



Chapter 9 Exercises

Review Questions

1. What property prevents the ligands of cell-surface receptors from entering the cell?
 - a. The molecules bind to the extracellular domain.
 - b. The molecules are hydrophilic and cannot penetrate the hydrophobic interior of the plasma membrane.
 - c. The molecules are attached to transport proteins that deliver them through the bloodstream to target cells.
 - d. The ligands are able to penetrate the membrane and directly influence gene expression upon receptor binding.
2. The secretion of hormones by the pituitary gland is an example of _____.
 - a. autocrine signaling
 - b. paracrine signaling
 - c. endocrine signaling
 - d. direct signaling across gap junctions
3. Why are ion channels necessary to transport ions into or out of a cell?
 - a. Ions are too large to diffuse through the membrane.
 - b. Ions are charged particles and cannot diffuse through the hydrophobic interior of the membrane.
 - c. Ions do not need ion channels to move through the membrane.
 - d. Ions bind to carrier proteins in the bloodstream, which must be removed before transport into the cell.
4. Endocrine signals are transmitted more slowly than paracrine signals because _____.
 - a. the ligands are transported through the bloodstream and travel greater distances
 - b. the target and signaling cells are close together
 - c. the ligands are degraded rapidly
 - d. the ligands don't bind to carrier proteins during transport
5. A scientist notices that when she adds a small, water-soluble molecule to a dish of cells, the cells turn off transcription of a gene. She hypothesizes that the ligand she added binds to a(n) _____ receptor.
 - a. intracellular
 - b. hormone
 - c. enzyme-linked
 - d. gated ion channel-linked
6. Where do DAG and IP₃ originate?
 - a. They are formed by phosphorylation of cAMP.
 - b. They are ligands expressed by signaling cells.
 - c. They are hormones that diffuse through the plasma membrane to stimulate protein production.
 - d. They are the cleavage products of the inositol phospholipid, PIP₂.
7. What property enables the residues of the amino acids serine, threonine, and tyrosine to be phosphorylated?
 - a. They are polar.
 - b. They are nonpolar.
 - c. They contain a hydroxyl group.
 - d. They occur more frequently in the amino acid sequence of signaling proteins.
8. Histamine binds to the H₁ G protein-coupled receptor to initiate the itchiness and airway constriction associated with an allergic response. If a mutation in the associated G protein's alpha subunit prevented the hydrolysis of GTP, how would the allergic response change?
 - a. There would be a more severe allergic response compared to normal G protein signaling.
 - b. There would be a less severe allergic response compared to normal G protein signaling.
 - c. There would be no allergic response.
 - d. There would be no change compared to normal G protein signaling.

9. A scientist observes a mutation in the transmembrane region of EGFR that eliminates its ability to be stabilized by binding interactions during dimerization after ligand binding. Which hypothesis regarding the effect of this mutation on EGF signaling is most likely to be correct?
- EGF signaling cascades would be active for longer in the cell.
 - EGF signaling cascades would be active for a shorter period of time in the cell.
 - EGF signaling cascades would not occur.
 - EGF signaling would be unaffected.
10. What is the function of a phosphatase?
- A phosphatase removes phosphorylated amino acids from proteins.
 - A phosphatase removes the phosphate group from phosphorylated amino acid residues in a protein.
 - A phosphatase phosphorylates serine, threonine, and tyrosine residues.
 - A phosphatase degrades second messengers in the cell.
11. How does NF- κ B induce gene expression?
- A small, hydrophobic ligand binds to NF- κ B, activating it.
 - Phosphorylation of the inhibitor I κ -B dissociates the complex between it and NF- κ B and allows NF- κ B to enter the nucleus and stimulate transcription.
 - NF- κ B is phosphorylated and is then free to enter the nucleus and bind DNA.
 - NF- κ B is a kinase that phosphorylates a transcription factor that binds DNA and promotes protein production.
12. Apoptosis can occur in a cell when the cell is _____.
- damaged
 - no longer needed
 - infected by a virus
 - all of these
13. What is the effect of an inhibitor binding an enzyme?
- The enzyme is degraded.
 - The enzyme is activated.
 - The enzyme is inactivated.
 - The complex is transported out of the cell.
14. How does PKC's signaling role change in response to growth factor signaling versus an immune response?
- PKC interacts directly with signaling molecules in both cascades but only exhibits kinase activity during growth factor signaling.
 - PKC interacts directly with signaling molecules in growth factor cascades but interacts with signaling inhibitors during immune signaling.
 - PKC amplifies growth factor cascades but turns off immune cascades.
 - PKC is activated during growth factor cascades but is inactivated during immune response cascades.
15. A scientist notices that a cancer cell line fails to die when he adds an inducer of apoptosis to his culture of cells. Which hypothesis could explain why the cells fail to die?
- The cells have a mutation that prevents the initiation of apoptosis signaling.
 - The cells have lost expression of the receptor for the apoptosis-inducing ligand.
 - The cells overexpress a growth factor pathway that inhibits apoptosis.
 - all of these
16. Which type of molecule acts as a signaling molecule in yeasts?
- steroid
 - autoinducer
 - mating factor
 - second messenger
17. Quorum sensing is triggered to begin when _____.
- treatment with antibiotics occurs
 - bacteria release growth hormones
 - bacterial protein expression is switched on
 - a sufficient number of bacteria are present

18. A doctor is researching new ways to treat biofilms on artificial joints. Which approach would best help prevent bacterial colonization of the medical implants?
 - a. an increase in antibiotic dosing
 - b. the creation of implants with rougher surfaces
 - c. vaccination of patients against all pathogenic bacteria
 - d. inhibition of quorum sensing

Critical Thinking Questions

19. What is the difference between intracellular signaling and intercellular signaling?
20. How are the effects of paracrine signaling limited to an area near the signaling cells?
21. What are the differences between internal receptors and cell-surface receptors?
22. Cells grown in the laboratory are mixed with a dye molecule that is unable to pass through the plasma membrane. If a ligand is added to the cells, observations show that the dye enters the cells. What type of receptor did the ligand bind to on the cell surface?
23. Insulin is a hormone that regulates blood sugar by binding to its receptor, insulin receptor tyrosine kinase. How does insulin's behavior differ from steroid hormone signaling and what can you infer about its structure?
24. The same second messengers are used in many different cells, but the response to second messengers is different in each cell. How is this possible?
25. What would happen if the intracellular domain of a cell-surface receptor was switched with the domain from another receptor?
26. If a cell developed a mutation in its *MAP2K1* gene (encodes the MEK protein) that prevented MEK from being recognized by phosphatases, how would the EGFR signaling cascade and the cell's behavior change?
27. What is a possible result of a mutation in a kinase that controls a pathway that stimulates cell growth?
28. How does the extracellular matrix control the growth of cells?
29. A scientist notices that a cancer cell line shows high levels of phosphorylated ERK in the absence of EGF. What are two possible explanations for the increase in phosphorylated ERK? Be specific in which proteins are involved.
30. What characteristics make yeasts a good model for learning about signaling in humans?
31. Why is signaling in multicellular organisms more complicated than signaling in single-celled organisms?
32. *Pseudomonas* infections are very common in hospital settings. Why would it be important for doctors to determine the bacterial load before treating an infected patient?