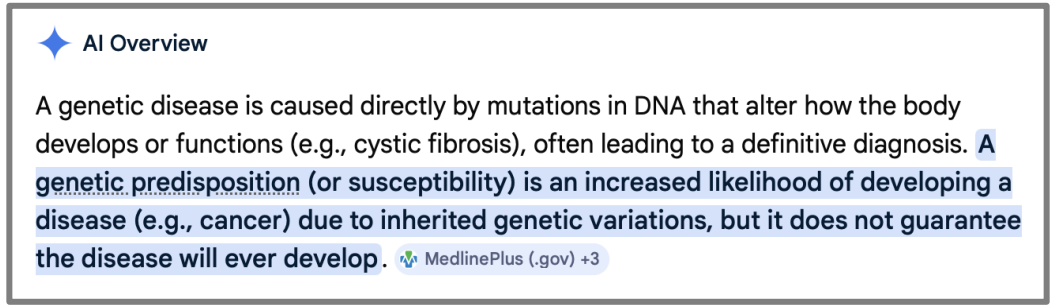
Schizophrenia

- At the end of Lecture 2, the Professor said that schizophrenia and psychosis are the same thing. However, psychosis is a symptom while schizophrenia is is a specific chronic mental disorder.
- It affects males more than females, in contrast to depression.
- There is overlap between schizophrenia, mania and paranoia, but the main difference is that schizophrenia is irreversible and that it is hard to deal with schizophrenic patients.
- Pathogenesis is unknown.
- Onset of schizophrenia is in the late teens - early '20s. In males, onset is in the early twenties (19-22). In females it's later (26-28). At the end of Lecture 2, the Professor correlated this with the major changes in the responsibilities of life that occur around these ages.
- Genetic predisposition -- Familial incidence. It runs in families, but it is not a genetic disease. Instead, it is associated with a genetic predisposition (risk factors), as are diabetes, hypertension, and asthma. Check the next page for more on this.
- Therefore, hereditary Influences may account only for 10% of schizophrenia cases. The main problem/influence is the environment.
- Multiple genes are involved.
- Afflicts 1% of the population worldwide and numbers are increasing.
- A thought disorder

Genetic Disease vs Genetic Predisposition to a Disease

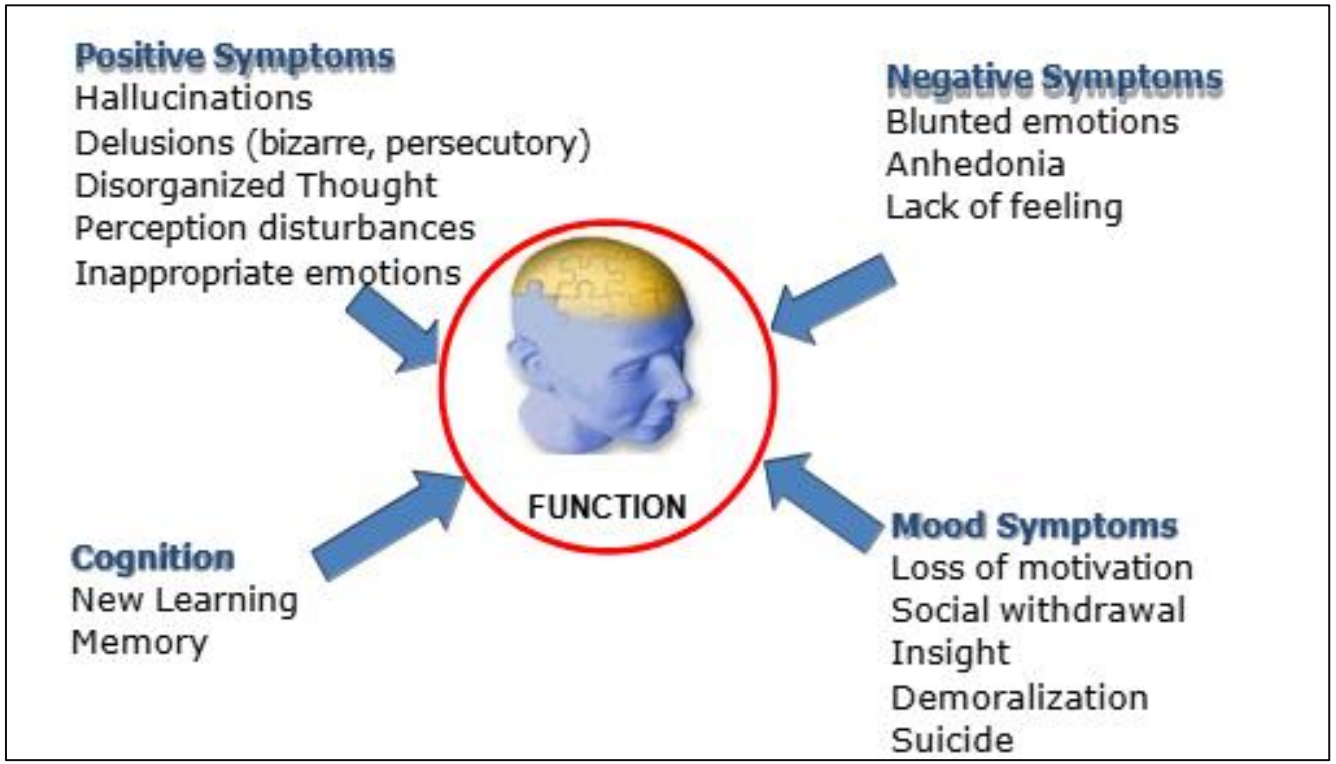
How do you know if it's a **genetic disease** or not?

- You follow **twins**: do they both develop schizophrenia, or does only one develop it?
- If not **both**, then it is not a genetic disease – there is a **genetic predisposition** involved instead.
- You may find that the father or other relatives develop it, but this is a result of genetic **risk factors** arising from **single nucleotide polymorphisms** rather than a mutation.
- Furthermore, there is what we call the **environment-gene interaction,** where the **co-existence** of an environmental factor and a genetic factor results in the development of schizophrenia.
- The relationship between genes is very complicated and is governed by **epigenetic** changes as well.
- Because schizophrenia is not a genetic disease, having a schizophrenic parent does not mean that a certain proportion of their children are expected to develop schizophrenia (i.e. we cannot use Punnett squares here).



