

# NPB110A Questions

## Lecture 2

**Question:** Which of the following is NOT an example of negative feedback?

- A. Sweating on a hot day
- B. Beta cells secreting insulin after a meal
- C. Entry of sodium into a cell to trigger more sodium entering during an action potential
- D. Shivering on a cold day
- E. When there is an excess of adrenaline, gland A "senses" this and inhibits its release of adrenaline.

**Answer:** C

**Explanation:**

Answers A and C are negative feedback loops to maintain homeostasis; we sense the change in temperature and our body responds by lowering/ increasing the temperature.

Answer E is negative feedback, when our body senses our low blood volume and increases it by stimulating our body to drink more water.

Answer B is when our body senses an increase in blood glucose and secretes insulin to decrease plasma glucose to normal.

Answer C is correct because this is positive feedback; entry of sodium triggers more sodium to enter after threshold potential, to trigger an action potential.

## Lecture 3

**Question:** In terms of Tindenberg's Four Questions, which of the following is NOT a proximate explanation on why humans urinate?

- A. The human bladder develops at Week 10 during fetal development.
- B. When human bladders are full, it sends a message to the hypothalamus to urinate.
- C. Urinating helps humans get rid of waste.
- D. Because of the enlargement of the prostate, older males urinate more frequently.

**Answer:** C

**Explanation:**

Choices A and D are 'Development' → Sequential changes in individuals across their lifespan

Choice B is 'Causation/ Mechanism' → how humans urinate

Choice C is 'Function/ Adaptation.'

**Question:** I am a scientist, and I want to conduct an experiment to determine whether chocolate improves mental well-being. Which of the following pairs is NOT correct?

- A. Null hypothesis/ Chocolate has no effect on mental well-being.
- B. Dependent variable/ amount of chocolate given
- C. Independent variable/ amount of chocolate given

D. Alternative hypothesis/ Chocolate has a positive effect on mental well-being.

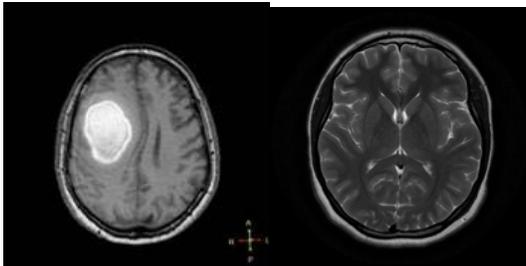
**Answer:** B

**Explanation:**

Dependent variables are the outcomes we are measuring. Since we can change the amount of chocolate given to each subject, that is the independent variable. The dependent variable is mental well-being.

## Lecture 4

**Question:** A resident has trouble reading a MRI for a patient with a brain hemorrhage. To help the poor new doctor, the attending gives the resident two MRIs: one of a person with a brain hemorrhage and one of a person without. Which of the following correctly matches the MRI with a positive/ negative control?



**Figure A:** Intracerebral Hemorrhage [Intracerebral Hemorrhage]. (2019). Retrieved January 14, 2019, from [https://prod-images.static.radiopaedia.org/images/3251974/11274cddb5fa0a305ab53bdf31ecc8\\_thumb.jpg](https://prod-images.static.radiopaedia.org/images/3251974/11274cddb5fa0a305ab53bdf31ecc8_thumb.jpg)

**Figure B:** Normal brain (MRI) [Normal Brain MRI]. (2018). Retrieved January 14, 2019, from [https://images.radiopaedia.org/images/13656005/bd937738ad6223a03f8aedcf4920a7\\_big\\_gallery.jpeg](https://images.radiopaedia.org/images/13656005/bd937738ad6223a03f8aedcf4920a7_big_gallery.jpeg)

- A. Figure A is the negative control because this shows an MRI with a hemorrhage.
- B. Figure A is the positive control because this shows the patient with a hemorrhage.
- C. Figure B is the negative control because this shows a patient without a hemorrhage
- D. B and C are correct
- E. A and B are correct

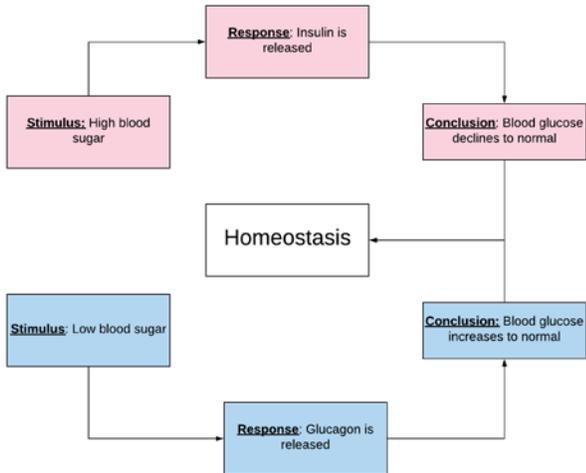
**Answer:** D

**Explanation:**

The positive control is somebody we know HAS a hemorrhage.

The negative control is somebody we know does NOT have a hemorrhage.

**Question:** The following diagram shows the physiological responses to abnormal amounts of glucose. Which of the following statements is INCORRECT?



- A. A rising blood glucose is **sufficient** to elicit a response from the pancreas.
- B. Insulin is **sufficient** to maintain blood glucose levels over all ranges.
- C. Glucagon is **necessary** to maintain blood glucose levels.
- D. Insulin is **necessary and sufficient** to maintain blood glucose levels after a meal.

**Answer:** B

**Explanation:**

Insulin is not sufficient because if we add it to a system with low blood glucose, it will not maintain blood glucose levels.

\*\*sucky Q\*\*

**Question:** An individual lost the ability to break the bonds between the second and third phosphate in ATP. Which of the following processes can the individual (theoretically) still perform?

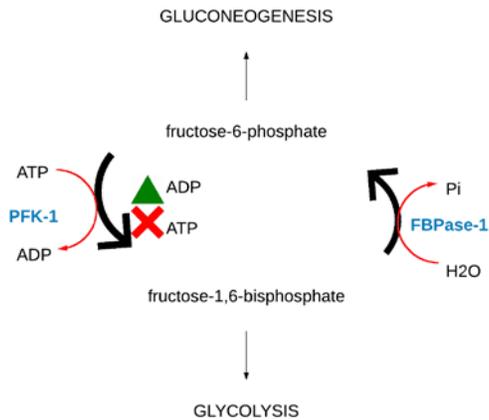
- A. Lift up heavy objects.
- B. Break down fat from adipose tissue.
- C. Communication via action potentials.
- D. Keep running the sodium/ potassium pump.

