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Membrane Receptors & GPCR Signaling

Study Guide — Cell Membrane

High school / pre-med / IB-style questions on membrane receptor types (GPCRs, ion channels, RTKs, intracellular receptors) with a focus on GPCR logic: why cells need membrane receptors, why cascades and amplification exist, second messengers (cAMP, IP3/DAG, Ca^{2+}), and how signals are turned off (desensitization, GTP hydrolysis, PDE).

50 items — Study Guide with Answers

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1 A peptide hormone is released into the blood and reaches many tissues, but only some tissues respond. What is the **MOST** direct reason a peptide hormone typically needs a membrane receptor to affect a cell?

- A** Peptide hormones are too large to exist in blood without receptors.
- B** Peptide hormones are usually polar, so they cannot freely diffuse through the lipid bilayer to reach intracellular targets. ✓
- C** Peptide hormones must be converted into ATP before a cell can respond.
- D** Peptide hormones can only act on cells that have mitochondria.
- E** Peptide hormones only work by binding directly to DNA in the nucleus.

► **Explanation:** Most peptide hormones are hydrophilic, so they can't cross the hydrophobic membrane core. A membrane receptor allows an external signal (first messenger) to be converted into an internal response. Blood transport and mitochondria are not the limiting issue here.



2 Which signaling molecule is **MOST** likely to act through a G-protein-coupled receptor (GPCR) on the cell surface?

- A** Cortisol (a steroid hormone)
- B** Thyroid hormone (lipid-soluble)
- C** Adrenaline/epinephrine (a catecholamine) ✓
- D** Nitric oxide (a gas)
- E** Oxygen

► **Explanation:** Catecholamines like adrenaline are water-soluble and typically signal via cell-surface receptors such as GPCRs. Steroids and thyroid hormone usually act via intracellular receptors, and gases like NO diffuse across membranes without needing a surface receptor.





3 A student says: “If a hormone binds a receptor, the hormone must enter the cell.” Which statement is the best correction?

- A Correct—binding always requires the hormone to pass through the membrane first.
- B Incorrect—many hormones bind membrane receptors and trigger intracellular signaling without entering the cell. ✓**
- C Incorrect—hormones never bind receptors; they bind enzymes.
- D Correct—only steroid hormones can bind receptors.
- E Incorrect—hormones act only by changing membrane cholesterol content.

► **Explanation:** Many signals (especially hydrophilic ones) bind to cell-surface receptors, which then start intracellular cascades. Only some hormones (like many steroids) cross the membrane to bind intracellular receptors.



4 Which structural feature is most characteristic of GPCRs?

- A A single transmembrane helix and a ligand-binding domain inside the nucleus
- B Seven transmembrane segments (typically alpha-helices) ✓**
- C A pore that directly conducts Na^+ and K^+ ions when the ligand binds
- D A cytosolic catalytic domain that phosphorylates tyrosine residues by itself
- E Two lipid bilayers surrounding the receptor protein

► **Explanation:** GPCRs are famous for their 7-transmembrane structure. Ion channels form pores; receptor tyrosine kinases have intrinsic kinase activity; none of those define GPCRs.



5 A heterotrimeric G protein is best described as:

- A A DNA-binding protein made of three chromosomes





- ✓ **B** A three-subunit protein (, ,) that binds GDP/GTP and relays signals from GPCRs
- C** A membrane lipid made of three fatty acids
- D** A receptor that directly phosphorylates glucose
- E** An ion channel with three gates

► **Explanation:** The “G” refers to guanine nucleotides (GDP/GTP). Heterotrimeric G proteins have , , and subunits and transmit signals from GPCRs to effector proteins.

6 In the classic GPCR cycle, which event **MOST** directly switches the G subunit into its active state?



- A** Phosphorylation of G by the receptor
- B** Exchange of GDP for GTP on G ✓
- C** Hydrolysis of ATP to ADP on G
- D** Binding of oxygen to G
- E** Removal of the membrane lipids around the receptor

► **Explanation:** Activated GPCRs act like a ‘GEF’ for G , promoting GDP release and GTP binding. It’s the GDP→GTP swap (not receptor phosphorylation) that activates G .