Schizophrenia - symptoms

There are two types of problems with schizophrenia: positive and negative.
Those that are positive are easier to deal with.

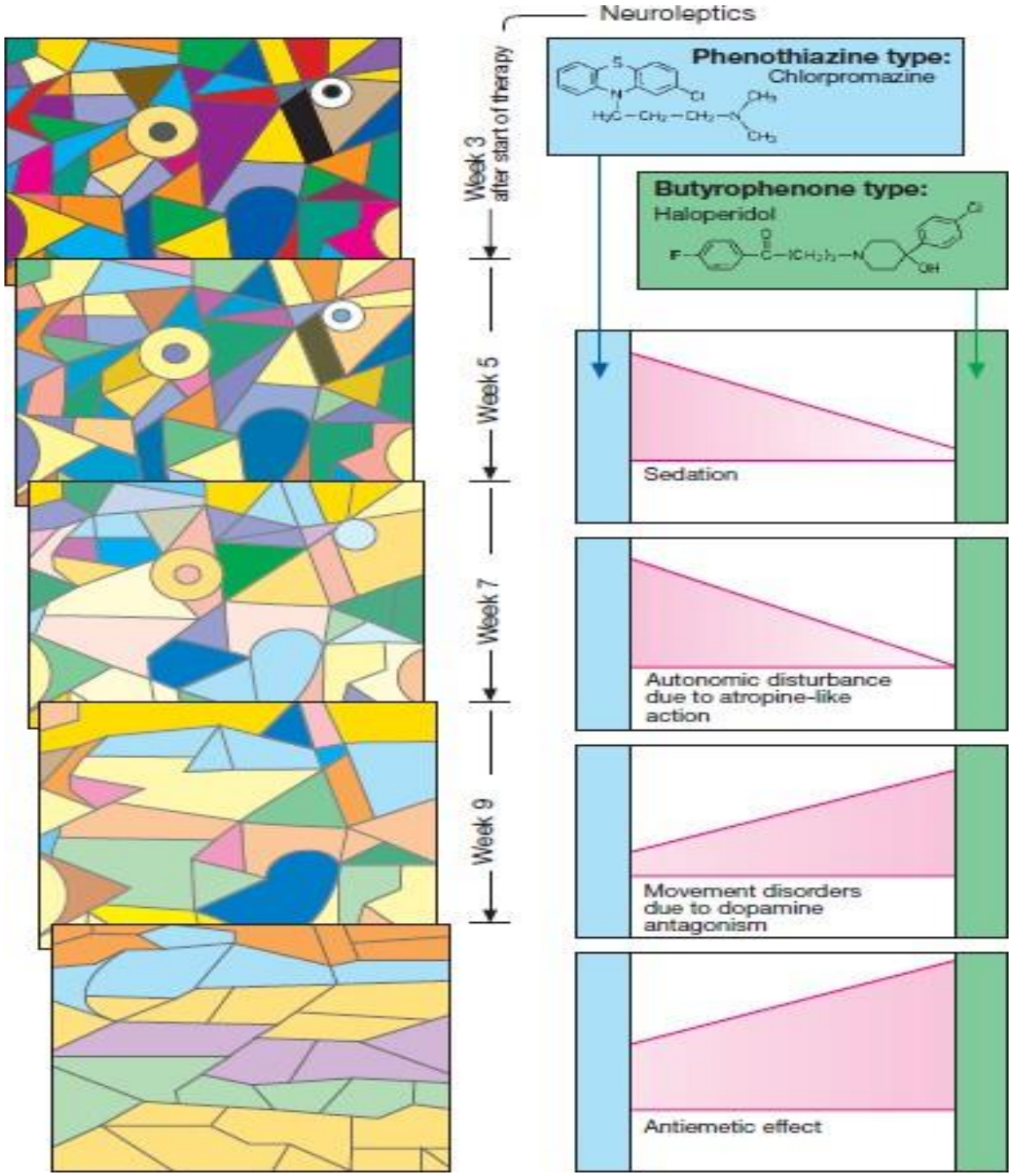


Their cognition is very low; they cannot learn anything new, have trouble performing calculations, etc...

Negative means major depression (sitting in the corner, doesn't want to continue life, suicidal thoughts, ...)

Patients can form logical sentences but cannot link them properly into comprehensive paragraphs.
This is known as thought disorder.
Thinking here is vastly impaired.

Typical picture of schizophrenia: patient is depressed with illusions and hallucinations.



This is what the brain of a schizophrenic looks like, as if it were a mix of colors merging together.

The idea of reference plays a huge role in their behavior. As with suicidal patients.
Ideas of reference are false beliefs that random or irrelevant occurrences in the world directly relate to oneself. Examples include believing TV characters are talking to you, newspapers contain secret messages for you, or that passersby are laughing specifically at you.

Ideas of reference usually arise from ideas taught to schizophrenic people in their childhoods, sometimes pertaining to ideas of grandeur (being a hero and saving the world). They begin to think that they are heroes and speak openly of their false achievements.
Schizophrenic people tend to feel as though they are being observed (by the FBI for example).
They may also start seeing things that don't exist (hallucinations), claiming that imaginary people or objects exist in the room with them.
Before knowing what to do, we need to know the theories explaining schizophrenia.

Schizophrenia

- Drugs currently used in the prevention of psychosis.

**** These drugs are not a cure ****

- Schizophrenics must be treated with medications indefinitely, in as much as the disease in lifelong and it is preferable to prevent the psychotic episodes than to treat them.