**Answer:** B

**Explanation:**

This question tries to establish the fact that ATP is needed for maintaining ion gradients (Choice C and D; but the student must know that the Na<sup>+</sup>/K<sup>+</sup> Pump requires energy). ATP is also needed for muscle contraction, active transport, and anabolic reactions.

Breaking down fat will yield energy (it is a catabolic reaction, and not an anabolic reaction)



The following two questions refer to the figure above.

Some background information:

The green indicates that the forward reaction is favored; while the red 'X' indicates that the forward reaction is NOT favored.

ATP and citrate are products following glycolysis,

**Question:** Based on the figure above, which of the following is true?

- PFK-1 is a phosphatase because ATP is required.
- FBP-ase is a phosphatase because ATP is required.
- FBP-ase is a kinase because it phosphorylates fructose 1,6-bisphosphate.
- PFK-1 is a kinase because it phosphorylates fructose 6-phosphate
- All of the statements above are false.

**Answer:** D

**Explanation:** Kinases add a phosphate to a protein. Phosphatases removes phosphates from a protein. PFK-1 uses the phosphate from ATP to phosphorylate Fructose 6-phosphate, and FBP-ase removes the phosphate from fructose 1,6 bisphosphate.

**Question:** Based on the figure above, which of the following is true?

- ATP is an inhibitor of PFK-1
- ATP works via feedback inhibition.
- ATP is an activator of FBP-ase-1.
- A and B are correct.
- A and C are correct.

**Answer:** D

**Explanation:**

Two key concepts in this question: activation/ inhibition and feedback inhibition (where an 'end product' is an inhibitory modulator of an enzyme).

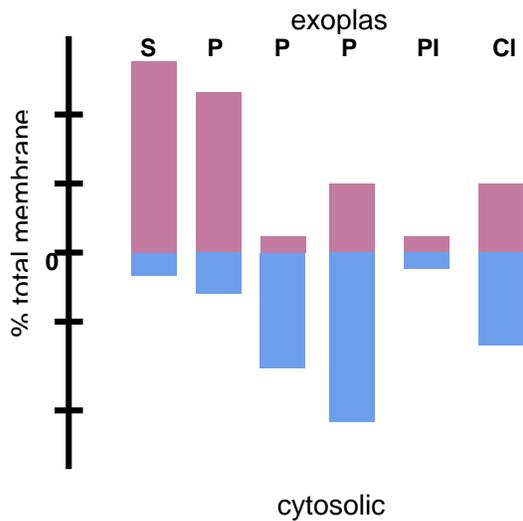
## Lecture 5

**Question:** Above shows a cell with a glycosylated protein depicted in red. Where can we NOT find this protein?

- A. In the cytosol
- B. In the ER
- C. In the Golgi
- D. In the extracellular matrix

**Answer:** A

**Explanation:** Proteins are glycosylated in the ER and modified in the Golgi, so it is always in contact with the extracellular membrane. Thus, it is never in contact with the cytosol.



**Question:** Based on the figure above, which statement is FALSE?

- A. SM and phosphatidylcholine (PC) help keep the membrane rigid.
- B. Phosphatidylserine (PS) and phosphatidylethanolamine (PE) help keep the membrane fluid.
- C. The asymmetrical membrane help with different interactions.
- D. Cholesterol (C) is found equally on both exoplasmic and cytosolic sides because it helps firm the membrane.
- E. All of the above are true.

**Answer:** E

**Explanation:** they're true

**Question:** I am a mad scientist trying to build a cell membrane for a new cold-blooded creature living in Antarctica. Which of the following will help this organism survive?

- A. Longer lipid chain tails

- B. Saturated lipid tails
- C. More sphingomyelin (SM) instead of phosphatidylcholine (PC)
- D. More van der Waals forces between the lipid chain tails
- E. None of the above will help the organism survive

**Answer:** E

**Explanation:** All of the above makes the membrane less fluid instead of more fluid, so it'll be easier for the membrane to freeze in such cold weather.

(Choice D is basically the explanation of Choice A)

**Question:** I am trying to study a transmembrane protein in the cell. Which of the following methods will help me isolate the protein?

- A. Using a detergent
- B. Adding a lipase
- C. Changing the pH
- D. A and C
- E. B and C

**Answer:** D

**Explanation:** Anything that can disrupt the membrane of the protein (i.e. detergents, salts, pH) will help isolate the protein. The lipase is used for GPI proteins, to cleave the bond between the protein and lipid.

\*\*not sure if lipase will also disrupt membrane\*\*?

**Question:** During a blood transfusion, a young resident accidentally gave a patient with Type B blood some Type AB blood. Which of the following is/ are reason(s) why this is detrimental to the patient?

- A. The red blood cell of the Type B patient is significantly smaller than Type AB red blood cell
- B. The different proteins on the red blood cells of Type B and AB will elicit an immune response
- C. The glycosylated proteins on the cytoplasmic side of the membrane of Type B patients elicits different intracellular messengers
- D. B and C are correct
- E. A and C are correct

**Answer:** B

**Explanation:** Choice C is tempting but the glycosylated proteins are not found on the cytoplasmic side but the exoplasmic side.

## Lecture 6

**Question:** The kidneys filter water, ions, and wastes. In one particular case, water flows by osmosis from the filtrate (future urine) back to the bloodstream. Which of the following is true?

- A. The filtrate is hypertonic compared to the blood.
- B. The filtrate is hypotonic compared to the blood.
- C. The filtrate is isotonic compared to the blood.
- D. None of the above is correct.

**Answer:** B

**Explanation:** Water travels from low concentration to high concentration of solutes. Thus, the filtrate must have less concentration than the bloodstream.

**Question:** A patient comes in the emergency room having lost a LOT of blood due to a gunshot wound. The resident cannot find the normal 0.9% saline IV solution, so he makes his own 10% saline. The patient passes away in the next hour. Why is that?