7 A student says: “GPCRs amplify signals because one hormone molecule can enter and directly activate thousands of enzymes.” What is the best correction?



- A** Correct—hormones enter cells and directly activate thousands of enzymes by themselves.
- B** Incorrect—amplification usually occurs because one activated receptor can activate many G proteins, each triggering downstream enzyme activity and second messengers. ✓
- C** Incorrect—amplification is impossible in biology; responses are always one-to-one.
- D** Correct—amplification is caused mainly by increased diffusion speed of hormones.





E Incorrect—GPCRs are channels, so no cascades are involved.

► **Explanation:** Amplification is a property of cascades: one receptor can activate multiple G proteins; each can activate an effector enzyme; each enzyme can generate many second messenger molecules. The hormone typically does not need to enter the cell.

8 Why do cells often use multi-step signaling cascades instead of a single-step “receptor → final response” system?



A Cascades always make responses slower and less controllable, which cells prefer.

B Cascades allow amplification and also provide multiple control points for regulation and integration of signals. ✓

C Cascades are needed because receptors cannot bind ligands tightly.

D Cascades are used only in plants, not animals.

E Cascades prevent the need for ATP in signaling.

► **Explanation:** Cascades do more than amplify: they create checkpoints, allow branching, integrate multiple inputs, and shape timing. Slower speed can be a tradeoff, not the goal.

9 A key trade-off of using signaling cascades (like GPCR → second messengers → kinases) is that cascades:



A Prevent signal amplification

B Can be slower than direct ion channel signaling, but allow amplification and fine-tuned regulation ✓

C Require the ligand to enter the nucleus

D Always produce identical responses in every tissue

E Work only in bacteria





► **Explanation:** Ligand-gated ion channels can change membrane potential in milliseconds. GPCR cascades are often slower (seconds) but provide amplification, regulation, and signal integration.

10 Which receptor type is most likely to produce the **FASTEST** response after ligand binding?



- A Intracellular nuclear receptor
- B Receptor tyrosine kinase (RTK)
- C **Ligand-gated ion channel** ✓
- D GPCR signaling through a kinase cascade
- E A receptor that only changes gene transcription after hours

► **Explanation:** Ligand-gated ion channels directly change ion flow, so they act fastest. GPCRs and RTKs usually act through multi-step signaling; nuclear receptors often change transcription and are slower.

11 Acetylcholine can act through two receptor families: nicotinic and muscarinic. Which statement is correct?



- A Nicotinic receptors are GPCRs; muscarinic receptors are ligand-gated ion channels.
- B **Muscarinic receptors are GPCRs; nicotinic receptors are ligand-gated ion channels.** ✓
- C Both nicotinic and muscarinic receptors are intracellular receptors.
- D Both are receptor tyrosine kinases.
- E Neither receptor binds acetylcholine directly.

► **Explanation:** Nicotinic ACh receptors are ion channels (fast). Muscarinic ACh receptors are GPCRs (often slower, modulatory).





12 Which statement best distinguishes an RTK (receptor tyrosine kinase) from a GPCR?



- A RTKs have 7 transmembrane helices; GPCRs have one.
- B RTKs often dimerize and autophosphorylate tyrosine residues; GPCRs signal mainly by activating heterotrimeric G proteins. ✓**
- C RTKs are always ion channels; GPCRs are always enzymes.
- D GPCRs can only respond to steroid hormones; RTKs respond only to light.
- E GPCRs work only in bacteria; RTKs work only in plants.

► **Explanation:** RTKs are typically single-pass receptors that activate kinase signaling via tyrosine phosphorylation (often after dimerization). GPCRs are 7-pass receptors that activate G proteins and second messengers.

13 Insulin is best matched with which receptor type?



- A Ligand-gated ion channel
- B GPCR (Gs)
- C Receptor tyrosine kinase (RTK) ✓**
- D Intracellular nuclear receptor
- E Voltage-gated sodium channel

► **Explanation:** At this level, insulin is classically linked to an RTK pathway (phosphorylation cascades). GPCRs (Gs/Gi/Gq) are used by many other hormones and neurotransmitters, but not the classic insulin receptor.





