GPAT

2027-28

PHARMACOLOGY

CLASS- 9

TIME- 8 : 30 P.M

PHARMACODYNAMICS

UPDATED QUESTIONS TILL GPAT 2026

GPAT

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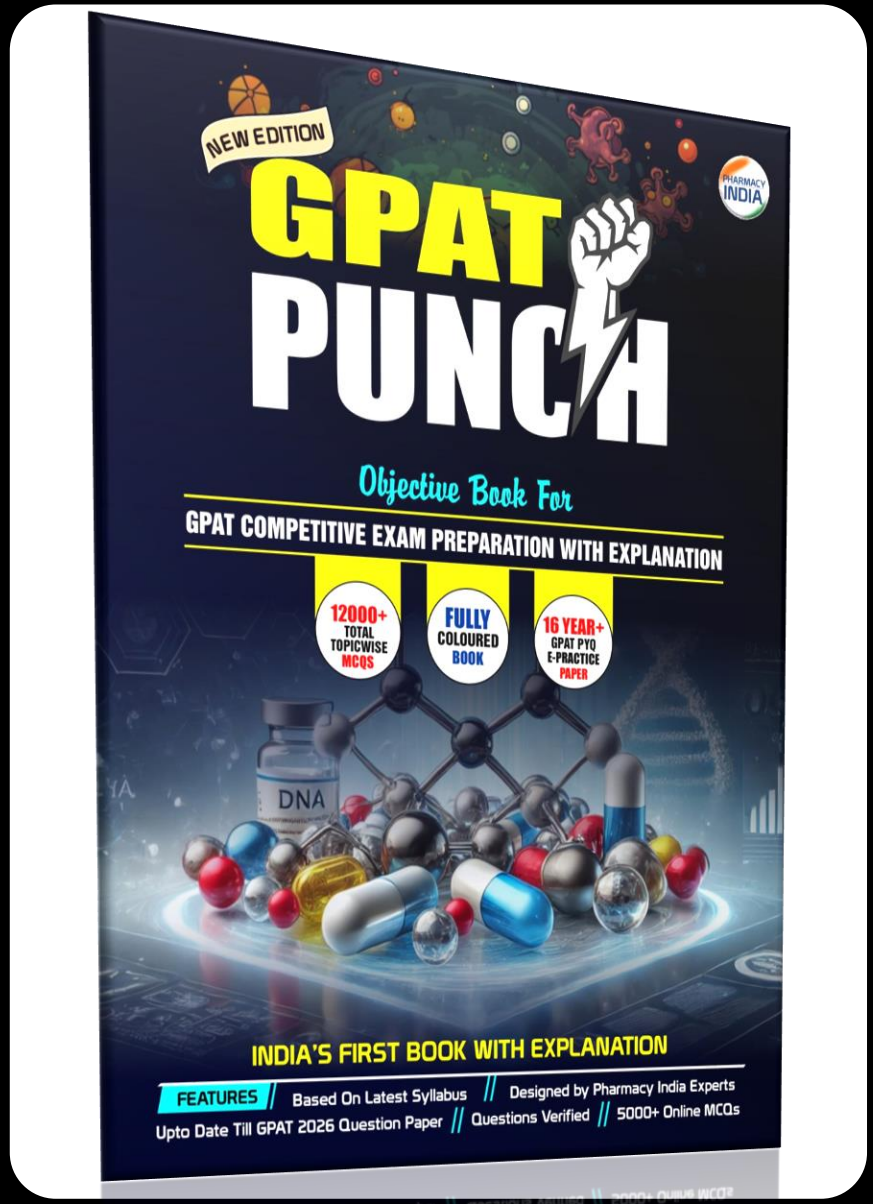


GPAT PUNCH OBJECTIVE BOOK

- Based on Latest Syllabus
- Up to Date Till GPAT 2026 Question Paper
- 5000+ Online Practice MCQs
- Total 12000+ MCQs
- 15 Year+ GPAT PYQ E-Practice Paper

DAILY UPDATES

जुड़िए PHARMACY INDIA

के साथ.....

WHATSAPP & TELEGRAM SE JUDNE KE LIYE ICONS PAR CLICK KARE

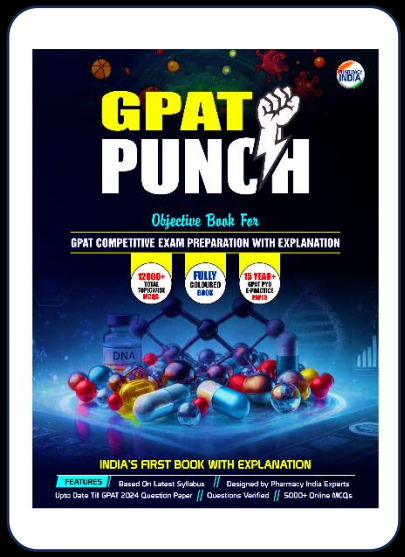
01. **G-proteins coupled with receptors are: [GPAT-2014, 2024]**

(a) Monomeric

(b) Dimeric

(c) Trimeric

(d) Tetrameric



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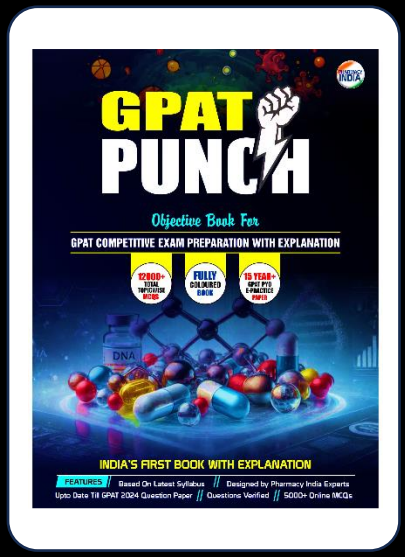
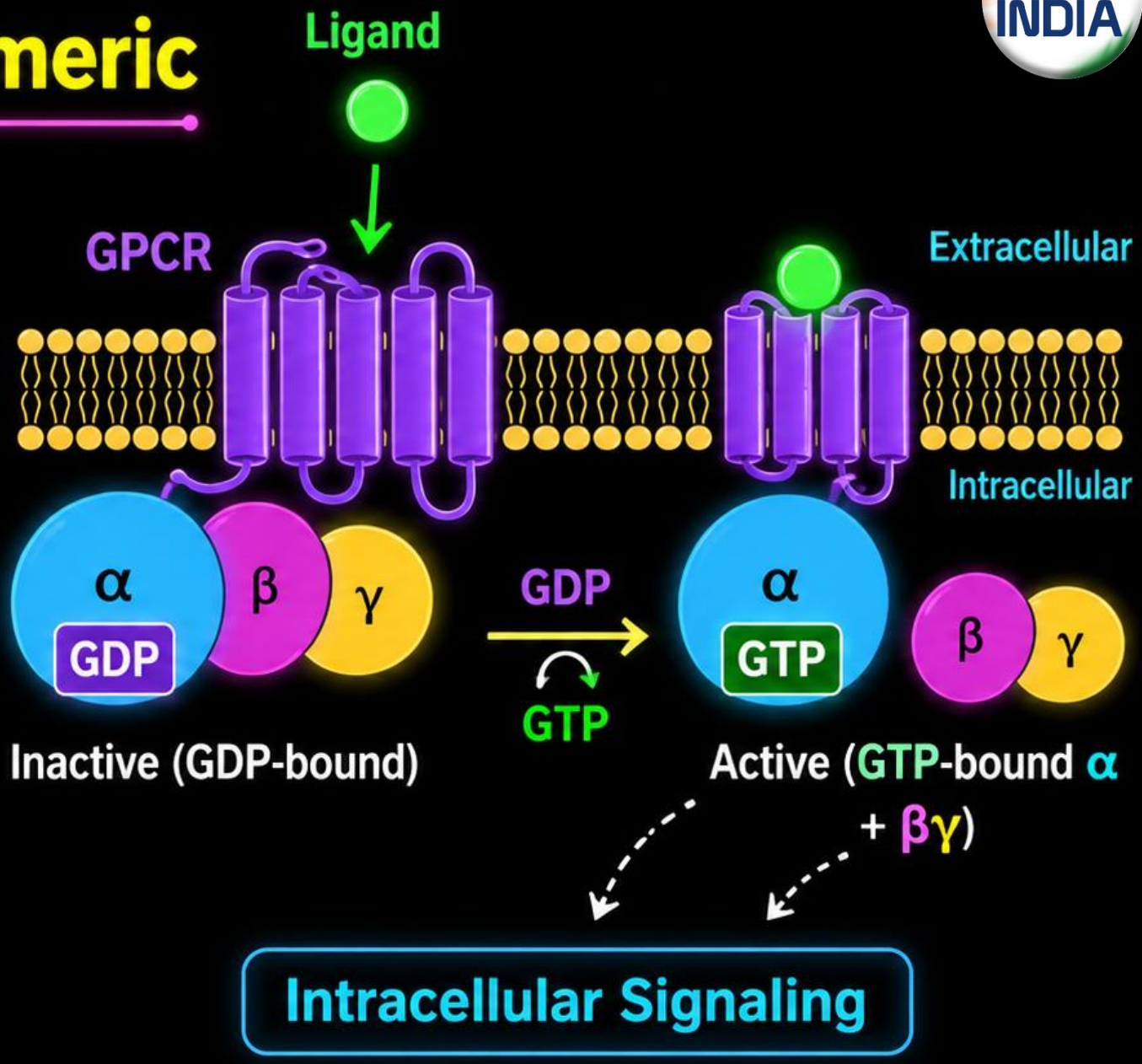


G-Proteins are Heterotrimeric

- Contain α, β, γ subunits.
- α subunit binds GDP/GTP.
- GPCR activation → GDP to GTP exchange on α.
- Subunits regulate intracellular signaling.

★ α has GDP/GTP binding; β and γ modulate and stabilize the complex.



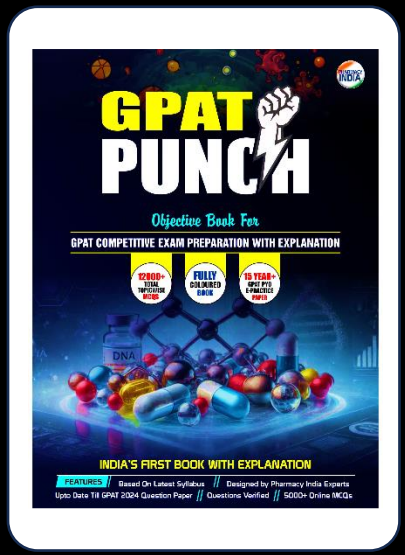
02. **Which of the following hyperlipidemia drugs acts via a GPCR? [GPAT-2023 SHIFT-I]**

(a) Nicotinic acid

(b) Fenofibrate

(c) Atorvastatin

(d) Ezetimibe



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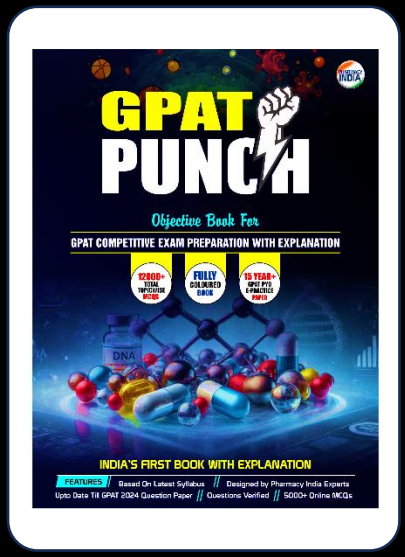
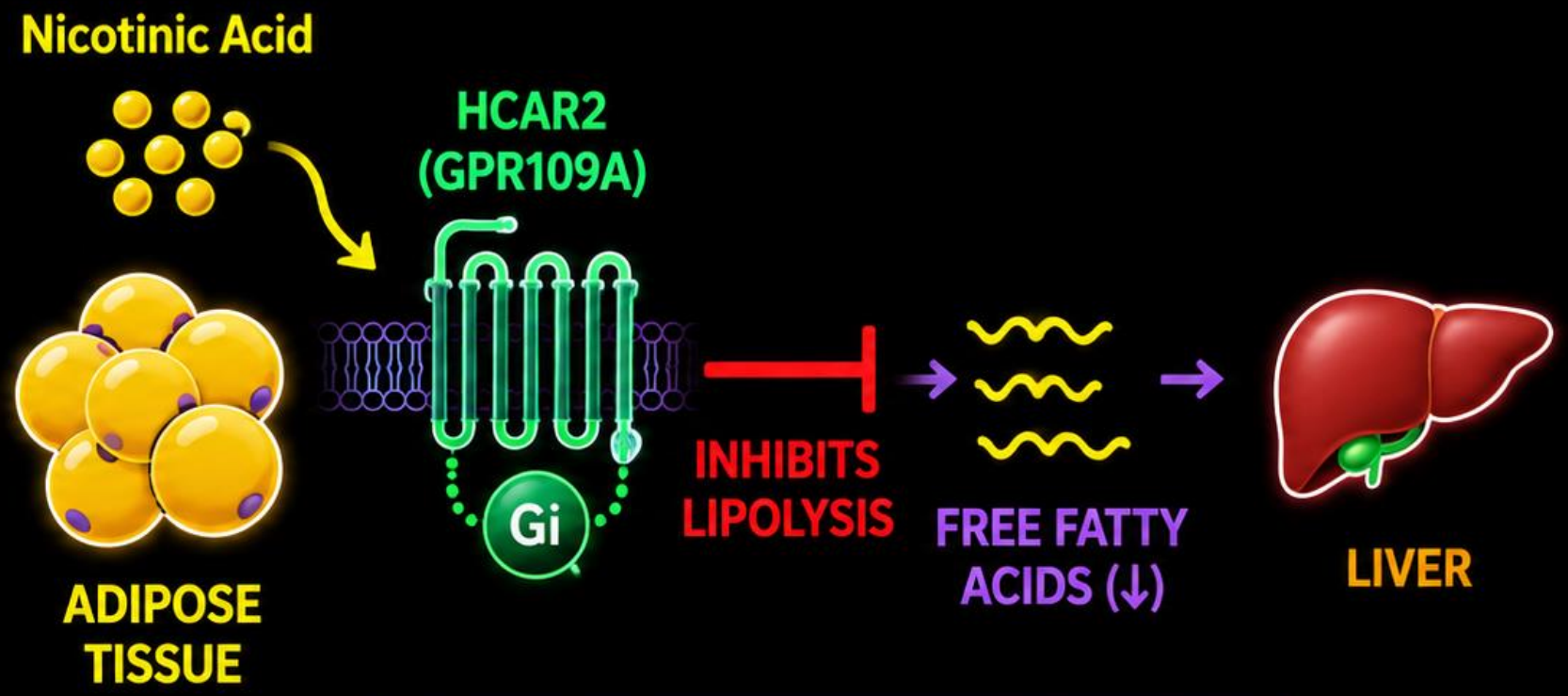


NICOTINIC ACID ACTS VIA GPCR

- Acts on HCAR2/GPR109A
- Inhibits lipolysis in adipose tissue
- Reduces free fatty acid delivery to liver
- Helps improve lipid profile



IMPROVES LIPID PROFILE
↓ LDL ↓ TG ↑ HDL

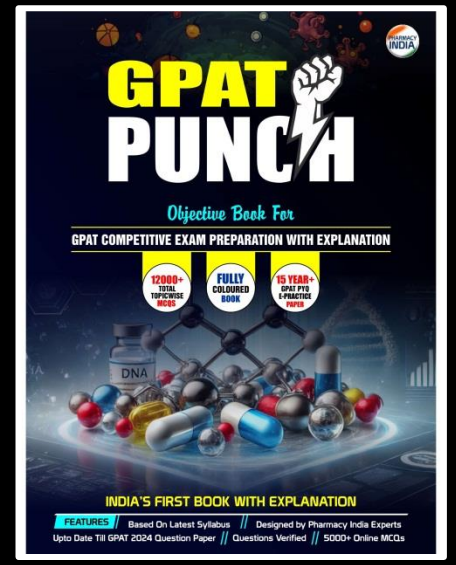
03. **Receptor for which no endogenous mediator or ligand is at present known is called: [GPAT-2015]**

(a) Silent receptors

(b) Spare receptors

(c) Orphan receptors

(d) False receptors



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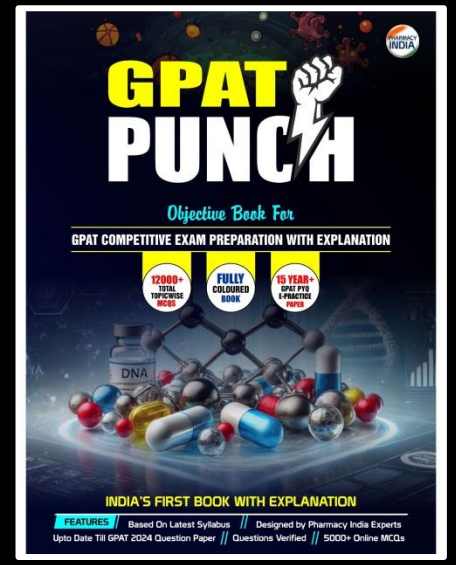
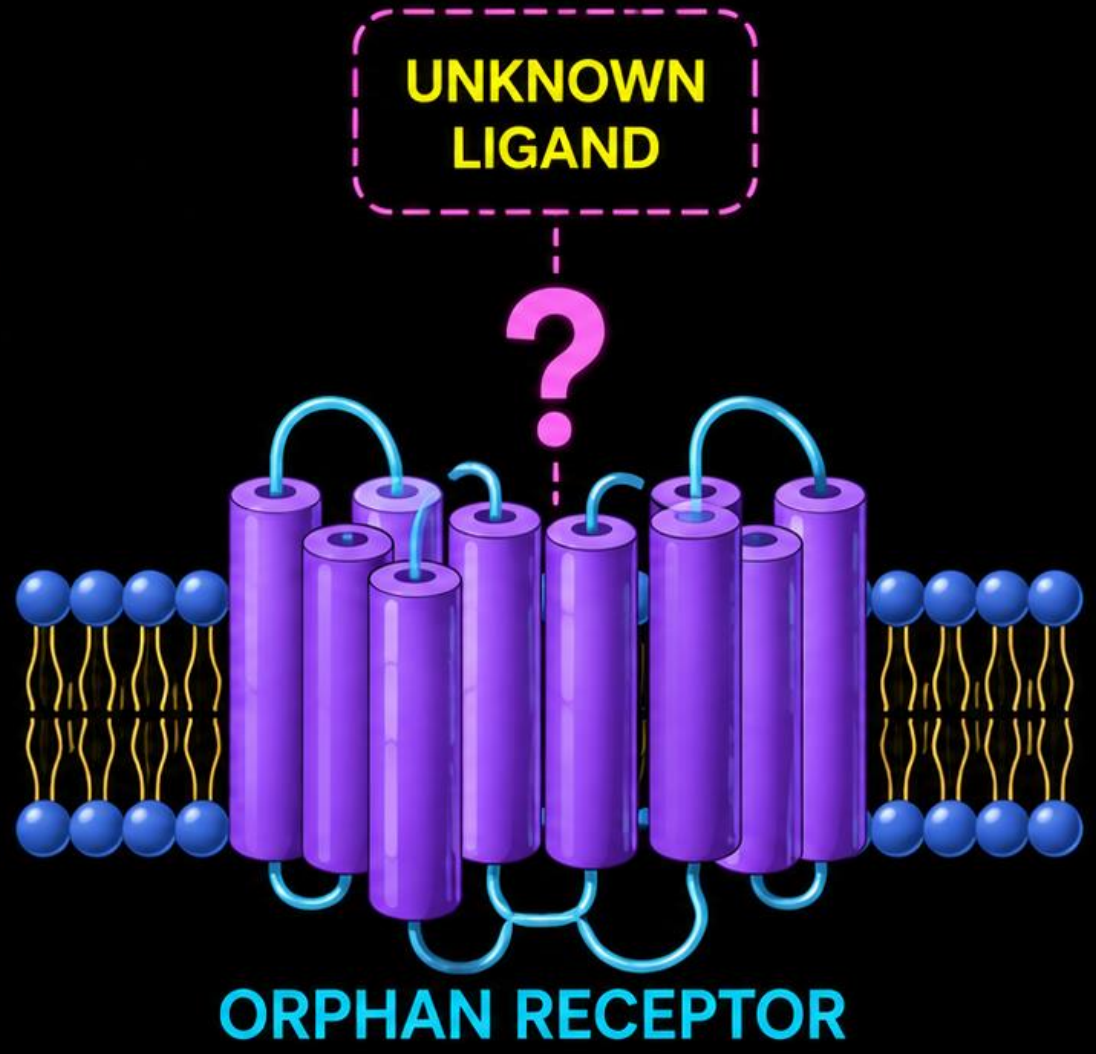


Orphan Receptor

- Receptor with no known endogenous ligand
- Identified by structure but ligand unknown
- Important targets in drug discovery
- When ligand is found, it becomes de-orphanized



→ Potential for novel therapeutic agents

04. A competitive antagonist is a substance that: [GPAT-2015]

[P] Interacts with receptors and produces submaximal effect

[Q] Binds to the same receptor site and progressively inhibits the agonist response

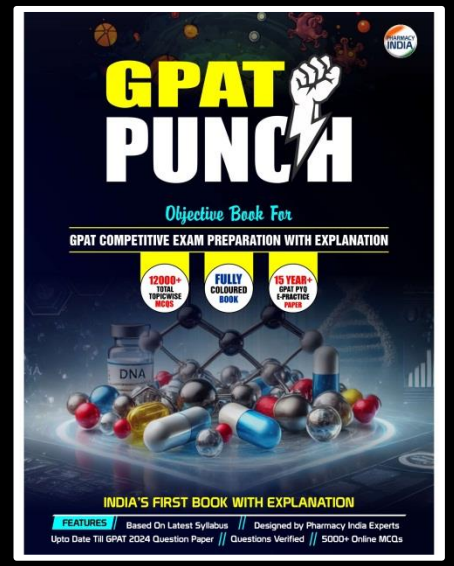
[R] Binds to the nonspecific sites of tissue

(a) [P] true, [Q] and [R] false

(b) [P] and [Q] true, [R] false

(c) [Q] true, [P] and [R] false

(d) [Q] and [R] true, [P] false



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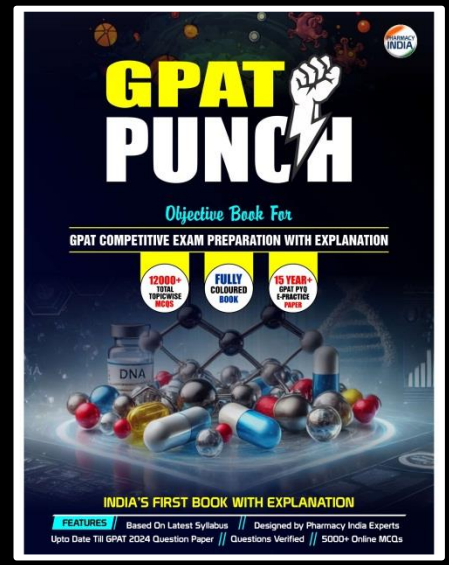
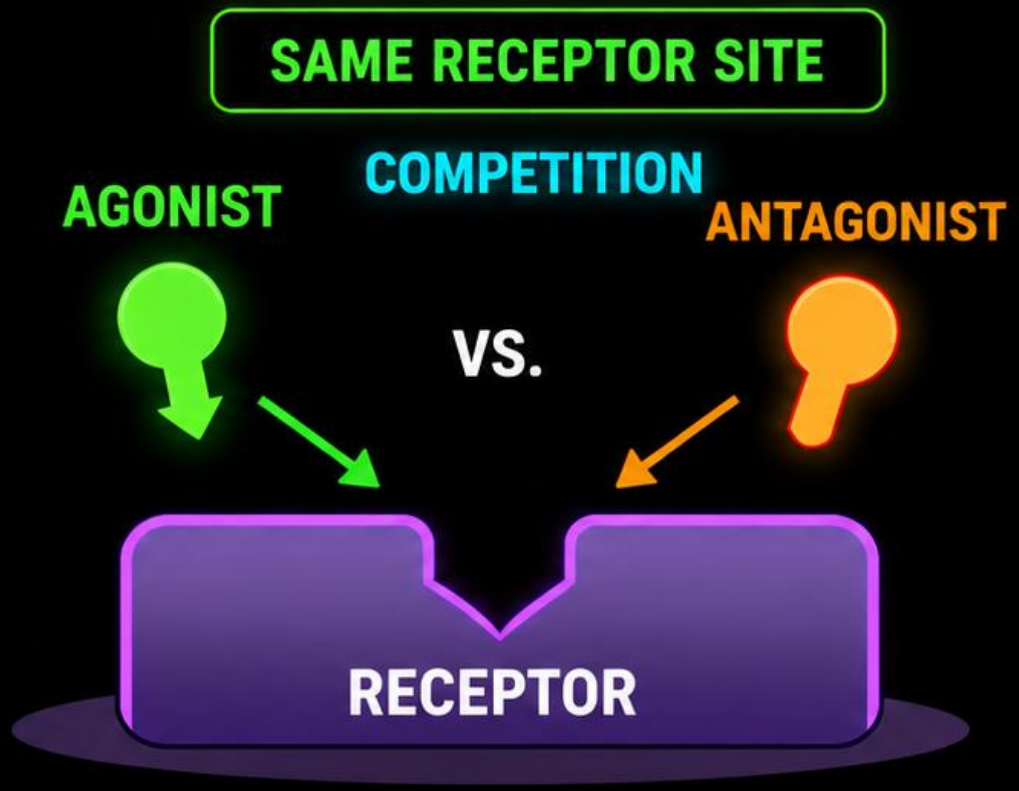
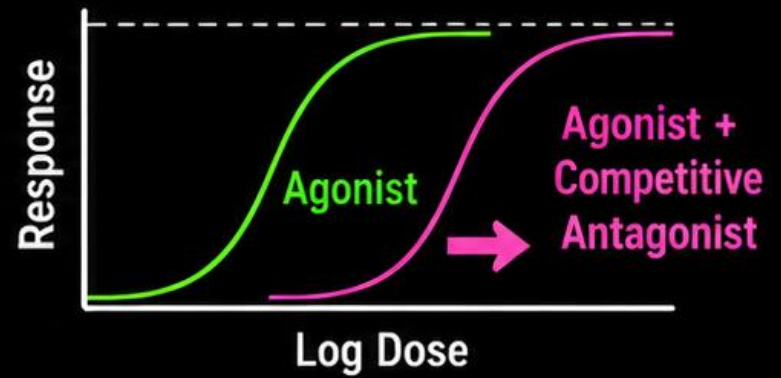


COMPETITIVE ANTAGONIST

- **BINDS** to the **SAME RECEPTOR SITE** as **AGONIST**
- HAS **AFFINITY** but **NO INTRINSIC ACTIVITY**
- **INHIBITS** AGONIST RESPONSE
- **SHIFTS DOSE–RESPONSE CURVE** to the **RIGHT** →



EFFECT: ↑ agonist dose needed to achieve same effect → **RIGHTWARD SHIFT (↓ POTENCY)**



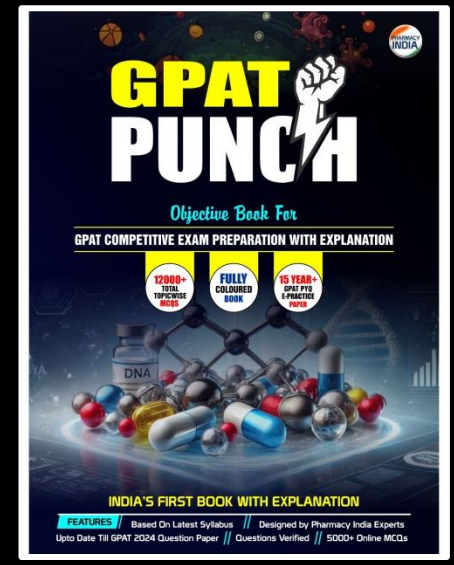
05. **The increase in concentration of second messengers such as cAMP, cGMP, Ca^{2+} etc. leads to: [GPAT-2013]**

(a) Inhibition of intracellular protein kinases and protein phosphorylation

(b) Protein kinase activation and protein phosphorylation

(c) Blocking of interaction between a receptor and an effector

(d) Antagonism with endogenous ligands



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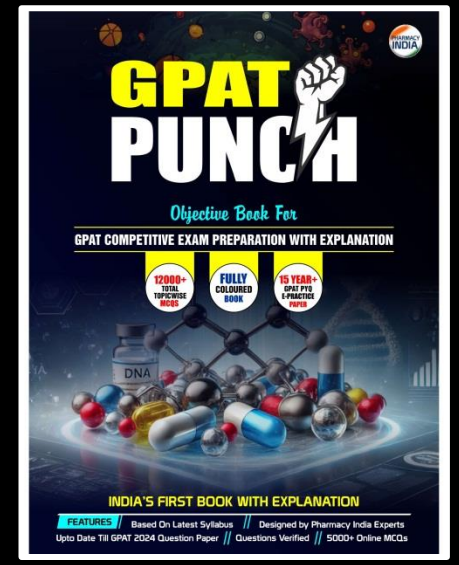
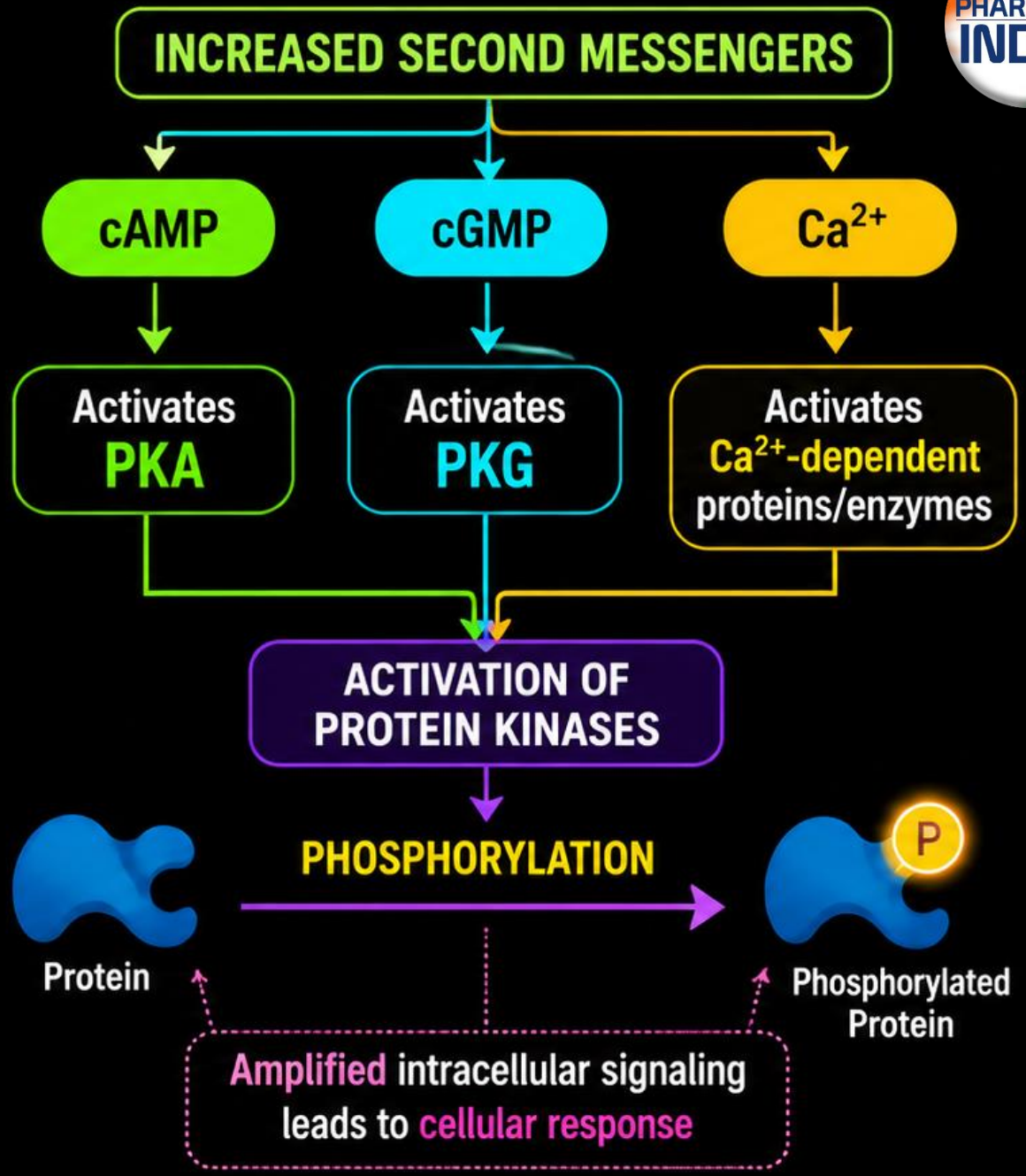


SECOND MESSENGERS ACTIVATE KINASES

- cAMP activates PKA
- cGMP activates PKG
- Ca^{2+} activates calcium-dependent proteins and enzymes
- Overall result: intracellular signaling amplification and protein phosphorylation



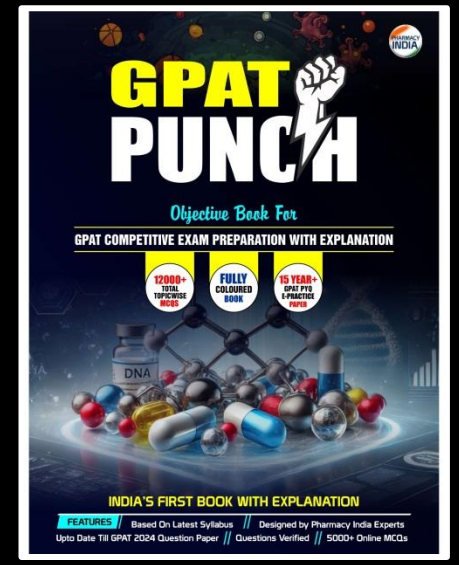
06. **Which one of the following is a false statement for competitive antagonists? [GPAT-2012]**

(a) They have affinity for the agonist binding site on receptor

(b) They have no intrinsic activity

(c) They cause parallel rightward shift of the control dose-response curve

(d) Maximum response of agonist cannot be achieved in their presence by increasing agonist concentration