- A. The patient's red blood cells burst due to the hypertonic solution.
- B. The patient's red blood cells shrink due to the hypertonic solution.
- C. The patient's red blood cells burst due to the hypotonic solution.
- D. The patient's red blood cells shrink due to the hypotonic solution.
- E. The patient died from another cause, because the resident followed the correct procedure.

**Answer:** B

**Explanation:** The resident gives the patient a hypertonic solution. Water will flow out of the blood cells, causing it to shrink.

**Question:** A person just drank carbonated grape soda. Which of the following is true?

- A. The carbon dioxide and sugar goes into the cell via a symporter.
- B. Carbon dioxide goes into the cell via diffusion and the sugar goes into the cell via special protein transporters.
- C. Carbon dioxide goes into the cell via diffusion and sugar goes into the cell via an ATPase.
- D. Both carbon dioxide and sugar enter the cell via ATP.
- E. Sugar is broken down into ions and diffuse into the membrane along with the carbon dioxide.

**Answer:** B

**Question:** Joanne hasn't slept in five days because she has five exams tomorrow and is stress eating. Her meals have only consisted of five boxes of chocolate chip cookies, ten handfuls of chocolate chips, and a tub of chocolate ice cream. The next day she takes a urine test and finds glucose in her urine. She finds this odd because her body is not diabetic and her body is supposed to absorb the glucose via GLUTs (glucose transporters)! What might be a reason why?

- A. She has no ATP to allow her GLUTs to work.
- B. So much glucose is diffusing into her cells that the concentration gradient is ruined
- C. All her GLUTs are being used and reached their maximum carrying capacity.
- D. She hasn't been eating any potassium so glucose cannot be taken in via a symporter
- E. None of the above are correct

**Answer:** C

**Explanation:** GLUTs can saturate!

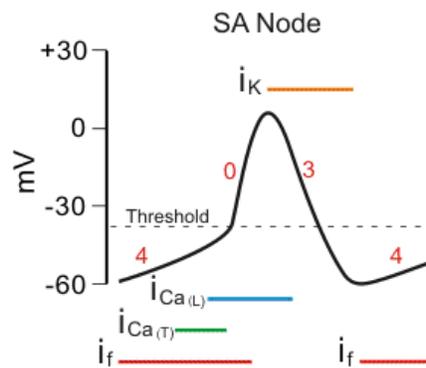
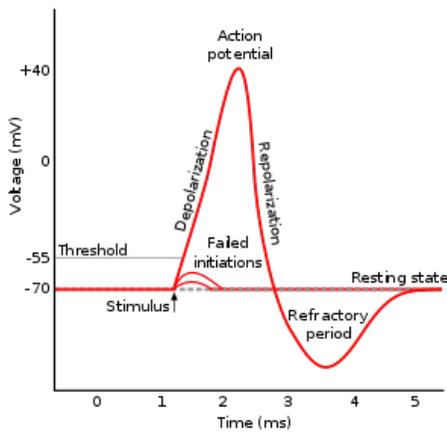
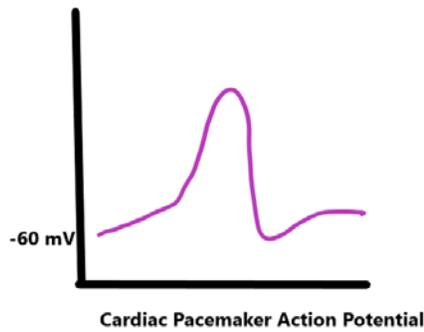
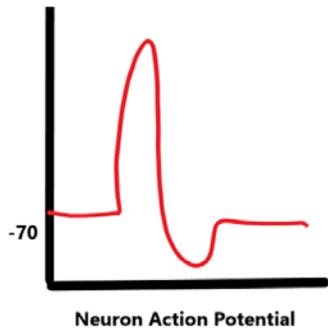
**Question:** The picture above shows a cell. The bigger the font, the more of the molecule there is. This cell wants to make proteins. What is a way to bring amino acids into the cell?

- A. Via an antiporter with sodium
- B. Via a symporter with sodium
- C. Via diffusion
- D. Via a symporter with potassium
- E. Two or more are correct

**Answer:** A

# Lecture 7

*\*\*ignoring calcium for the following questions, because that's what we did\*\**



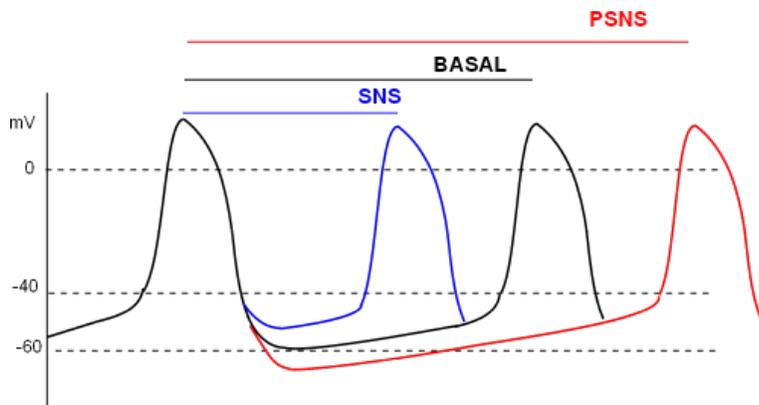
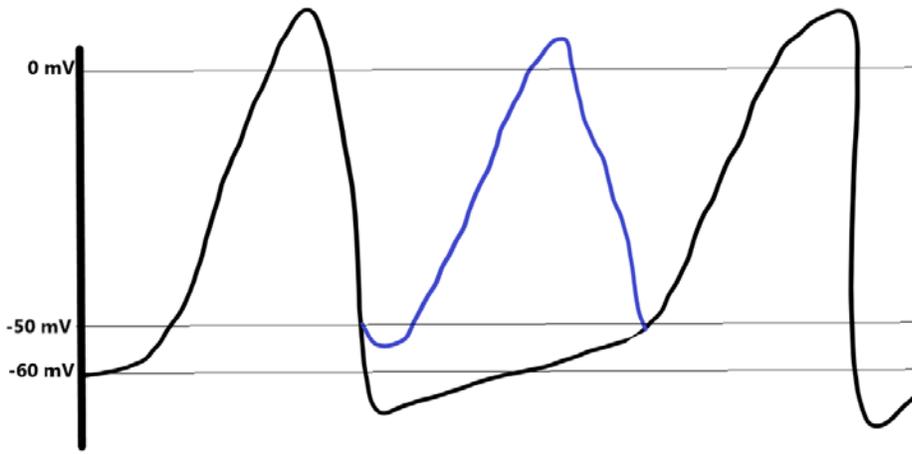
**Question:** Above shows the action potential of a neuron and an action potential of a cardiac pacemaker. Compared to the normal neural action potential (where resting potential is -70mV) what does this mean about membrane permeability? Recall that  $E_{K^+} = -90mV$  and  $E_{Na^+} = 50mV$ . Ignore calcium.