SCHIZOPHRENIA IS FOR LIFE

There is no remission

Schizophrenia is a horrible disease lasting for life. We can try to manage the disease , but we cannot cure it. The problem with schizophrenic patients is that the way the disease is managed renders them disabled (a person needs to live with him and take care of them)

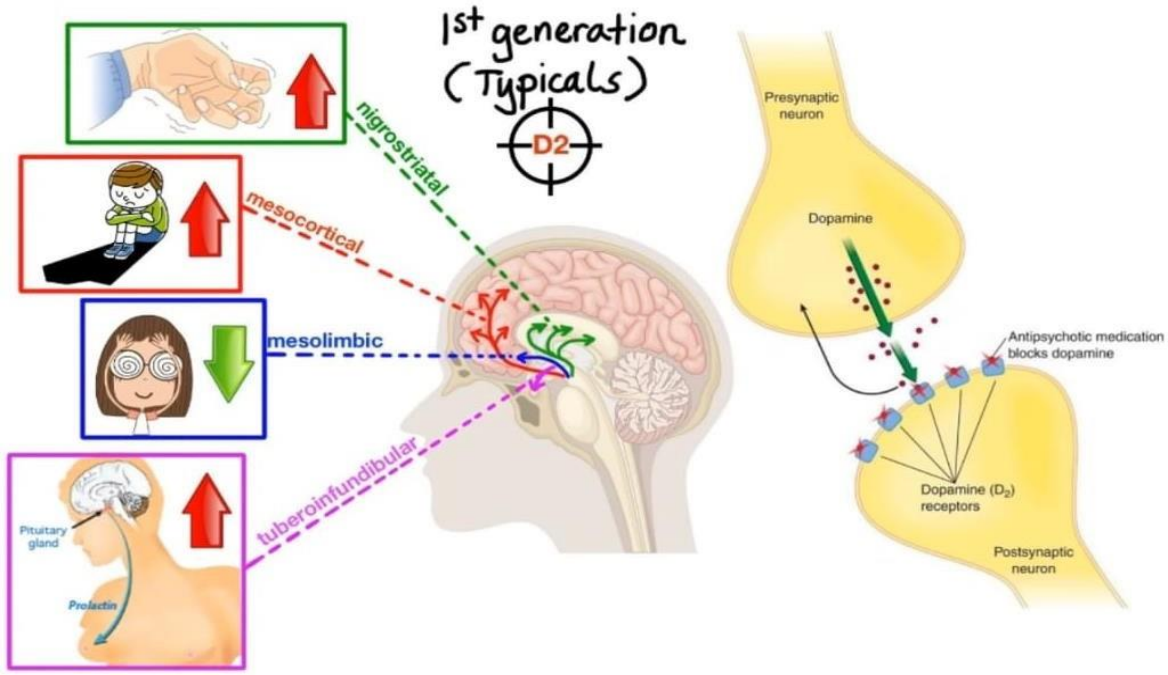
Dopamine Theory of Schizophrenia

- Many lines of evidence point to the aberrant increased activity of the dopaminergic system as being critical in the symptomatology of schizophrenia.
- There is a greater occupancy of D2 receptors by dopamine => greater dopaminergic stimulation

The dopamine theory states that schizophrenia involves an increase in dopamine and a resulting increase in the activation of D2 receptors. This leads to the positive symptoms of schizophrenia. The presence of negative symptoms may seem ironic as dopamine is associated with pleasure, but this can be explained by the interplay between dopamine and serotonin; in certain areas of the brain, dopamine can cause the suppression of serotonin. However, this theory was later proven false (see next slide).

To test this theory, we tried to do the opposite of what happens in the disease: we antagonized dopamine (e.g. by administering anti-D2 drugs). One would expect that antagonizing dopamine would reverse the all of the symptoms of the disease, but the following effects actually came about:

1) Mesolimbic: fewer hallucinations, illusions, and disorganized thoughts, and limited motor effects.
2) Nigrostriatal: extrapyramidal side effects.
3) **Mesocortical: patients became more depressed.** This particular finding disproved the dopamine theory, as antagonizing dopamine should reverse depression if the disease was truly caused by increased dopamine. In other words, the D2 antagonists should've improved their mood, but instead it did the opposite. This caused scientists to adopt a new theory based on the balance between serotonin and dopamine instead of dopamine alone (see next slide).
4) Tuberoinfundibular: this pathway has to do with the effect of dopamine on the anterior pituitary gland. Remember from endocrinology that dopamine inhibits prolactin release. Inhibition of dopamine leads to an increase in prolactin, causing gynecomastia in men and menstrual irregularities in women.



Schizophrenia Pathophysiology

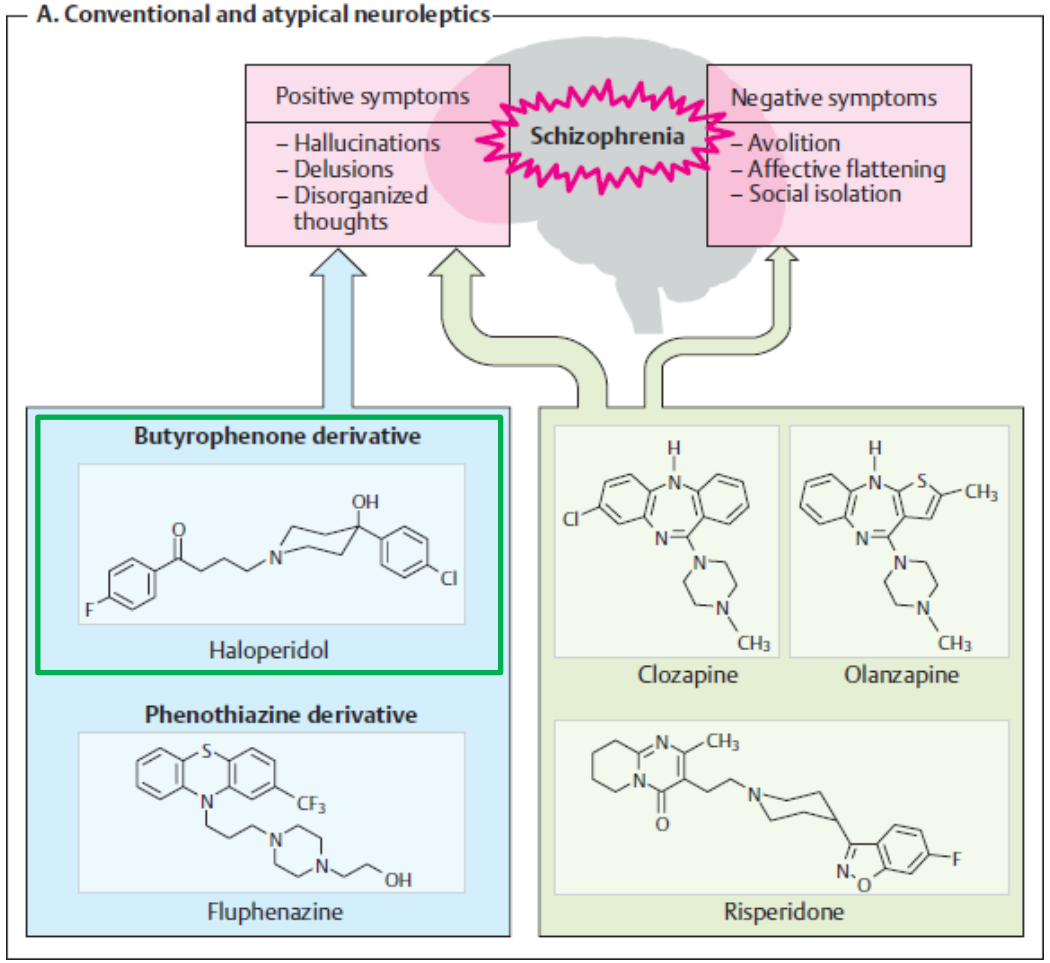
Schizophrenia Pathophysiology		Pharmacologic Profile of APDs	
Past			
Excess dopaminergic activity		Dopamine antagonists	D_2-receptor
Present			
Renewed interest in the role of serotonin (5-HT)		Combined antagonists	5-HT_2/D_2