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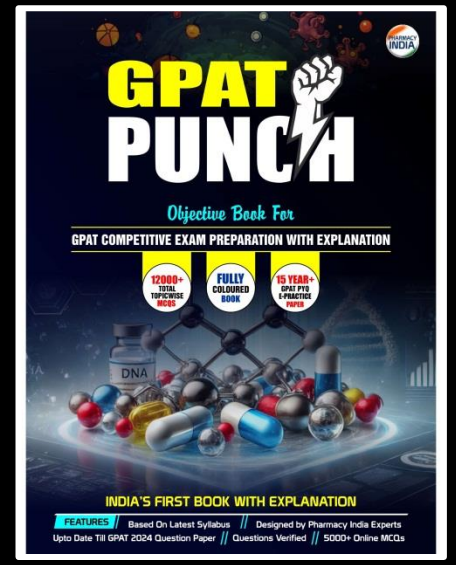
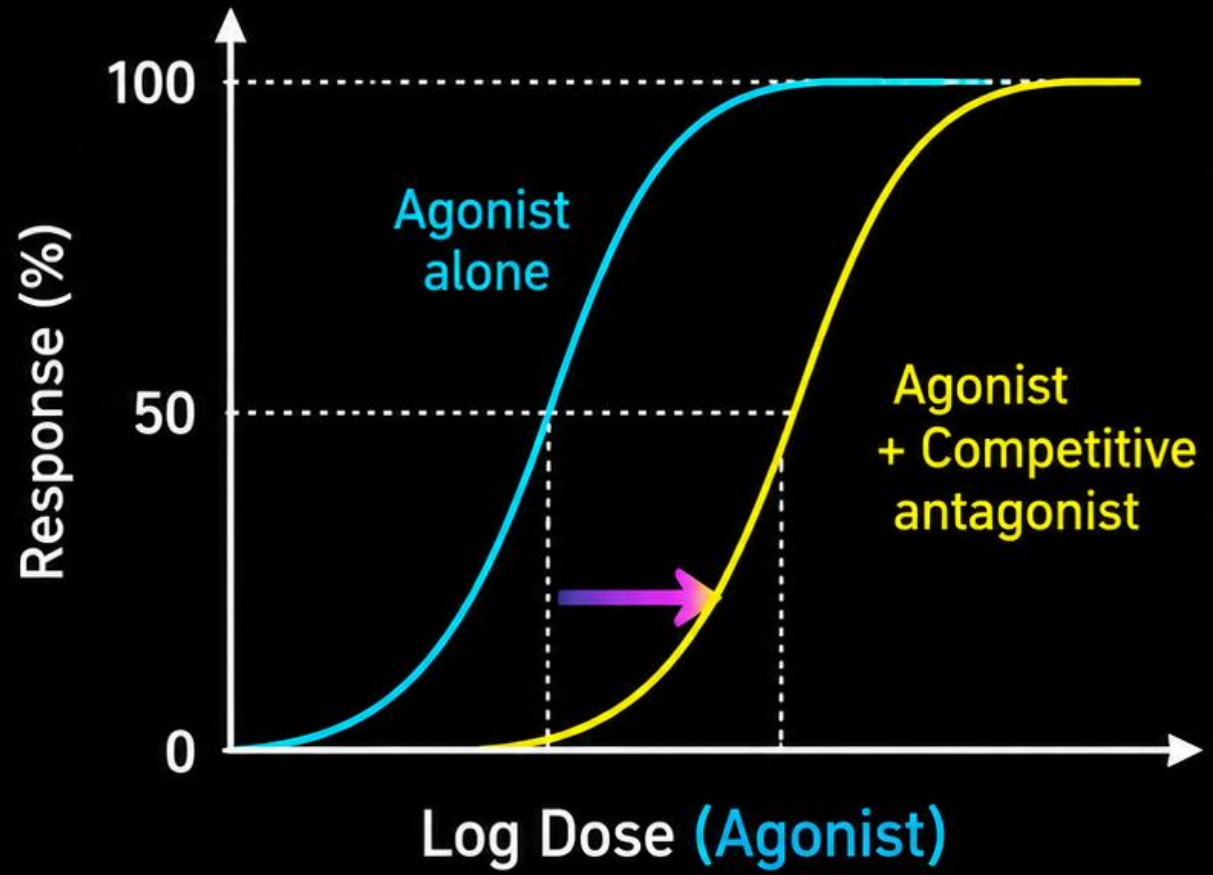


COMPETITIVE ANTAGONISM

PHARMACY INDIA

- Affinity for agonist binding site
- No intrinsic activity
- Causes parallel rightward shift of dose-response curve
- E_{max} remains unchanged because antagonism is surmountable



Higher agonist concentration is needed → same maximal effect

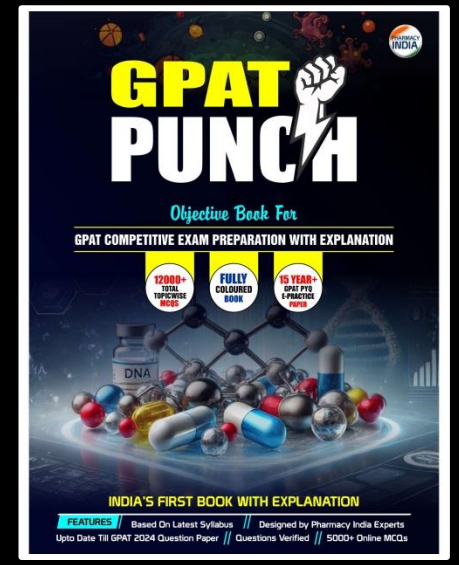
07. **Which one of the following receptors is NOT a ligand-gated ion channel receptor? [GPAT-2012]**

(a) Nicotinic receptor

(b) 5-HT receptor

(c) GABA-A receptor

(d) H2 receptor



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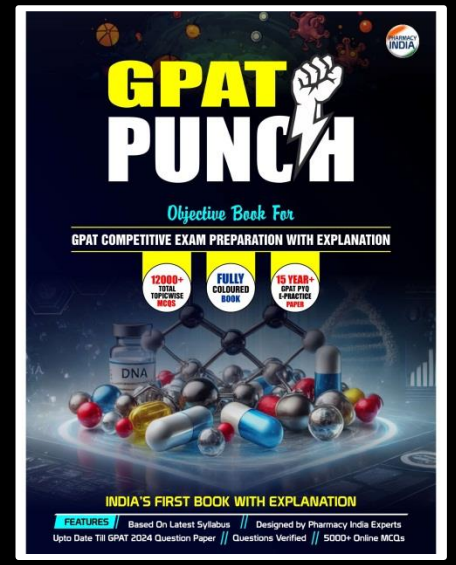
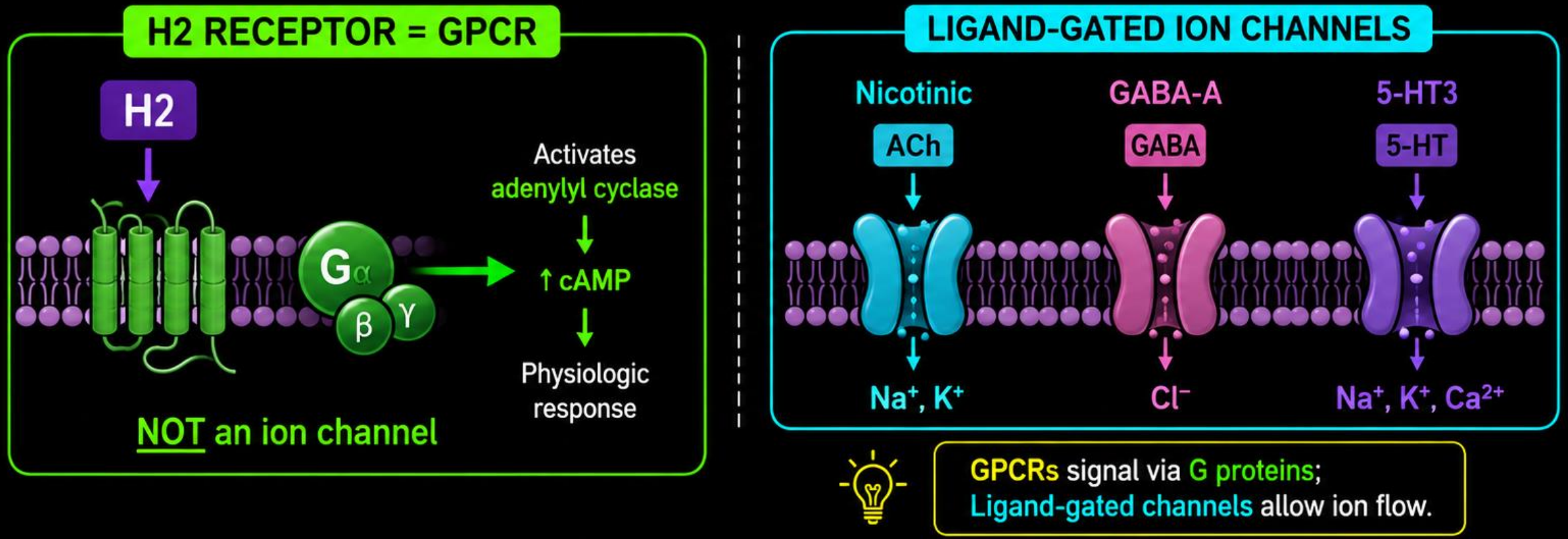


H2 Receptor = GPCR

- H2 receptor is G-protein coupled.
- Nicotinic receptor is ligand-gated ion channel.
- GABA-A is a chloride ion channel.
- 5-HT3 is also a ligand-gated ion channel.



GPCRs signal via G proteins;
Ligand-gated channels allow ion flow.

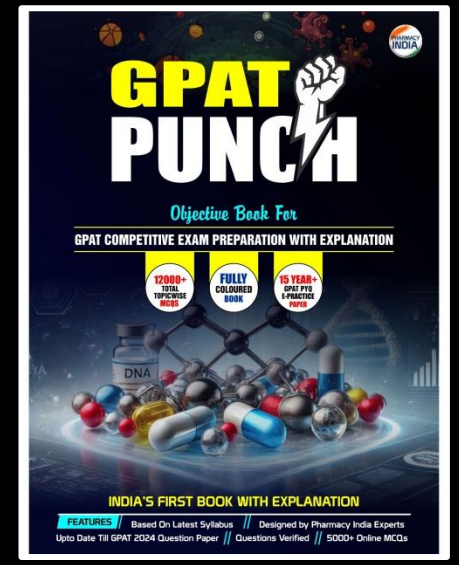
08. Which one of the following is NOT an example of G-protein coupled receptor? [GPAT-2011]

(a) Muscarinic cholinergic receptor

(b) Alpha adrenoceptor

(c) Nicotinic cholinergic receptor

(d) Beta adrenoceptor



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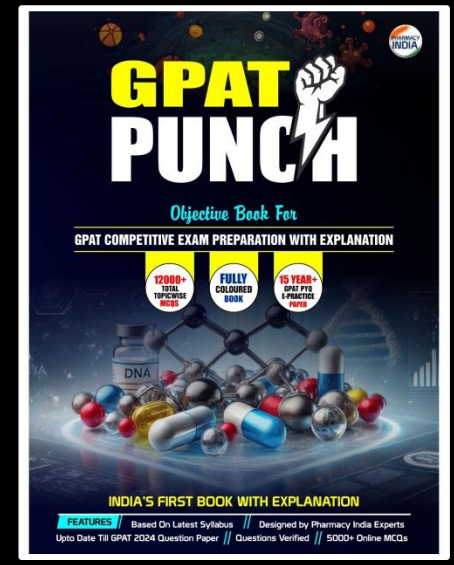
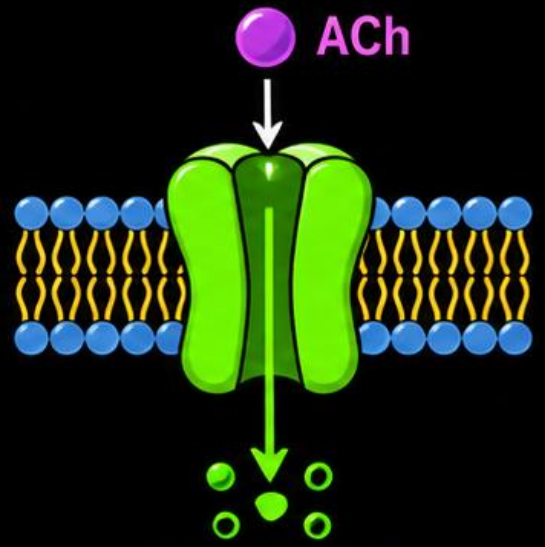


Nicotinic Receptor is an Ion Channel

- Nicotinic receptor is a ligand-gated ion channel.
- Muscarinic receptors are GPCRs.
- Alpha adrenoceptors are GPCRs.
- Beta adrenoceptors are GPCRs.

NICOTINIC RECEPTOR (ION CHANNEL)

Ligand-Gated Ion Channel

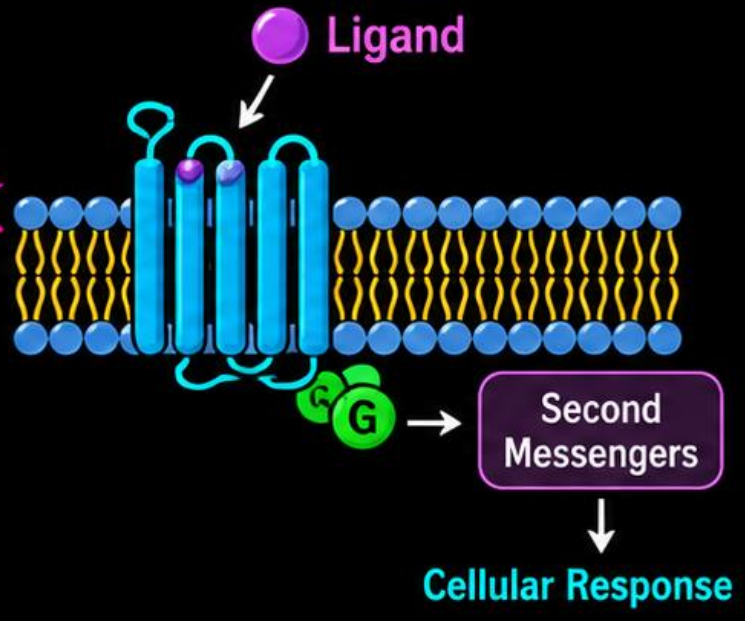


Direct ion flow → Fast response (ms)

VS

MUSCARINIC / ADRENOCEPTORS (GPCRs)

G Protein–Coupled Receptors



Indirect via second messengers → Slower response (s–min)

☆ Key Takeaway: Nicotinic = Ion Channel (Fast) | Muscarinic, α & β Adrenoceptors = GPCRs (Slower)

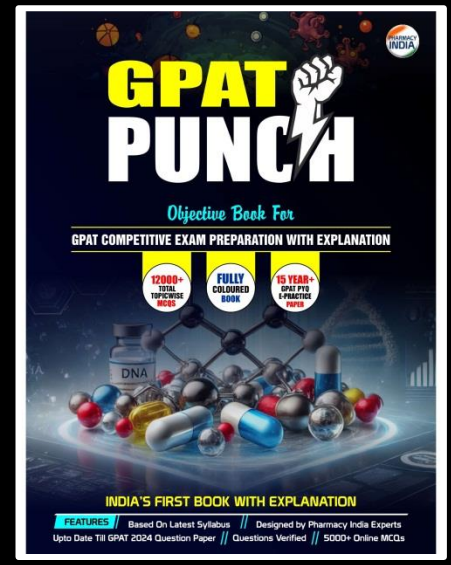
09. **Which one of the following drugs does NOT act through G-protein coupled receptors? [GPAT-2010]**

(a) Epinephrine

(b) Insulin

(c) Dopamine

(d) TSH



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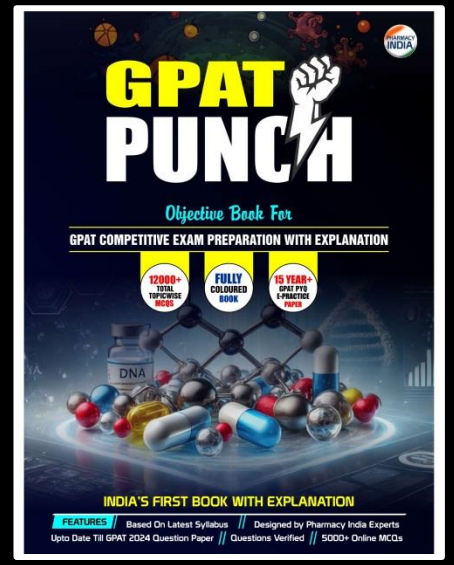
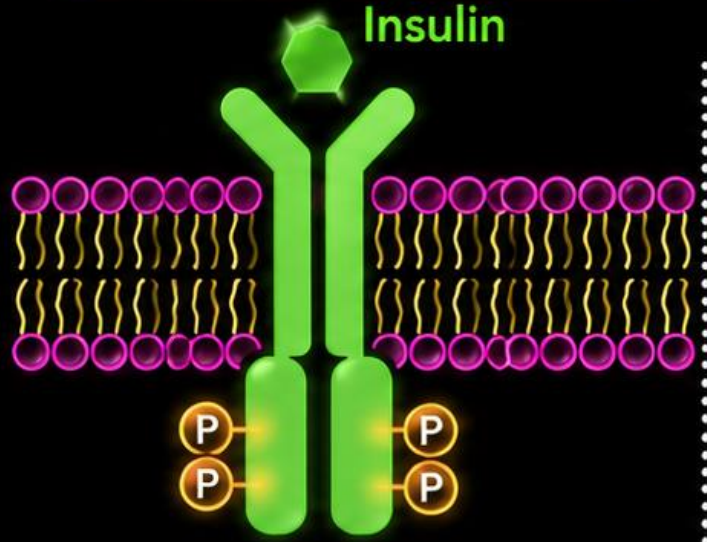


Insulin Uses a Tyrosine Kinase Receptor

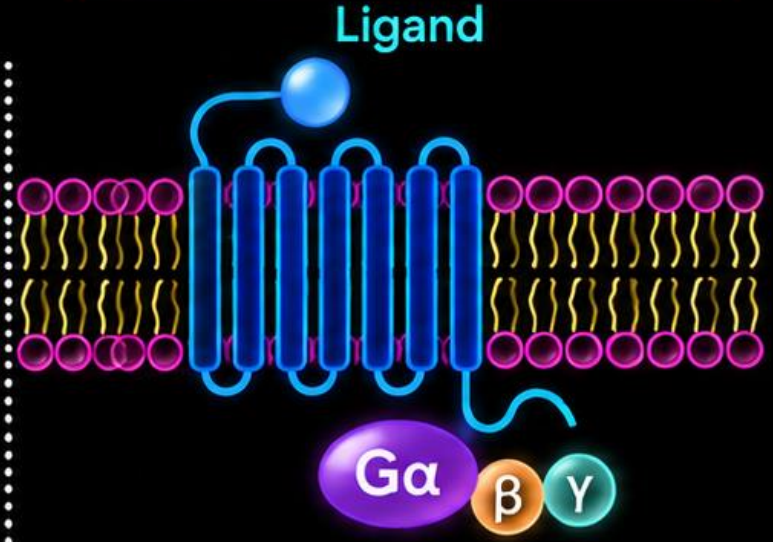
- **Insulin** acts through **receptor tyrosine kinase**.
- **Epinephrine** acts through **adrenergic GPCRs**.
- **Dopamine** acts through **dopamine GPCRs**.
- **TSH receptor** is **GPCR-linked**.

INSULIN RECEPTOR
(Tyrosine Kinase)



Autophosphorylation
↓
Tyrosine kinase signaling
(e.g., PI3K–Akt, MAPK)

GPCR EXAMPLES
(Epinephrine, Dopamine, TSH)



G protein activation
↓
Second messenger pathways
(e.g., cAMP, IP_3/DAG, Ca^{2+})

Key Point: Insulin ≠ GPCR → Insulin uses a **receptor tyrosine kinase**.

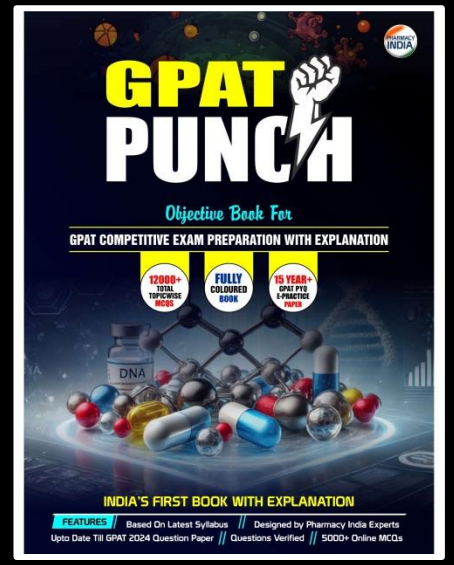
10. **Identify the receptor which demonstrates the fastest onset of response when stimulated. [GATE-2007]**

(a) Nuclear receptors

(b) Ionotropic receptors

(c) G-protein coupled receptors

(d) Insulin receptor



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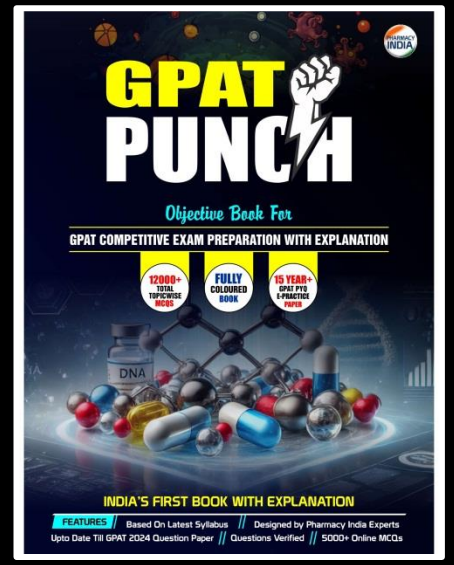
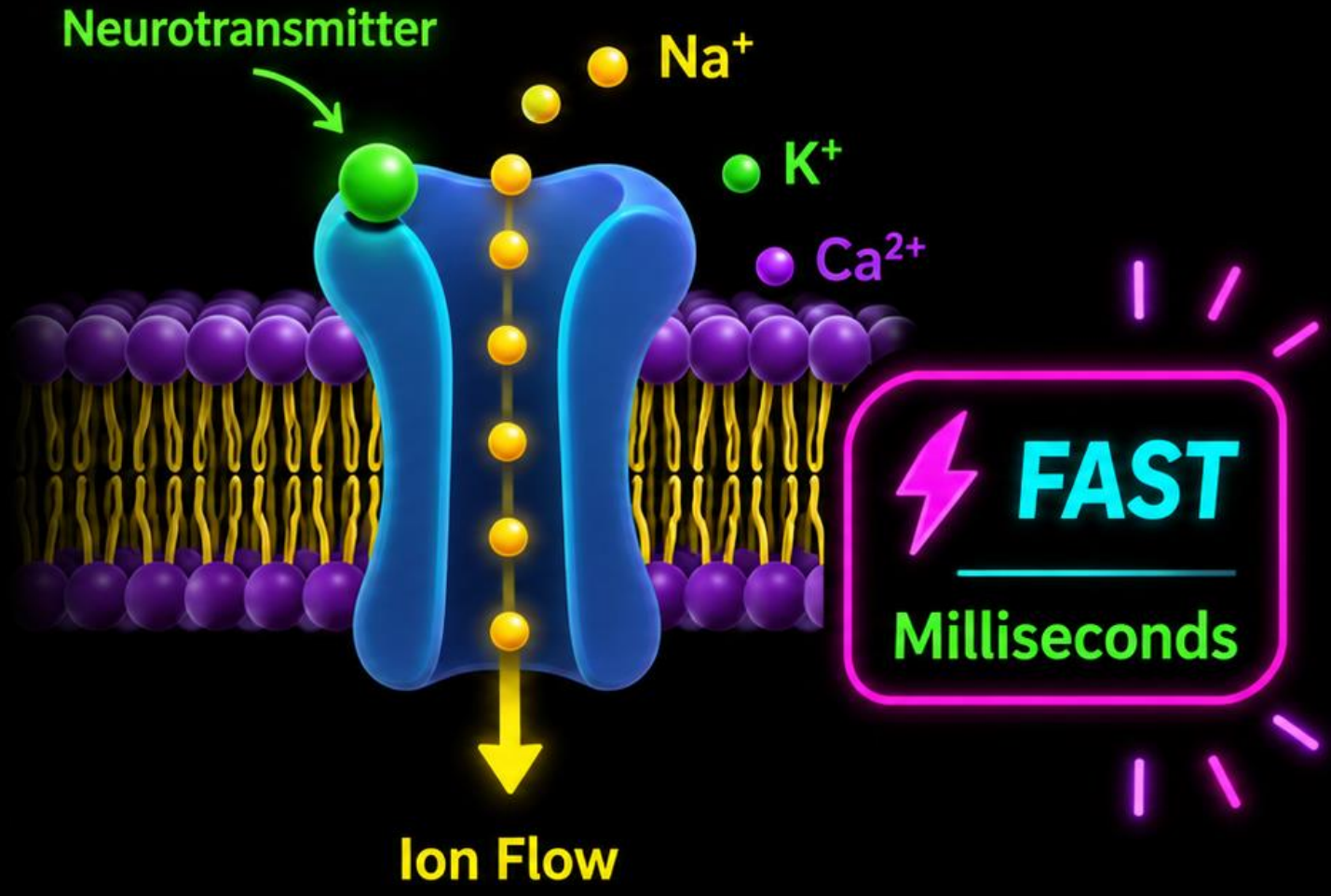


Ionotropic Receptors: Fastest Response

- They are **ligand-gated** ion channels.
- Response occurs within **milliseconds**.
- They allow **direct ion flow** across membrane.
- Nuclear receptors are **slower** because they alter gene expression.



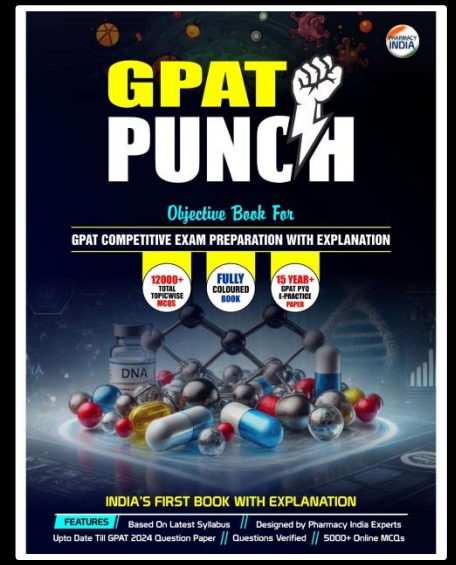
11. **Bioassays are carried out to: [GATE-2004]**

(a) Measure the pharmacological activity of a drug

(b) Avoid clinical trials for new drugs

(c) Detect impurity in a given drug

(d) Screen for pharmacogenetic influences of new drugs



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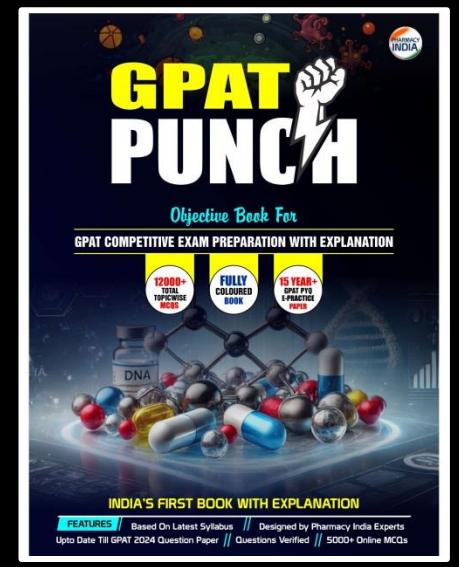
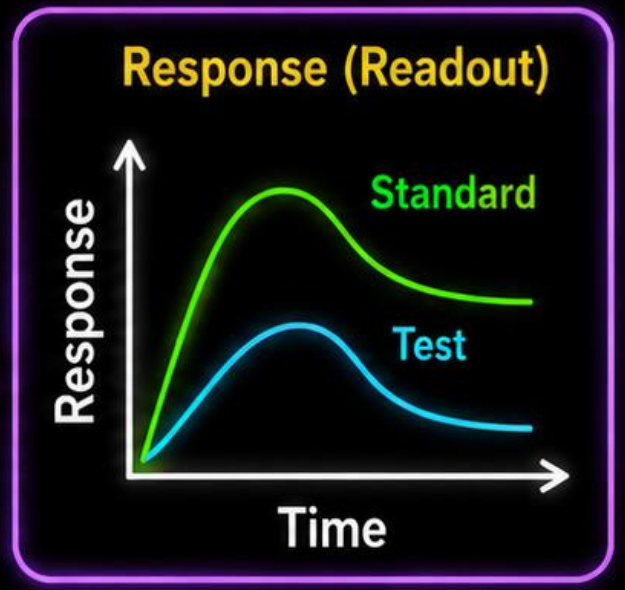


Bioassay Measures Pharmacological Activity

- Bioassay measures the biological activity of a drug or substance.
- It uses living tissues, cells, or whole organisms.
- It is useful when chemical assay alone is not sufficient.
- Response of a test sample is compared with a standard.



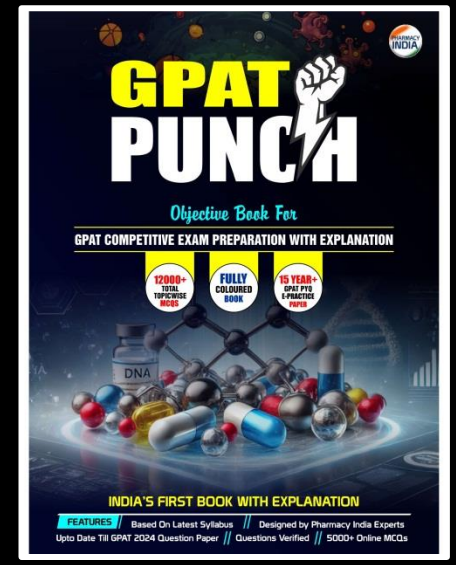
12. **One of the following statements for adenyl cyclase is wrong. Identify it. [GATE-1999]**

(a) It is a membrane-bound enzyme

(b) It is inactivated by phosphodiesterase

(c) It catalyzes cyclic AMP formation

(d) It is active only when associated with G-protein



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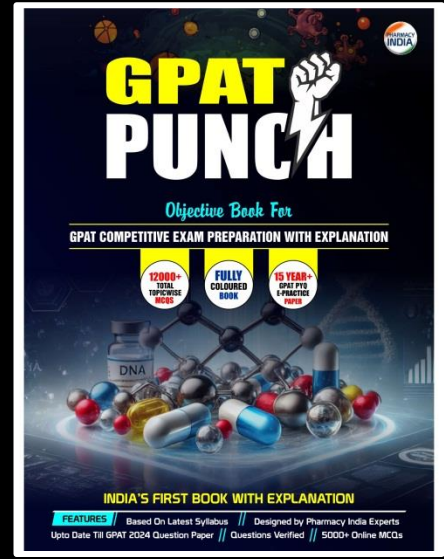
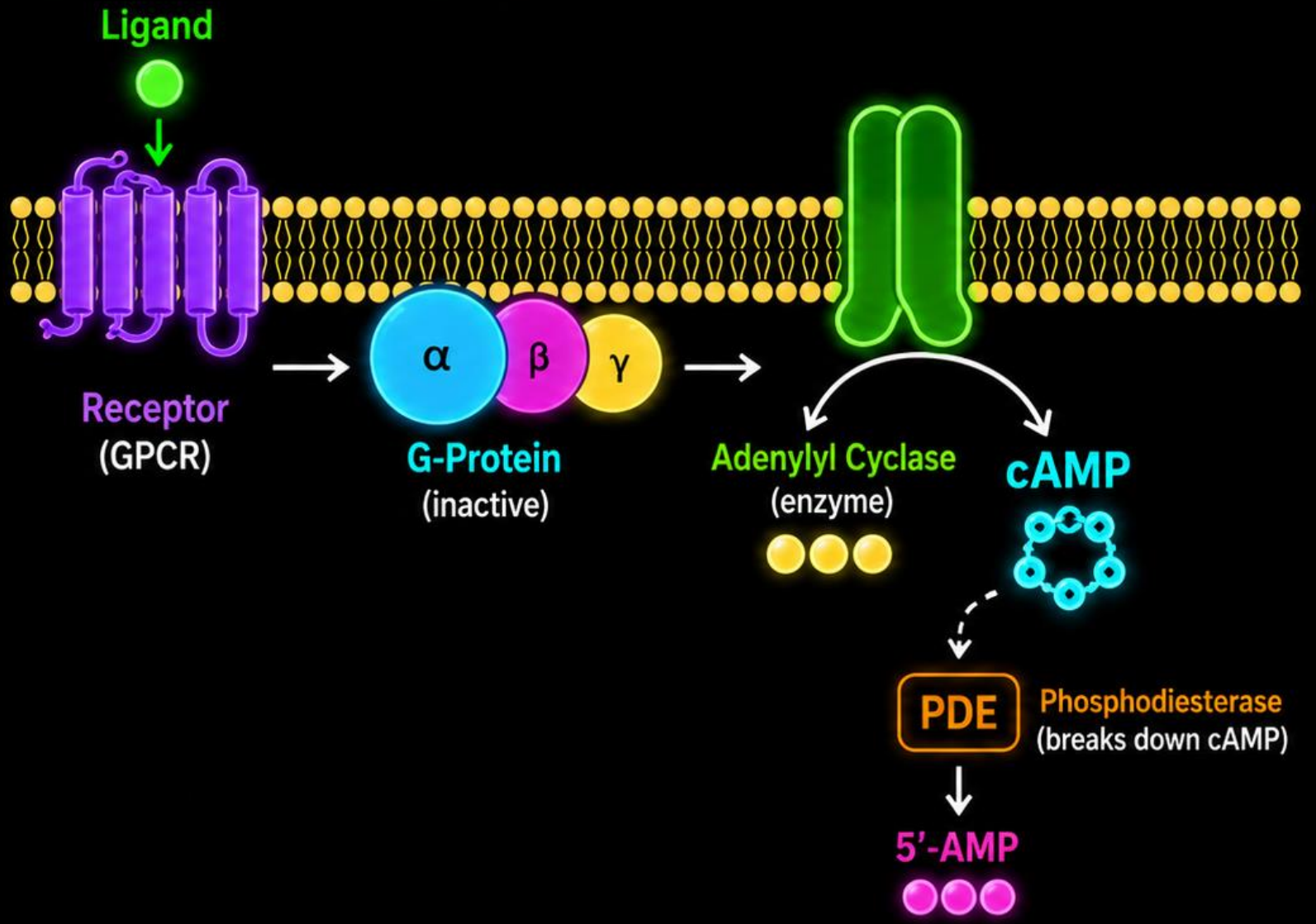


Adenylyl Cyclase Forms cAMP

- **Adenylyl cyclase** is a **membrane-bound enzyme**.
- It converts **ATP** into **cAMP**.
- It is regulated by **stimulatory** or **inhibitory G-proteins.**
- **Phosphodiesterase** breaks down **cAMP**, not adenylyl cyclase.