14 A hormone binds a GPCR and causes a rapid increase in intracellular cAMP. Which G type is the BEST match?



- A G s ✓
- B G i
- C G q
- D Tubulin
- E Actin

► **Explanation:** G s stimulates adenylyl cyclase, increasing cAMP production. G i inhibits adenylyl cyclase, and G q activates phospholipase C (IP3/DAG pathway).

15 A GPCR activation leads to a DECREASE in cAMP levels. Which G type is the BEST match?



- A G s
- B G i ✓
- C G q
- D RNA polymerase
- E Myosin

► **Explanation:** G i inhibits adenylyl cyclase, lowering cAMP. G s does the opposite, and G q mainly drives IP3/DAG and Ca²⁺ signaling.

16 A signal activates a GPCR that increases intracellular Ca²⁺ by releasing Ca²⁺ from the endoplasmic reticulum. Which pathway is most consistent with this?



- A G s → adenylyl cyclase → cAMP





- B** G i → inhibition of adenylyl cyclase
- C** G q → phospholipase C → IP3 + DAG ✓
- D** RTK → direct opening of Na⁺ channels
- E** Steroid receptor → DNA binding → immediate Ca²⁺ release

► **Explanation:** G q activates phospholipase C, producing IP3 and DAG. IP3 binds ER receptors and triggers Ca²⁺ release. cAMP (Gs) signaling is a different branch.

17 Which statement about IP3 and DAG is correct?



- A** IP3 remains in the membrane and activates protein kinase C directly.
- B** DAG diffuses through the cytosol to bind receptors on the ER.
- C** IP3 diffuses in the cytosol and can trigger Ca²⁺ release from the ER; DAG stays in the membrane and helps activate protein kinase C. ✓
- D** Both IP3 and DAG are steroid hormones.
- E** IP3 is a membrane channel; DAG is a nuclear transcription factor.

► **Explanation:** IP3 is soluble and travels to the ER to open Ca²⁺ channels. DAG is hydrophobic and stays in the membrane, helping activate PKC (often together with Ca²⁺).

18 Which molecule is a classic SECOND messenger produced/used inside cells during signaling?



- A** Insulin (in the bloodstream)
- B** cAMP ✓
- C** A GPCR protein
- D** A hormone receptor gene





E Hemoglobin

► **Explanation:** Second messengers (like cAMP, Ca^{2+} , IP3) are intracellular signaling molecules that spread/amplify the signal after receptor activation. Insulin is an extracellular first messenger in this context.

19 Which option is NOT a second messenger in the typical GPCR sense?



A Ca^{2+}

B cAMP

C IP3

D DAG

E Adrenaline/epinephrine (the extracellular signal) ✓

► **Explanation:** Adrenaline is a first messenger (outside the cell). cAMP, Ca^{2+} , IP3 and DAG act inside the cell to relay/amplify the signal.

20 A drug inhibits phosphodiesterase (PDE), the enzyme that breaks down cAMP. In a cell using a cAMP pathway, the MOST likely result is:



A cAMP signals become weaker and shorter

B cAMP signals become stronger and/or last longer ✓

C cAMP can no longer be produced by adenylyl cyclase

D The GPCR is converted into an ion channel

E The hormone must now enter the nucleus to work

► **Explanation:** If breakdown is blocked, cAMP accumulates and persists, so downstream signaling tends to be stronger/longer. Production is still controlled by adenylyl cyclase upstream.





21 A mutation makes phosphodiesterase (PDE) much more active than normal. A GPCR pathway that relies on cAMP would most likely show:



- A Exaggerated cAMP signaling because cAMP is preserved
- B Reduced cAMP signaling because cAMP is broken down too quickly ✓**
- C No change because PDE only affects DNA replication
- D A switch from GPCR signaling to RTK signaling
- E Immediate opening of ligand-gated ion channels

► **Explanation:** Faster cAMP breakdown shortens and weakens the signal. This is a common logic trap: “more enzyme activity” doesn’t always mean more signaling—here it removes the messenger.

22 A G subunit cannot hydrolyze GTP back to GDP (its “timer” is broken). After receptor activation, what is the MOST likely effect?