Recall 5-HT2A antagonists. If we antagonize the 5-HT2A receptor, we produce antidepressant activity.

Rather than using exclusively D2 antagonists, the new protocol is to combine serotonin and dopamine receptor antagonists. Therefore, we antagonize the serotonergic activity (the feedback loop discussed previously) as well as antagonize the D2 receptor.

Based on the new and old theories, we made two types of drugs:

The typical type (old):
- Only control positive symptoms.
- Building on the dopamine theory.
- Only used in acute attacks of schizophrenia.



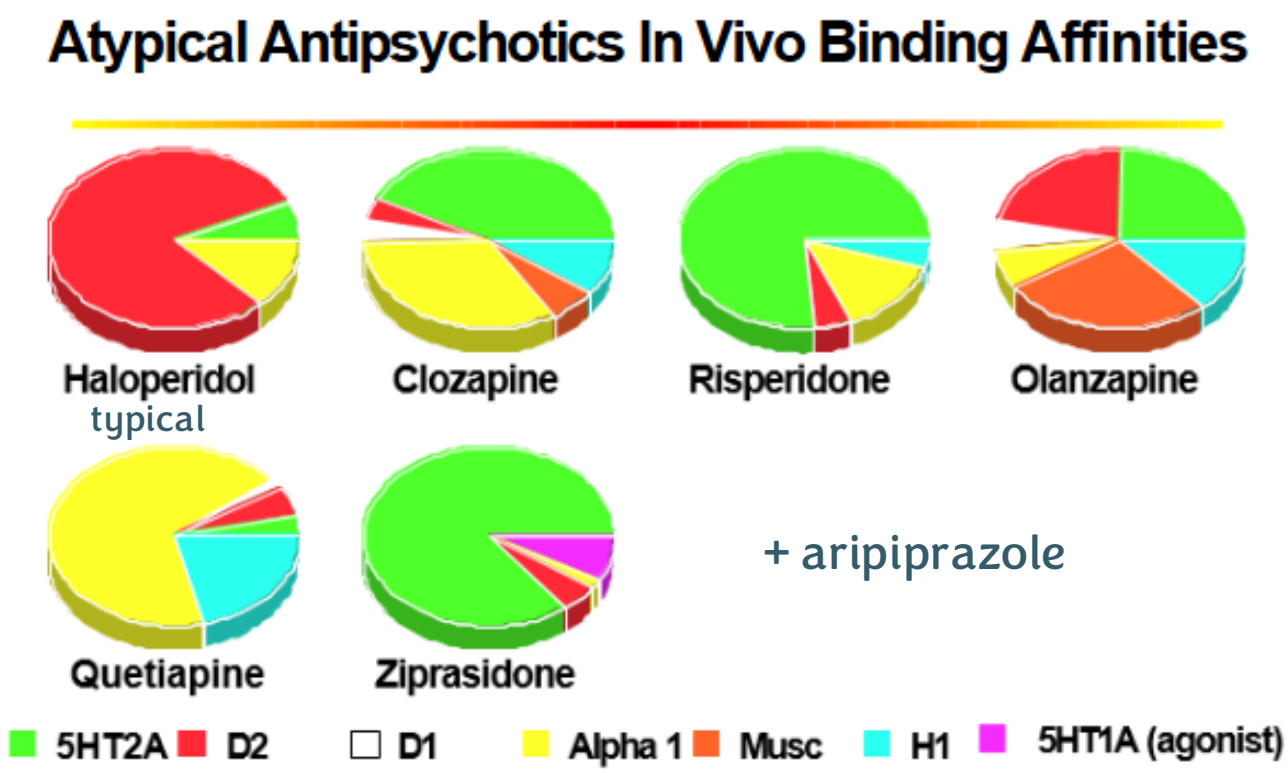
The atypical type (new):
- Have effects on both the negative and positive symptoms.
- Based on the theory that both dopamine and serotonin are involved.

Our success in managing the negative symptoms is still limited, so we can't alleviate the depression using those drugs, but it can help the patient slightly.

More on the atypical drugs in the next slide.