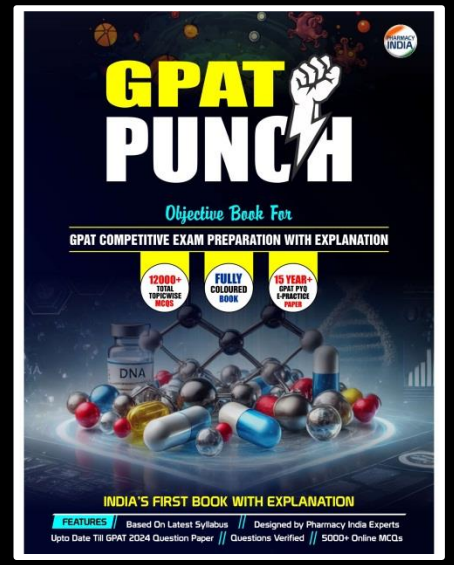
13. **In order to produce characteristic pharmacological action, a drug must always: [GATE-1995]**

(a) Reach high blood vessels

(b) Be absorbed from GIT readily

(c) Achieve adequate concentration at the site of action

(d) Be excreted unchanged in urine



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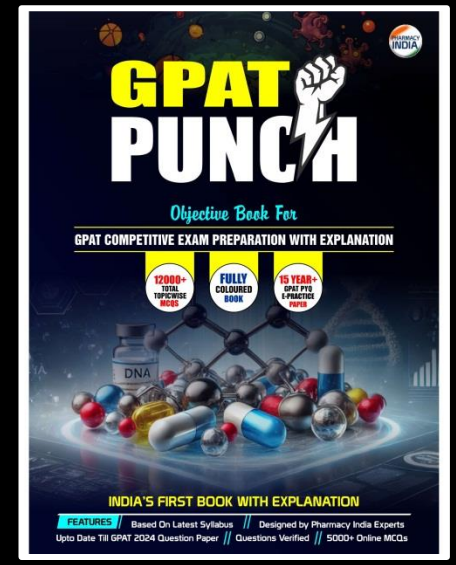
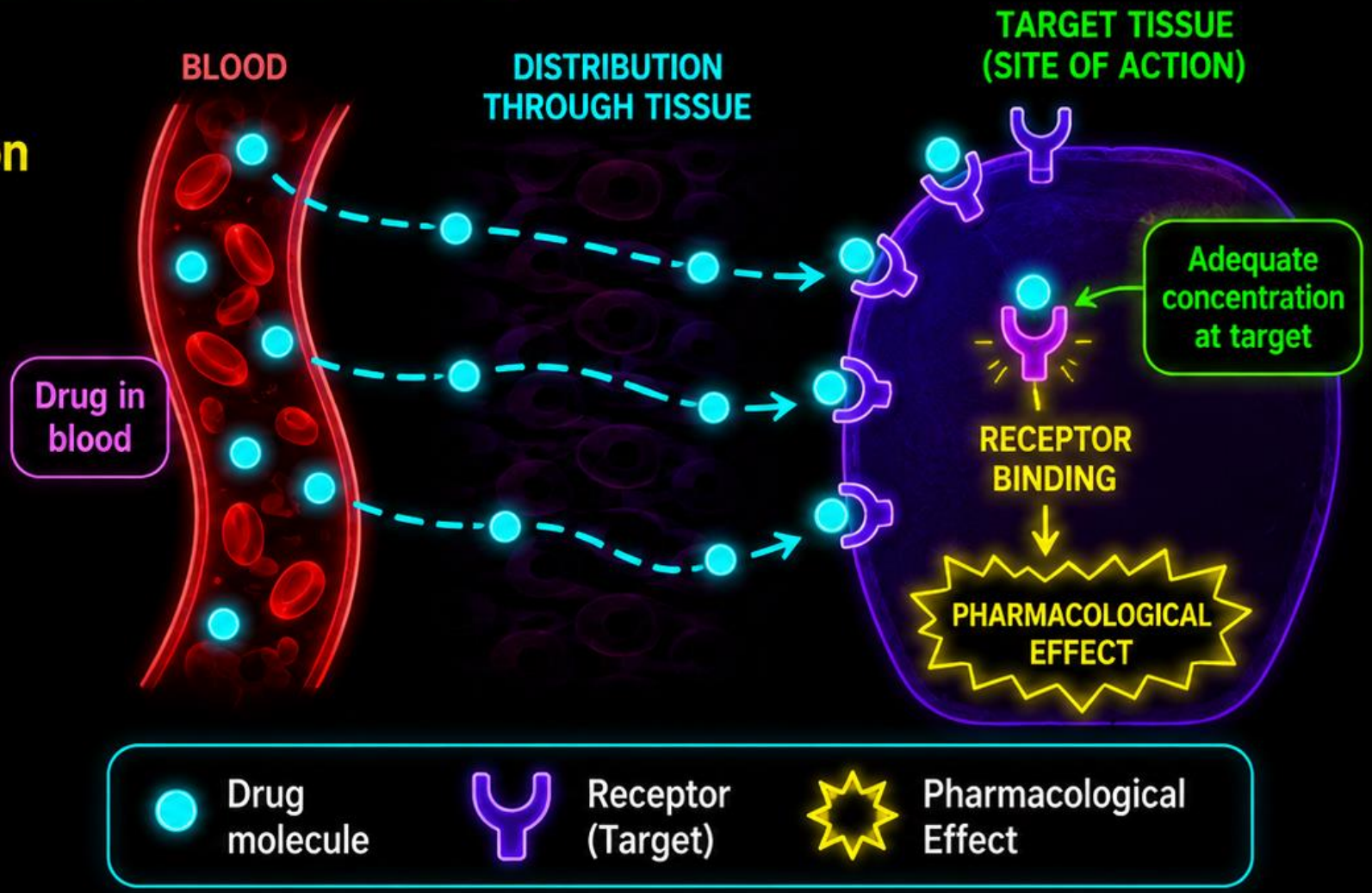


Drug Action Needs Adequate Site Concentration

PHARMACY INDIA

- **Pharmacological** effect depends on **drug concentration** at the **site of action.**
- **Blood level** alone does not always guarantee **response.**
- Some drugs act **locally** without major **systemic absorption.**
- **Adequate concentration** at the **receptor/target** is **essential.**



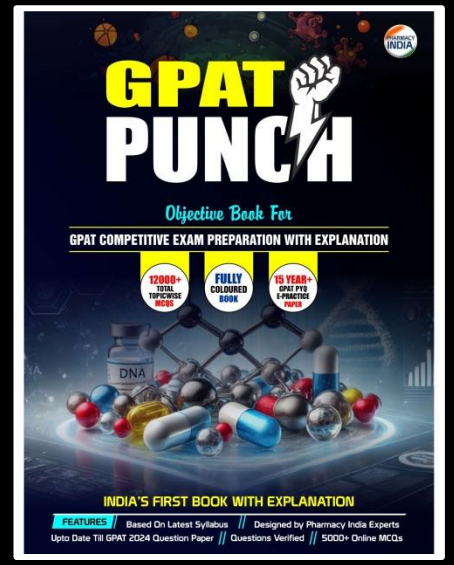
14. **To understand drug-receptor interaction, it is necessary to quantify the relation between: [GATE-1988]**

(a) Drug and its toxicity

(b) Drug and its absorption

(c) Drug and its biological effect

(d) Drug and intermediate product



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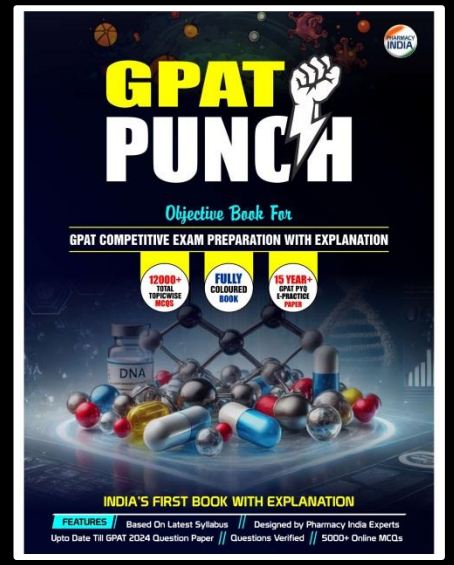
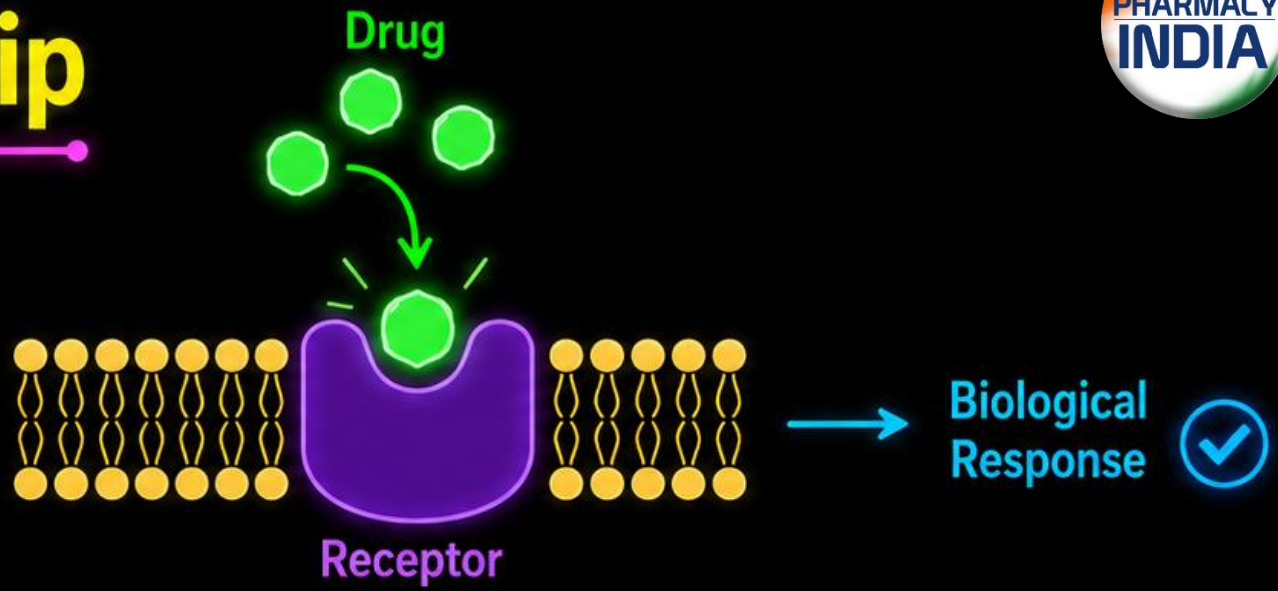
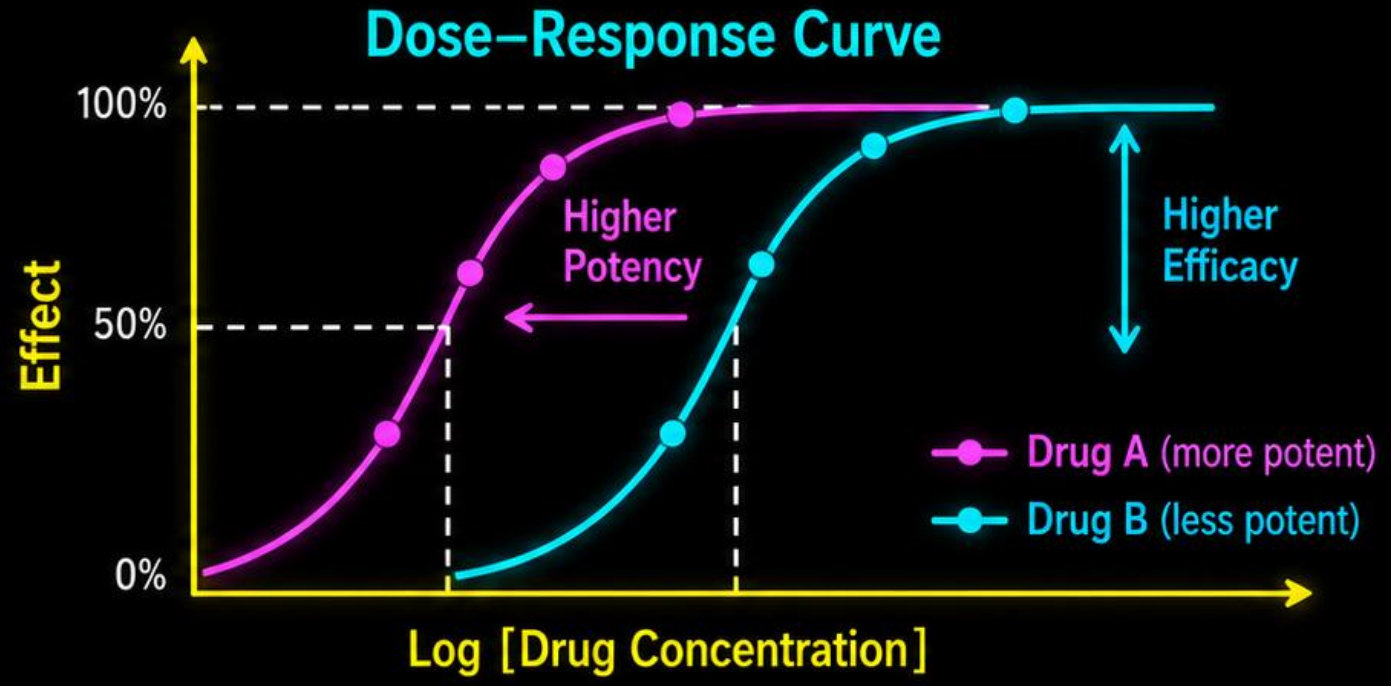


Drug–Effect Relationship

- **Pharmacodynamics** relates drug **dose** or **concentration** to **biological effect**.
- **Drug–receptor** interaction is assessed by the **response** produced.
- **Dose–response** curves help **quantify** this relationship.
- **Potency** and **efficacy** are derived from this **comparison**.





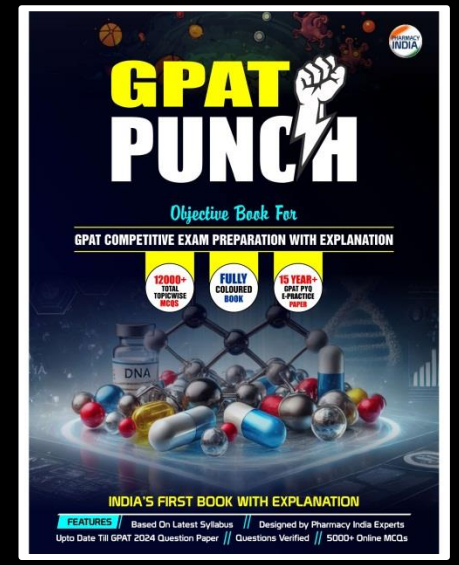
15. **The osmotic activity of mannitol is an example of:**

(a) Physical action

(b) Chemical action

(c) Enzymatic action

(d) Receptor action



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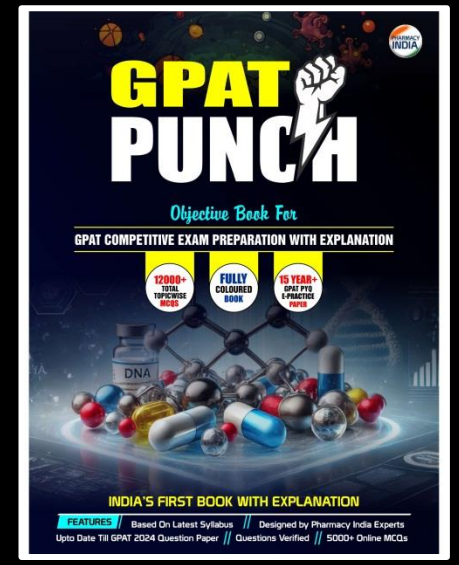
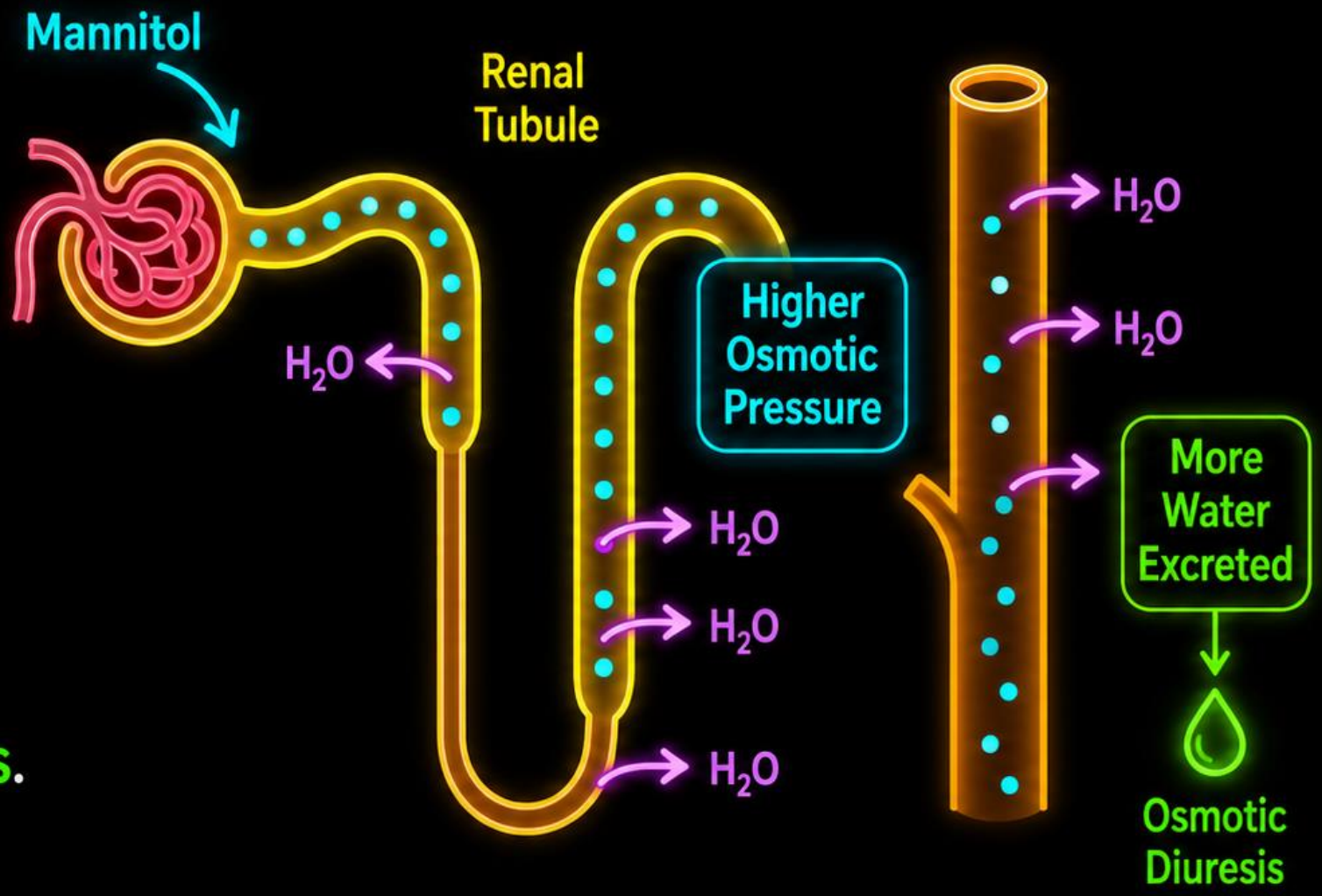


Mannitol Shows Physical Action

- Mannitol acts by an **osmotic** effect.
- It does **not** require **receptor binding**.
- It increases **osmotic pressure** in **renal tubules**.
- This causes **osmotic diuresis**.



16. **Chelating agents sequestering heavy metals is an example of:**

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(b) Chemical action

(c) Physical action

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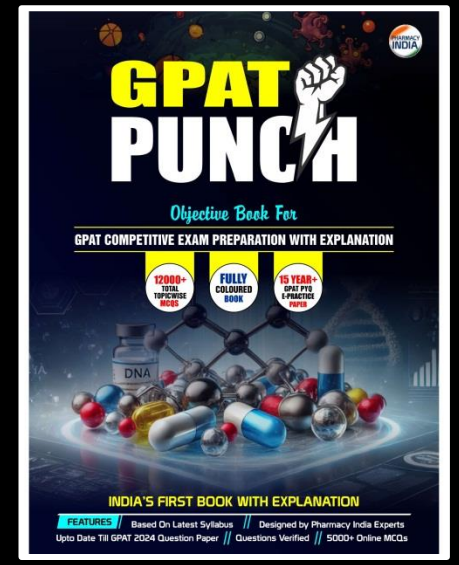

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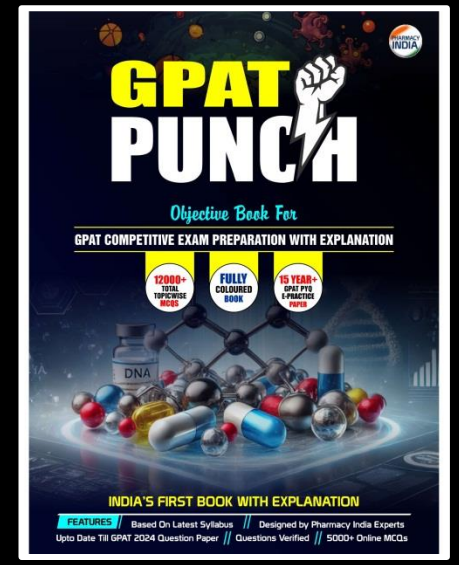
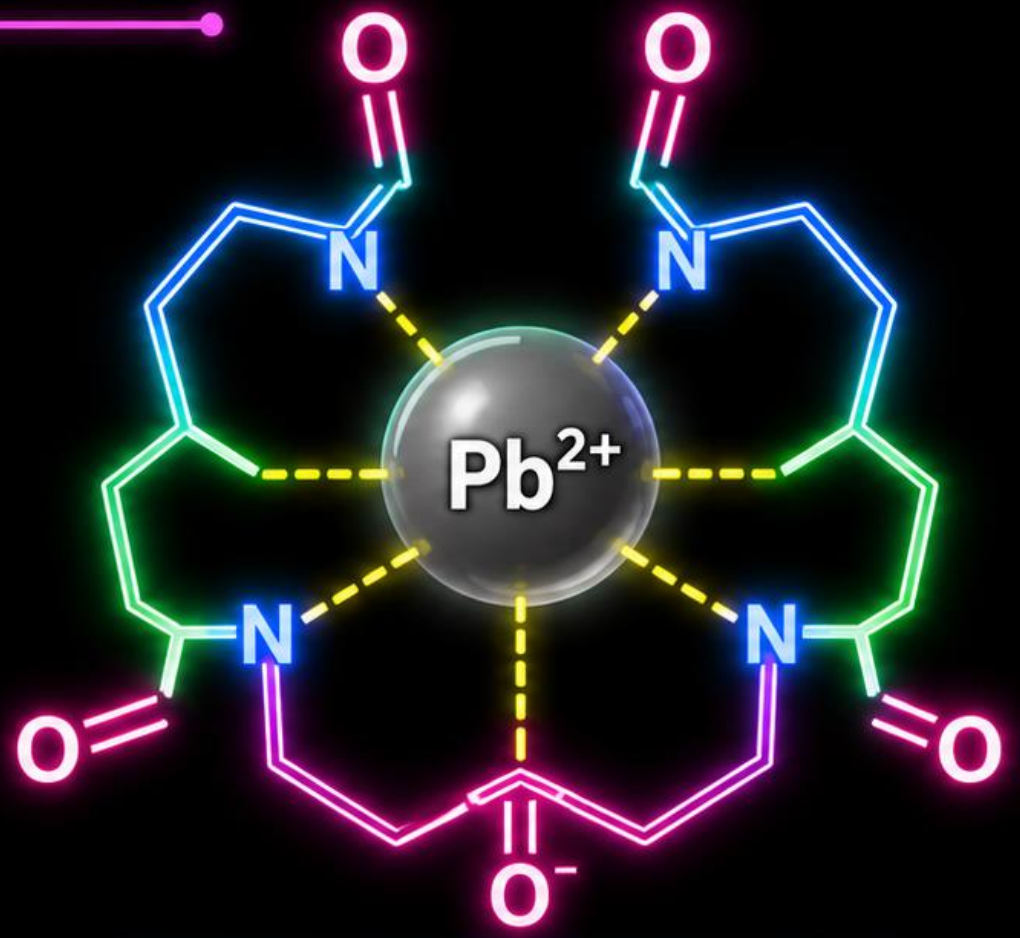


Chelation Is Chemical Action

- Chelating agents bind heavy metals chemically.
- They form stable complexes.
- The complex becomes less toxic and can be excreted.
- Example: EDTA chelates lead.



EDTA–Pb^{2+} Complex
(Less toxic & excretable)

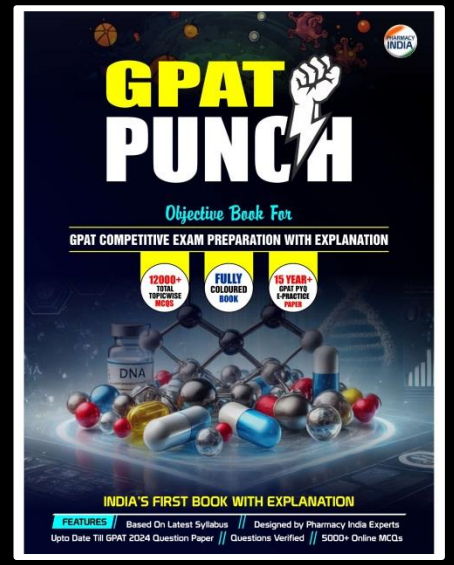
17. **Which of the following is an example of competitive enzyme inhibition?**

(a) Acetazolamide—carbonic anhydrase

(b) Digoxin—Na^+/K^+ ATPase enzyme

(c) Sulphonamides—folate synthase pathway

(d) Theophylline—phosphodiesterase



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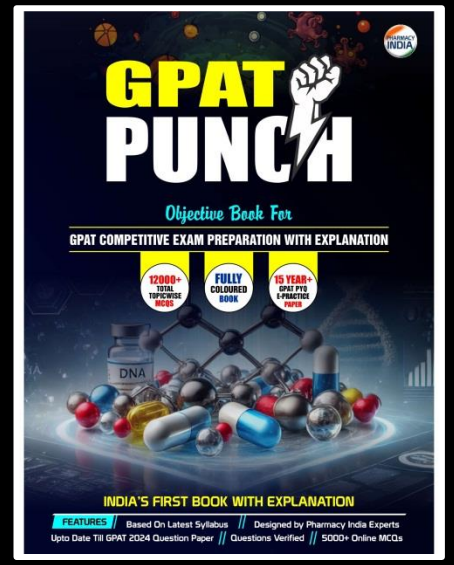
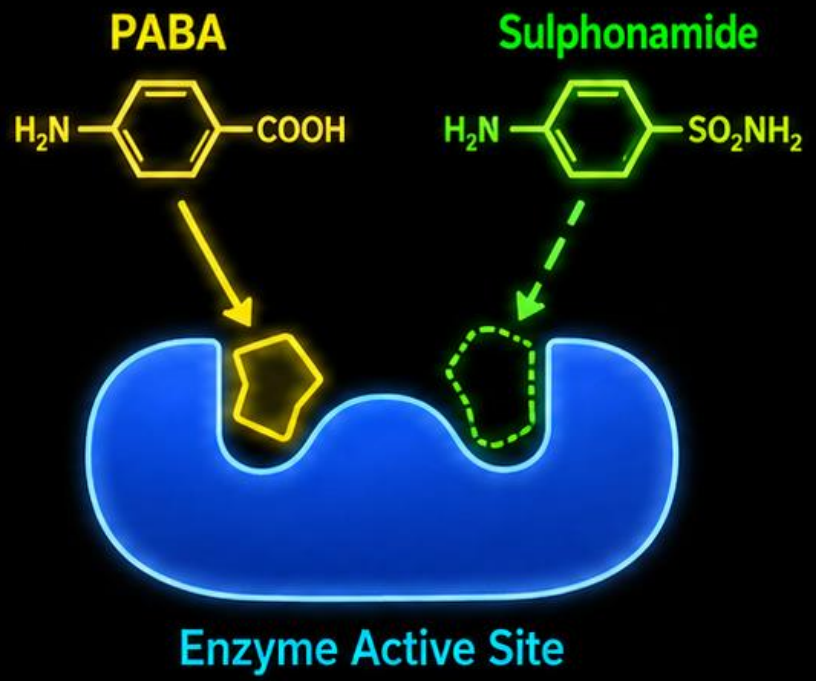
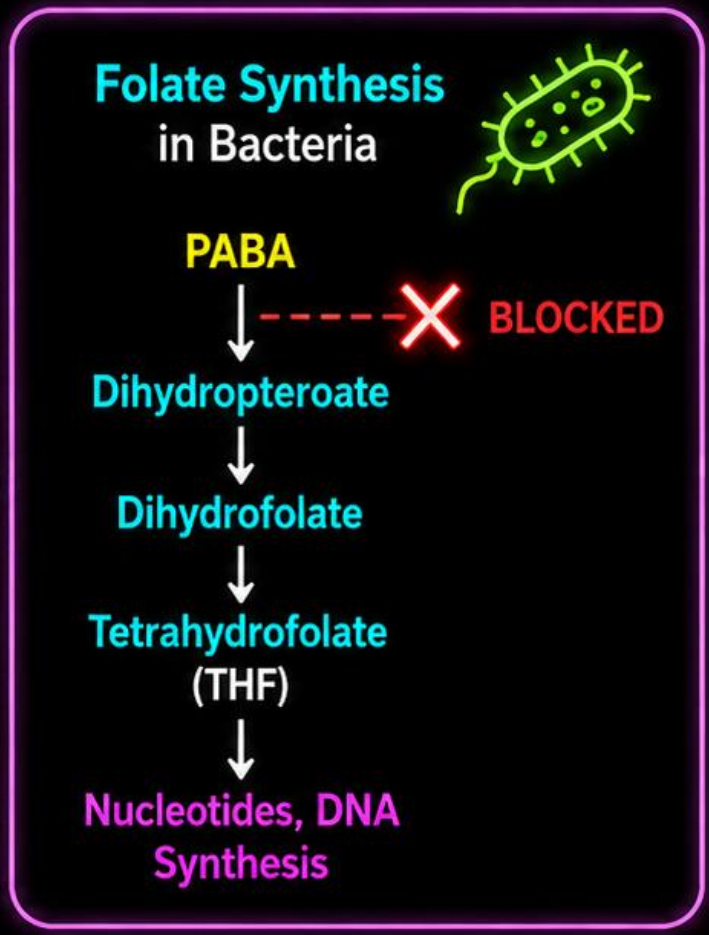


Sulphonamides: Competitive Enzyme Inhibition

- Sulphonamides are structural **analogues** of **PABA**.
- They **compete** at the **active site** in the folate synthesis pathway.
- This **blocks** bacterial **folic acid formation**.
- It is a classic example of **competitive enzyme inhibition**.



Sulphonamide **competes** with **PABA** for the active site.



★ **Sulphonamides** block folate synthesis by **competing** with **PABA** at the enzyme active site.

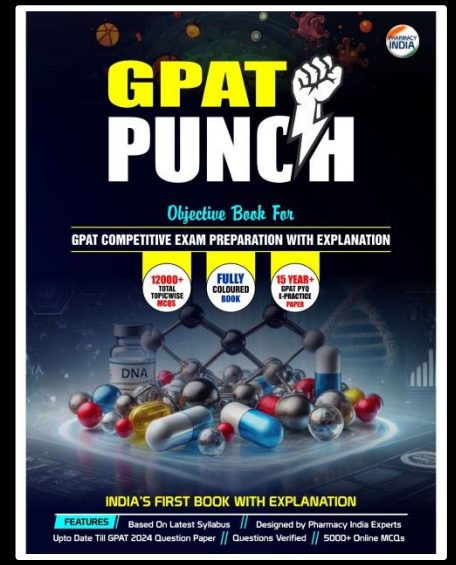
18. **A drug that activates a receptor to produce an effect in the opposite direction to that of recognized agonist is:**

(a) Agonist

(b) Antagonist

(c) Partial agonist

(d) Inverse agonist



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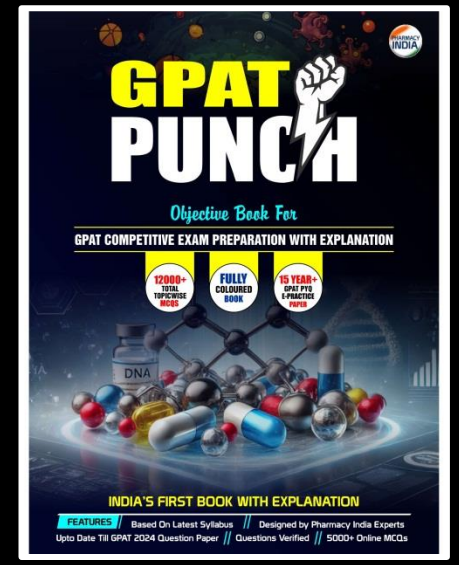
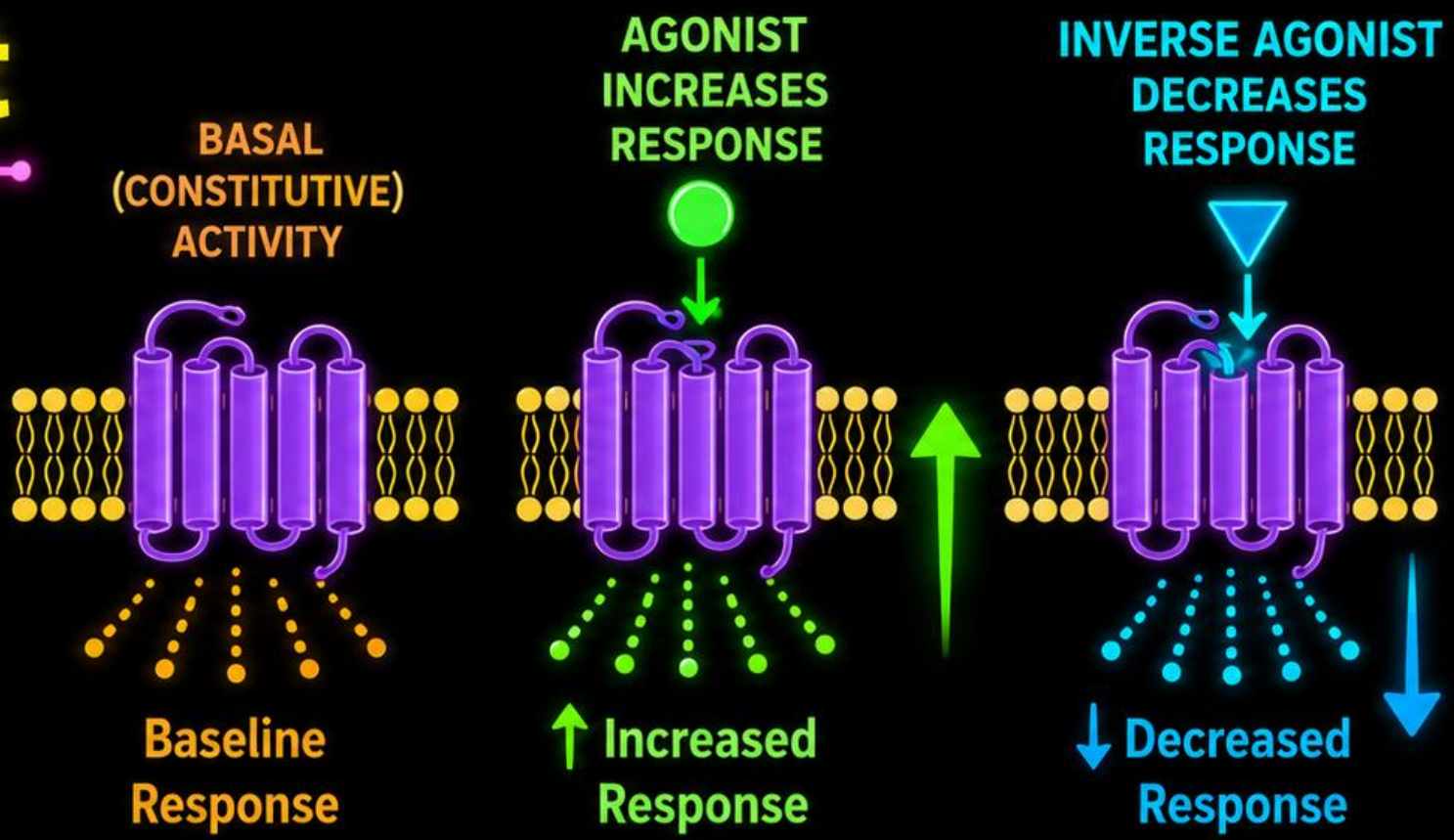
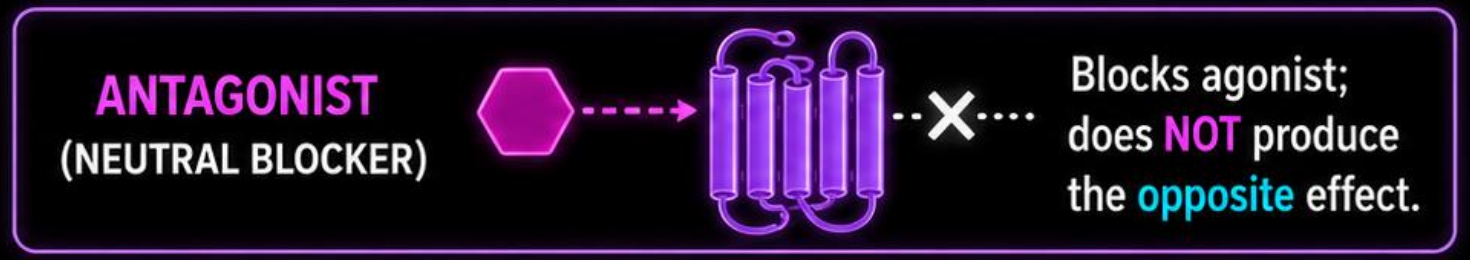


Inverse Agonist Gives Opposite Effect

- It **binds** to the receptor with **affinity**.
- It **reduces basal** or **constitutive** receptor activity.
- Its effect is **opposite** to that of an **agonist**.
- **Antagonists** only **block**; they do not produce the **opposite** effect.