- A The signal turns off faster than normal
- B The G protein stays active longer, prolonging downstream signaling ✓**
- C The receptor cannot bind ligand anymore
- D Second messengers cannot be produced under any conditions
- E The membrane becomes impermeable to water

► **Explanation:** GTP hydrolysis normally terminates G signaling. If hydrolysis fails, G remains in the GTP-bound active state longer, so effector activation (like adenylyl cyclase or PLC) persists.





23 A different mutation prevents GDP from leaving G, even after a GPCR is activated. What happens to signaling through that G protein?

- A Signaling increases because GDP locks G in the active form
- B Signaling decreases because G cannot bind GTP and therefore cannot activate downstream effectors ✓**
- C Signaling becomes independent of receptors
- D The GPCR turns into a transcription factor
- E The cell produces more DNA to compensate

► **Explanation:** G is inactive when GDP-bound. If GDP can't be released, GTP can't bind, so the G protein stays "off" even if the receptor is activated.



24 A patient has been exposed to high levels of the SAME hormone continuously for days. The tissue response gradually decreases even though hormone concentration stays high. Which mechanism best explains this?

- A Random mutation eliminates all receptors in every cell instantly
- B Receptor desensitization/downregulation (e.g., phosphorylation, arrestin binding, internalization) ✓**
- C The hormone becomes lipid-soluble over time
- D The hormone is converted into ATP
- E The nucleus moves outside the cell

► **Explanation:** Continuous stimulation often triggers desensitization: receptors become less able to signal (e.g., via arrestin) and may be internalized, reducing responsiveness even with high hormone levels.



25 Which description best matches GPCR desensitization in basic terms?





- A The ligand is destroyed by ribosomes
- B The receptor is modified so it couples less effectively to G proteins, often followed by receptor internalization ✓**
- C The cell membrane dissolves, preventing signaling
- D The receptor moves into the nucleus to bind DNA
- E All GPCRs permanently stop working after one signal

► **Explanation:** Desensitization reduces signaling from a receptor that has been stimulated repeatedly/continuously. It's often reversible and helps prevent overstimulation.

26 Chronic exposure to a strong agonist often leads to fewer receptors on the cell surface. This is best termed:



- A Upregulation
- B Downregulation ✓**
- C Depolarization
- D Translation
- E Independent assortment

► **Explanation:** Downregulation means fewer receptors (often due to internalization and decreased expression). It reduces sensitivity to constant stimulation.

27 A tissue is exposed to a receptor blocker (antagonist) for a long time. The blocker is suddenly removed. The tissue now responds excessively to normal hormone levels. The best explanation is:



- A The hormone has become more concentrated automatically
- B Receptor upregulation occurred during blockade, increasing receptor number/sensitivity ✓**





- C The cell ran out of ATP, making responses stronger
- D The nucleus moved closer to the membrane
- E Ion channels became irrelevant because GPCRs turned into DNA

► **Explanation:** When receptors are blocked chronically, cells can compensate by increasing receptor number or signaling components. Removing the blocker can then produce an exaggerated response (hypersensitivity).

28 Two different tissues are exposed to the same hormone at the same concentration. Tissue A responds strongly; Tissue B does not respond at all. Which is the MOST direct explanation?



- A Tissue B has DNA, but Tissue A does not.
- B Tissue B lacks the appropriate receptor (or key downstream signaling components) for that hormone. ✓
- C Tissue B has too much oxygen, preventing signaling.
- D Tissue A has no membrane, so hormones enter freely.
- E The hormone must be converted into glucose only in Tissue A.

► **Explanation:** Cells respond only if they express the receptor (and the pathway machinery) needed to detect and transduce the signal. Oxygen/DNA presence is not the defining reason.

29 A common student confusion is: “If two pathways both raise cAMP, they must cause the same effect.” Why is this often false?



- A cAMP cannot activate any proteins, so it has no effects.
- B Different cell types express different downstream targets (enzymes, transcription factors), so the same second messenger can produce different outcomes. ✓
- C cAMP always leaves the cell immediately.
- D Only one hormone in the body uses cAMP.





- E** cAMP signals only in the nucleus, never in the cytoplasm.