- A. The pacemaker membrane is more permeable to  $Na^+$  than the neural membrane.
- A. The membrane is more permeable to  $K^+$  than the neural membrane.
- B. The pacemaker membrane most likely has more  $K^+$  leak channels than the neural membrane.

- C. A and C
- D. B and C

**Answer:** A

If it was equally permeable to  $\text{Na}^+$  and  $\text{K}^+$ , the resting membrane potential would be  $-40\text{mV}$ .  
 In the neural action potential,  $\text{K}^+$  is more permeable so the resting membrane potential is  $-70\text{mV}$ .  
 Since  $-60\text{mV}$  is closer to  $\text{Na}^+$  membrane potential, it is more permeable to sodium.



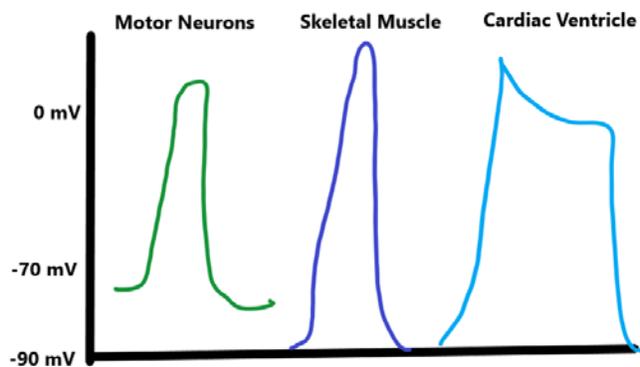
\*\*a better picture will be used if you like this question\*\*

**Question:** Above shows a picture of a cardiac pacemaker action potential, which is an action potential that regulates heartbeat. The black action potential, starting at  $-60\text{mV}$ , shows Joanne's normal

pacemaker activity. When Joanne sees her crush, her pacemaker activity is shown in blue, which starts at -50mV. What is a possible mechanism of the change in action potential?

- A. The pacemaker membrane is more permeable to  $K^+$
- B. The pacemaker membrane is more permeable to  $Na^+$
- C. The  $Na^+/K^+$  ATPase is not working.
- D. There are more  $K^+$  leak channels

**Answer:** B



**Question:** Above shows the action potentials of a neuron, skeletal muscle, and cardiomyocyte, respectively. What does this show about resting membrane potentials?

- A. Skeletal muscle and cardiac ventricle membranes have membrane proteins with relatively similar permeability to ions.
- B. Motor neuron membranes have more  $K^+$  leak channels.
- C. Skeletal muscle and cardiac ventricle muscles have more  $K^+$  leak channels than motor neurons.
- D. A and B are correct
- E. A and C are correct

**Answer:** E

**Explanation:** Cardiac ventricle and skeletal muscles have a resting membrane potential similar to each other, and both are much closer to  $E_{K^+} = -90mV$ .

**Question:** The figure above shows an alien cell. Instead of 'bananas in the ocean' (more  $K^+$  inside and more  $Na^+$  outside), this cell has more  $Ba^{2+}$  inside the cell and more  $Be^{2+}$  outside the cell. The  $E_{Ba^{2+}} = -1000mV$  and the  $E_{Be^{2+}} = 3000mV$  and the resting potential is 2000 mV. What does this mean for the membrane permeability of the alien cell?

- A. The membrane is more permeable to  $\text{Be}^{2+}$  than  $\text{Ba}^{2+}$
- B. The membrane is more permeable to  $\text{Ba}^{2+}$  than  $\text{Be}^{2+}$ .
- C. The membrane is equally permeable to both ions.
- D.  $\text{Be}^{2+}$  is stronger than  $\text{Ba}^{2+}$  because it is heavier.
- E.  $\text{Ba}^{2+}$  is stronger than  $\text{Be}^{2+}$  because it is lighter.

**Answer:** C

**Explanation:**  $-1000 + 3000 \text{ mV} = 2000 \text{ mV}$ .

**Question:** The figure above shows an alien cell. Instead of 'bananas in the ocean' (more  $\text{K}^+$  inside and more  $\text{Na}^+$  outside), this cell has more  $\text{Ba}^{2+}$  inside the cell and more  $\text{Be}^{2+}$  outside the cell. The  $E_{\text{Ba}^{2+}} = -1000 \text{ mV}$  and the  $E_{\text{Be}^{2+}} = 3000 \text{ mV}$  and the resting potential is  $2000 \text{ mV}$ . If we open more  $\text{Ba}^{2+}$  leak channels...

- A. The resting potential will become more positive.
- B. The resting potential will become more negative.
- C. The resting potential will stay the same.
- D. The cell will burst.
- E. The cell will shrink.

**Answer:** B

**Explanation:** More  $\text{Ba}^{2+}$  leak channels will move the resting potential closer to  $E_{\text{Ba}^{2+}} = -1000 \text{ mV}$

## Lecture 8

**Question:** In the membrane, there are not only voltage gated channels but also leak channels. What would happen if we added  $\text{Na}^+$  leak channels in addition to the voltage gated  $\text{Na}^+$  channels?

- A. The resting membrane potential would start at a more hyperpolarized state.
- B. The resting membrane potential would start at a more depolarized state.
- C. It would be harder to reach threshold.
- D. A and C
- E. B and C

**Answer:** B

**Explanation:** More  $\text{Na}^+$  channels would make the cell more positive.

**Question:** We have learned that the voltage-gated channels have a 'ball and chain' to activate or inactivate the channel. In one neuron, the ball and chain never developed (and thus cannot block the channel). The activation gates, however, are working properly. What will this do to the cell?