19. **Partial agonists are drugs which:**

(a) Have affinity but low intrinsic activity

(b) Have only affinity

(c) Have only intrinsic activity

(d) Have low affinity but high intrinsic activity

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(a) Have affinity but low intrinsic activity

(b) Have only affinity

(c) Have only intrinsic activity

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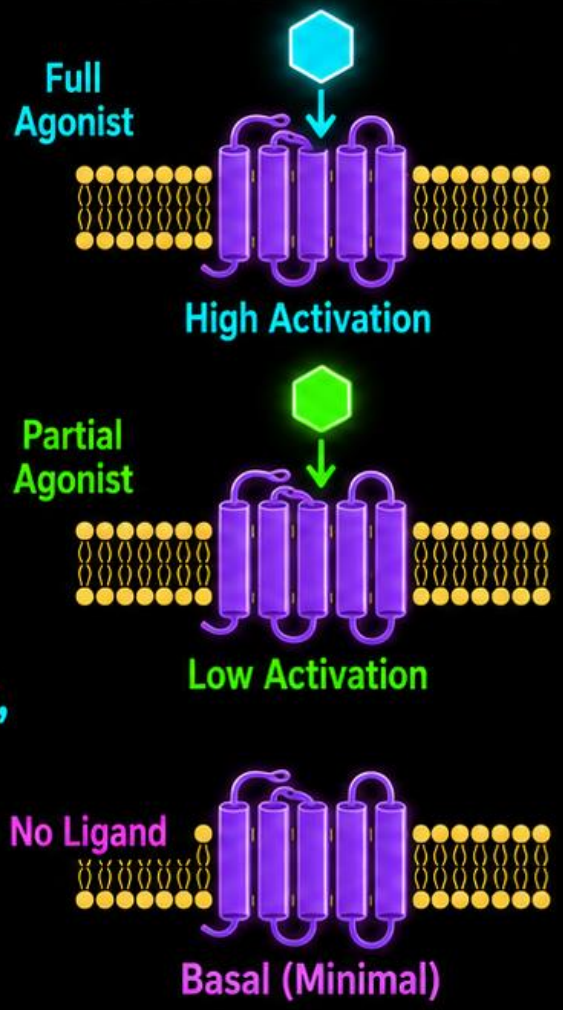
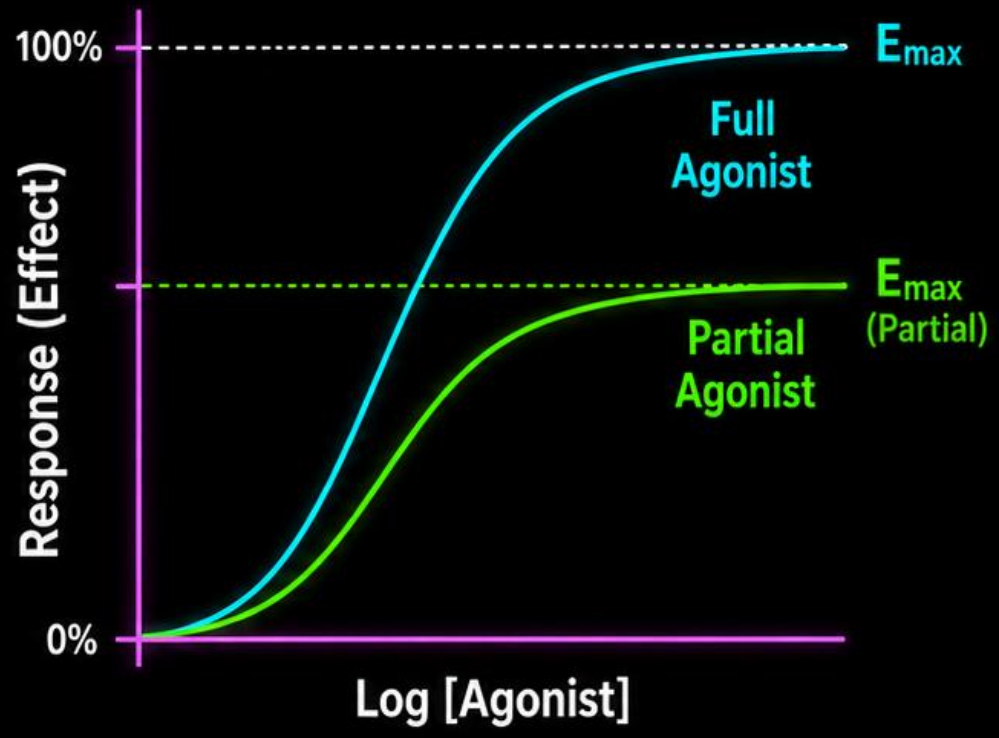
Partial Agonist: Affinity + Low Intrinsic Activity

- Partial agonists bind to receptors.
- They produce a submaximal response.
- Their intrinsic activity is lower than full agonists.
- In the presence of a full agonist, they may act functionally like antagonists.

Receptor Activation



Dose–Response Curve



Binds well, but activates less.
Lower E_{max} = lower intrinsic activity.

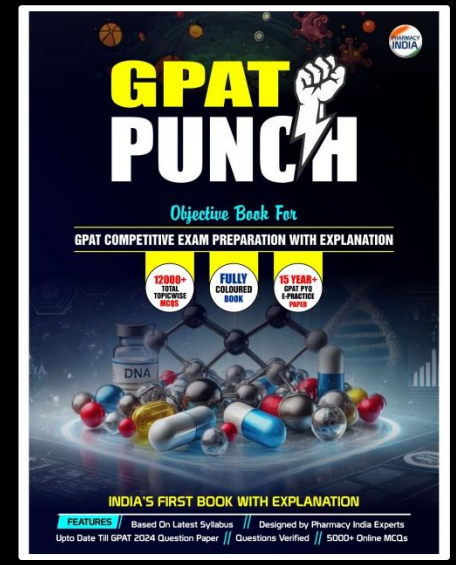
20. Agonists are drugs having:

(a) Only affinity for the receptor

(b) Only intrinsic activity

(c) Affinity and maximum intrinsic activity

(d) Affinity and very low intrinsic activity



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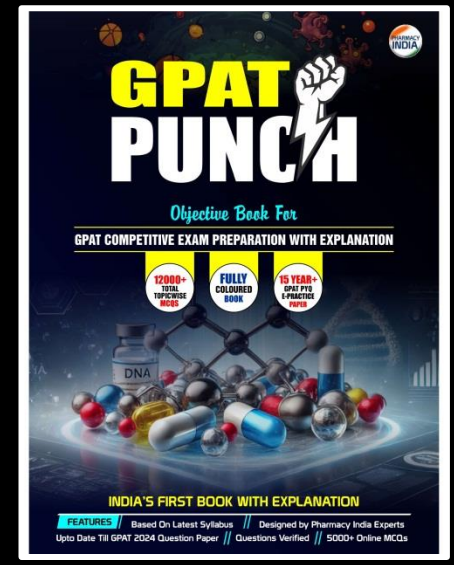
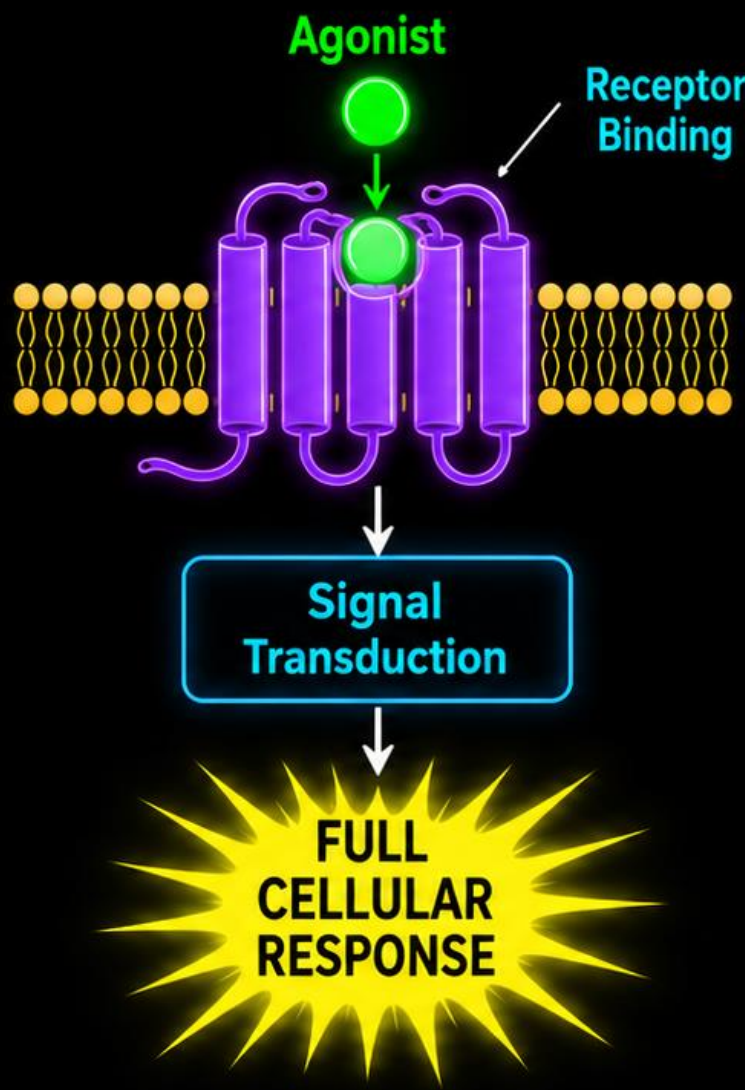
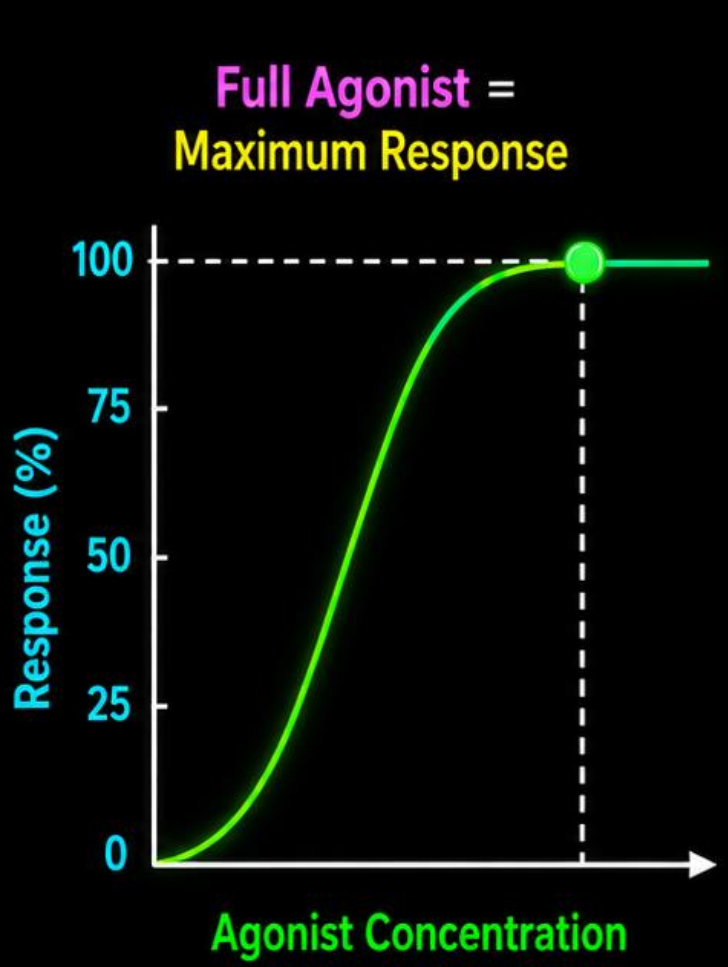


Agonist = Affinity + Maximum Intrinsic Activity

- **Agonists** bind to receptors because they have **affinity**.
- They **activate** receptors because they have **intrinsic activity**.
- A **full agonist** produces the **maximum response**.
- Both **affinity** and **efficacy** are required for **agonist action**.





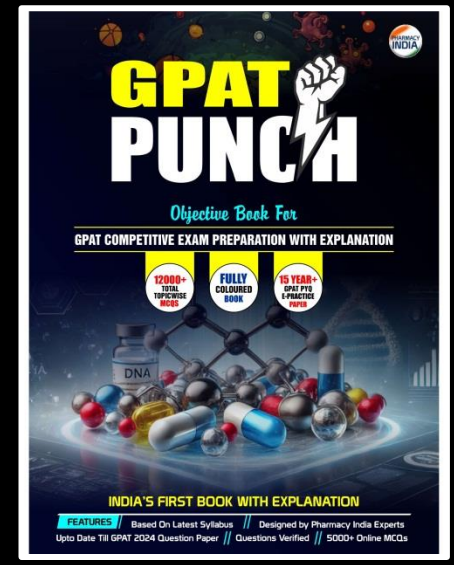
21. **Which of the following acts by inhibiting enzyme in the body?**

(a) Atropine

(b) Allopurinol

(c) Levodopa

(d) Metoclopramide



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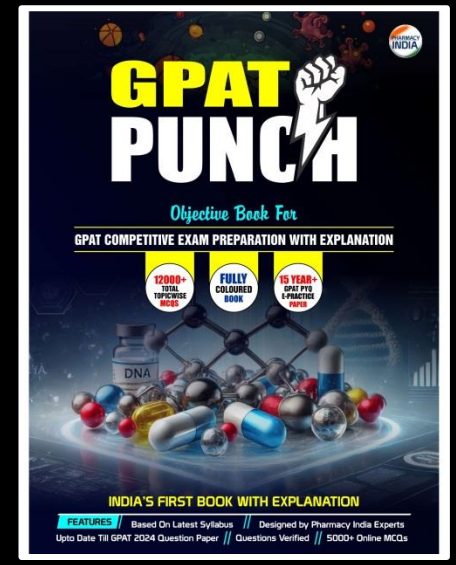
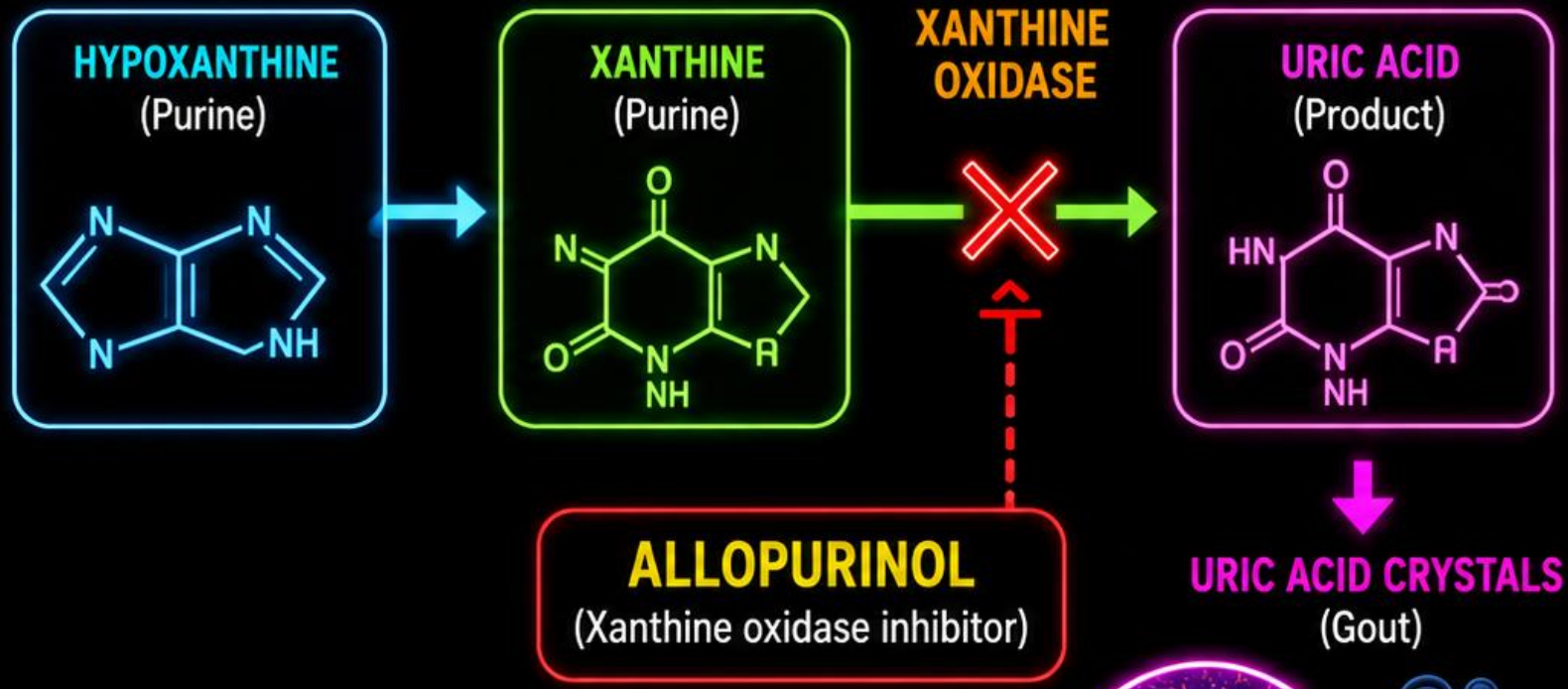


ALLOPURINOL INHIBITS XANTHINE OXIDASE

- **Blocks** xanthine oxidase enzyme
- **Decreases** uric acid formation
- **Used in** gout and hyperuricemia
- **Atropine** acts on receptors, not enzyme inhibition



Allopurinol reduces uric acid production by inhibiting xanthine oxidase.

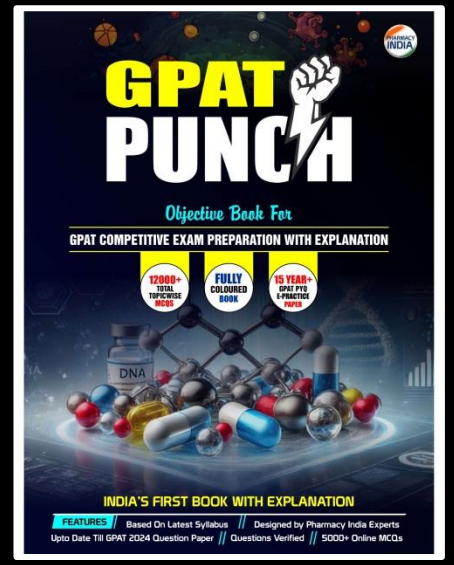
22. **Following agent is a competitive type of enzyme inhibitor:**

(a) Acetazolamide

(b) Disulfiram

(c) Physostigmine

(d) Theophylline



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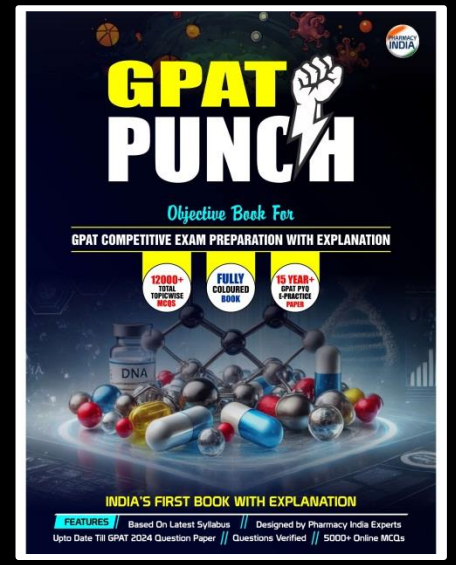


THEOPHYLLINE INHIBITS PHOSPHODIESTERASE

- Competitive enzyme inhibition
- Blocks phosphodiesterase (PDE)
- Raises cAMP level
- Increased cAMP contributes to bronchodilation

1. THEOPHYLLINE

2. BLOCKS PDE

cAMP ------> 5′-AMP

Competitive inhibition

3. ↑ cAMP LEVEL

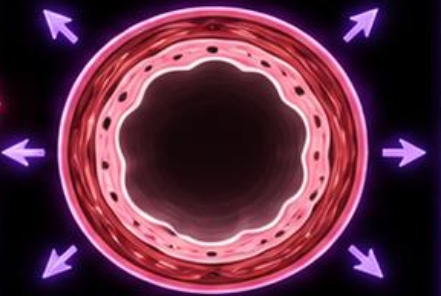
cAMP increases

4. BRONCHIAL SMOOTH MUSCLE RELAXES

Reduced Ca^{2+}

Smooth muscle relaxes

5. BRONCHODILATION



Airway opens

easier breathing

Theophylline competitively **inhibits PDE** → **prevents cAMP breakdown** → **↑ cAMP** → **bronchial smooth muscle relaxes** → **bronchodilation.**

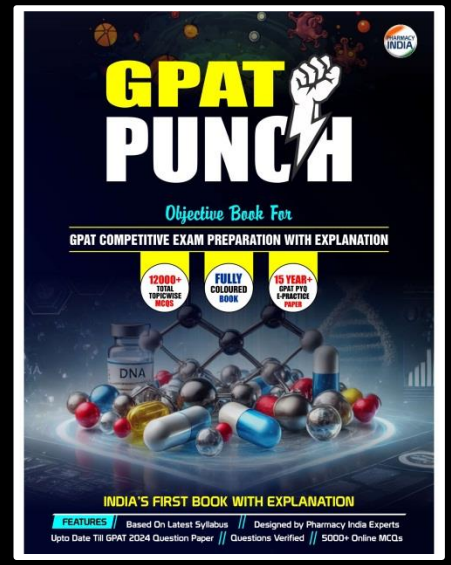
23. Receptor which has seven helical membrane structures is:

(a) Tyrosine kinase receptor

(b) Gene expression regulating receptor

(c) Intrinsic ion channel receptor

(d) G-protein coupled receptor



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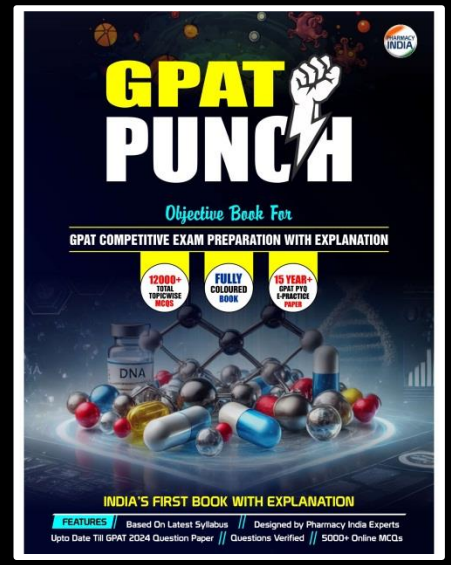
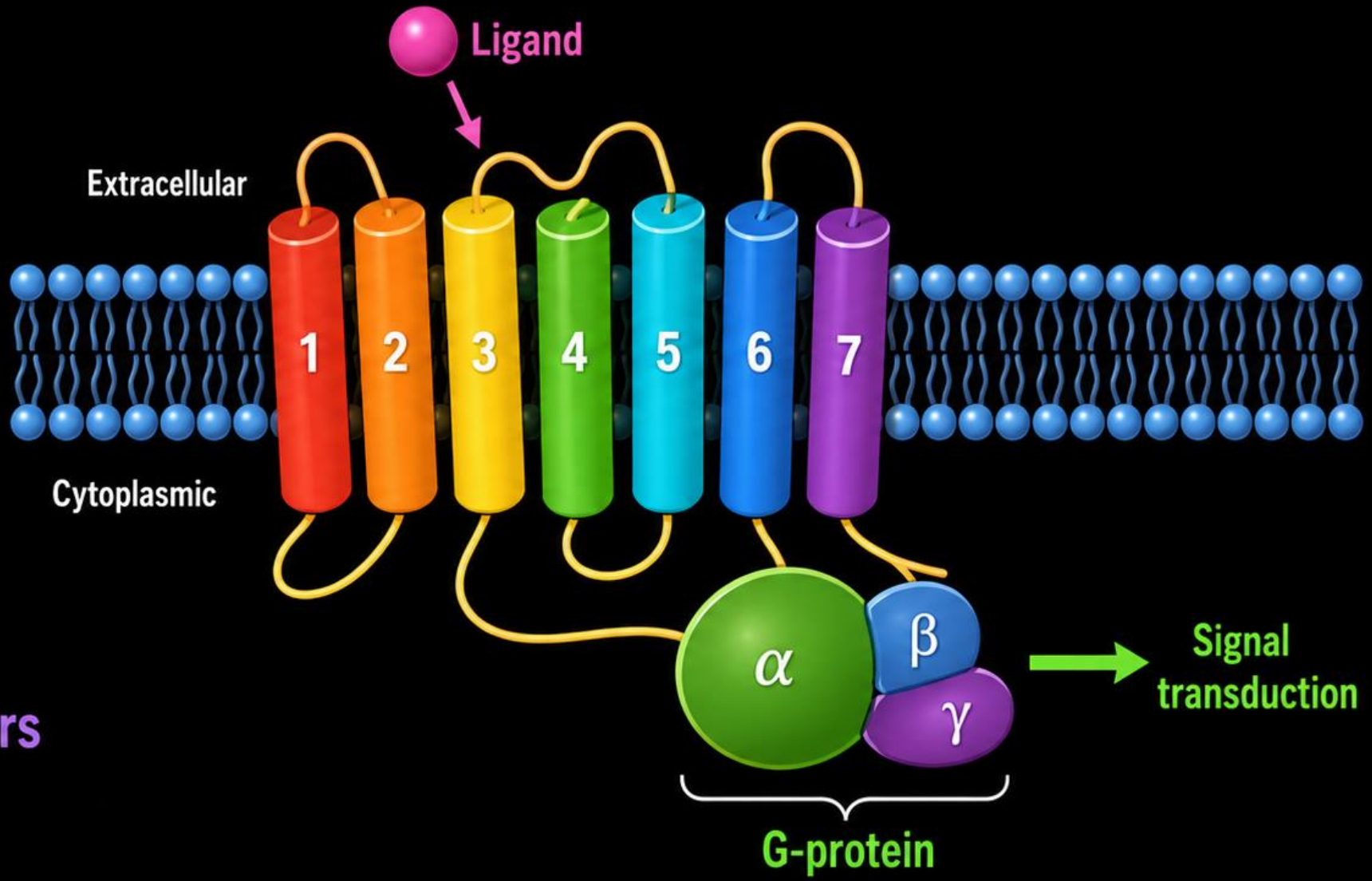


GPCR = 7 Transmembrane Helices

- Seven membrane-spanning alpha-helices
- Also called serpentine receptor
- Ligand binding activates G-proteins
- Examples: adrenergic and muscarinic receptors



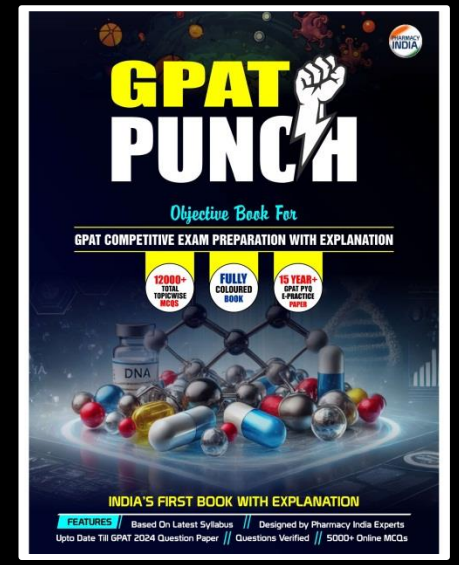
24. All are intracellular second messengers in receptor-mediated signal transduction except:

(a) Cyclic AMP

(b) Inositol triphosphate

(c) Diacylglycerol

(d) G-protein



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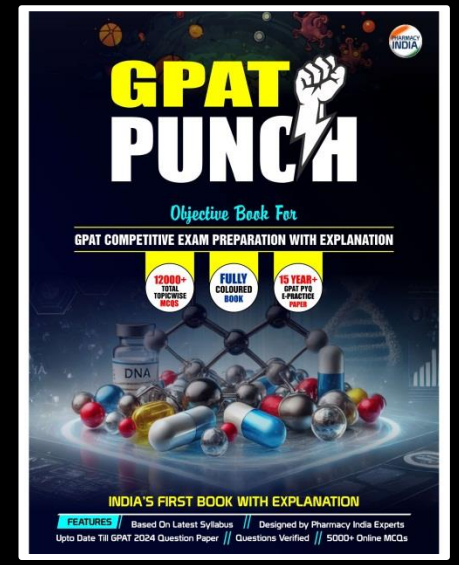


Second Messengers vs G-Protein

PHARMACY INDIA

- cAMP, IP_3 and DAG are second messengers
- They carry signals inside the cell
- G-protein is a signal transducer
- It links receptor to enzyme or ion channel

SECOND MESSENGERS

cAMP

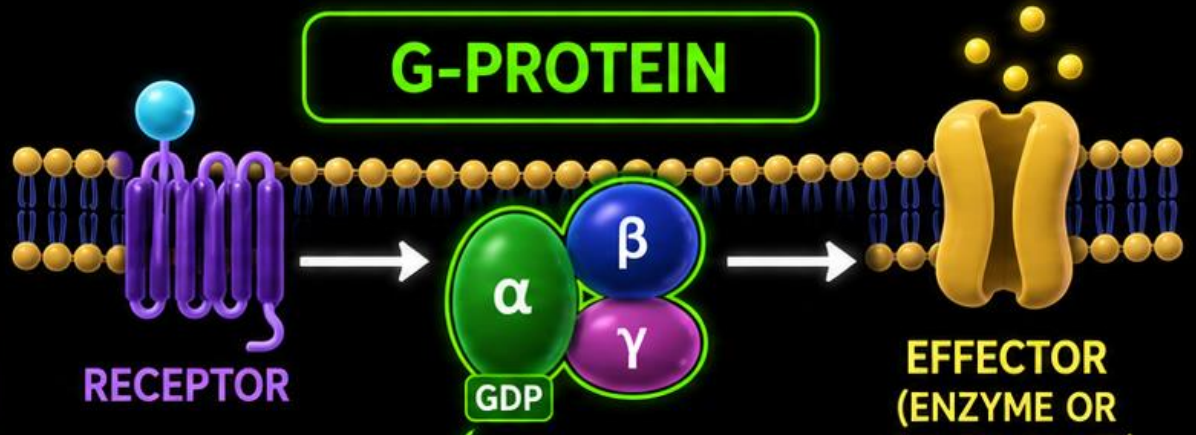
IP_3

DAG

Carry signals inside the cell

VS

G-PROTEIN



Signal transducer
Links receptor to enzyme or ion channel

NOT A SECOND MESSENGER

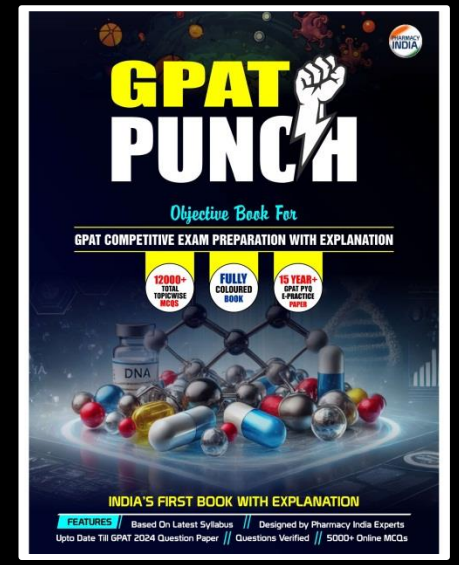
25. A receptor with enzymatic property is:

(a) Insulin receptor

(b) Progesterone receptor

(c) Thyroxine receptor

(d) Histamine receptor



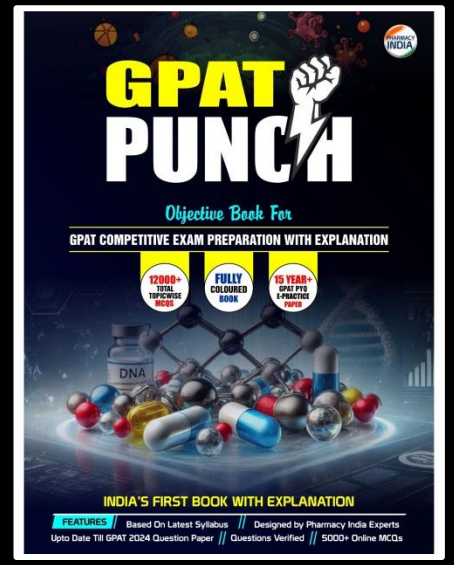
25. **A receptor with enzymatic property is:**