Haloperidol (the main old drug) – Think "Hello"peridol

- The main **old** drug you have to know is Haloperidol.
- Since it is an old drug (only blocks D2 receptors), it exerts all four effects we talked about in slide 9. We use it for its mesolimbic effect: **decreasing hallucinations.**
- It is used to control patients during **acute** attacks of **psychosis**, whether due to schizophrenia or drug-induced psychosis, when we are afraid that the patient will **harm** himself or others.
- It is given **one, two, or three** times in the hospital, and could be **injectable or oral.**
- **Important note:** In the event of an acute attack, we are only concerned with dampening the positive effects. This is not the time to try and control the negative symptoms, hence why we use a D2 receptor antagonist rather than an antagonist for both D2 and serotonin receptors (which is what the 'new' drugs do).
- Key takeaway: old drugs (including haloperidol) are used only in acute attacks of psychosis.



Haloperidol is a typical/old drug. Clozapine, risperidone, olanzapine, quetiapine, ziprasidone, and aripiprazole are all atypical/new drugs.

When comparing haloperidol to the new/atypical drugs, we notice that the D2 effect is preserved to some extent in each drug, but the affinities to the other types of brain receptors changes. This creates drugs with new side effects. Side effects depend on the receptor blocked:

- H1 :cause sleepiness.
- Muscarinic receptors: cause dry mouth, blurry vision, urinary retention and constipation.
- Anti-serotonin activity and its effects.

Clozapine

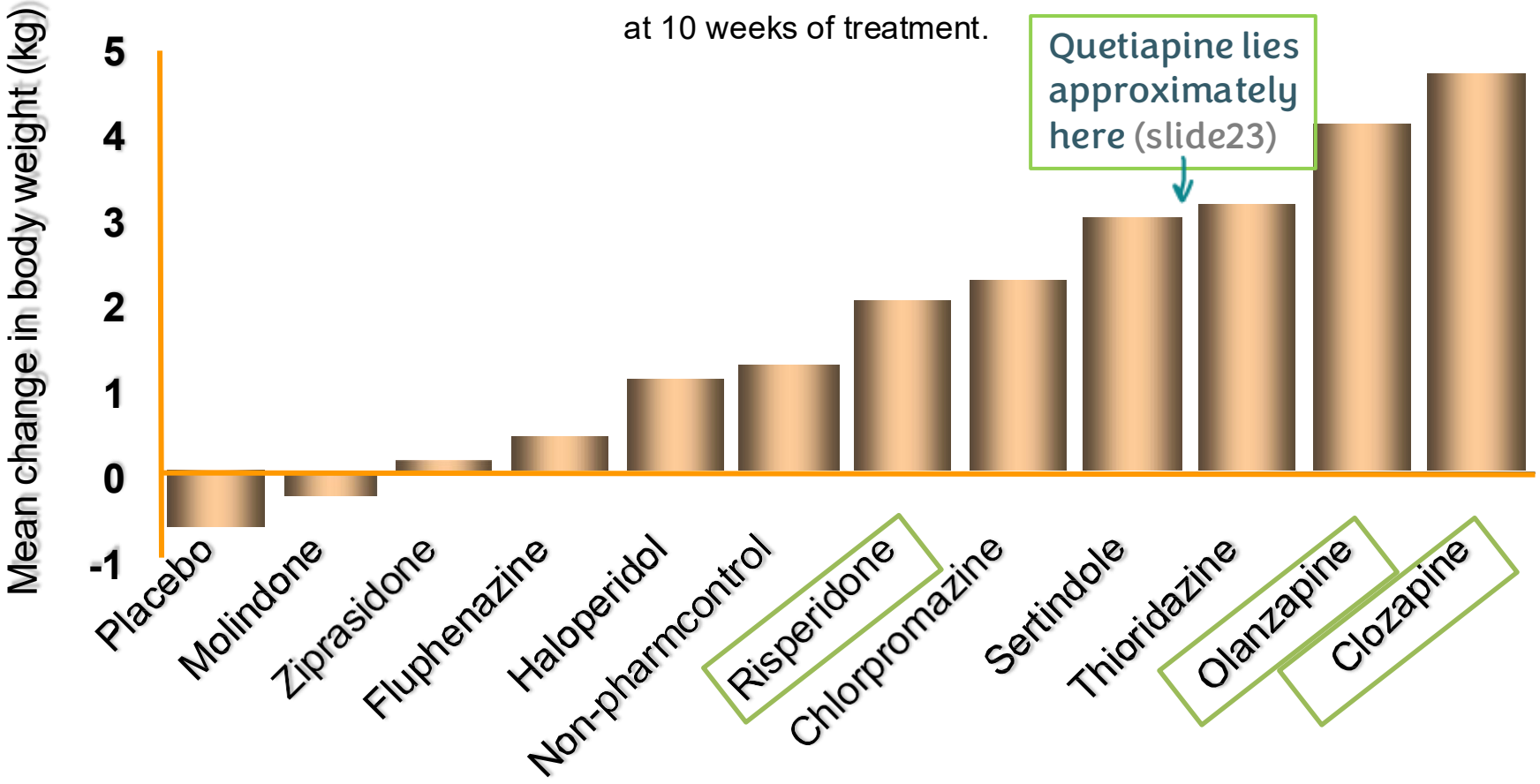
- binds 5HT2,alpha, D2, muscarinic, H1 receptors...etc..
- It is the best drug for negative symptoms, yet it is the last choice drug (discussed later).
- The problem is when it antagonizes 5HT2A it also antagonizes 5HT2C, leading to diabetes mellitus (due to imbalance in the serotonin-insulin axis), and weight gain (next slide).
- It also causes agranulocytosis (unknown mechanism) which can be fatal.

Risperidone

- The majority of its effect is concentrated on serotonin receptors, with some effect on alpha, D2, and H1 receptors. So it is largely based on the new theory.

ESTIMATED MEAN WEIGHT GAIN AT 10 WEEKS

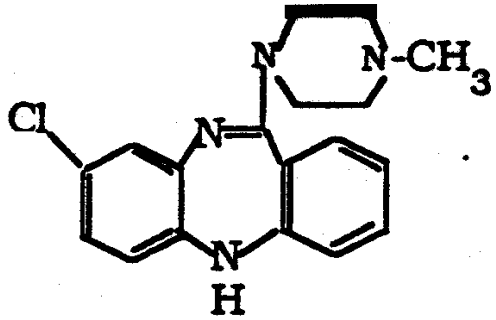
- A comprehensive literature search identified 78 studies that included data on weight change in patients treated with a specific antipsychotic.
- For each agent a meta-analysis and random effects regression estimated the change in weight at 10 weeks of treatment.