► **Explanation:** Second messengers are like a 'common language,' but cells have different 'readers.' Which proteins are present and where they are located determines what the cell does with the message.

30 Which statement best captures why amplification is useful in physiology?



- A** It allows a tiny extracellular signal to create a large intracellular response, even at very low hormone concentrations. ✓
- B** It prevents receptors from binding ligands, protecting the cell from signals.
- C** It makes membranes less selective so more molecules can cross.
- D** It eliminates the need for feedback control.
- E** It ensures every tissue responds identically to every hormone.

► **Explanation:** Amplification increases sensitivity: small changes in ligand can produce noticeable physiological changes. But it still requires regulation and does not remove tissue specificity.

31 A student argues: "Amplification is always good, so cells should amplify as much as possible." What is the best counterargument?



- A** Amplification has no cost and never causes problems.
- B** Too much amplification can increase noise/overreaction and must be balanced by feedback and shut-off mechanisms. ✓
- C** Amplification prevents any cellular response from happening.
- D** Amplification only exists in plants.
- E** Amplification requires hormones to bind DNA directly.





► **Explanation:** Amplification can make cells hypersensitive: tiny fluctuations could cause large responses. That's why cells also evolved brakes—GTP hydrolysis, second messenger breakdown, receptor desensitization, and feedback loops.

32 Which event is the **MOST** immediate “amplification step” in a classic GPCR → cAMP pathway?



- A** One GPCR activating multiple G proteins over time ✓
- B** A hormone being synthesized in the endocrine gland
- C** A ribosome translating a receptor protein
- D** Oxygen binding hemoglobin
- E** DNA replication in S phase

► **Explanation:** The first major amplification is that one activated receptor can activate many G proteins. Later amplifications include one adenylyl cyclase making many cAMP molecules and kinases phosphorylating many targets.

33 A cell is injected with cAMP directly (bypassing the receptor). Which result is **MOST** likely in a pathway where cAMP activates protein kinase A (PKA)?



- A** No response, because receptors are always required for any signaling.
- B** Downstream effects may still occur, because cAMP is downstream of the receptor and can activate PKA directly. ✓
- C** The GPCR will automatically bind more hormone.
- D** The cell membrane will become impermeable to ions.
- E** DNA will be cut into pieces as part of normal signaling.

► **Explanation:** Second messengers are downstream of receptors. If you artificially provide the messenger, you can trigger downstream steps even without ligand binding (though regulation normally prevents this).





34 A signaling pathway uses cAMP, but two different hormones produce opposite effects in the same cell. Which explanation is MOST plausible?



- A** cAMP sometimes becomes DNA, causing opposite effects.
- B** The hormones may activate different receptors that engage different downstream branches, or they may regulate different target proteins despite both involving cAMP. ✓
- C** Opposite effects are impossible if cAMP is involved.
- D** cAMP always increases every cellular process.
- E** The cell must be dead if it shows opposite responses.

► **Explanation:** Even if cAMP rises, the final outcome depends on which targets are affected (different enzymes, ion channels, gene regulators) and how the pathway is wired. Same messenger does not guarantee same physiological effect.

35 Which event most directly turns OFF a G-mediated signal at the G protein level?



- A** G binding GDP and releasing Pi after hydrolysis ✓
- B** The ligand binding to the receptor more strongly
- C** The receptor moving into the nucleus
- D** The cell wall thickening
- E** Ribosomes converting GTP into ATP

► **Explanation:** G has intrinsic GTPase activity: it hydrolyzes GTP → GDP, switching itself off and allowing the heterotrimer to re-form. Stronger ligand binding doesn't directly "switch off" the G protein.





36 Which mechanism MOST directly ensures that a second messenger signal (like cAMP) does not last forever?



- A DNA replication
- B Enzymatic breakdown/removal (e.g., PDE breaks down cAMP; pumps remove Ca^{2+}) ✓**
- C Formation of microtubules
- D Random mutation
- E Protein synthesis in ribosomes

► **Explanation:** Signals are terminated by specific “off” enzymes and transporters. For example, PDE breaks down cAMP; Ca^{2+} is pumped back into stores/out of the cell; IP3 is degraded.

