Bio121
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Review Questions
Lectures 17 & 18

1. What are the different names used to refer to signal producing and signal responding cells?

2. What kind of cellular activity can be controlled by cell to cell communication?

3. What are the four components common to all signal transduction pathways? What would happen to the pathway if any of these components were absent?

4. Briefly describe the following types of signaling:
   Juxtacrine signaling:

   Paracrine signaling:

   Endocrine signaling:

   Synaptic signaling:

5. Describe the ligands involved in each type of signaling.
   Juxtacrine ligands:

   Paracrine ligands:

   Endocrine ligands:

   Synaptic ligands:

6. What is autocrine signaling? How is it different from paracrine signaling?

7. How are endocrine and synaptic signaling similar? How are they different?

8. What type of molecules are endorphins? What type of signaling do they participate in? What cell type releases them? What happens when endorphins are released by these cells? How are they involved in the placebo effect? What happens if endorphins are misregulated?

9. What type of molecule is ghrelin? What type of signaling does it participate in? What cell type releases ghrelin? Where are the cells that respond to ghrelin located? What is the function of ghrelin?

10. What type of molecule is leptin? What type of signaling does it participate in? What cell type releases leptin? Where are the cells that respond to leptin located? What is the function of leptin? How is leptin similar and different from ghrelin?

11. What is the observed phenotype of mice with mutations in the gene that encodes leptin? What is the molecular basis of this phenotype?

12. Leptin is produced in adipose cells, therefore obese individuals tend to have higher levels of leptin in circulation. However, in these individuals high leptin does not correspond to a reduction of food intake. Explain what might be going on.

13. What type of molecule is oxytocin? What type of signaling does it participate in? What cell type releases oxytocin? Where are the cells that respond to oxytocin located? What is the function of oxytocin?

14. What are GPCRs? What are G proteins? How do GPCRs function?

15. If a cell had a loss of function mutation in a G protein subunit that led to the production of G proteins that could not bind GPCRs, could GPCRs still signal? Why or why not? Could ligands still bind GPCRs?

16. Describe how enzyme-linked receptors function.

17. How do ion-channel-coupled receptors work? What is the most common ligand of these receptors? What cell types heavily rely on these receptors and why?

18. What are the two types of intracellular receptors?

19. How do nuclear receptors work? What domain is common to all of these proteins? Why?

20. What is the nitric oxide (NO) receptor called and what are its two functions? Why is NO important in blood vessels? What would happen in the absence of NO or if guanylyl cyclase had an inactivating mutation?

21. Why is it advantageous for signal transduction cascades to be so complex?

22. What is a scaffold protein?

23. What are two common mechanisms used as molecular switches/relays?

24. What enzymes are responsible for phosphorylation and dephosphorylation? Does phosphorylation always lead to activation and dephosphorylation always lead to inactivation?

25. What are GEFs and GAPs? Which would activate a GTPase? Which would speed up the inactivation of a GTPase? (As a part of this answer you need to know when GTPases are active – in the GTP or GDP bound state?)

26. What are second messengers and what are they used for?

27. What are the five common second messengers?

28. Where does cytosolic calcium come from? If calcium participates in so many different signaling events, how does the cell ensure inappropriate calcium-mediated signaling events do not occur?

29. How does calmodulin function as a relay? Explain how calmodulin activates CaM kinase.

30. Do cells usually achieve target mechanisms in response to just one type of signaling molecule?

    Why is this advantageous? *(Hint: think about your answer to #21)*