(a) Insulin receptor

(b) Progesterone receptor

(c) Thyroxine receptor

(d) Histamine receptor

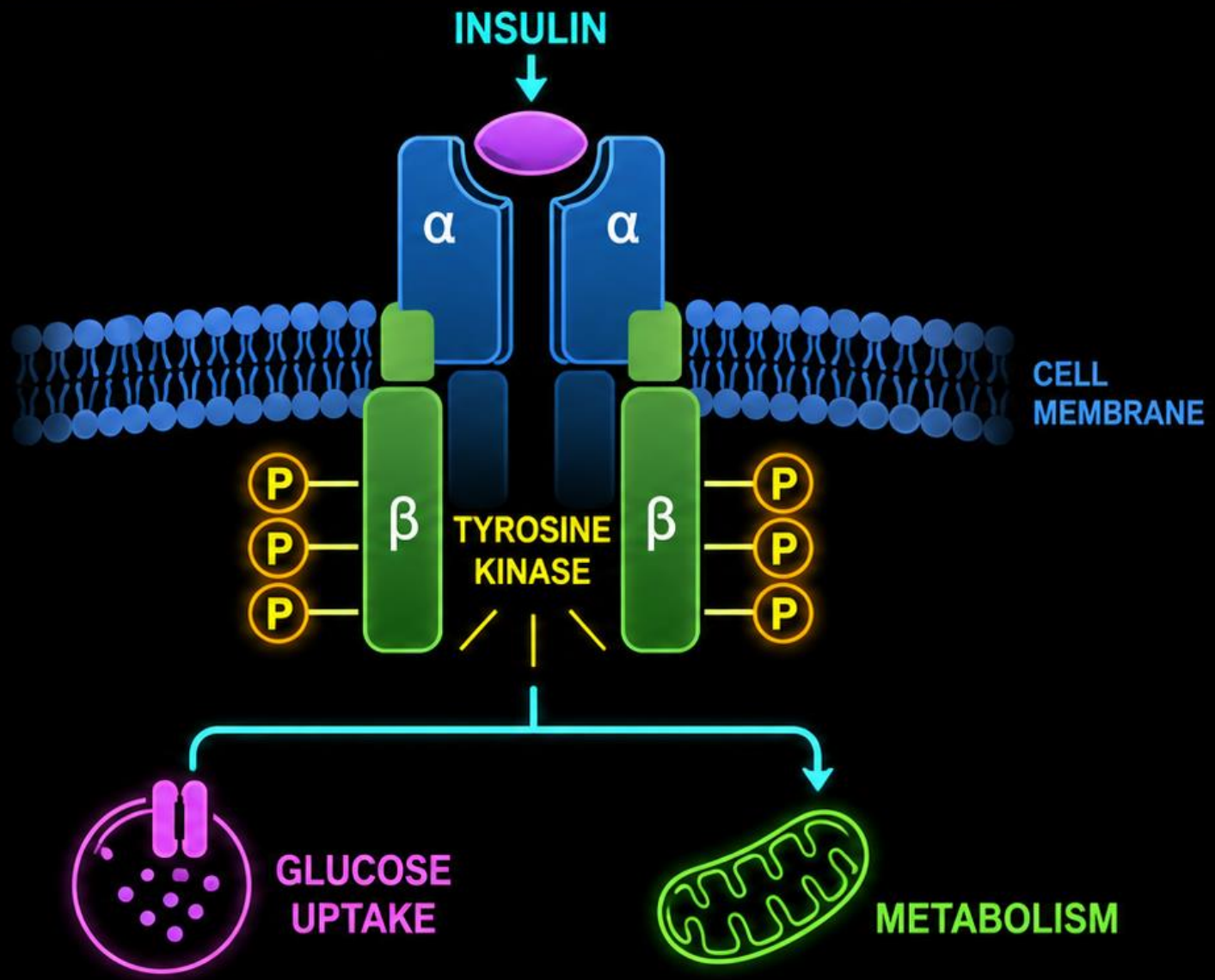


25

Insulin Receptor = Tyrosine Kinase

PHARMACY INDIA

- Receptor has intrinsic enzymatic activity
- Tyrosine kinase phosphorylates proteins
- Helps regulate glucose uptake and metabolism
- Steroid and thyroid receptors mainly regulate gene transcription



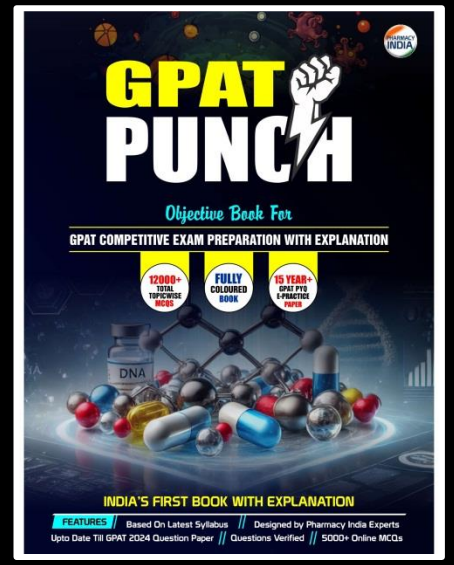
26. **When optimal therapeutic effect becomes suboptimal above and below a narrow range of doses, the drug exhibits:**

(a) Supramaximal effect

(b) Desensitization phenomenon

(c) Therapeutic window phenomenon

(d) Narrow margin of safety



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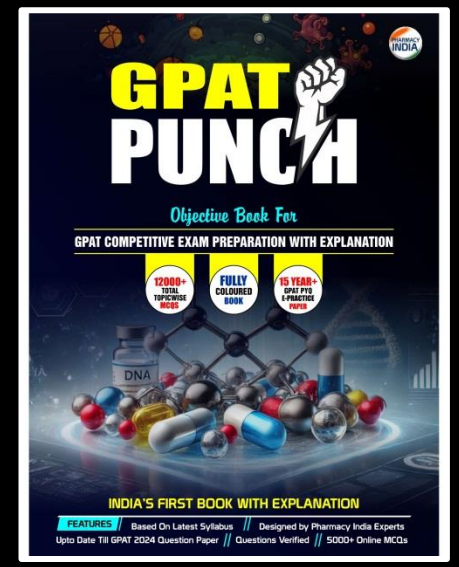


26

Therapeutic Window

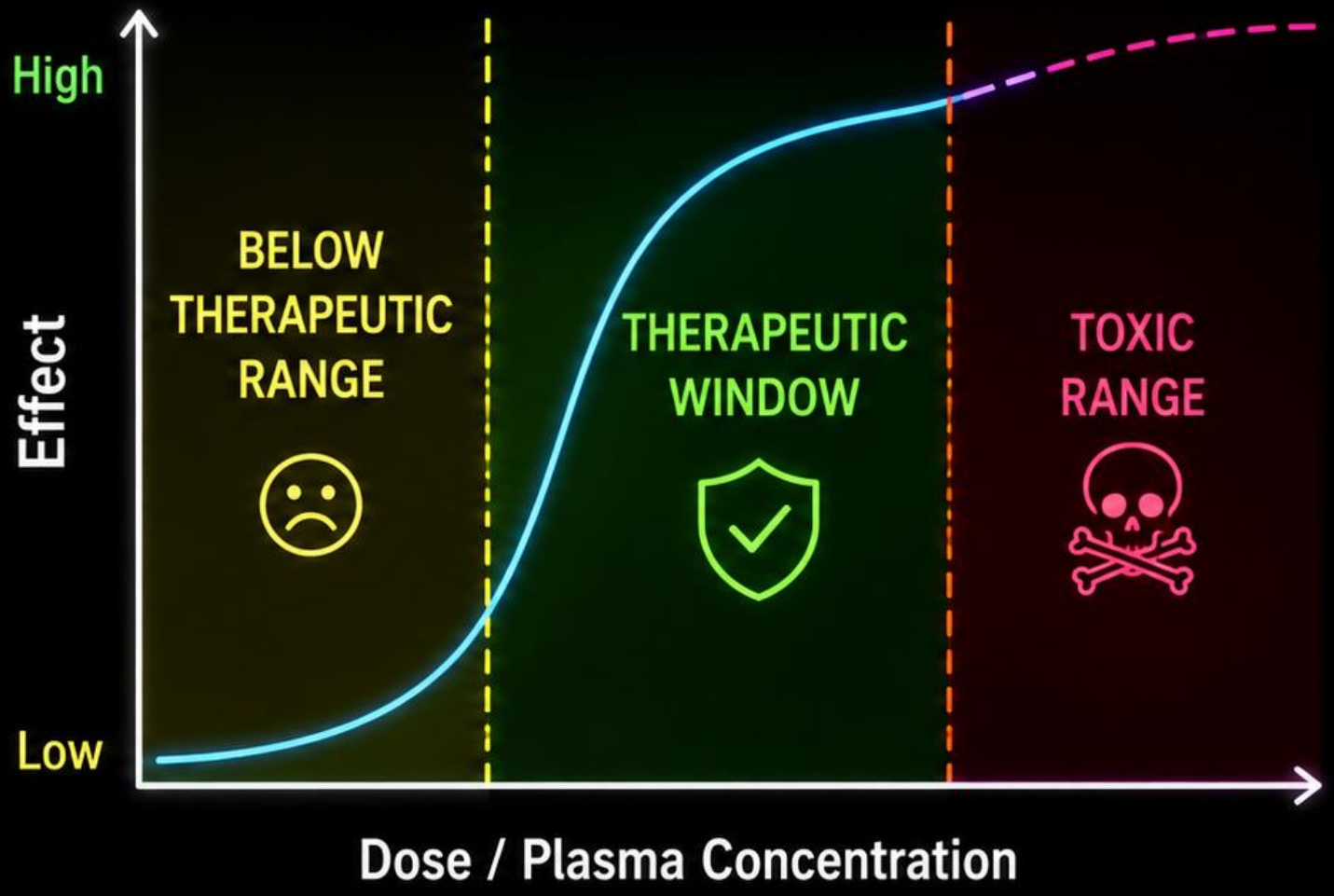
PHARMACY INDIA

Optimal effect occurs within a narrow dose range

Below range: subtherapeutic / inadequate effect

Above range: toxicity risk increases

Narrow-window drugs need close monitoring



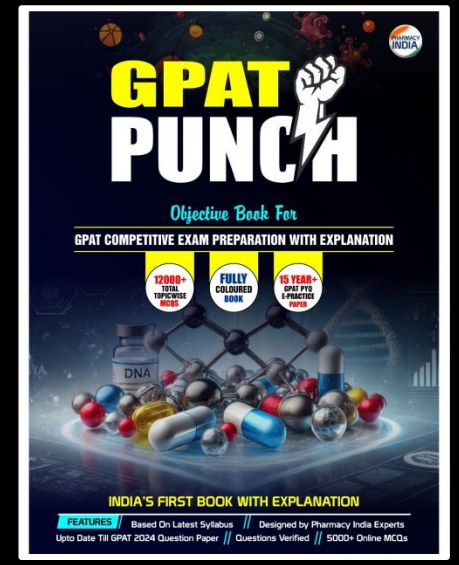
27. **Therapeutic drug monitoring is particularly useful in the following situation:**

(a) Minimal inter-individual variation in response

(b) Drug with irreversible action

(c) Drug actions last longer than blood levels

(d) Cases of poisoning



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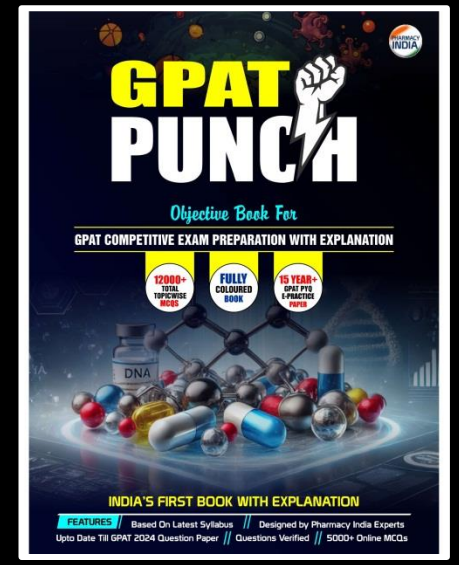
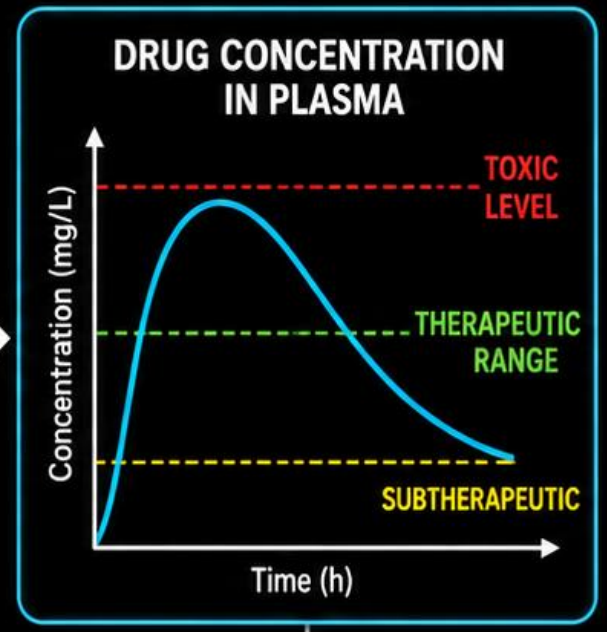


TDM Helps in Poisoning

PHARMACY INDIA

- **Measures** drug concentration in **plasma**
- Useful in **poisoning** and **toxicity**
- **Guides** dose **adjustment** and treatment
- **Very important** for **narrow therapeutic index** drugs



TOXICITY RISK

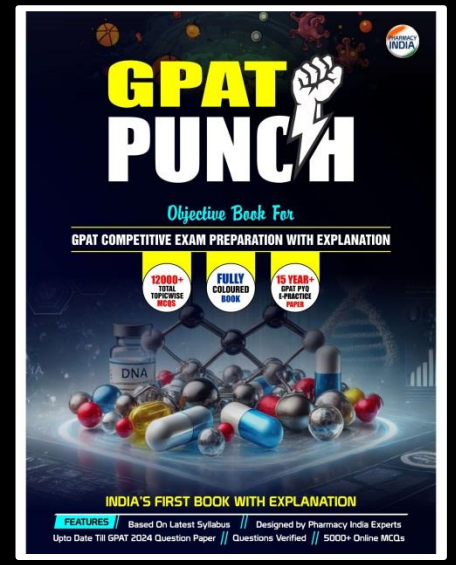
28. **Which of the following drugs exhibits therapeutic window phenomenon?**

(a) Captopril

(b) Furosemide

(c) Diazepam

(d) Imipramine



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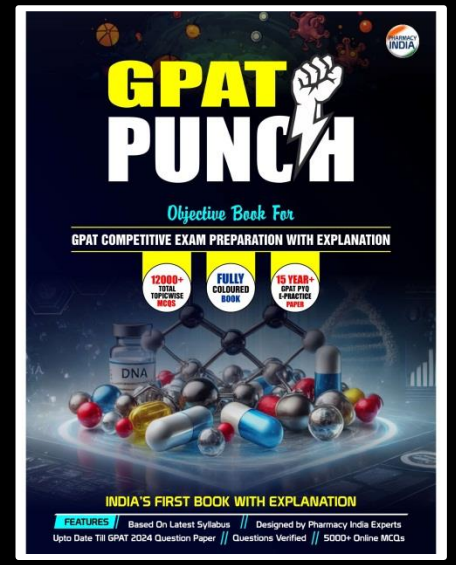


Imipramine: Narrow Therapeutic Window

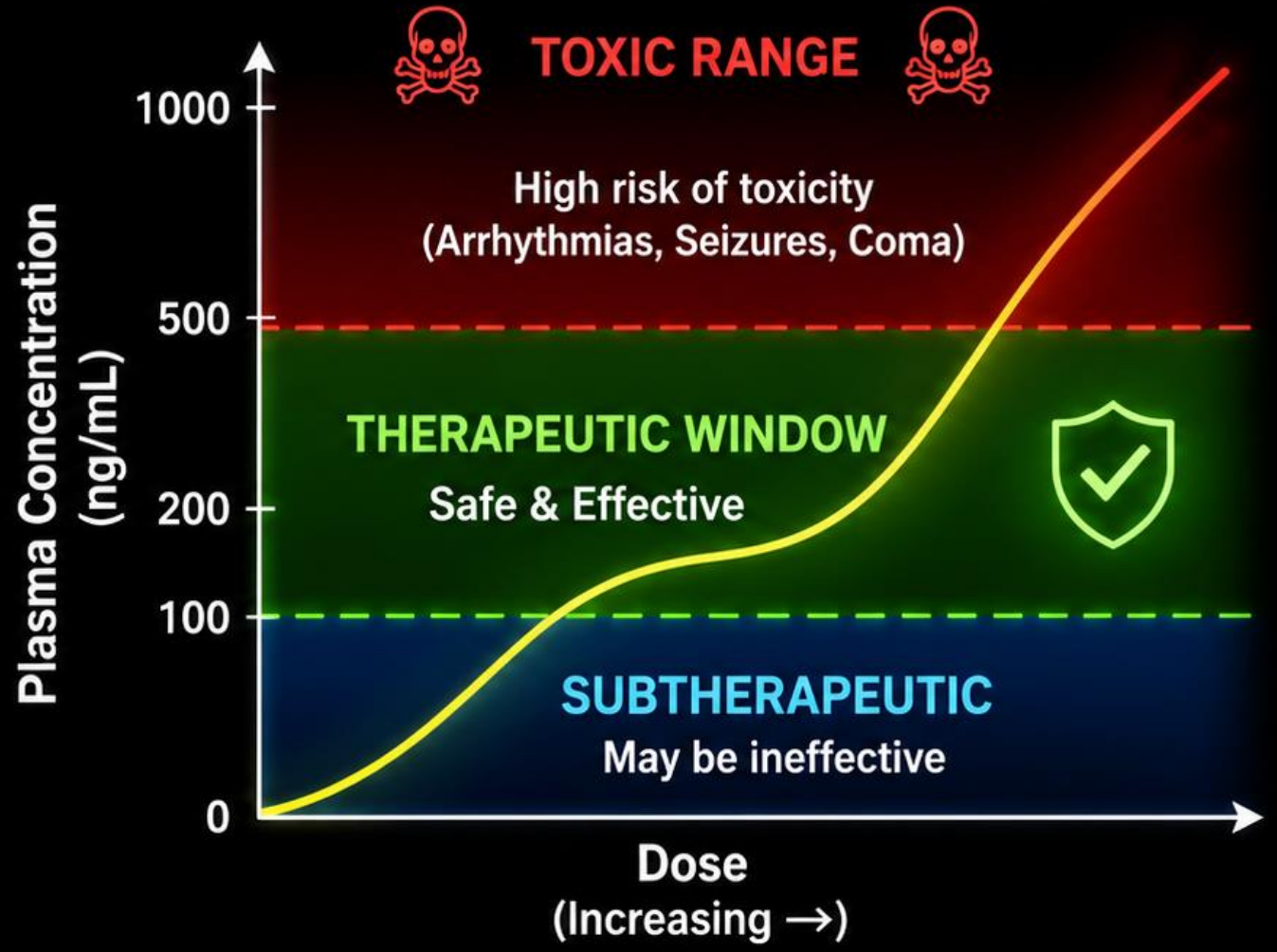
PHARMACY INDIA

Imipramine is a **tricyclic antidepressant**

High levels can cause **significant toxicity**

Dose must remain in a **controlled range**

Represents a **narrow therapeutic window** drug



Small increase in dose → large increase in toxicity

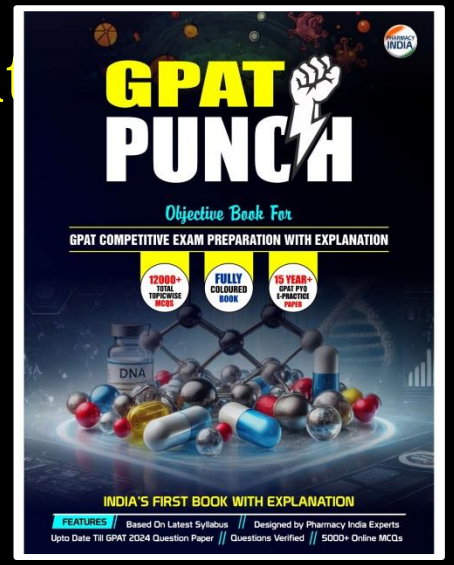
29. **Therapeutic drug monitoring is done for following agent**

(a) Levodopa

(b) Omeprazole

(c) Phenytoin

(d) Enalapril



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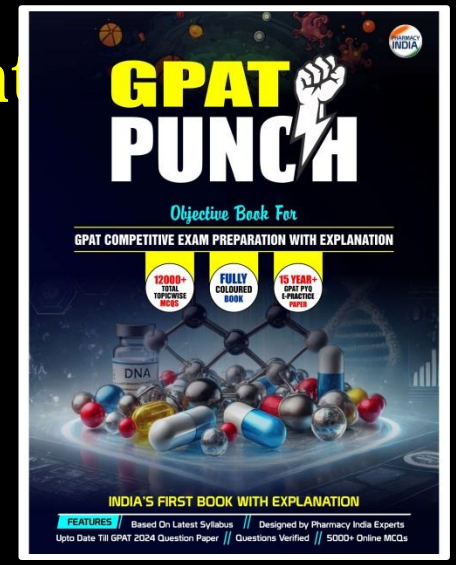
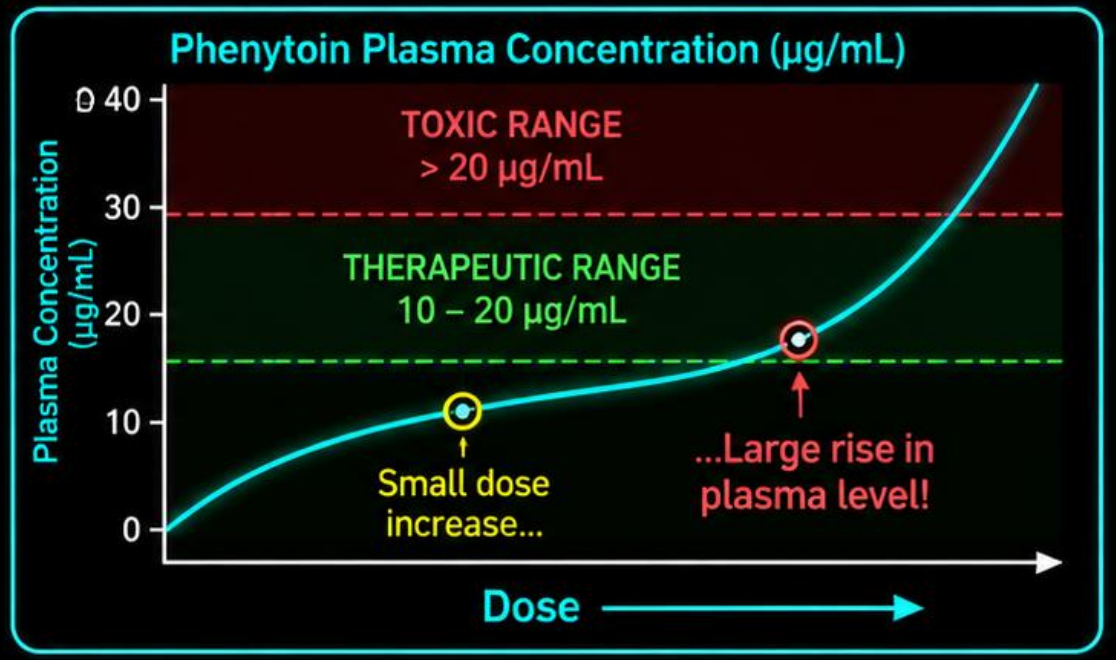


Phenytoin Needs TDM

PHENYTOIN

- **Narrow** therapeutic index
- **Nonlinear** pharmacokinetics
- **Small dose changes** can greatly alter plasma level
- **TDM** helps **avoid toxicity** and **treatment failure**



TOXICITY

- Nystagmus
- Ataxia
- Dysarthria
- Confusion
- Coma

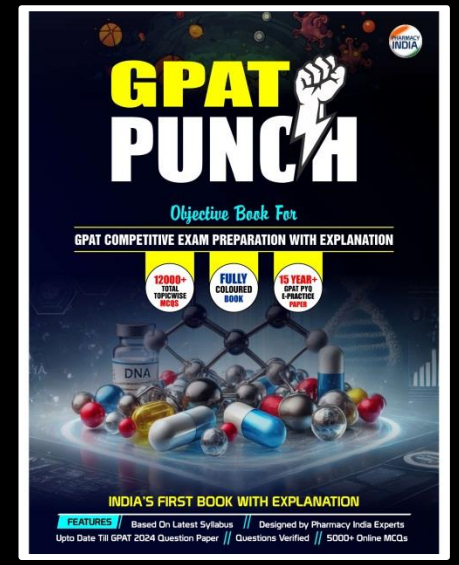
30. **The therapeutic index is a measure of:**

(a) Safety

(b) Potency

(c) Efficacy

(d) Dose variability



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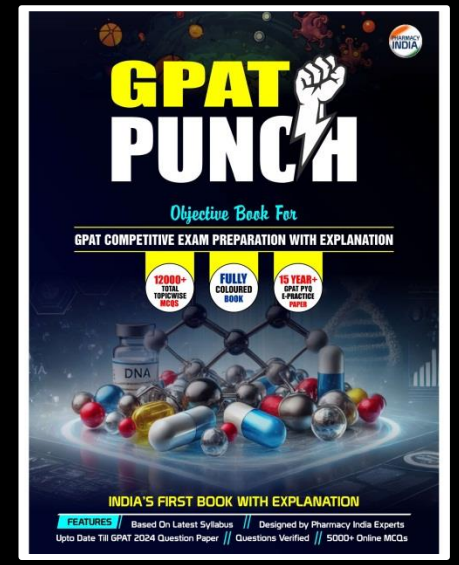


THERAPEUTIC INDEX = SAFETY

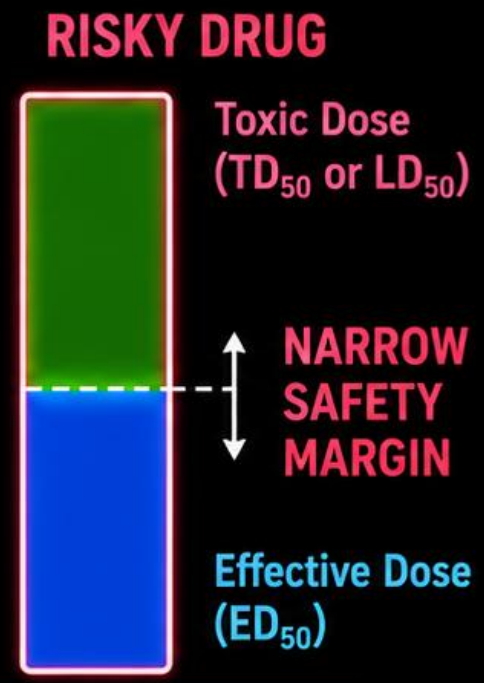
Compares toxic dose with effective dose

Usually expressed as TD_{50}/ED_{50} or LD_{50}/ED_{50}

Higher therapeutic index = safer drug

Lower therapeutic index = greater toxicity risk

$TI = TD_{50}/ED_{50}$ (High)



$TI = TD_{50}/ED_{50}$ (Low)

$$\text{THERAPEUTIC INDEX (TI)} = \frac{\text{Toxic Dose } (TD_{50} \text{ or } LD_{50})}{\text{Effective Dose } (ED_{50})}$$

31. **The term “chemical antagonism” means that:**

(a) Two drugs combine with one another to form an inactive compound

(b) Two drugs combine with one another to form a more active compound

(c) Two drugs combine with one another to form a more water-soluble compound

(d) Two drugs combine with one another to form a more fat-soluble compound

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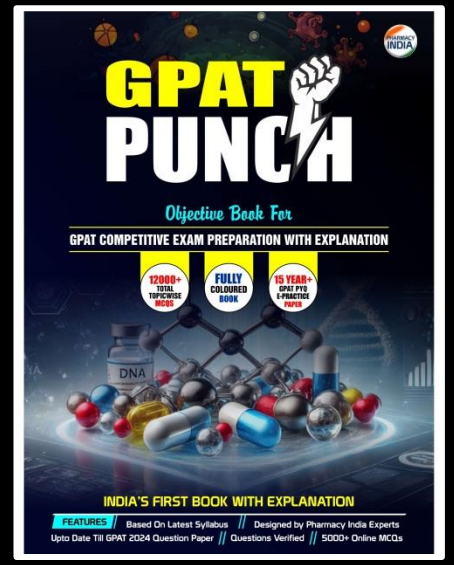


PHARMACY INDIA

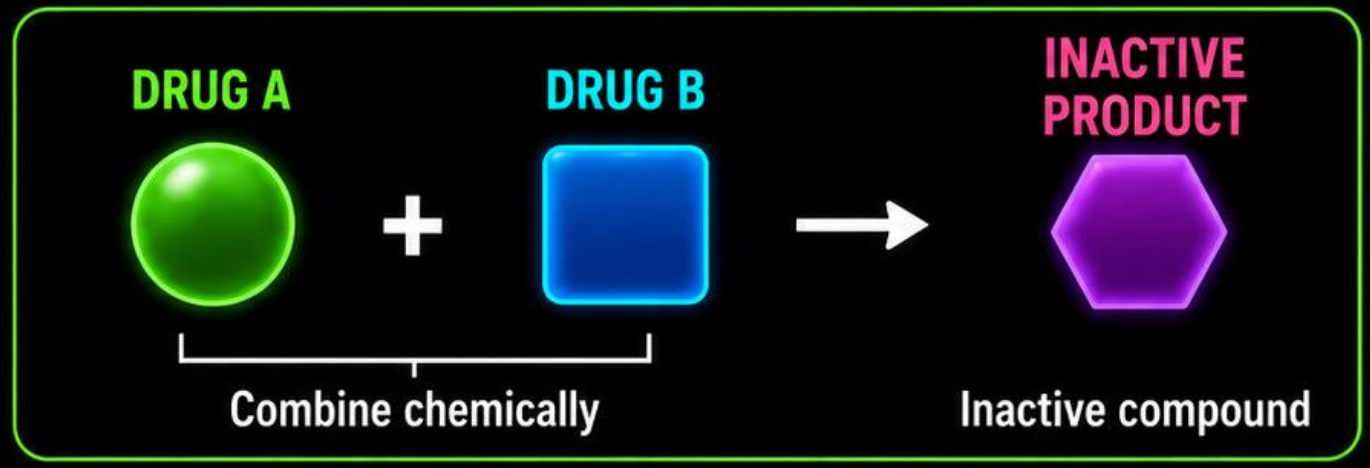
CHEMICAL ANTAGONISM

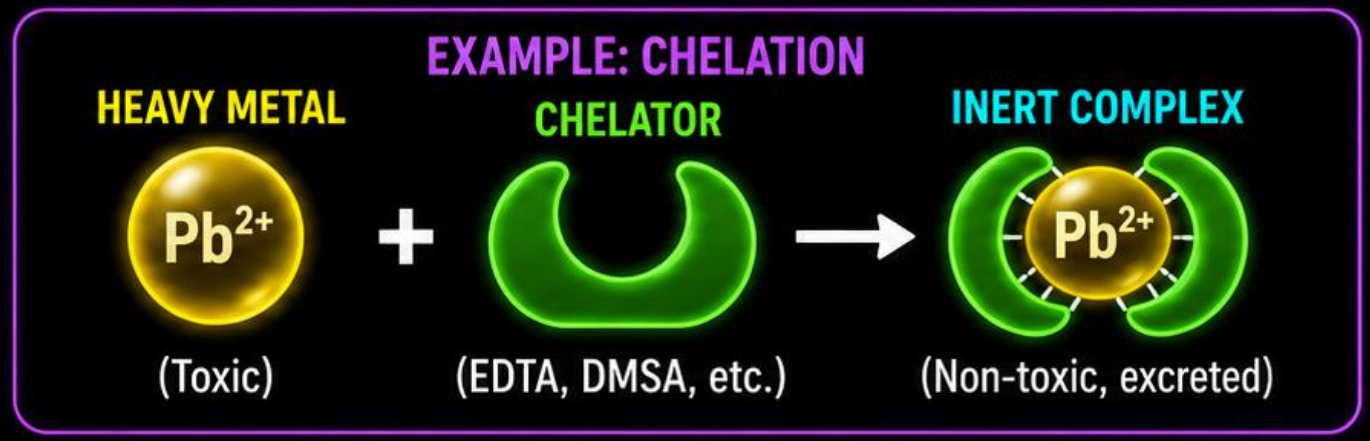
Two **substances** combine chemically.

The **reaction** forms an **inactive compound.**

No **receptor** is involved.

Example: chelators neutralize **heavy metals.**





Chemical antagonism reduces activity by **chemical inactivation, not receptor blockade.**

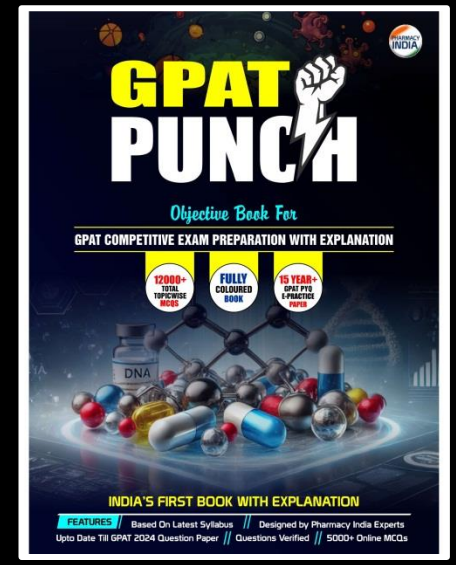
32. Antagonism between two drugs having opposite physiological effects at the same time is called:

(a) Physical antagonism

(b) Chemical antagonism

(c) Functional antagonism

(d) Competitive antagonism



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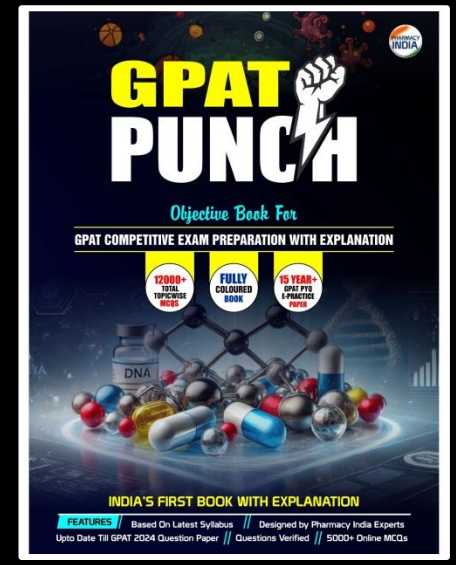


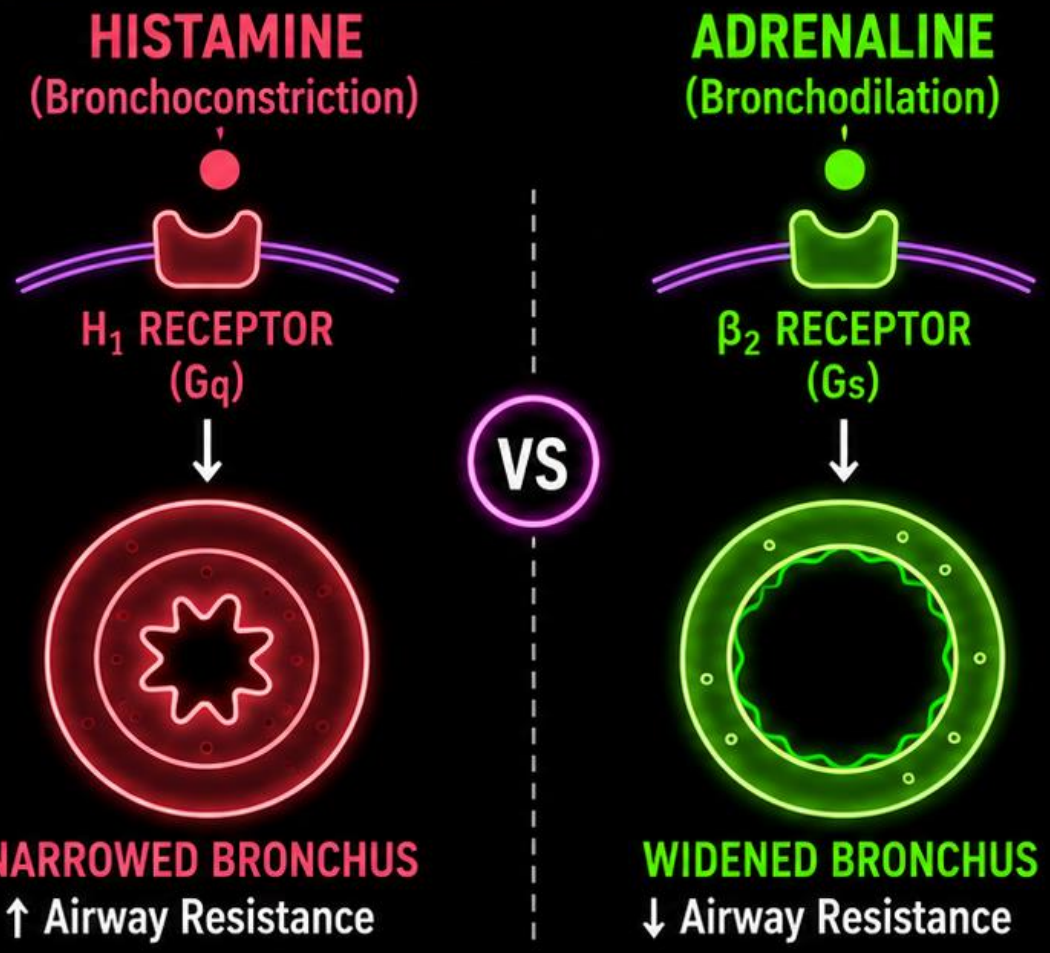
FUNCTIONAL ANTAGONISM

Two drugs act on **different receptors.**

Their physiological effects **oppose** each other.