37 Which statement best explains how GPCR signaling can be BOTH amplified AND specific?



- A Amplification always destroys specificity.
- B Specificity comes from receptor type, cell-specific downstream proteins, and where/when the signal occurs—even if steps are amplified. ✓**
- C Specificity occurs only because hormones are different colors.
- D Specificity occurs because all cells have identical pathways.
- E Specificity occurs only in the nucleus; membrane signals are never specific.

► **Explanation:** Cells ‘choose’ responses by expressing specific receptors, G proteins, effectors, and target proteins. Amplification increases magnitude, while specificity is controlled by molecular wiring and localization.

38 A student asks: “If the cell could just let hormones enter, why evolve receptors at all?” Which answer is most accurate?





- A Cells evolved receptors mainly to make membranes thicker.
- B Receptors allow controlled detection, amplification, and regulation of signals—especially for hydrophilic signals that cannot cross membranes. ✓**
- C Receptors exist only because cells cannot produce hormones.
- D Receptors prevent all signaling from happening accidentally.
- E Receptors are only used for transporting oxygen.

► **Explanation:** Receptors aren't just "entry points"; they are control systems. They allow external signals to be sensed without the signal entering, and they give the cell knobs to tune sensitivity, amplification, timing, and shut-off.

39 Which example best illustrates amplification in sensory physiology using a GPCR?



- A A single photon activates a visual GPCR (rhodopsin), triggering many downstream molecules and a measurable electrical response. ✓**
- B A single ion passes through an ion channel and instantly creates a whole muscle contraction.
- C A steroid hormone binds DNA directly and creates an immediate action potential in milliseconds.
- D A ribosome translates one protein and the cell becomes invisible.
- E A mitochondrion divides and that directly activates a receptor.

► **Explanation:** Vision is a classic amplification story: one activated rhodopsin can activate many G proteins (transducin), leading to a cascade that changes ion channel activity and produces a detectable signal.

40 Olfactory (smell) receptors are famous for being:



- A Ligand-gated chloride channels found only in bacteria





- B A large family of GPCRs that detect many different odor molecules ✓**
- C Receptor tyrosine kinases that directly phosphorylate odor molecules
- D Nuclear receptors that bind odor molecules inside the nucleus
- E Enzymes that break down odors in blood plasma

► **Explanation:** Smell relies on GPCRs. The diversity of receptor genes helps detect a huge range of odorants, and signaling cascades amplify detection.

41 Which statement about the β subunits of a heterotrimeric G protein is most accurate?



- A They have no role and are just “packaging” for the α subunit.
- B They can also participate in signaling (for example, by modulating certain ion channels) in addition to the α subunit. ✓**
- C They are DNA molecules, not proteins.
- D They are only found inside the nucleus.
- E They replace ATP synthase in mitochondria.

► **Explanation:** Both G_{α} and G_{β} can interact with downstream targets. The common student trap is thinking only G_{α} matters— G_{β} can be signaling-active too.

42 A GPCR pathway changes a cell’s behavior within seconds, while a steroid hormone pathway changes gene expression over hours. Which explanation best accounts for this time difference?



- A GPCRs use DNA replication, which is faster than transcription.
- B GPCRs often modify existing proteins via second messengers and phosphorylation, while steroid receptors often change transcription and protein production. ✓**
- C Steroid hormones cannot enter cells, so they take longer to find receptors.





- D GPCR signals must always wait for mitosis, which is fast.
- E Steroid hormone signaling is faster because it bypasses receptors.

► **Explanation:** Fast signaling often comes from changing activity of existing proteins (e.g., phosphorylation, ion channel modulation). Slower signaling often involves changing gene expression and synthesizing new proteins.

43 A student asks: “If amplification exists, why doesn’t a cell respond maximally to just one hormone molecule every time?” Which is the best answer?



- A Cells never amplify signals; amplification is a myth.
- B Cells have thresholds, feedback, messenger breakdown, and limited receptor activation time; amplification is real but controlled. ✓**
- C A single hormone molecule always causes maximal response in all tissues.
- D Hormone molecules are too heavy to bind receptors one at a time.
- E Amplification only happens during meiosis.