- A. This will influence the 'falling phase' of the action potential.
- B. This will influence the absolute refractory period of the cell.
- C. There will no longer be a relative refractory period.
- D. A and B are correct.
- E. A and C are correct.

**Answer:** D

**Explanation:**

Characteristics of the absolute refractory period includes: inactivation of  $\text{Na}^+$  channels, opening of voltage gated  $\text{K}^+$  channels .

Characteristics of the relative refractory period includes: too much  $K^+$  traveled out of the voltage gated  $K^+$  channels, inactivation gate of  $Na^+$  channels open and activation gates of  $Na^+$  channels close. Thus, since the relative refractory period has the inactivation gate of  $Na^+$  open and relies on the activation gates of  $Na^+$  closing, C is not correct.

**Question:** The figure above shows an action potential traveling through a normal neuron and a damaged neuron. What might be the cause of the action potential pathway of the damaged neuron?

- A. Damaged voltage gated sodium channels
- B. Damaged voltage gated potassium channels
- C. Damaged myelin sheath
- D. Damaged nodes of Ranvier
- E. Damaged leak channels

**Answer:** C

**Explanation:** The photo shows an action potential traveling through the neuron in one direction and the damaged one shows the action potential traveling through the membrane.

**Question:** Phenytoin is used with patients who have seizures (or repetitive firing of action potentials). Which statement below can explain Phenytoin's mechanism of action?

Hint: You want to PREVENT or mitigate the repetitive firing or action potentials.

- A. Phenytoin binds to voltage gated sodium channels to keep it open longer.
- B. Phenytoin binds to the membrane which increases its permeability to sodium.
- C. Phenytoin binds to the inactive form of voltage gated sodium channels to prolong the falling period.
- D. Phenytoin lowers the threshold so it'd be easier to fire action potentials.

**Answer:** C

**Explanation:** We want to prevent the firing of action potentials so we prolong the inactive form to prolong the absolute refractory period.

**Question:** You go to the dentist and he gives you local anesthesia for your root canal. He tells you that the anesthesia inhibit nerve conduction; i.e. slows down/ prevents action potentials. As a smart NPB major, you know that this really means that the anesthesia...

- A. Increases permeability to sodium
- B. Highly depolarizes the membrane
- C. Block sodium channels
- D. Lowers threshold potential

**Answer:** C

**Explanation:** blocking sodium channels will prevent action potentials.

## Lecture 9

**Question:** hi

## Lecture 10 Questions

Which of the following mechanisms can be ways to treat Staphylococcus Aureus (MRSA in hospitals)?

- A. Antifungal agents
- B. An agent that deactivates the virulence factors
- C. An agent that binds to communication receptors of the pathogen
- D. A and B
- E. B and C

Answer: E

Explanation: Quorum Sensing

Inspired by the Polyjuice Potion from Harry Potter, in which the wizard drinking the potion quickly transforms into another individual, you invent a pill that makes fast changes to keratin (skin proteins), biotin (hair proteins) so the person drinking can quickly look like someone else. You're a smart scientist and you know that you have to alter protein function and signaling. Which of the following methods can you NOT use to make this quick transformation?

- A. Altering nuclear receptors
- B. Altering protein kinases
- C. Altering surface receptors
- D. Altering ion channel receptors
- E. Altering protein phosphatases

Answer: A

Explanation: QUICK transformation

You're at the dentist and he injects Lidocaine, a local anesthesia, in your gums. This makes your gum numb. He says that this drug blocks the signal from one neuron to another nearby neuron. As an intelligent NPB major, you know this this means that Lidocaine affects....

- A. Autocrine signaling
- B. Paracrine signaling
- C. Contact-dependent signaling
- D. Endocrine Signaling

Answer: B

Explanation: Neurons are paracrine signaling

There is a mistake in DNA transcription of the testosterone receptor. Testosterone receptor is supposed to have a 184 bp DNA binding domain, but it is transcribed as the 168bp binding domain of estrogen instead.

- A. Estrogen will stimulate the transcription of testosterone receptor.
- B. Testosterone will stimulate the transcription of estrogen receptor.
- C. Testosterone will stimulate the transcription of testosterone receptor (nothing changes).
- D. Testosterone will increase the affinity of the cell to estrogen.

Answer: A

Explanation: hormones are transcription factors and bind to DNA. In this case, the transcription factor domain is altered so estrogen will be the TF for transcription of testosterone receptor.

You're an astronaut on Mars and discover an organism that, instead of a phospholipid bilayer, has a membrane with phosphate heads and polar ethanolic tails. Which type of signaling will this affect?

- A. G-protein coupled signaling
- B. Ion channel signaling
- C. Steroid hormone signaling
- D. Receptor tyrosine kinase signaling

Answer: C

Explanation: Steroid hormones must diffuse in the cell membrane into the nucleus to make the effect.

## Lecture 11 Questions

**Question:** A patient presents severe allergies so he has to take Loratadine (histamine1 agonist). Let's assume that increased activity of the  $G_s$  pathway leads to allergies. Analyzing his etiology, we see that he has a hyperactive Protein Kinase A that has a higher sensitivity to cAMP. Besides taking a histamine agonist, what else can stop his allergies?

- A) Making sure there is excess GTP in the cell.
- B) Weakening the bonds between the alpha and beta/gamma subunits.
- C) Increasing the rate of PDE (phosphodiesterases)
- D) Making sure Adenyl Cyclase is extra receptive to an alpha subunit bound to GTP.
- E) Increasing phosphorylation of CREB (a transcription factor) by PKA.

Answer: C

The point of this question is to remember the pathway of  $G_s$  (the stimulatory pathway). Not sure the mechanism of allergies, but if we assume that increased activity of  $G_s$  pathway leads to allergies, we want to PREVENT this  $G_s$  pathway.

- A- Excess GTP will increase alpha/beta/gamma association, which will lead it to bind to Adenyl Cyclase more often which will further the pathway
- B- Weakening the bonds between the subunits will increase rate of dissociation, which will activate AC.
- D- If AC is extra receptive, pathway will continue,
- E- Increasing phosphorylation is an alternative downstream effect and will not have anything to do with phosphorylation of our target.

C is the correct answer because PDE will have breakdown cAMP. With less cAMP, less PKA is activated.