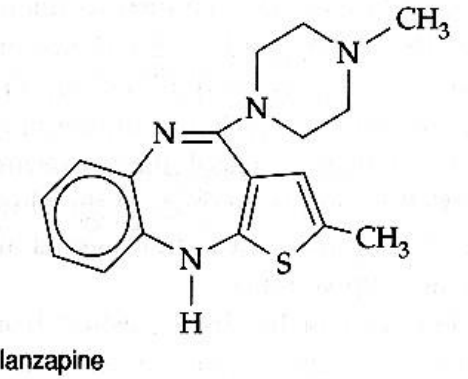


Clozapine increases weight the most, followed by olanzapine Respiridone causes some weight gain.

Allison DB, Mentore JL, Heo M, et al: Weight gain associated with conventional and newer antipsychotics: a meta Analysis. AJP, 1999.

Clozapine and olanzapine





Olanzapine

- VERY low EPS
- Blocks D1, D2, D4, α□adrenergic, 5HT2, muscarn ic, and histamine H1 receptors
- May show greater efficacy against negative symptoms than other antipsychotic drugs
- Agranulocytosis is a potentially **fatal** side effect for **clozapine**
- Both drugs have high efficacy, but cause significant weight gain and diabetes

Olanzapine

- Similar to clozapine but not as strong
- Doesn't cause agranulocytosis
- Side effects related to sleepiness and metabolic syndrome → high weight gain due to 5HT2C antagonism.

Clozapine is the last resort due to agranulocytosis.

Takeaway from clozapine and olanzapine: clozapine is the last choice drug due to the risk of agranulocytosis despite being the best acting. Olanzapine is a decent choice, but it causes significantly more weight gain compared to the other drugs due to its action on 5-HT2C.

Risperidone

Endocrine effect

- One of the most prescribed drugs in Jordan.
- In **women**, these disturbances include:
  - **galactorrhea**
  - **loss of libido**
  - delayed ovulation and menstruation or amenorrhea.
- In **men**, these disturbances include:
  - **gynecomastia**
  - **impotence.**

- Causes some weight gain, but not as much as clozapine or olanzapine.
- Good for negative symptoms
- **Very** good for positive symptoms.
- It doesn't cause metabolic syndrome like the other drugs, and doesn't cause agranulocytosis
- It has a strong effect on D2 receptors, therefore:
  - It has strong endocrine effects, such as gynecomastia in men and amenorrhea in women, as well as loss of libido which schizophrenic people are already experiencing due to dopamine imbalances.
  - Worse yet, it exerts extrapyramidal side effects at high doses (remember the effects of D2 antagonism from slide 9). There are four types of extrapyramidal side effects (see next slide).

We recommend you read the whole table

Neurological Side Effects of antipsychotics

REACTION	FEATURES	TIME OF MAXIMAL RISK	PROPOSED MECHANISM	TREATMENT
Acute dystonia	Spasm of muscles of tongue, face, neck, back; may mimic seizures; *not* hysteria	1 to 5 days	Unknown	Antiparkinsonian agents are diagnostic and curative
Akathisia	Motor restlessness; *not* anxiety or "agitation"	5 to 60 days	Unknown	Reduce dose or change drug: antiparkinsonian agents,b benzodiazepines or propranololc may help
Parkinsonism	Bradykinesia, rigidity, variable tremor, mask facies, shuffling gait	5 to 30 days	Antagonism of dopamine	Antiparkinsonian agents helpful
Tardive dyskinesia	Oral-facial dyskinesia; widespread choreoathetosis or dystonia	After months or years of treatment (worse on withdrawal)	Excess function of dopamine hypothesized	Prevention crucial; treatment unsatisfactory

Check the next slide

Extrapyramidal Side Effects (EPS)

- Extrapyramidal side effects are motor disturbances that result from **dopamine D2 receptor blockade**, particularly in the **nigrostriatal pathway**.

- They were **first recognized** with the **long-term** use of **high-potency first-generation antipsychotics** such as **haloperidol** (haloperidol main side effect), which strongly antagonize D2 receptors. However, in current practice, haloperidol is often used **short-term (e.g., in acute psychosis or agitation)**, and brief use (such as one or two injections) is **unlikely to produce significant EPS**.

- Despite this, EPS can still occur with **second-generation antipsychotics**, especially **risperidone at high doses**, due to its **moderate (so-called "half") D2 receptor antagonism**. Therefore, it is important to **monitor patients closely for early signs of EPS**, particularly during dose escalation or prolonged therapy.

Extrapyramidal Side Effects (EPS)

Extrapyramidal side effects are classified into four main types: **acute dystonia, akathisia, parkinsonism, and tardive dyskinesia**, with tardive dyskinesia being the most serious and potentially irreversible form.

1) **Acute dystonia** presents as painful muscle spasms involving the tongue, face, and neck, and in severe cases may mimic seizures. It typically occurs early, within **1–5 days** of starting treatment and is managed with **antiparkinsonian agents**.
2) **Akathisia** is characterized by a subjective feeling of inner restlessness, where the patient is unable to sit still and may appear anxious. It usually develops within the **first few days to weeks** of treatment. Management includes **dose reduction** or the use of **antiparkinsonian agents**.
3) **Drug-induced parkinsonism** (day 5–30) resembles Parkinson's disease, with symptoms such as bradykinesia, rigidity, and tremor, resulting from dopamine blockade in the nigrostriatal pathway.

It is important to note that patients do not necessarily develop all these forms; they may experience **one, more than one, or none**.

Extrapyramidal Side Effects (EPS)

Antiparkinsonian agents:

- **All the 3 previous types can be treated similarly with antiparkinsonian agents**

Parkinsonian symptoms in general can be treated by **two approaches**:
1. **Dopaminergic approach** → increasing dopamine (e.g., levodopa + carbidopa)
2. **Anticholinergic approach** → decreasing acetylcholine activity

However, in **EPS (drug-induced parkinsonism)**, we **avoid the dopaminergic approach** because increasing dopamine can **worsen psychosis**. Therefore, treatment relies mainly on the **anticholinergic approach**, which restores the dopamine–acetylcholine balance.