► **Explanation:** Amplification increases sensitivity, but cells still control magnitude and duration. Off-switches (GTP hydrolysis, PDE, pumps), receptor desensitization, and limited pathway components prevent unlimited responses.

44 A tissue reaches its maximal response when only ~10% of receptors are occupied by hormone. This BEST illustrates:



- A Receptor reserve (“spare receptors”) due to amplification ✓**
- B That receptors are unnecessary
- C That the hormone must enter the nucleus
- D That the membrane has become leaky
- E That enzymes have stopped working





► **Explanation:** If maximal effect occurs with partial occupancy, the system has more receptors than needed for max response (receptor reserve). This often happens because downstream amplification means you don't need to activate every receptor to saturate the response.

45 Half the GPCRs in a tissue are irreversibly blocked, but the tissue can still reach the same maximal response (though it takes more hormone). What is the BEST interpretation?



- A** There were spare receptors; losing some shifts sensitivity but not necessarily the maximum ✓
- B** Blocking receptors always increases sensitivity
- C** GPCRs are not involved in this tissue at all
- D** The hormone changed into a steroid hormone
- E** The cell wall compensates for receptor loss

► **Explanation:** With receptor reserve, fewer receptors still can produce maximal downstream activation. But the tissue becomes less sensitive: you need more hormone to achieve the same response.

46 A competitive antagonist binds the same receptor site as the hormone but does not activate signaling. Which outcome is most expected?



- A** The hormone response becomes stronger at every concentration.
- B** More hormone is required to get the same effect, because the antagonist competes for receptor binding. ✓
- C** The antagonist permanently destroys the receptor by definition.
- D** The receptor becomes a nuclear receptor.
- E** Competitive antagonists always increase cAMP.





► **Explanation:** If the antagonist competes for the same binding site, it reduces receptor occupancy by the hormone at a given concentration. Increasing hormone concentration can outcompete the antagonist (classic competition logic).

47 A student thinks GPCR signaling is “pointless” because the receptor does not make the final response directly. Which analogy best captures why GPCR signaling is useful?



- A A light switch that directly builds a house
- B A door key that opens a single door and nothing else
- C A microphone connected to an amplifier: a small input can create a large, controllable output ✓**
- D A rock falling randomly down a hill
- E A book that cannot be read

► **Explanation:** GPCR cascades let tiny signals become big responses (amplification) and allow many control points (volume knobs, filters, shut-off switches). That’s why they’re so widely used in physiology.

48 Which statement is the BEST definition of “signal transduction”?



- A The conversion of glucose to ATP
- B The process of converting an external signal into an internal cellular response through receptors and intracellular pathways ✓**
- C The movement of red blood cells through capillaries
- D The production of hormones in endocrine glands
- E The replication of DNA during S phase

► **Explanation:** Signal transduction is about information flow: receptor detection → intracellular messengers/cascades → response. It is not metabolism or replication.





49 Which option correctly identifies a **FIRST** messenger vs a **SECOND** messenger?



- A First: cAMP; Second: adrenaline
- B First: IP₃; Second: hormone receptor
- C First: adrenaline (outside cell); Second: cAMP (inside cell) ✓**
- D First: ATP synthase; Second: mitochondria
- E First: DNA; Second: RNA

► **Explanation:** First messengers are extracellular signals (hormones, neurotransmitters). Second messengers are intracellular molecules made/used after receptor activation (like cAMP, Ca²⁺, IP₃).

50 A GPCR is activated, but adenylyl cyclase (the enzyme that makes cAMP) is nonfunctional. Which result is most likely for a G_s-coupled pathway?



- A cAMP still rises because cAMP is made directly by the receptor
- B cAMP does not rise; downstream cAMP-dependent effects are greatly reduced even if the receptor and G protein are normal ✓**
- C The pathway automatically switches to IP₃/DAG even with no changes
- D The hormone becomes lipid-soluble and enters the nucleus
- E The cell immediately undergoes mitosis

► **Explanation:** In a G_s pathway, adenylyl cyclase is the key effector that produces cAMP. If it's broken, the signal cannot be amplified through cAMP even if the upstream receptor is fine.