Albuterol is a drug that targets G-proteins. It is taken by those who have asthma. In asthma attacks, we want to increase the amount of cAMP in the cell to inhibit inflammatory cells. Which of the following paths will help with the mechanism of

- A. Increase protein kinase A activity
- B. Increase phospholipase C activity
- C. Increase diacylglycerol activity
- D. Stimulate adenylyl cyclase activity

Answer: D

Explanation: only AC converts ATP to cAMP

Cyclic AMP can be used for a variety of functions. In eukaryotic cells, it has a role in glycogen, lipid, and sugar metabolism. You are a scientist and you are studying a cell that has low fatty acid levels. You also find low levels of cAMP. Which of the following can be an explanation to this problem?

- A. There is increase in phosphodiesterase activity
- B. There is a problem with adenylyl cyclase.
- C. There is an increase in  $G_i$  ( $G$  inhibitory) activity.

- D. A and B are correct
- E. All of the above

Answer: E

Explanation: A, B and C can result in a decrease in cAMP.

The G-protein fails to convert GDP to GTP. Which of the following can occur?

- A. There will be a buildup of ATP
- B. There will be a decrease in protein kinase activity
- C. There will be a decrease in adenylyl cyclase activity
- D. There will be a decrease in cAMP levels
- E. None of the above

Answer: E

Explanation: G-proteins don't convert GDP to GTP; intracellular GTP replaces GDP in a cell

Most drugs inhibit the G-protein cascade at the level of the ligand (i.e. dopamine, hormone, initiator).

What is a possible explanation of this?

- A. Drugs should target at the beginning of the cascade because signal amplification will make it difficult to stop the cascade
- B. Drugs should target at the beginning of the cascade because we have to wait for the cell for 1-2 hours before it will reach the level of the effector
- C. Drugs should target at the beginning of the cascade because drugs have more affinity to the G-protein than adenylyl cyclase
- D. Drugs should not target at the beginning of the cascade-- drug companies have it wrong.

Answer: A

Explanation: this is true

## Lecture 12

Phosphorylase A stimulates the breakdown of glycogen, and glycogen synthase inhibits the synthesis of glycogen. Since both phosphorylase A and glycogen synthase is phosphorylated during hyperglycemia, how would this both help decrease glucose in the bloodstream?

- A. Phosphorylation of proteins can be stimulatory or inhibitory.
- B. It's difficult to remove the phosphate off these proteins, so once we phosphorylate one, the other one must also have been phosphorylated during the last signal.
- C. Phosphorylase A is from a G<sub>s</sub> pathway and Glycogen synthase is from a G<sub>i</sub> pathway.
- D. Phosphorylase A is from a G-protein pathway but glycogen synthase is from an ion-gated channel pathway.

Answer: A

Phosphorylase A stimulates the breakdown of glycogen and phosphorylation of this enzyme stimulates the activity. On the other hand, glycogen synthase phosphorylation inhibits its activity.

You are studying a cell and you accidentally put detergent on it, which only ruins the membrane. Which of these signaling molecules will it affect?

- A. G-protein

- B. IP3
- C. Diacylglycerol
- D. A and B
- E. A and C

Answer: E

Explanation: The G-protein and DAG are embedded in the membrane.

There is something wrong with the signaling pathway and now there's a buildup of cyclic AMP. Which of the following can be reasons why?

- A. The subunits on PKA are morphed, and now there is no longer a binding spot for cAMP.
- B. Adenylyl cyclase is enhanced.
- C. The Gi pathway is inhibited.
- D. None of the above are correct
- E. All of the following are correct

Answer: E

Explanation: We can enhance stimulation or inhibit inhibition. If the binding site of cAMP is no longer

CREB is a protein found in the nucleus of the cell, and can be one of the downstream targets of the G-protein pathway. Once activated, it will increase the transcription of the CRE genes. How will an increase in phosphodiesterase (PDE) activity in the cytosol affect the expression of the CRE genes?

- A. It will increase activity of PKA, which will increase the transcription factor activity of CREB.
- B. It will increase activity of GTP hydrolysis by the G-protein, which will decrease transcription factor activity of CREB
- C. It will decrease activity of adenylyl cyclase, which will decrease transcription factor activity of CREB.
- D. It will increase breakdown of cAMP, which will decrease transcription factor activity of CREB.

Answer: D

Explanation: PDE will break down cAMP so it cannot go on to stimulate PKA to set of a chain of signals to activate CREB in the nucleus.

Which of the following diseases is NOT correctly matched with a potential cure?

- A. Cholera toxin- a drug that degrades externally degrades GTPase.
- B. Pertussis toxin- a drug that activates alpha subunit.
- C. Hypoglycemia- increasing cAMP in the cytosol
- D. Hyperglycemia- decreasing cAMP in the cytosol

This one was kind of mean. Trimmer mentioned in the diabetes lecture that cAMP has two pathways; one in which increases glycogen breakdown, and one that inhibits glycogen synthesis.

A- cholera toxin has a GTPase that cannot degrade GTP, so it has a hyperactive alpha subunit which leads to increased cAMP levels.

B. pertussis toxin also leads to increased cAMP levels, but it is a "broken brake" so Gi does not work.

Thus if we activate the alpha subunit then we will increase inhibitory activity.

C- increasing cAMP can lead to glycogen breakdown.

D- cAMP can inhibit or activate glycogen breakdown.

## Lecture 13 Questions

(integrating concepts from Lecture 30)

We see that Per and Cry is being transcribed at a higher level. What is not a possible explanation?

- A. Increased cAMP because increased AC activity
- B. Increased calcium concentration inside of the cytosol
- C. CaMK's ability to bind calmodulin
- D. Inability of RTKs to dimerize

Per and Cry can increase in activation with the transcription factor, CREB. CREB is activated by phosphorylation in the Gs or Gq pathway. Thus, if Per and Cry is being transcribed more, the Gs or Gq pathway must be working. Thus, increased cAMP, increased calcium, will lead to the pathway working. CaMK's ability to bind to calmodulin will activate it, which will activate CREB.

Since RTKs ability to dimerize doesn't have to do with CREB (it is a pathway of GPCR), that is the answer.