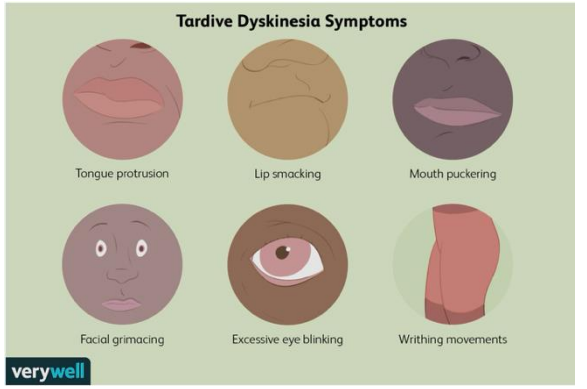
Tardive Dyskinesia (TD)

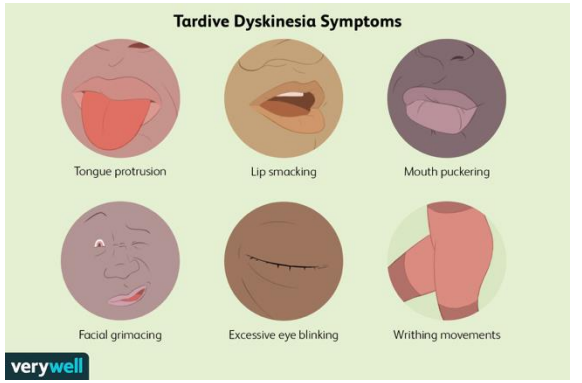
- Tardive dyskinesia is a **late-onset extrapyramidal side effect** caused by **chronic D2 receptor blockade**, usually appearing after **months to years of antipsychotic use**. It is characterized by **involuntary, repetitive, and purposeless movements**, primarily involving the **face and mouth** (lip smacking, tongue protrusion, chewing movements, grimacing), but movements of the **limbs and trunk** can also occur. The main problem with tardive dyskinesia is that it is **irreversible**.

- The underlying mechanism is **dopamine receptor supersensitivity**. With chronic D2 blockade, the brain compensates by **upregulating D2 receptors**, increasing both their number and sensitivity. While the drug is being taken, these new receptors are largely blocked or masked, making the adaptation **"invisible"**. Over time, normal dopamine binds to these hypersensitive receptors, producing **exaggerated involuntary movements**, which manifest as TD.

- This mechanism is similar to what happens with **chronic beta-blocker use**, where β-receptors are upregulated. While the beta-blocker is present, the effect is hidden, but abrupt withdrawal allows normal catecholamines to act on the newly upregulated receptors, causing **tachycardia, hypertension, and other exaggerated responses**. This is why **gradual tapering** is essential. Unlike opioid **agonists**, where **tolerance develops through receptor downregulation**, the effect of D2 **antagonists** occurs due to receptor **upregulation**, and increased dopamine production.
- However, some sources says that there is no increase in dopamine production.

Tardive Dyskinesia (TD)





Clinical points:

· TD is the **most dangerous EPS** because it can be **irreversible.**

· Unlike other EPS, TD may **persist even after stopping the drug.**

· It is more common with **long-term use and high-potency typical antipsychotics** (e.g., haloperidol ~10%), but can also occur with **high (and even low) doses of the atypical antipsychotic** risperidone (~1–2%) and, less frequently, with other atypical drugs.

· **Never stop the drug abruptly**, as symptoms can worsen; **tapering is key.**

Management:

- TD is managed by long term tapering of risperidone to manage the symptoms and to comfort the patient, starting with **4 mg** of risperidone for 2 months, then 3mg for the second 2 months then 2 mg for few months **until we reach the dose of 1 mg daily as a maintenance dose**, and the dose **increases** as needed when psychotic **episodes recur**. (We **don't** co-administer antiparkinsonian drugs in treating **TD**, however, in other EPS, we do)

Meet Jeff, living with TD

Second Generation Antipsychotic Drugs

Compound	Sedation	Hypo-tension	Motor effects
Risperidone	++	+++	+/++ Dose dependent
Clozapine	++	++	-
Aripiprazole	0/+	0/+	0/+

Most of the antipsychotics cause **sedation** and **hypotension** due to the **alpha 1 receptor** involvement

First Generation Antipsychotic Drugs

Compound	Seda-tion	Hypo-tension	Motor (EP) Effects
Phenothiazines			
Chlorpromazine	+++	++	++
Fluphenazine	+	+	++++
Haloperidol	+	+	++++

Quetiapine

- No increased risks for extrapyramidal symptoms
- Causes Diabetes Miletus but less frequently
- Shares sedation (has high sedation effect), orthostatic hypotension, weight gain (check slide 12)
- Does cause anticholinergic side effects– dry mouth, constipation
- **Does not elevate prolactin**

Ziprasidone - 2001

- Similar to advantages of others, but argued **not to cause weight gain**, however, its effectiveness is less than clozapine and olazipine.
- It is said to have a good therapeutic effect on the negative symptoms, but not as much as clozapine.
- We do not use it in Jordan.

Clozapine – 1.7 kg/month

Olanzipine – 2.3 kg/month

Quetiapine - 1.8 kg/month

Risperidone – 1 kg/month

Ziprasidone – 0.8 kg/month

Aripiprazole

- Our main concern in the previous drugs is that the positive symptoms are well controlled, in contrast to the negative ones, to solve this problem, the Japanese invented this drug.
- **Partial agonist at D2 receptor**

- Affinity for muscarinic, α_1-adrenergic, serotonin and histamine receptors (but not that strong of an affinity)
- Few extrapyramidal side effects

- **Weight gain** (mild)
- Doesn't cause DM

feeling dizzy (main side effect)

Aripiprazole

- Aripiprazole is a **partial agonist at D2 receptors**, meaning it **stabilizes dopamine activity** rather than fully blocking it. Because it is a **partial agonist**, its clinical effect may take time to fully develop, so careful initiation is important.