Also called **physiological antagonism.**

Example: adrenaline opposes **histamine-induced bronchoconstriction.**



Different Receptors → Opposite Effects → Functional (Physiological) Antagonism

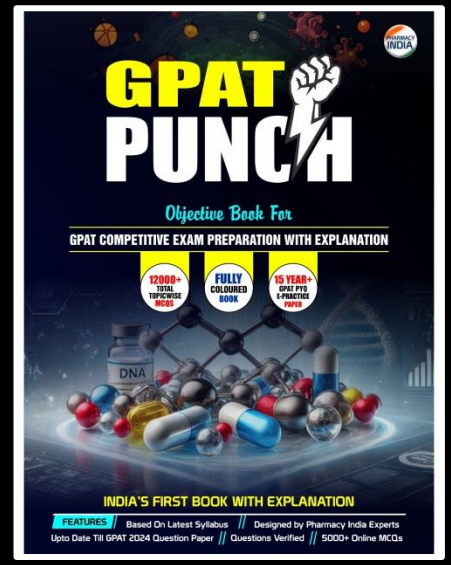
33. Drug antagonism between adrenaline and histamine is:

(a) Competitive

(b) Physiological

(c) Physical

(d) Chemical



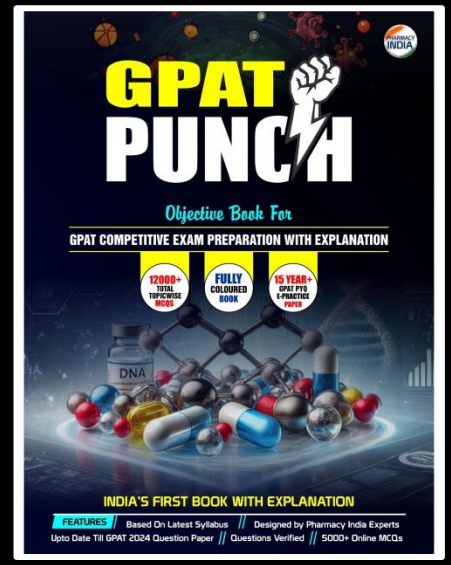
33. **Drug antagonism between adrenaline and histamine is:**

(a) Competitive

(b) Physiological

(c) Physical

(d) Chemical



ADRENALINE vs HISTAMINE

PHARMACY INDIA

They act on **different receptors.**

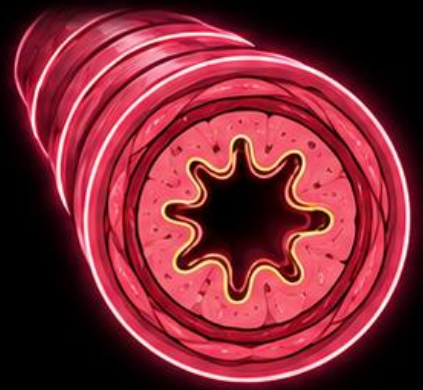
Histamine causes **bronchoconstriction.**

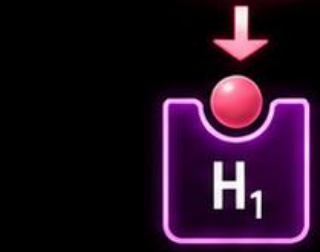
Adrenaline causes **bronchodilation.**

This is **physiological antagonism.**

HISTAMINE

Bronchoconstriction (NARROW AIRWAY)



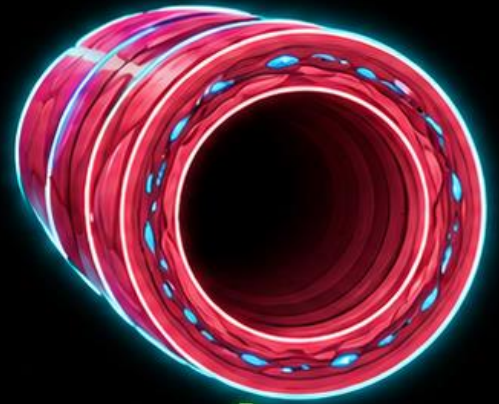


HISTAMINE RECEPTOR

VS

ADRENALINE

Bronchodilation (WIDE AIRWAY)



β_2 ADRENERGIC RECEPTOR

ADRENALINE (β_2) RELAXES AIRWAYS ⟷ HISTAMINE (H_1) CONSTRICTS AIRWAYS

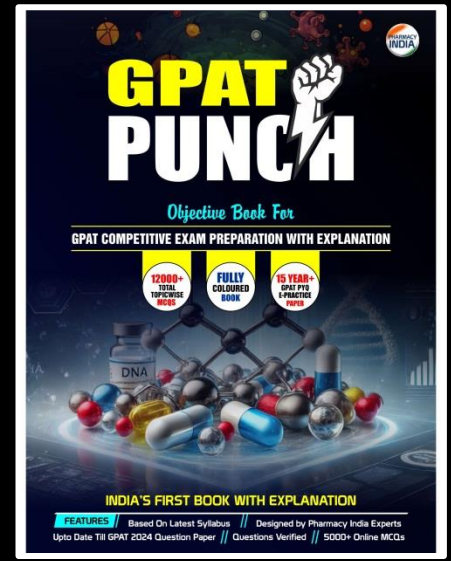
34. **Atropine in organophosphorus compound poisoning is an example of:**

(a) Physical antagonism

(b) Physiological antagonism

(c) Competitive receptor antagonism

(d) Non-competitive antagonism



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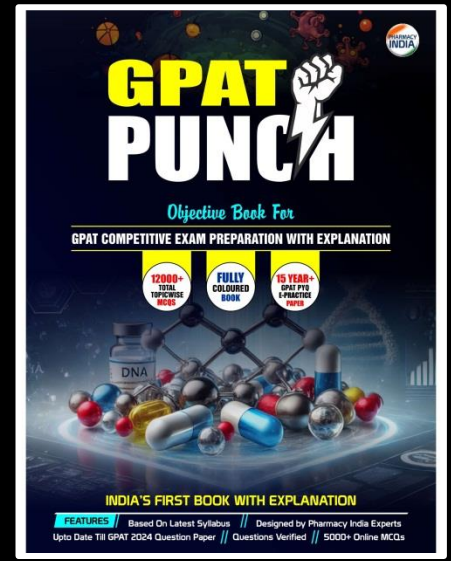


ATROPINE BLOCKS MUSCARINIC RECEPTORS

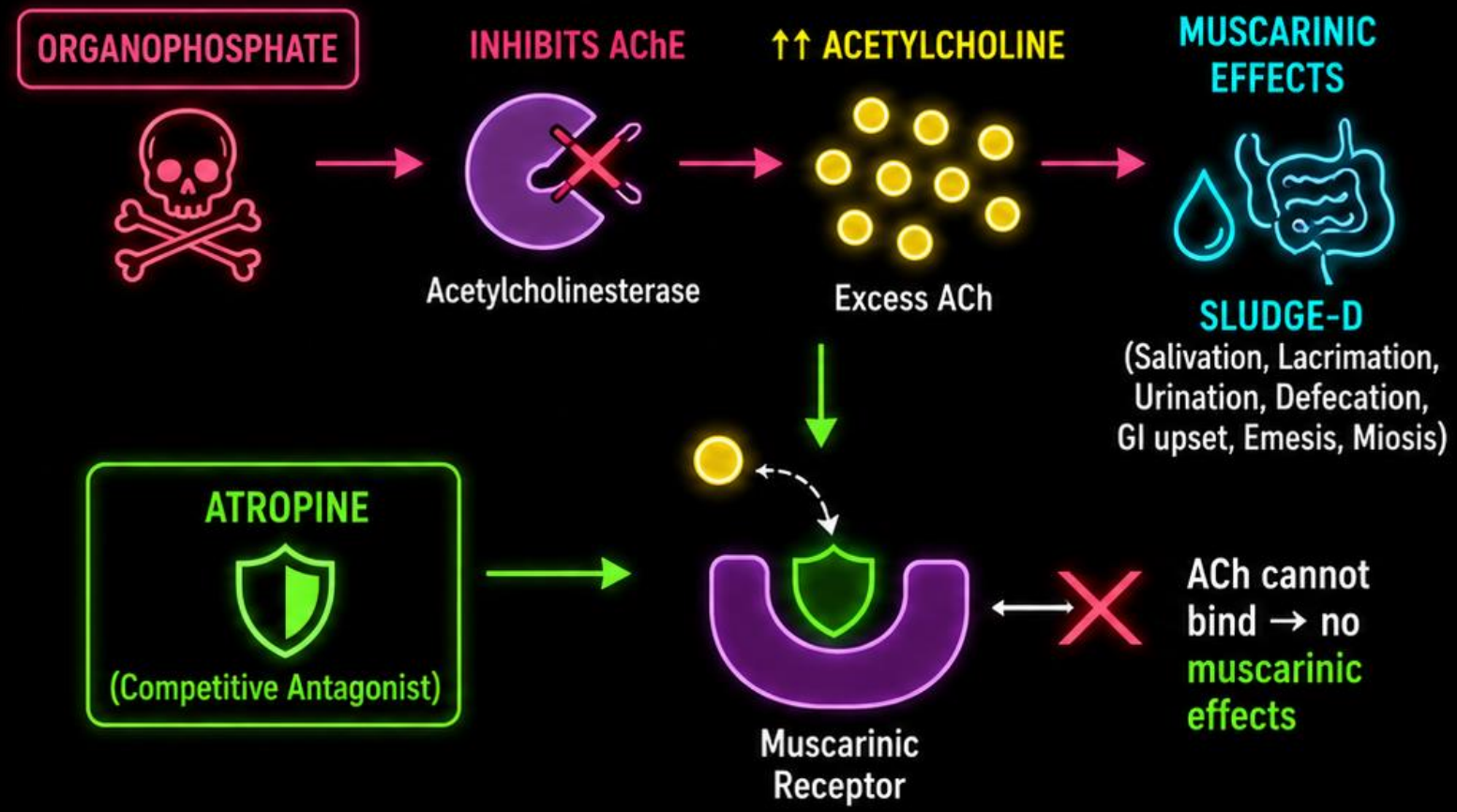
PHARMACY INDIA

Organophosphates increase acetylcholine.

Atropine competitively blocks muscarinic receptors.

It prevents acetylcholine action at muscarinic sites.

This is competitive receptor antagonism, not chemical antagonism.



ATROPINE COMPETITIVELY BLOCKS MUSCARINIC RECEPTORS → PREVENTS ACh ACTION IN ORGANOPHOSPHORUS POISONING

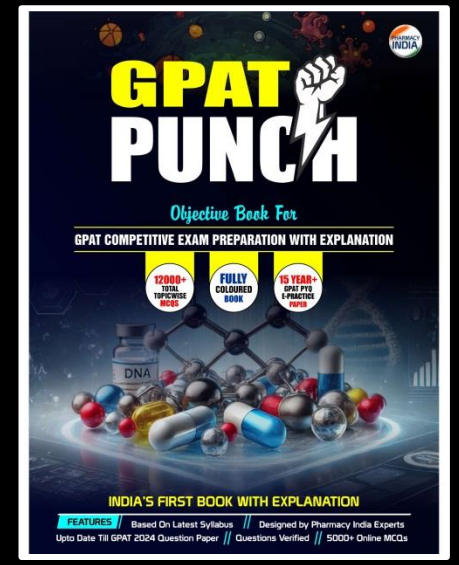
35. **The correct statement applicable to LD50 is:**

(a) The dose producing maximum response

(b) The dose which produces mortality in 50%

(c) The dose which produces desired effect in 50%

(d) The dose which is half of dose producing 100% mortality



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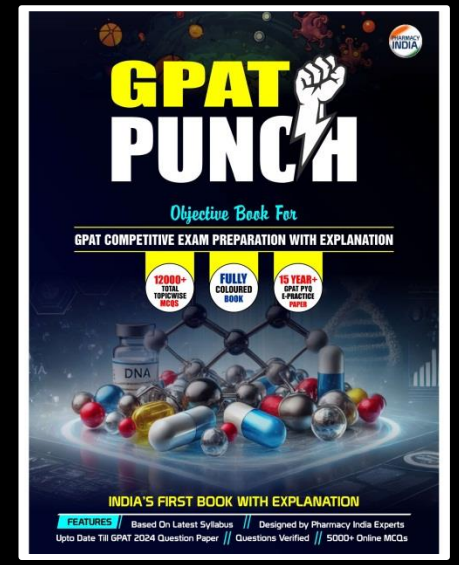
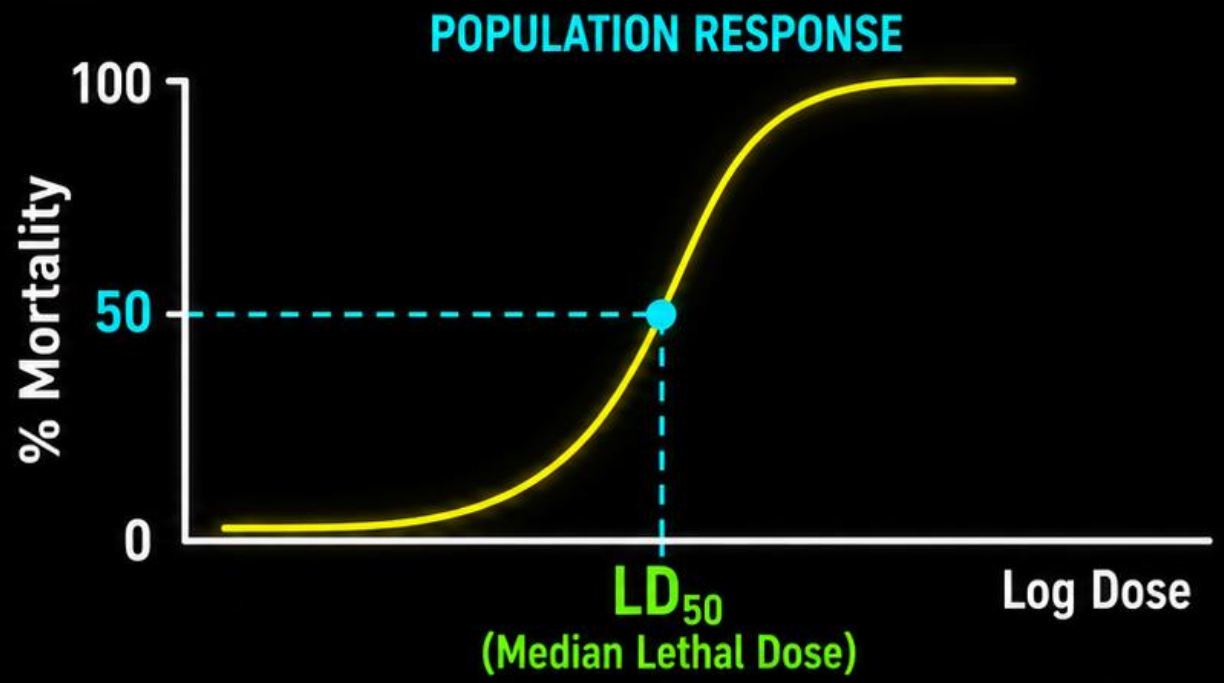


LD_{50} = MEDIAN LETHAL DOSE

PHARMACY INDIA

- LD_{50} causes death in 50% of test animals.
- Used in toxicity assessment.
- It reflects lethality, not therapeutic effect.
- ED_{50} refers to effect in 50% subjects.



50% SURVIVE

50% DIE

At LD_{50}, 50% of the population dies.

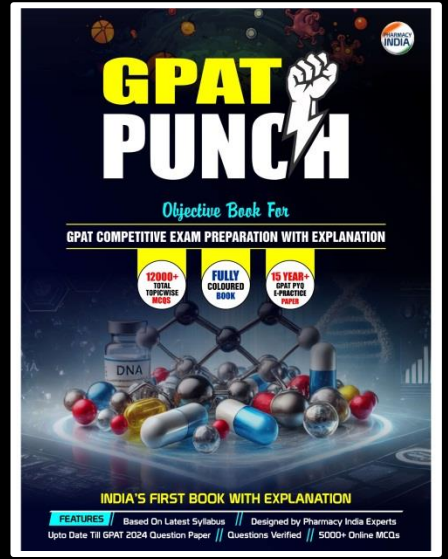
36. **The dose of following class of drugs has to be adjusted by repeated measurement of affected physiological parameter:**

(a) Oral contraceptives

(b) Antiepileptics

(c) Antidepressants

(d) Oral anticoagulants



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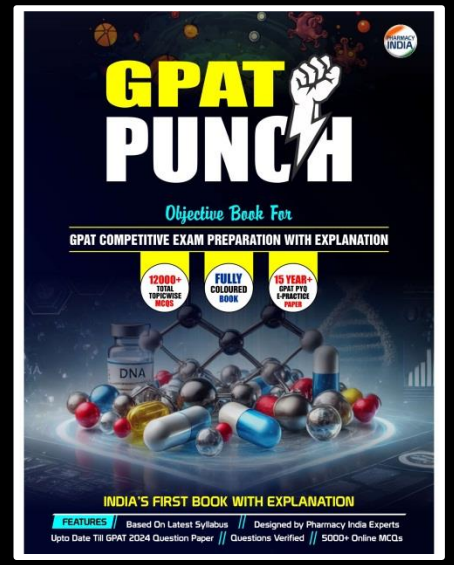
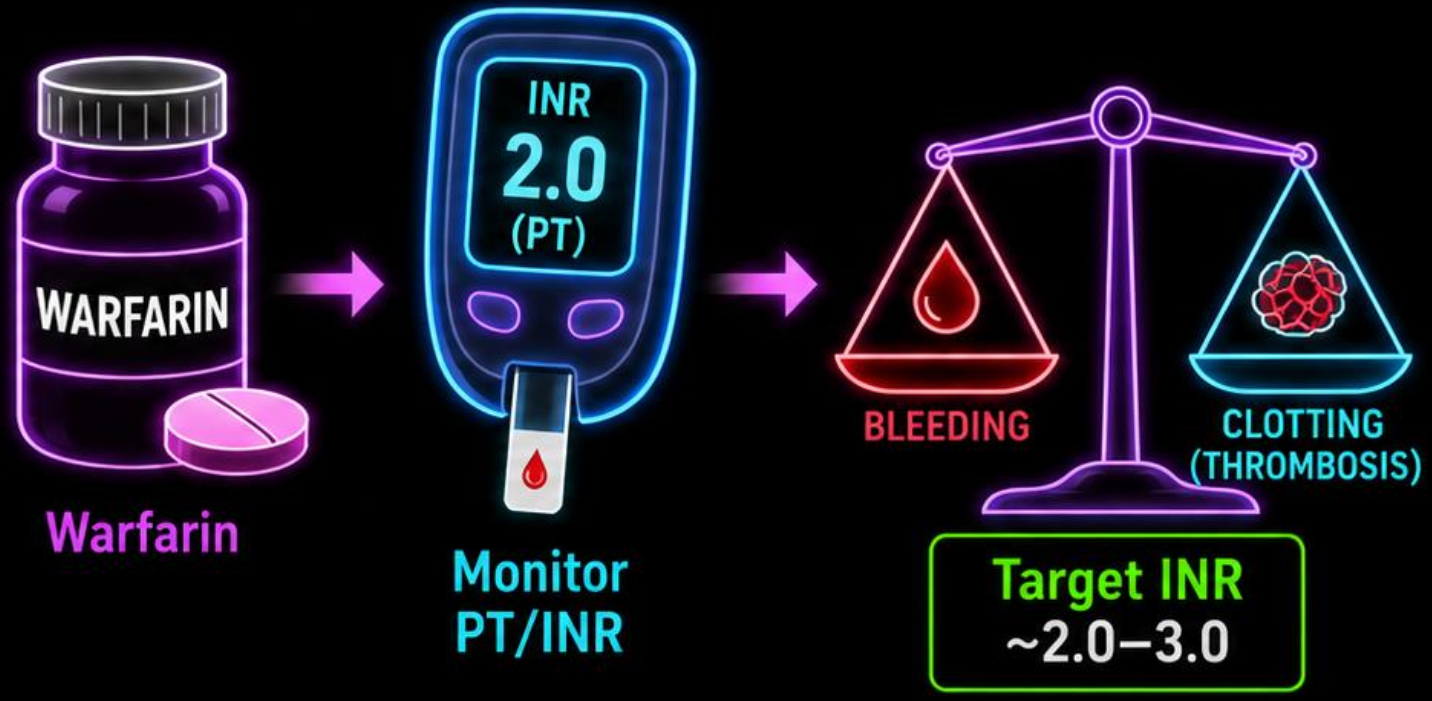


ORAL ANTICOAGULANTS NEED MONITORING

PHARMACY INDIA

- Dose is adjusted by **repeated physiological measurement.**
- Warfarin is monitored with **PT/INR.**
- High dose may cause **bleeding.**
- Low dose may fail to **prevent thrombosis.**



Goal: Keep anticoagulation in the **therapeutic window**
(Minimize **bleeding risk** while preventing **thrombosis**)

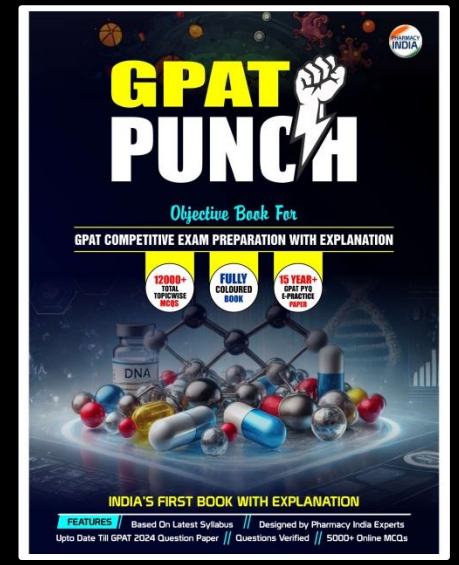
37. Phenomenon of tachyphylaxis can be seen with:

(a) Noradrenaline

(b) Adrenaline

(c) Phenylephrine

(d) Tyramine



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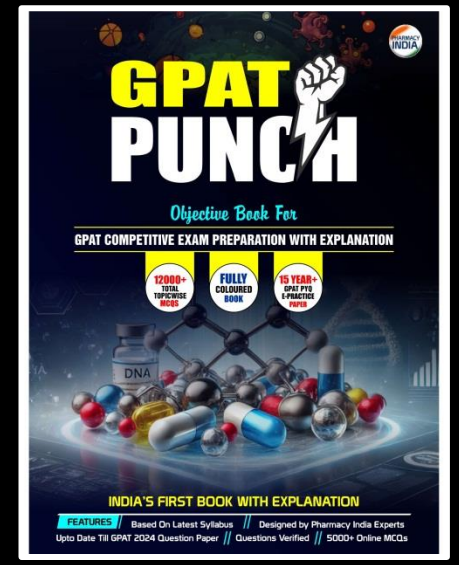


TYRAMINE CAUSES TACHYPHYLAXIS

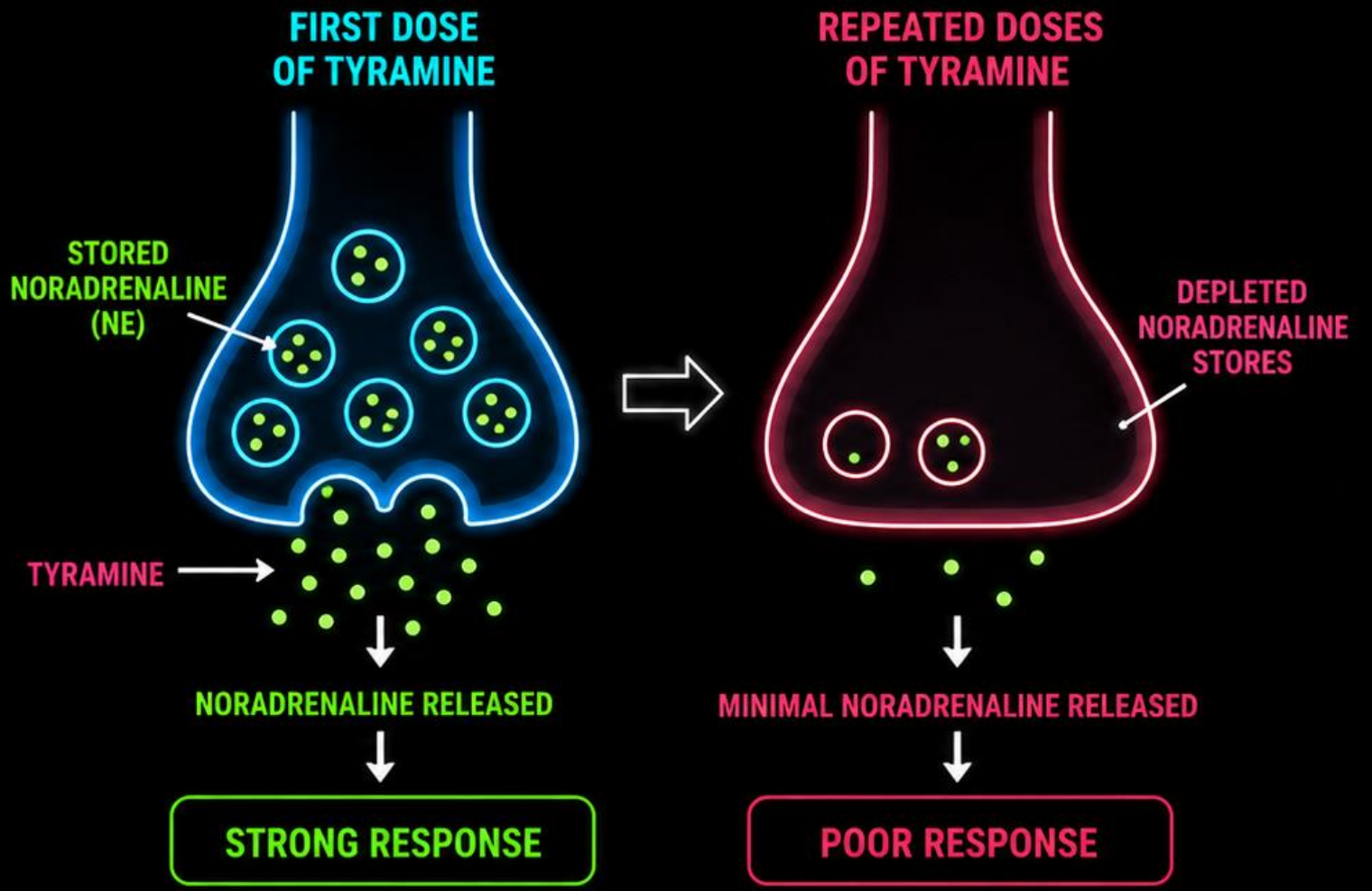
PHARMACY INDIA

1. Tyramine is an **indirectly acting** sympathomimetic.

2. It releases stored **noradrenaline.**

3. Repeated doses **deplete stores.**

4. Response falls rapidly = **tachyphylaxis.**



REPEATED TYRAMINE → DEPLETED NE STORES → RAPIDLY FALLING RESPONSE = TACHYPHYLAXIS

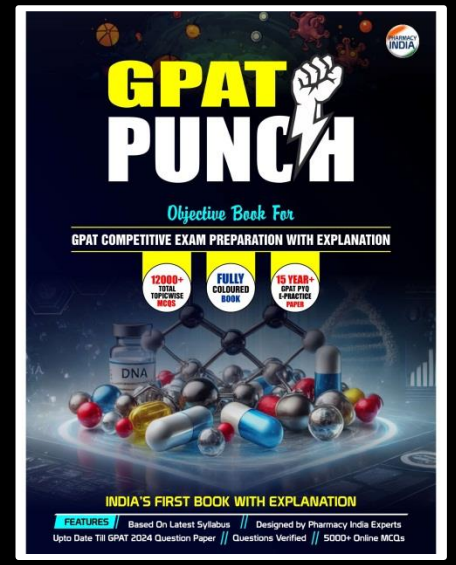
38. Pharmacogenetics deals with:

(a) Clinically significant genetically mediated variations in drug response

(b) Harmful effects on foetus

(c) Drug-induced genetic defect

(d) Development of drugs by DNA recombinant technology



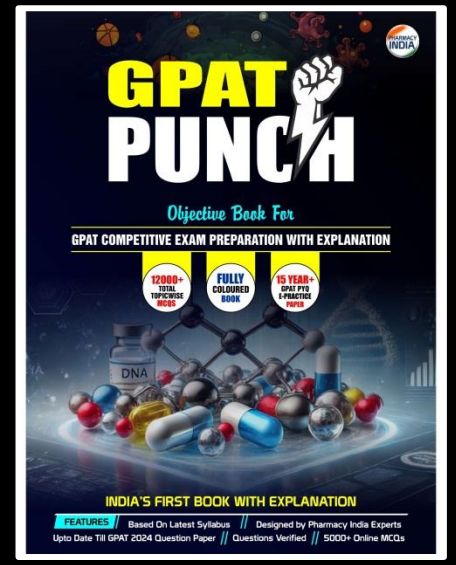
38. **Pharmacogenetics deals with:**

(a) Clinically significant genetically mediated variations in drug response

(b) Harmful effects on foetus

(c) Drug-induced genetic defect

(d) Development of drugs by DNA recombinant technology



PHARMACOGENETICS

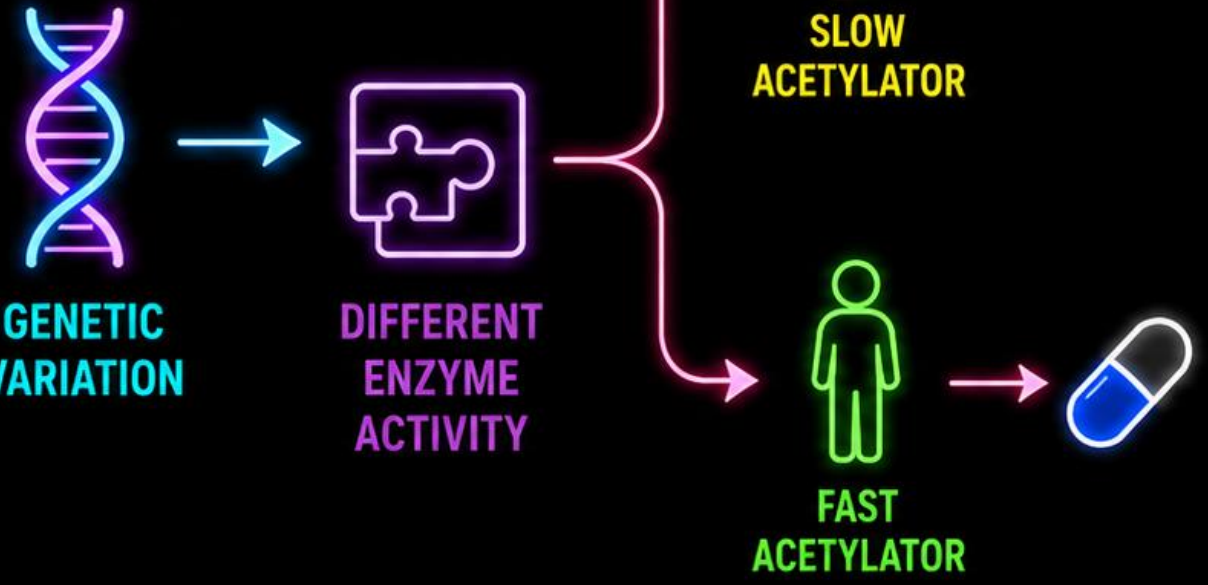
PHARMACY INDIA

Studies genetically mediated variation in drug response.

Explains differences in efficacy and toxicity.

Enzyme polymorphism alters metabolism.

Example: slow and fast acetylators of isoniazid.



RIGHT DRUG, RIGHT DOSE, RIGHT PATIENT
BASED ON YOUR GENES.

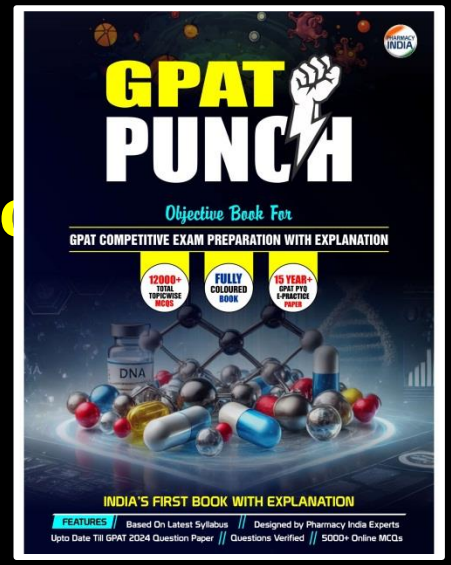
39. **Drug tolerance due to decreased availability of drug at receptor site because of hepatic enzyme induction is kn as:**

(a) Cellular tolerance

(b) Drug dispositional tolerance

(c) Physiological tolerance

(d) Pseudo tolerance



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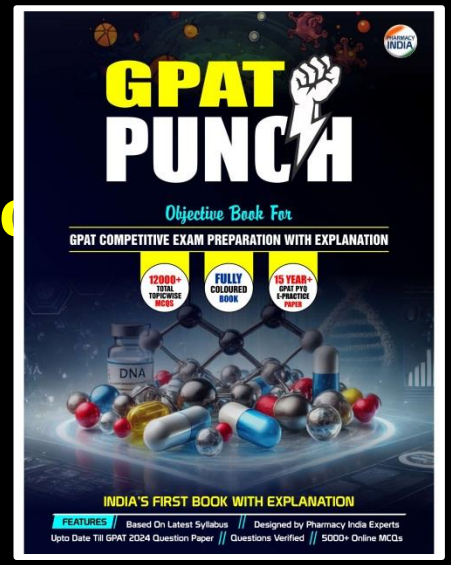


DISPOSITIONAL TOLERANCE

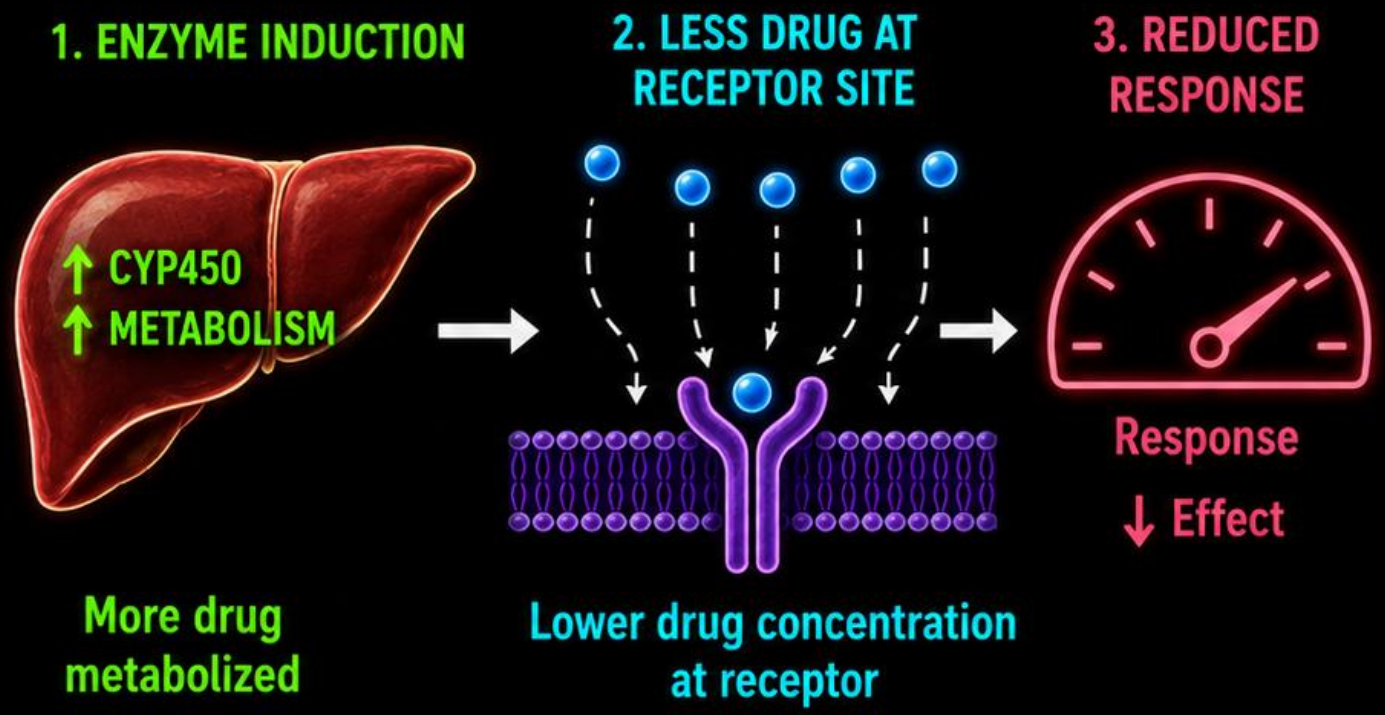
PHARMACY INDIA

Also called **pharmacokinetic tolerance.**

Hepatic enzyme **induction** increases **drug metabolism.**

Less drug **reaches** the **receptor site.**

Higher dose is needed for the same effect.