Trastuzumab (Herceptin) is a drug that targets the HER-2 Receptor (a receptor tyrosine kinase). Assuming that activation of this RTK causes breast cancer, what is a possibility to explain its mechanism?

- A. Preventing dimerization of HER-2 receptor
- B. Preventing pKA from phosphorylating the tyrosines
- C. Making sure that HER-2 creates binding domains for other signal proteins after it transautophosphorylates
- D. Increasing Ca<sup>2+</sup> concentration

This is a question testing the RTK pathway. If RTK pathway causes breast cancer, we need to inactivate it.

- A- Preventing dimerization will stop the pathway.
- B- pKA doesn't phosphorylate the tyrosines; it transautophosphorylates.
- C- If we make sure that it transautophosphorylates, we will be ensuring the pathway.
- D- Ca<sup>2+</sup> doesn't have to do with anything.

Oh no! A genetic defect causes the Cam Kinase in smooth muscles to be unable to phosphorylate and now the smooth muscles cannot contract. What will be the result of this?

- A. Calcium can no longer bind to calmodulin
- B. The calmodulin can no longer bind to the Cam kinase.
- C. The Cam Kinase cannot be fully activated.
- D. The Cam kinase cannot be activated at all.

Answer: C

Explanation: Calmodulin will activate the CaM Kinase, but autophosphorylation will fully activate it.

The dimer that is supposed to bind to inactive RTKs is broken down by a drug in the cytosol. What will this effect on the RTK pathway?

- A. The inactive RTKs cannot dimerize
- B. The RTKs cannot transautophosphorylate

- C. The proteins cannot bind to the phosphorylated tyrosines
- D. A and B are correct
- E. All of the above are correct.

Answer: E

Explanation: all of these are downstream of the dimer binding to inactive RTKs

The figure above shows the pathway of insulin and insulin receptor (an RTK), which play an important role in diabetes. You are a doctor and you find that in a diabetic patient, they have high plasma insulin but glucose is not entering the cell. You also notice that the insulin receptor is not phosphorylated. With this information, what is the MOST LIKELY explanation on why this person has diabetes (Type II)

- A. The insulin receptor is not receptive to insulin.
- B. There is a problem with the p13 kinase.
- C. The IRS protein is not phosphorylated
- D. The GLUT4 transporter cannot sense glucose in the plasma.

Answer: A

Explanation: This is upstream of the phosphorylation of the insulin receptor.

## Lecture 14

**Question:** I really need to relax, so I drink some of my grandpa's wine. Which of the following describes the mechanism of how I can relax?

List as:: (type of potential-- transmitter-gated ion channels that are activated -- Voltage gated cation channels activated)

- A. EPSP- Ach- Na<sup>+</sup> and Ca<sup>2+</sup> channels
- B. IPSP- GABA- K<sup>+</sup> and Cl<sup>-</sup> channels.
- C. EPSP- GABA- K<sup>+</sup> and Cl<sup>-</sup> channels.
- D. IPSP- Ach- Na<sup>+</sup> and Ca<sup>2+</sup> channels

Answer: B

Explanation: We want an inhibitory signal because I want to relax, not get excited!!

The only correctly matched one is B, because K<sup>+</sup> and Cl<sup>-</sup> leads to inhibitory responses.

**Question:** Anticonvulsants are anti-seizure medications, used for treatment in disorders like epilepsy and bipolar disorder. Which of the following can explain its mechanism of action? (Hint: seizures are spontaneous excitatory stimulations).

- A. Inhibit Na<sup>+</sup> channels
- B. Enhance GABA channels
- C. Inhibit K<sup>+</sup> channels
- D. A and B
- E. All of the above

Answer: D

Explanation: Sodium is excitatory, GABA and K<sup>+</sup> are inhibitory. From a normal action potential, we know that efflux of K<sup>+</sup> will lower the membrane potential so we should enhance K<sup>+</sup> channels and not inhibit it.

**Question:** SSRIs, Selective Serotonin Reuptake Inhibitors, can be used to keep a high concentration of extracellular serotonin for treatment of disorders like depression and anxiety. What will be affected by this high concentration of serotonin?

- A. There will be less interaction with G-proteins
- B. There will be a longer absolute refractory period.
- C. There will be increased activity of adenylyl cyclase.
- D. It will lower the threshold potential.

Answer: C

Explanation:

I had the misconception that neurotransmitters all operate under ionotropic and not metabotropic mechanisms. This question gets at neurotransmitters in metabotropic signaling (i.e. it can activate G-proteins and thus increase AC). With increased serotonin, it will interact more with G-proteins. It will have nothing to do with threshold potential and absolute refractory period.

**Question:** Acetylcholine is an important neurotransmitter in muscular contraction. What will be the fastest way to get rid of acetylcholine from extracellular spaces?

- A. Diffusion
- B. Endocytosis at the postsynaptic terminal
- C. Acetylcholinesterase, an enzyme that breaks down acetylcholine
- D. A or B can be correct
- E. B or C can be correct

Answer: E

Explanation: We should know that diffusion takes a heck of a long time.

**Question:** Which of the following mechanisms/ actions will stop an action potential from propagating?

- A. Inhibit calcium from entering the presynaptic terminal
- B. Inhibit fusion of vesicles with presynaptic membrane
- C. Inhibit chlorine from entering the presynaptic terminal
- D. A and B
- E. B and C

Answer: D

**Explanation:** Action potentials include electrical and chemical signaling. The choices above are for chemical signaling. Chlorine doesn't have to do anything with action potential propagation.

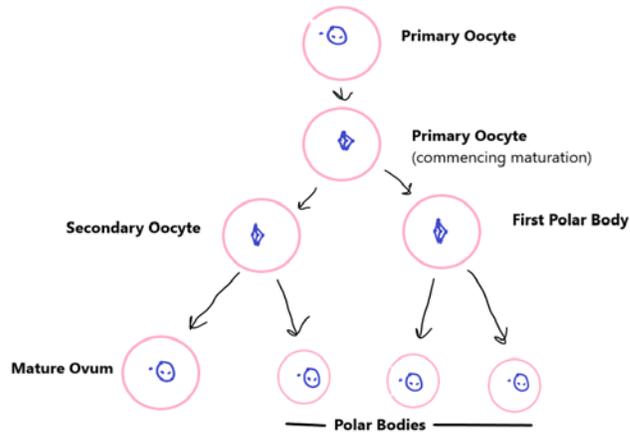
## Lecture 15

**Question:** You are studying a cell that has a mistake in a chromosome and one of its duplicates. Which checkpoint missed this mistake?