Oral and Long-Acting Injection (LAI) Use:

Aripiprazole can be administered as an **oral tablet formulation** or as a **long-acting injectable (LAI)**:

- Treatment is usually **started with the oral form first** to assess tolerability and minimize side effects such as dizziness.
- Once tolerated, the patient can be switched to an **injectable form** (monthly or every 3 months in extended-release (XR) formulations).
- When initiating the injection, it is **co-administered with the oral form for 14–21 days** to maintain therapeutic levels until the depot effect/loading dose is established.

Clinical Considerations

Long-acting injectable formulations are particularly useful in **patients with poor adherence**, as they ensure consistent drug delivery and reduce relapse risk. However, only the oral form is available in Jordan.
In some clinical settings, the **injection** may be presented to patients in a simplified or reassuring way (for example, described as a **"vitamin injection"** such as vitamin B12) to improve acceptance and adherence, especially in patients who are reluctant to take psychiatric medications due to stigma.
Similarly, risperidone is available also as an **oral solution** can be useful in patients with **stigma or poor cooperation**, as it may be mixed with a drink to facilitate administration when appropriate.

Dosage adjustments - interactions

Memorizing the doses is not required!

Check the next slide

	Adjusted Dose
CYP2D6 Poor Metabolizers	
CYP2D6 Poor Metabolizers	300 mg
CYP2D6 Poor Metabolizers taking concomitant CYP3A4 inhibitors	200 mg
Patients Taking 400 mg of ABILIFY MAINTENA	
Strong CYP2D6 or CYP3A4 inhibitors	300 mg
CYP2D6 and CYP3A4 inhibitors	200 mg
CYP3A4 inducers	Avoid use
Patients Taking 300 mg of ABILIFY MAINTENA	
Strong CYP2D6 or CYP3A4 inhibitors	200 mg
CYP2D6 and CYP3A4 inhibitors	160 mg
CYP3A4 inducers	Avoid use

Aripiprazole and Pharmacogenetics

Aripiprazole is metabolized by **CYP2D6 and CYP3A4**. Because it can be administered as a **long-acting injection that lasts for months**, it is essential to **strictly follow the pharmacogenetic dosing recommendations in the drug leaflet**.
According to these guidelines:

- **Poor CYP2D6 metabolizers** should receive a **reduced dose (300 mg instead of 400 mg)**
- If there is **combined impairment of CYP2D6 and CYP3A4**, the dose is further reduced to **around 200 mg**

Thus, **dosing depends on the patient's genetic makeup**, and adherence to the leaflet is crucial for safety and efficacy.

Clinical Note

These recommendations are particularly emphasized in populations where pharmacogenetic variability is well studied (e.g., Japan). Although the injectable form may not be available in our region (Jordan), the concept remains important.
Notably, around **13.5% of Jordanians are ultra-rapid CYP2D6 metabolizers**, meaning the drug may be **metabolized too quickly**, potentially reducing its effectiveness before reaching steady state, especially with long-acting formulations (codeine-like)

Tolerance and dependence to antipsychotic drugs

- Not addicting
- Physical dependence occurs
- Relapse in psychosis if discontinued abruptly, if tapered, psychosis recurs after couple of months.
- **Tolerance** develops to **sedative** effects (the doctor doesn't believe that this is true)
- **No tolerance** to **antipsychotic** effect
- Key takeaways for aripiprazole: dizziness is a major side effect but extrapyramidal effects, weight gain and diabetes mellitus are not, dosing is a lengthy and complicated process, it is a partial agonist rather than an antagonist, and the implications of being metabolized by different CYP450 enzymes.
- Important note: never stop any of the drugs in this lecture. Schizophrenic patients need these drugs for a lifetime. However, we can gradually reduce the dose until we get to a stable maintenance dose, increasing the dose again only when psychotic episodes recur. An example is the management plan for resperidone (slide 22).

Withdrawal-like syndrome

1. **Symptoms: nausea, vomiting, insomnia, and headache**
2. **Symptoms may persist for up to 2 weeks.**
3. **Symptoms can be minimized with a tapered reduction of drug dosage.**

Classification of Antipsychotic drugs

- Main categories are:
  - *Typical antipsychotics*

    Phenothiazines (**chlorpromazine**, **perphenazine**, **fluphenazine, thioridazine et al**) Thioxanthenes (**flupenthixol**, **clopenthixol**) Butyrophenones (**haloperidol**, **droperidol**)
  - *Atypical antipsychotics* (e.g. **clozapine**, **risperidone**, **sulpiride**, **olanzapine**)

Classification of Antipsychotic drugs

- Distinction between ‘typical’ and ‘atypical’ groups is not clearly defined, but rests on:
  - Incidence of extrapyramidal side-effects (less in ‘atypical’ group)
  - Efficacy in treatment-resistant group of patients
  - Efficacy against negative symptoms.

رسالة من الفريق العلمي

قَالَ رَسُولُ اللَّهِ صَلَّى اللَّهُ عَلَيْهِ وَسَلَّمَ: مَنْ كَانَتِ الآخِرَةُ هَمَّهُ جَعَلَ اللَّهُ غِنَاهُ فِي قَلْبِهِ وَجَمَعَ لَهُ شَمْلَهُ، وَأَتَتْهُ الدُّنْيَا وَهِيَ رَاغِمَةٌ، وَمَنْ كَانَتِ الدُّنْيَا هَمَّهُ جَعَلَ اللَّهُ فَقْرَهُ بَيْنَ عَيْنَيْهِ، وَفَرَّقَ عَلَيْهِ شَمْلَهُ، وَلَمْ يَأْتِهِ مِنَ الدُّنْيَا إِلَّا مَا قُدِّرَ لَهُ

«اللهم اغفر لحينا وميتنا، وصغيرنا وكبيرنا، وذكرنا وأنثانا، وشاهدنا وغائبنا»

PHARMACOLOGY QUIZ

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Corrections from previous versions:

Versions	Slide # and Place of Error	Before Correction	After Correction
V0 → V1	22	Management plan included co-administering antiparkinsonian agent for TD patients	Management plan in corrected
V1 → V2			