Enzyme Induction ↑ → **↑ Drug Metabolism** → **↓ Drug at Receptor Site** → **↓ Response** → **Higher Dose Needed**

Key Idea: Tolerance due to changes in **drug disposition**, not receptor sensitivity.

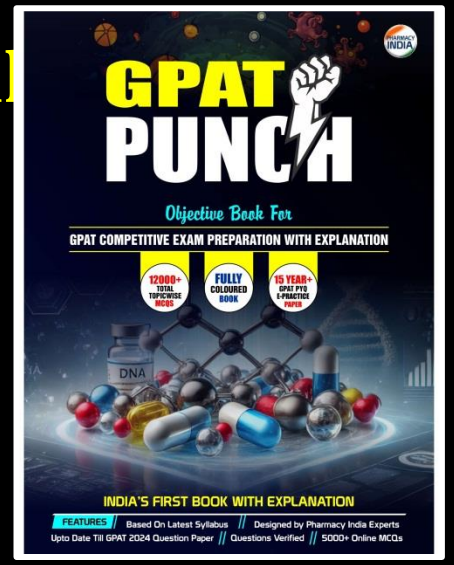
40. When there is gradual decrease in response to a drug ta
days or weeks to develop, the phenomenon is called:

(a) Desensitization

(b) Tachyphylaxis

(c) Tolerance

(d) Idiosyncrasy



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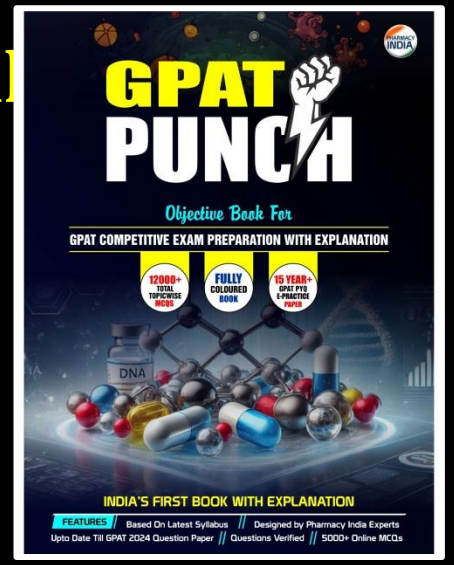
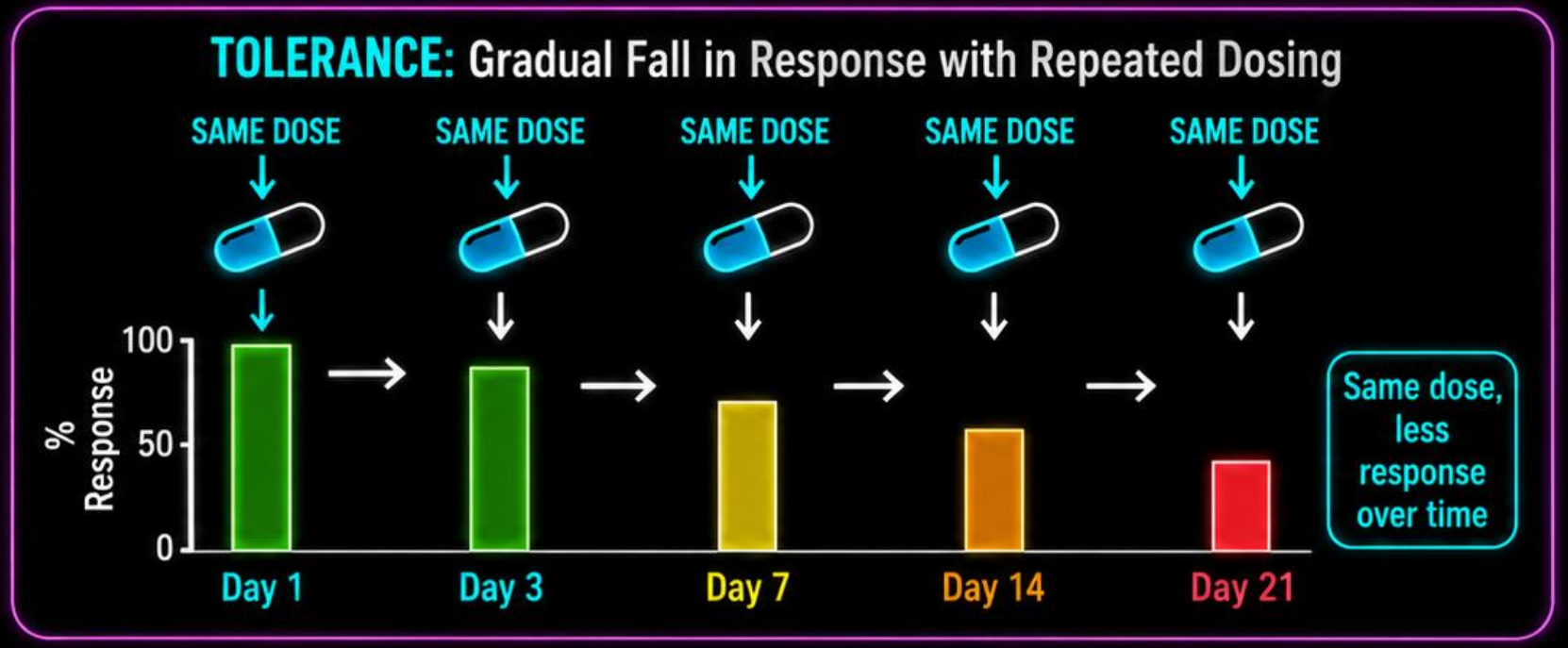


TOLERANCE DEVELOPS GRADUALLY

PHARMACY INDIA

1. Response decreases over days or weeks.
2. The same dose gives less effect.
3. A higher dose is needed for the same response.
4. It is slower than tachyphylaxis.



TOLERANCE = GRADUAL ↓ in response → Higher dose needed

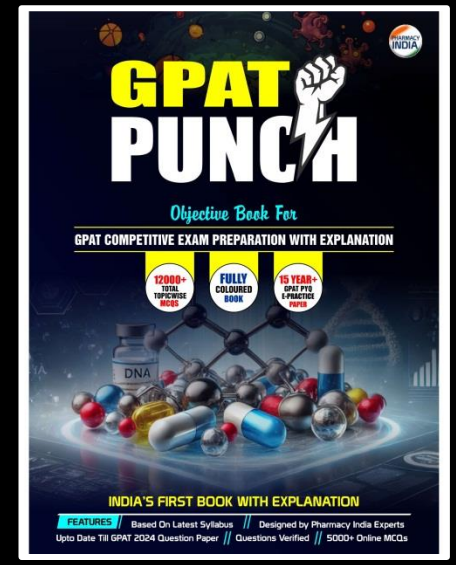
41. **When an inert chemical produces psychopharmacological effect, it is called:**

(a) Prodrug

(b) Placebo

(c) Idiosyncratic response

(d) Adjuvant



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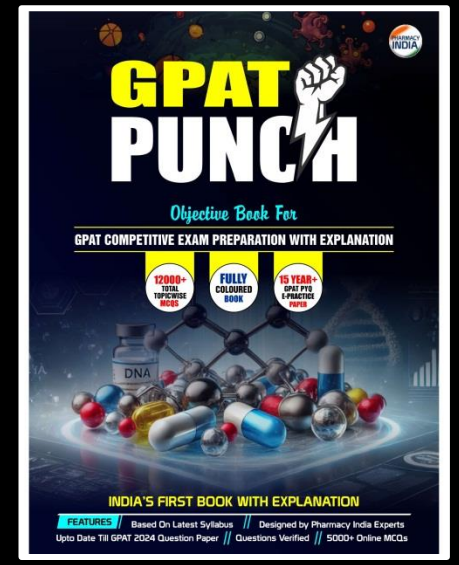
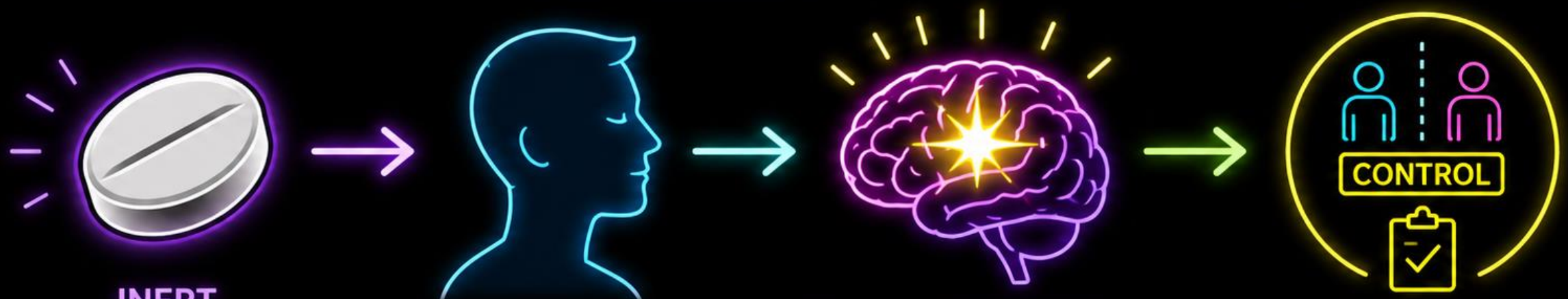


PLACEBO EFFECT



INERT SUBSTANCE
(Pharmacologically Inert)

PATIENT
(Expectation Activated)

BRAIN & EXPECTATION
Mind–Body Response Triggers Beneficial Effects

CLINICAL TRIALS
Used as a Control to Compare Efficacy

- Placebo is a **pharmacologically inert** substance.
- It can still produce a **perceived psychopharmacological** or **therapeutic effect.**
- The effect depends on **patient expectation** and **mind–body response.**
- It is important as a **control** in **clinical trials.**

42. Which of the following is a JAK-STAT linked receptor?

(a) Prolactin

(b) Estrogen

(c) TSH

(d) TRH

42. **Which of the following is a JAK-STAT linked receptor?**

(a) Prolactin

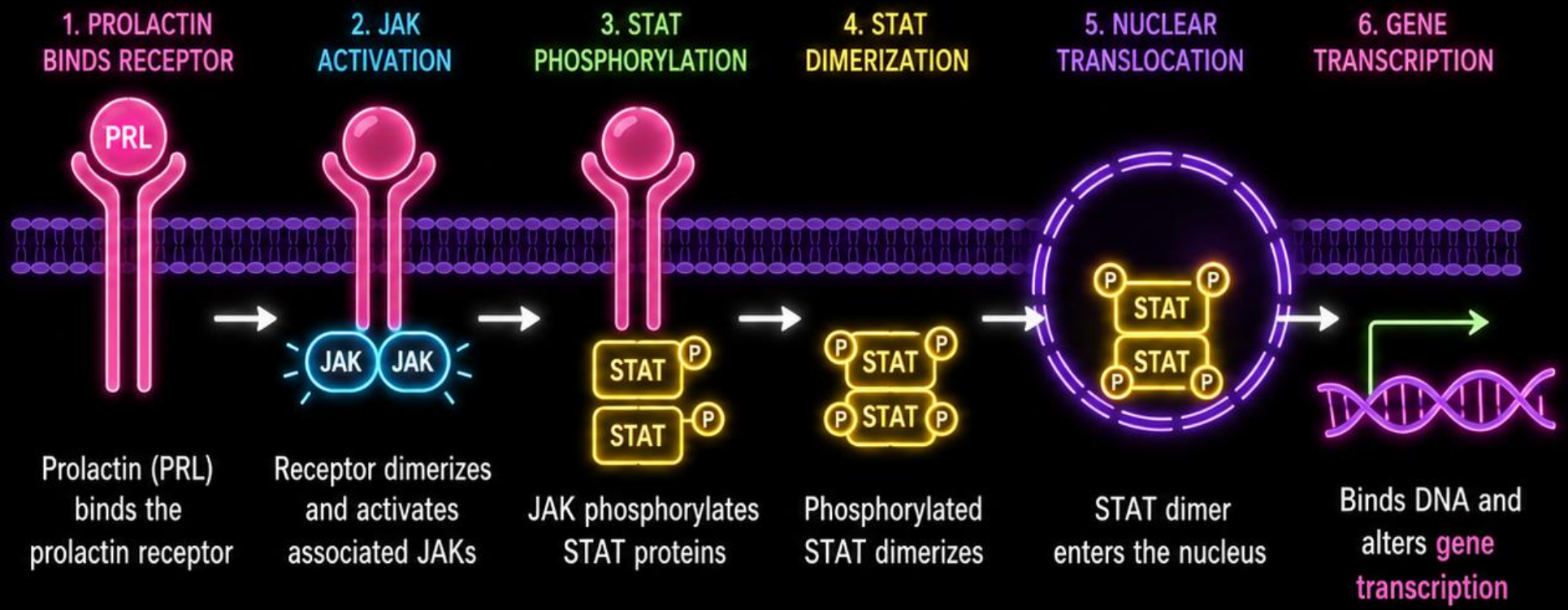
(b) Estrogen

(c) TSH

(d) TRH

PROLACTIN RECEPTOR = JAK–STAT

- Prolactin receptor signals through the JAK–STAT pathway.
- Ligand binding activates JAK.
- JAK phosphorylates STAT proteins.
- Phosphorylated STAT enters the nucleus and alters gene expression.



SIDE NOTE: Estrogen action is mainly nuclear-receptor mediated. | TSH and TRH mainly use GPCR pathways.

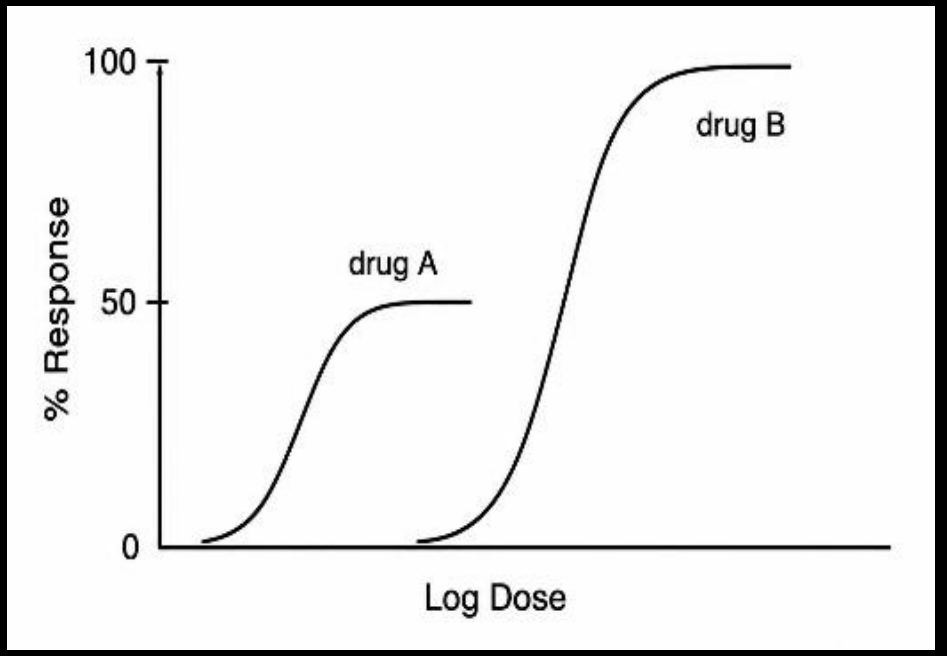
43. **The curves in the figure represent isolated tissue responses to two drugs. Which statement about efficacy is accurate?**

(a) Drug A has greater efficacy than drug B

(b) Drug A is more potent than drug B

(c) Drug B is more potent than drug A

(d) Drug B has greater efficacy than drug A



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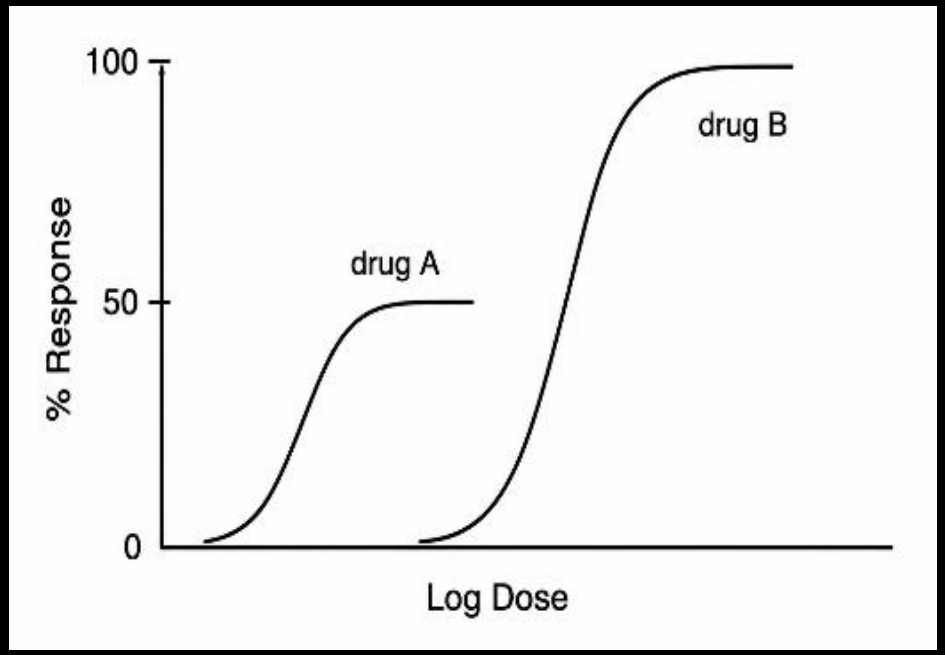
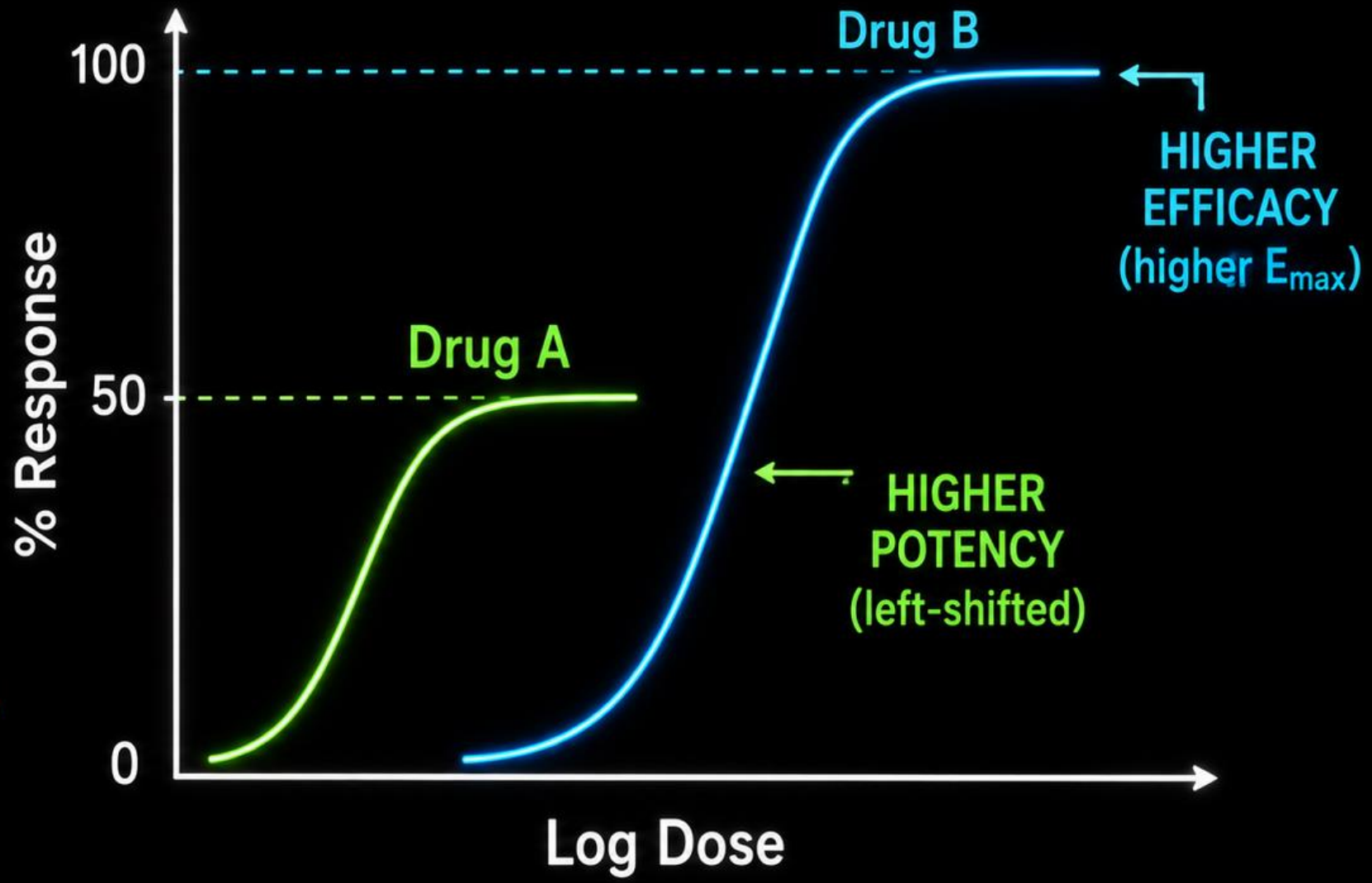


DRUG B HAS GREATER EFFICACY

1. Efficacy = maximum response a drug can produce.
2. Drug B reaches a higher maximum response than Drug A.
3. Drug A is left-shifted, so it is more potent.
4. For efficacy comparison, Drug B is superior to Drug A.



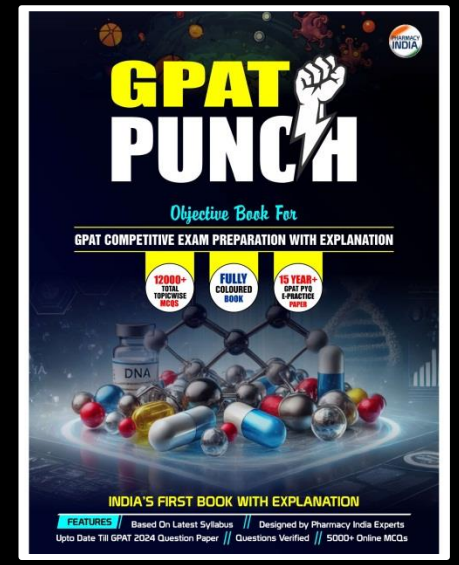
44. **Which receptor is NOT a G-protein coupled receptor?**

(a) 5-HT

(b) D2

(c) GABA-A

(d) GABA-B



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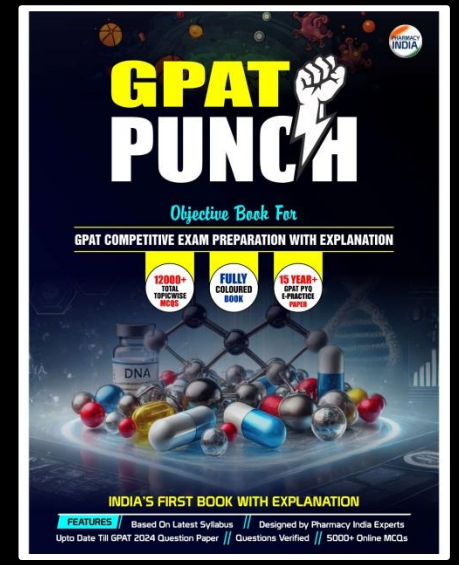
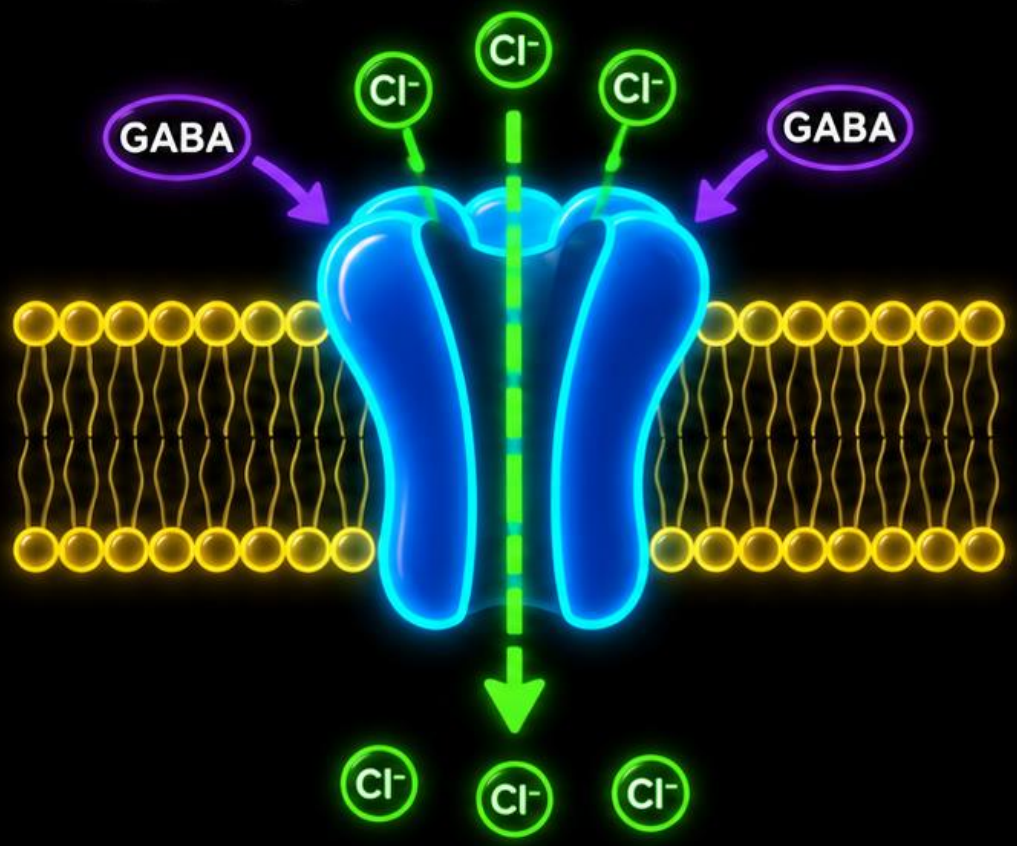


GABA-A IS AN ION CHANNEL

GABA-A (ION CHANNEL)

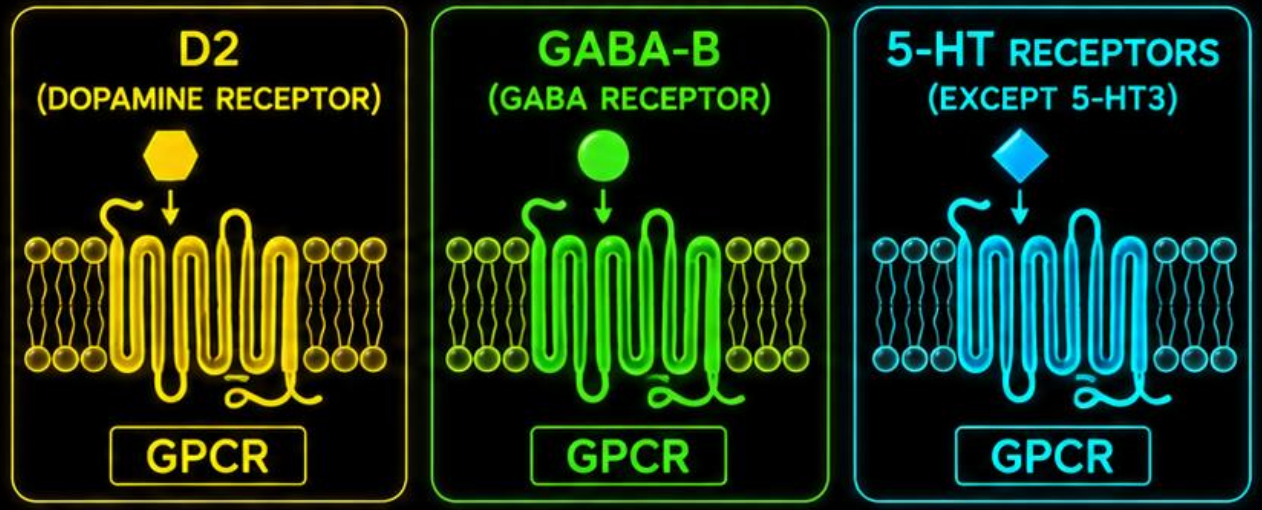
Ligand-gated chloride ion channel



Cl⁻ FLOWS IN → INHIBITION

- GABA-A is a ligand-gated chloride ion channel.
- It is not a G-protein coupled receptor.
- D2 and GABA-B are GPCRs.
- Most 5-HT receptors are GPCRs except 5-HT3.

COMPARISON: GPCRs (not ion channels)



GPCRs signal via G-proteins → second messengers

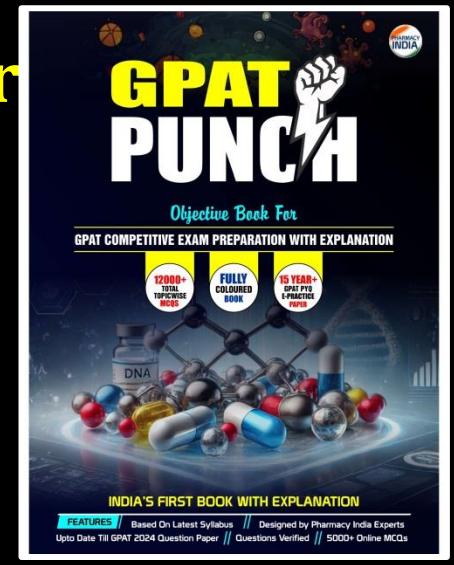
45. **A rapid reduction in effect of a given dose of a drug after only one or two doses is called:**

(a) Supersensitivity

(b) Tachyphylaxis

(c) Tolerance

(d) Hyposensitivity



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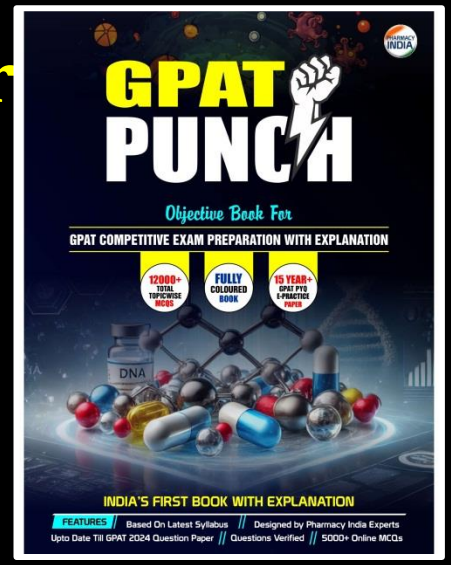
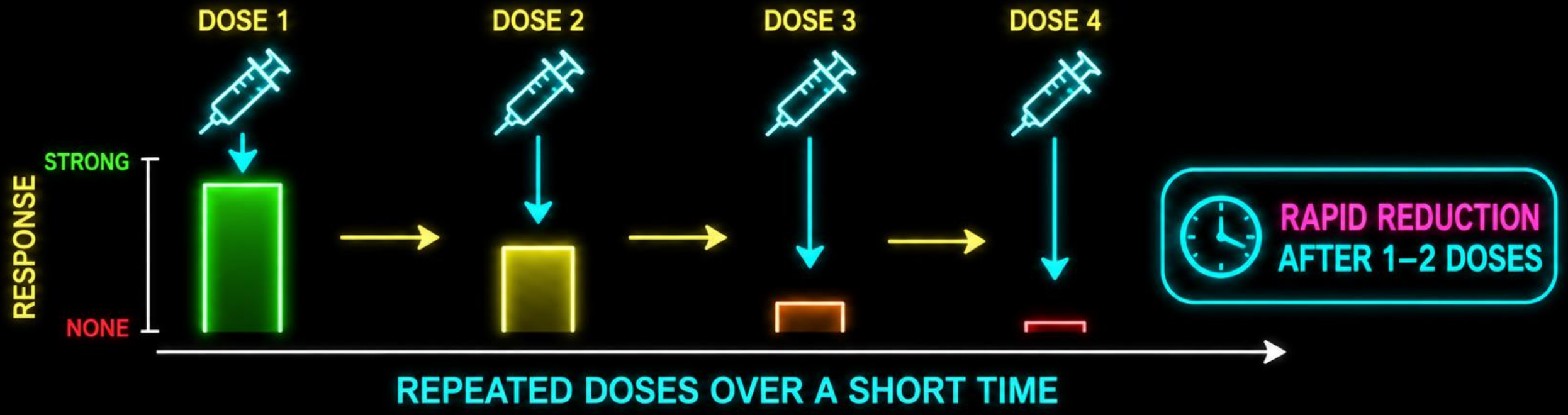


TACHYPHYLAXIS = RAPID FALL IN RESPONSE

PHARMACY INDIA

- **Tachyphylaxis** is **acute tolerance**.
- It develops after **one or a few** repeated doses.
- Response **falls rapidly**.
- Common with indirectly acting sympathomimetics such as **tyramine**.



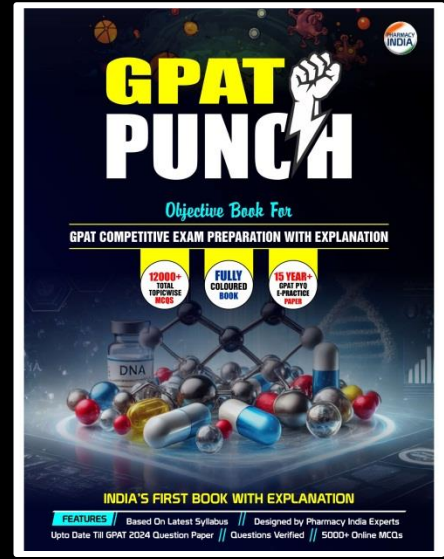
46. **True statement regarding inverse agonists is:**

(a) Binds to receptor and causes intended action

(b) Binds to receptor and causes opposite action

(c) Binds to receptor and causes no action

(d) Binds to receptor and causes submaximal action



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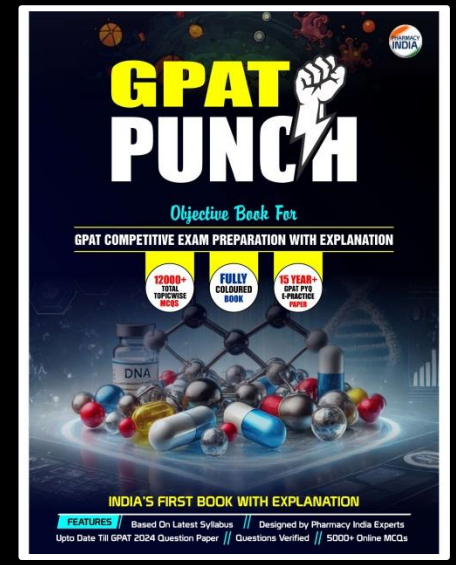
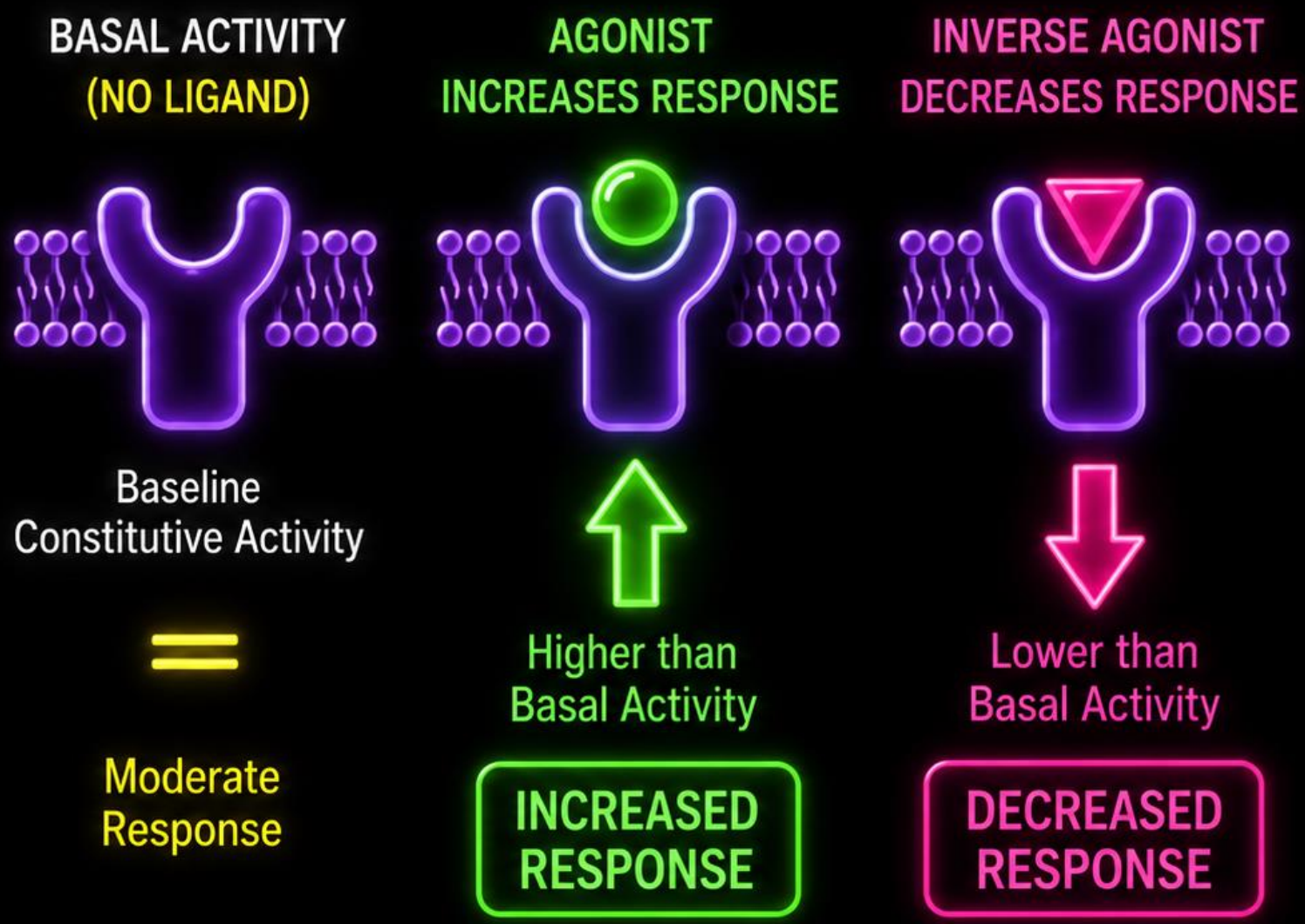


INVERSE AGONIST CAUSES OPPOSITE ACTION

PHARMACY INDIA

- **Inverse agonists** bind to the receptor.
- They **reduce** basal or constitutive receptor activity.
- Their effect is **opposite** to an agonist.
- **Neutral antagonists** bind but **do not change** basal activity.



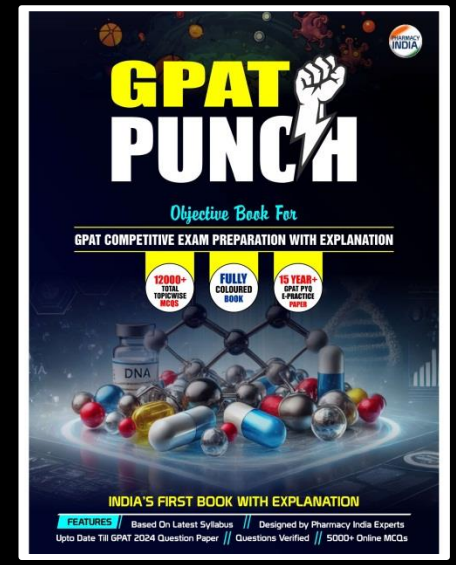
47. **True statement regarding dose-response curve is:**

(a) Cannot determine the potency of a drug

(b) Log dose-response curve is sigmoid shaped

(c) Cannot find response to antagonist

(d) A wide range of doses cannot be plotted



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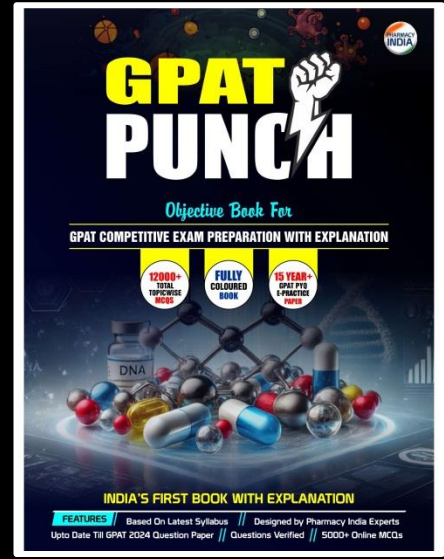


LOG DOSE–RESPONSE CURVE IS SIGMOID

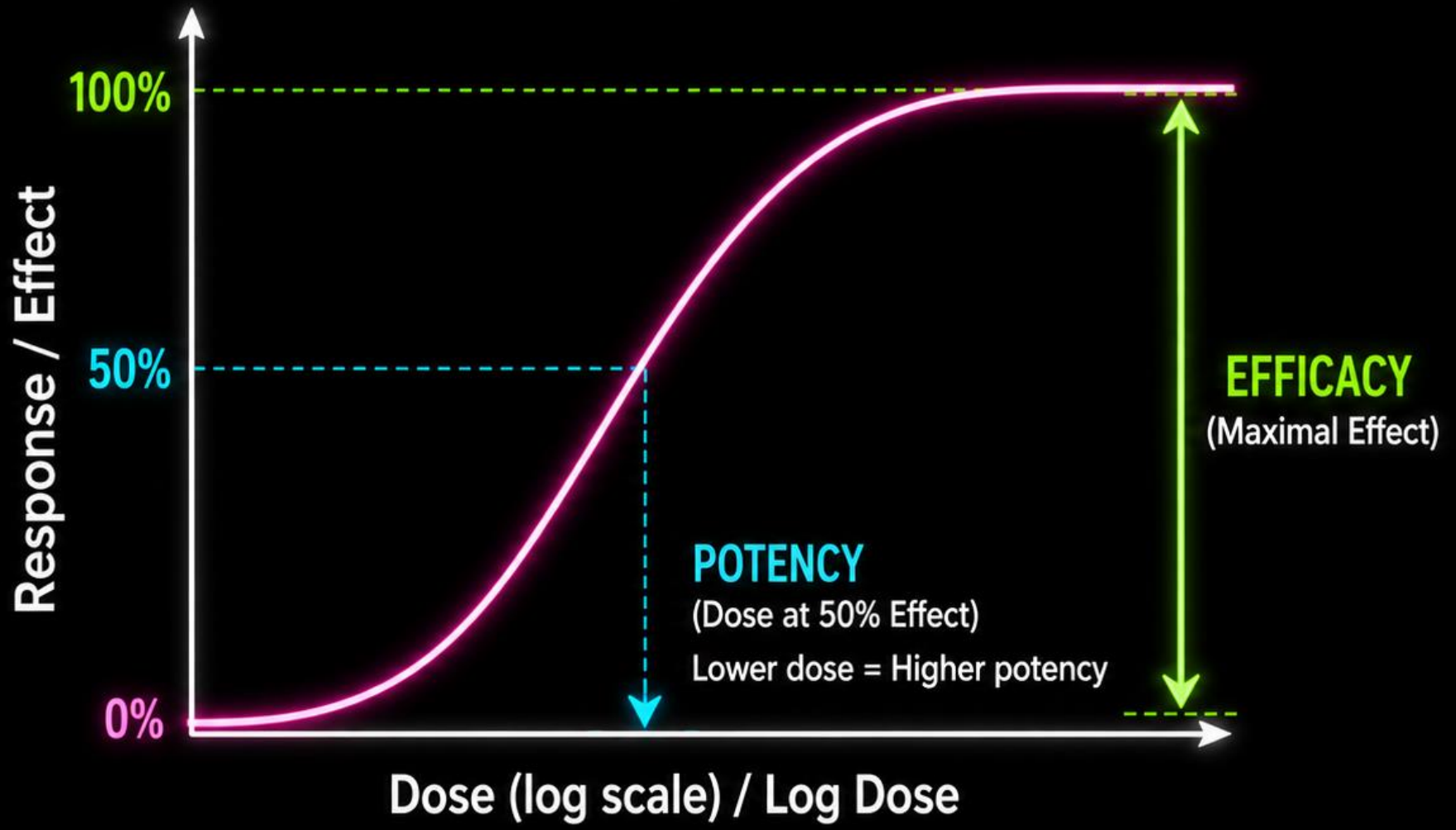
PHARMACY INDIA

A dose-response curve shows the relation between dose and effect.

On a log scale, the curve is sigmoid shaped.

It helps compare potency and efficacy.

It is useful for studying agonists and antagonists.



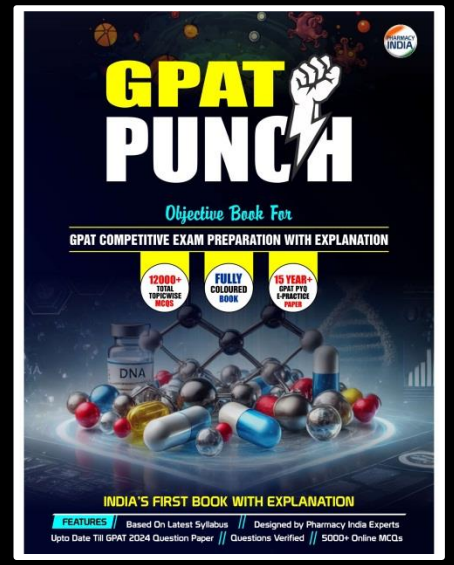
48. **True about G-protein coupled receptors is:**

(a) G-proteins bind to hormones on the cell surface

(b) All three subunits alpha, beta and gamma should bind to each other for G-proteins to act

(c) G-proteins act as inhibitory and excitatory because of difference in alpha subunit

(d) G-protein is bound to GTP in resting state



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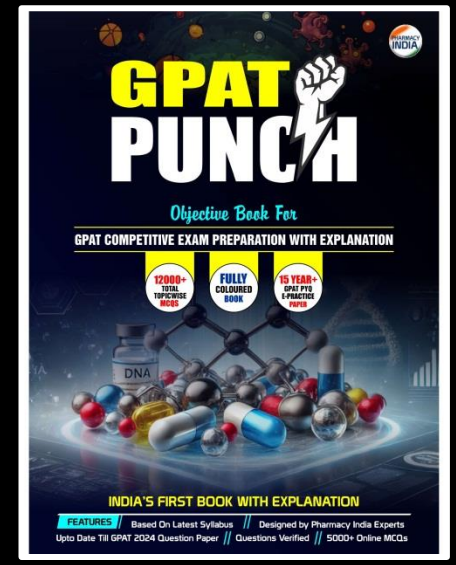
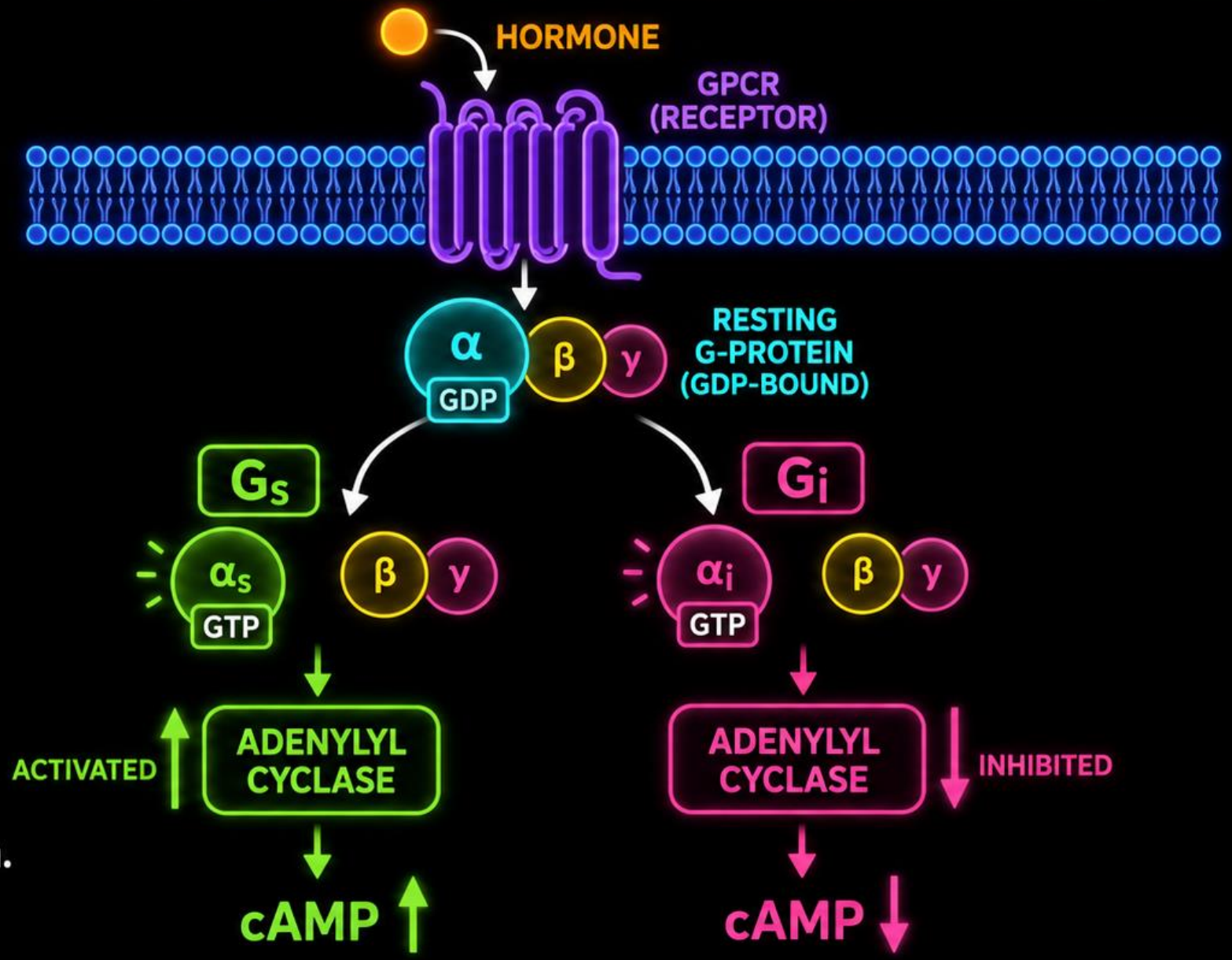


G-PROTEIN α SUBUNIT DECIDES RESPONSE

- G_s alpha stimulates adenylyl cyclase.
- G_i alpha inhibits adenylyl cyclase.
- Resting G-protein is GDP-bound, not GTP-bound.
- Hormone binds the receptor, not directly the G-protein.



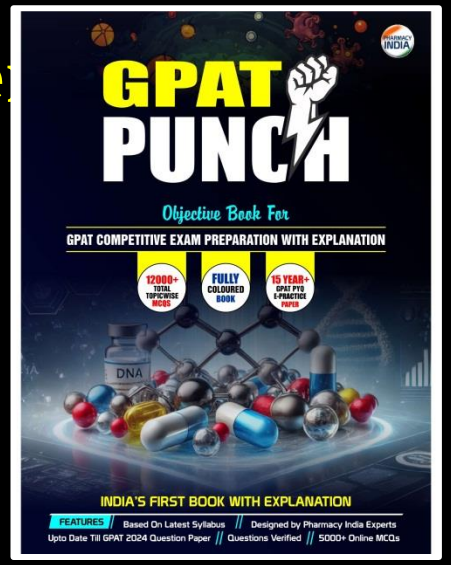
49. **Hormonal stimulation of formation of second messenger inositol 1,4,5-triphosphate quickly releases which intracellular messenger?**

(a) Cyclic AMP

(b) Prostaglandin

(c) Calcium

(d) Leukotriene



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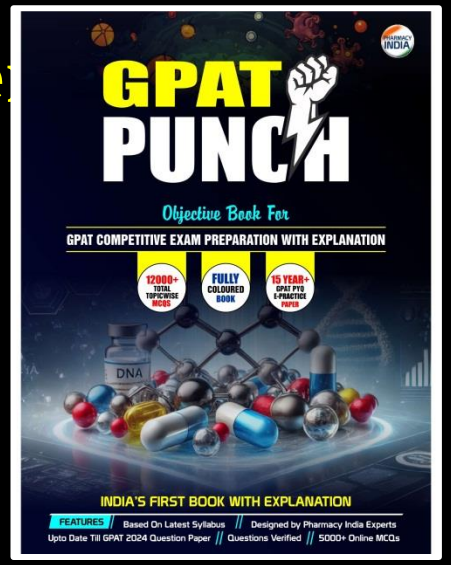
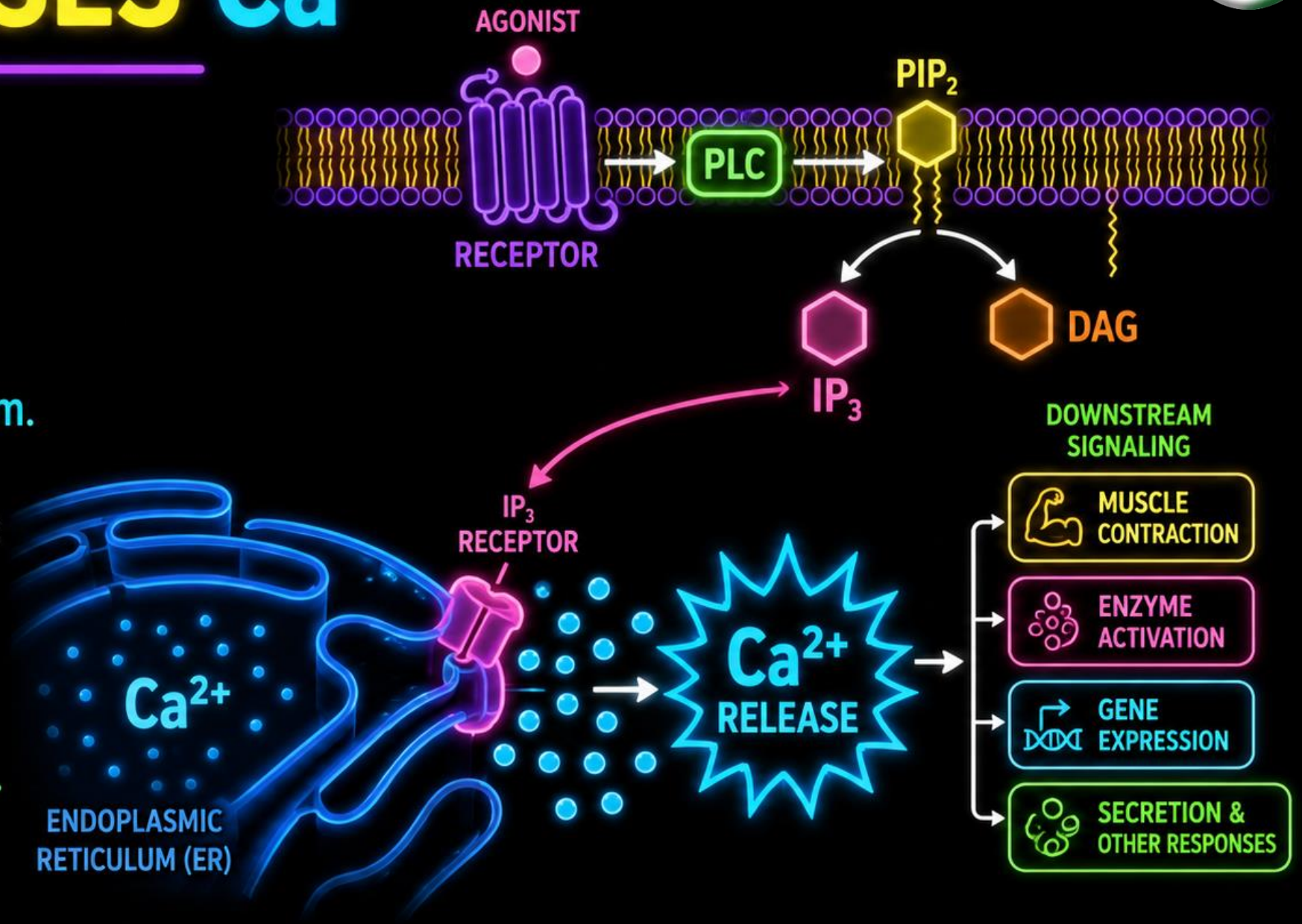


IP_3 RELEASES Ca^{2+}

1. IP_3 is formed from PIP_2 by phospholipase C.
2. IP_3 acts on receptors on the endoplasmic reticulum.
3. This causes rapid release of intracellular calcium.
4. Calcium then triggers many cellular responses.



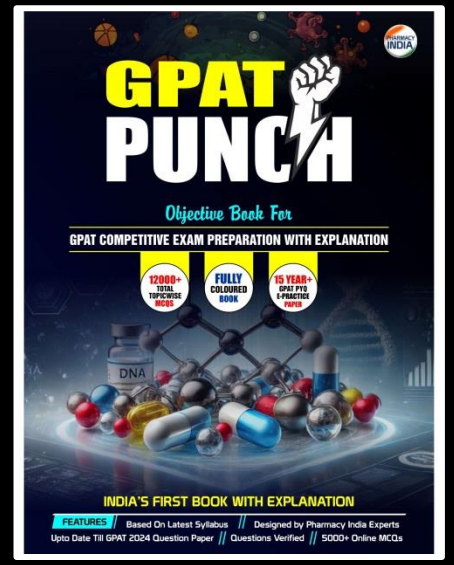
50. **Steroid hormones mainly act through which type of receptor?**

(a) Ligand-gated ion channel receptor

(b) G-protein coupled receptor

(c) Nuclear receptor

(d) JAK-STAT receptor



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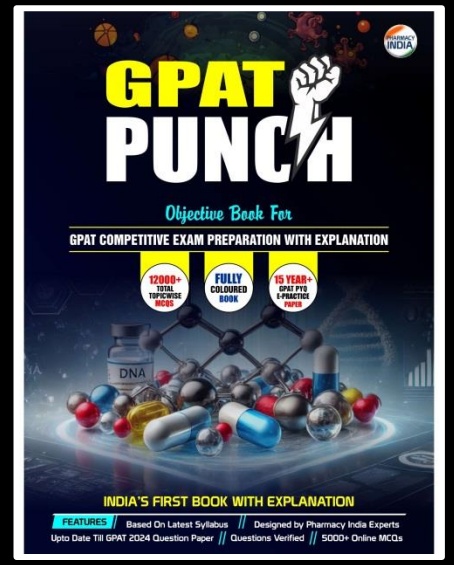
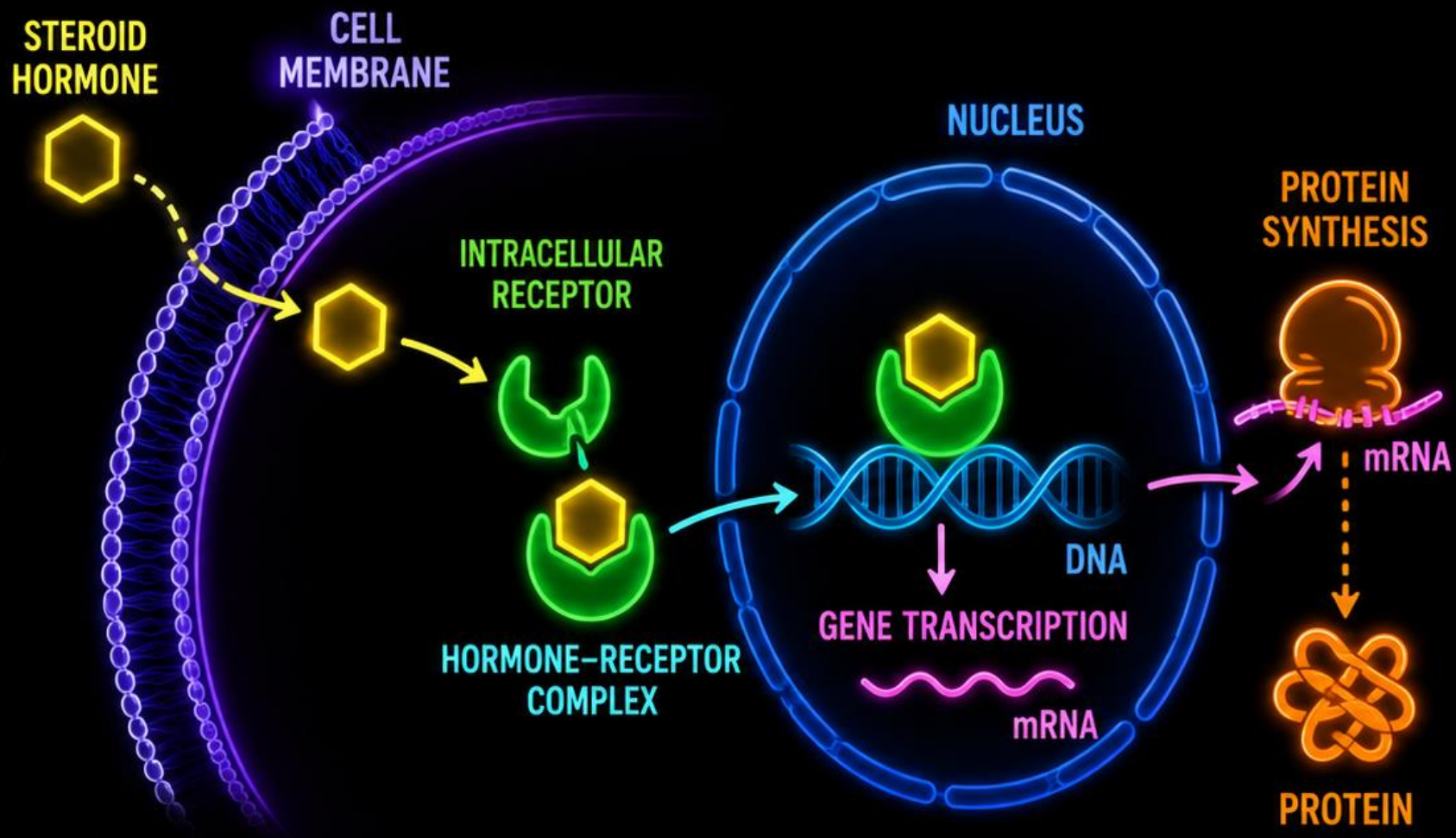


STEROID HORMONES ACT ON NUCLEAR RECEPTORS

1. Steroid hormones are lipid-soluble.
2. They cross the cell membrane easily.
3. They bind intracellular or nuclear receptors.
4. They regulate gene transcription and protein synthesis.



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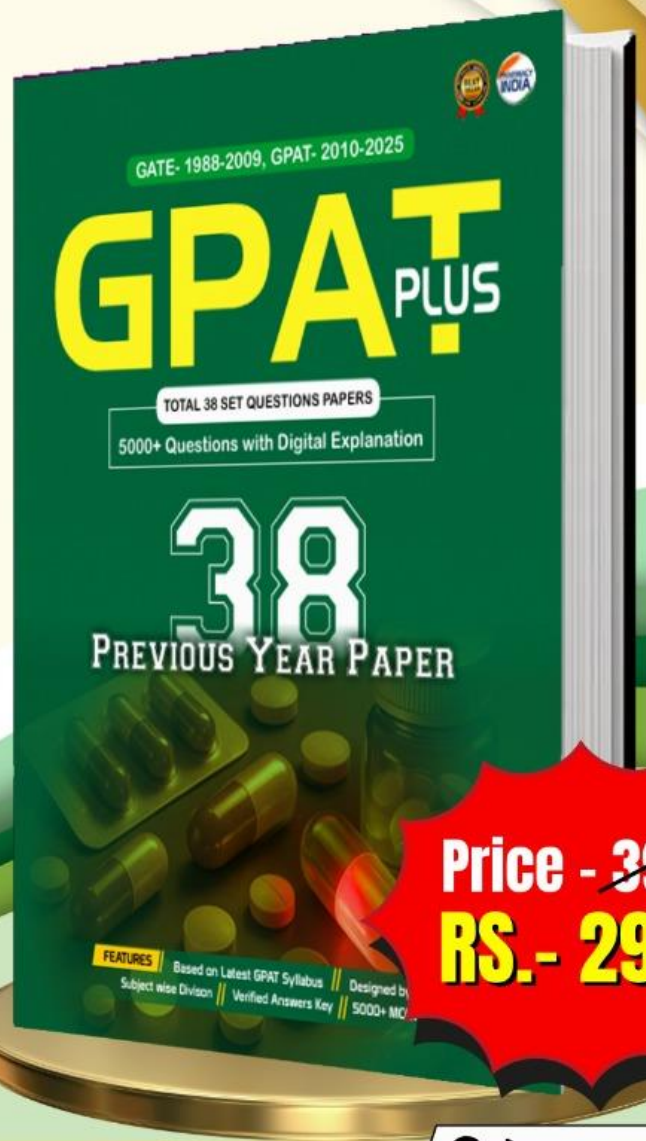

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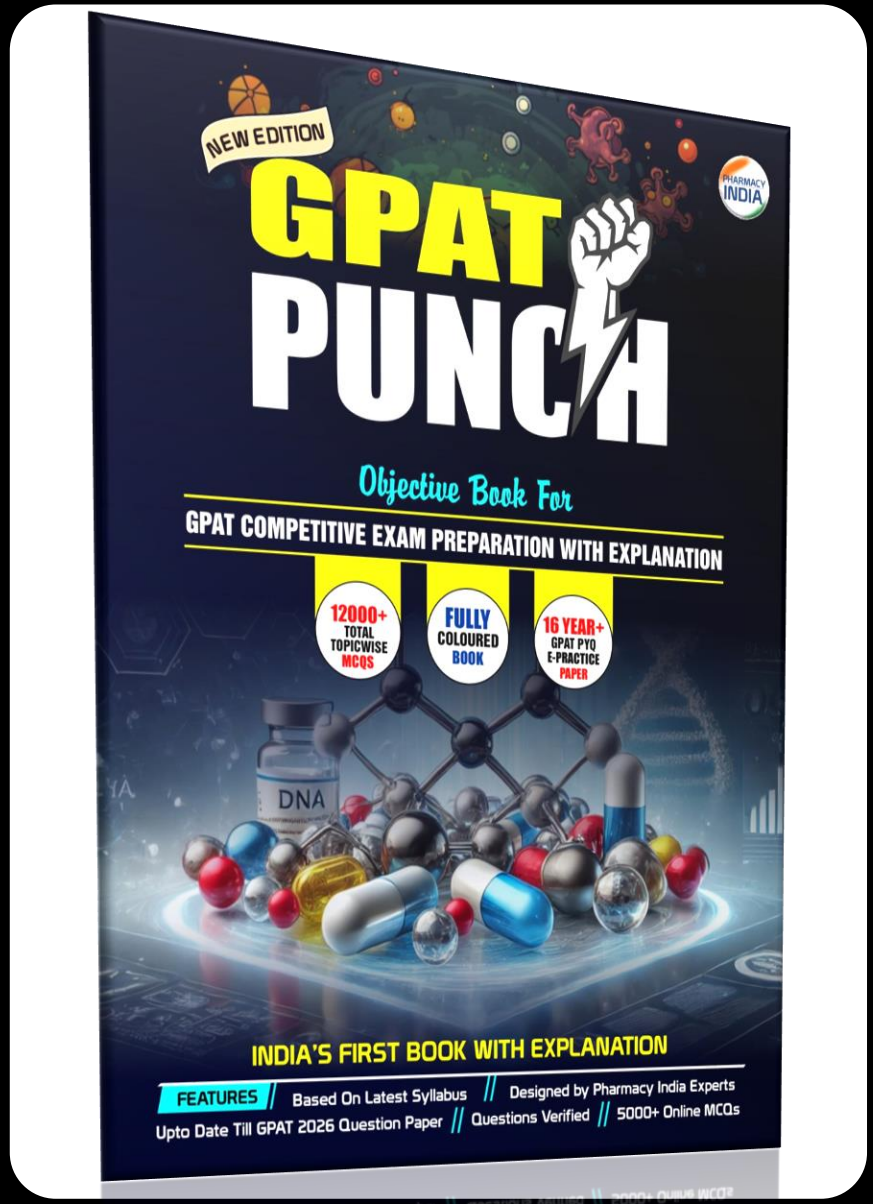

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