- A. G1 checkpoint
- B. G2 checkpoint
- C. Anaphase to Metaphase
- D. G0 checkpoint

Answer: A

Explanation: The G1 checkpoint is supposed to check whether all the chromosomes are good to divide before it actually divides. It also checks to see if the environment is conducive to division.



**Question:** Above shows a picture of oocyte cell division. Considering the size and DNA amount of each structure, where does oocyte cell division and normal cell division differ?

- A. G1 phase
- B. G2 phase
- C. S phase
- D. Prophase
- E. Cytokinesis

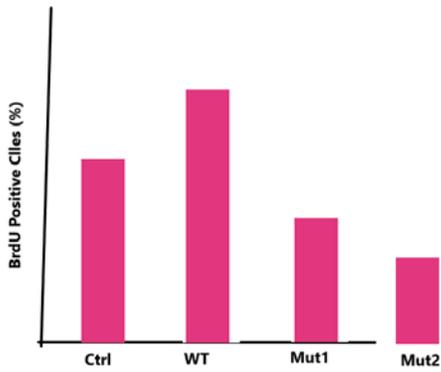
Answer: E

Explanation: Uneven division but same amount of DNA.

The oocytes are stuck in Prophase/ Metaphase for a longer period of time but we are considering size and DNA amount and not time.

**Question:** A patient with MadeUpDisease keeps on replicating DNA with a mutation on gene number 44. What is NOT a sufficient explanation about why the patient's cells keeps on dividing?

- A) There's something wrong in the microtubules in mitosis that separate the chromosomes.
- B) There is something wrong at the G1-S phase checkpoint.
- C) There is something wrong with in CDK activity.
- D) There is something wrong with transcription and translation.



**Question** We do a BRDU assay and we see this graph. What of the following is *most likely* corresponds to a neuron?

- A) Ctrl
- B) Wild type
- C) Mut1
- D) Mut 2

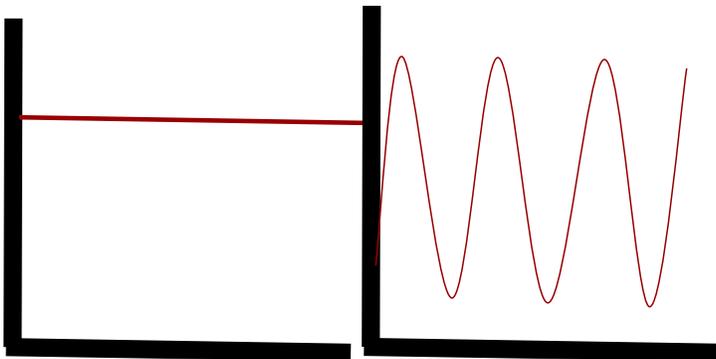
Answer: D

Explanation:

Mutant 1 could be accepted if there was a good explanation.

But neurons are *nullipotent* cells and they do not divide. BRDU is a test in living cells (inject living cells with BRDU to and stain it red; purple ones show if the cell incorporated it into the DNA).

However, there were some evidence in lecture that some neural cells can divide, so there will be some purple (some BRDU positive cells).



**Question:** Below shows two graphs. Which answer choice matches the graph to the correct description?

- A. The left graph shows number of cells and the right graph shows levels of DNA
- B. The left graph shows levels of CDK and the right graph shows levels of cyclin

- C. The left graph shows levels of CDK and the right graph shows activity level of CDKs
- D. A and B are correct
- E. B and C are correct

Answer: E

Explanation: Levels of CDK are always constant, but it is not always active because it depends on the level of cyclin. Cyclin are constantly being degraded and made, so it cycles like the right graph.

## Lecture 16

**Question:** A cell has made it to G1 checkpoint but decides not to divide due to a mistake in one of the chromosomes. Which of the following statements is false?

- A. Levels of Cyclin A are low.
- B. Levels of CDK are low.
- C. Activity levels of Wee1 kinase are high
- D. Levels of inhibitory proteins are high
- E. All of the above are false.

Answer: B

Explanation: levels of CDK are constant, activity levels change due to cyclins and phosphates.

**Question:** A cell is dividing uncontrollably. What is associated with this phenomenon?

- A. Ubiquitin ligases for cyclin not working properly
- B. High activity levels of wee1 Kinase
- C. Low activity levels of CDC25 phosphatase
- D. High levels of inhibitory proteins

Answer: A

Explanation: Ubiquitin ligases targets cyclin for degradation, which influences its fluctuating levels throughout the cell cycle.

**Question:** Angelina Jolie is one of millions of Americans who is affected by breast cancer. Which of the following statements can describe her cells at the time of her diagnosis?

- A. High levels of Mitogen-activated protein kinase (MAPK) which activates mitogens
- B. High levels of Wee1 kinase
- C. High levels of CAK
- D. A and B
- E. A and C

**Answer:** E

Explanation: CAK puts the activating phosphate, mitogens encourage mitosis  
Shows that phosphates can be activating or inhibitory

**Question:** High levels of proteasome activity will prevent cancer. Is this statement true or false, and mention why.

- A. True, because proteasomes will target cyclins which is necessary for mitosis.

- B. True, because proteasomes will target p53 which will inhibit mitosis.
- C. False, because proteasomes will target certain pro-apoptotic factors which kill cancer cells
- D. False, proteasomes target ubiquitinated proteins, and these proteins can either encourage or inhibit mitosis.

**Answer:** D

**Explanation:**

In class we learned two ubiquitin ligases that target proteins (i.e. cyclins and p53) which will result in inhibition of division. However, proteasomes will degrade any ubiquitinated proteins which can be pro- or anti-mitosis. An example is Bortezomib, a proteasome inhibitor used in cancer therapy.

**Question:** During World War II, atomic bombs were dropped in Hiroshima and Nagasaki. These atomic bombs set up waves of radiation that damaged DNA. This DNA damage is associated with...

- A. High levels of p21 transcription
- B. High levels of p53/ mdm2 degradation
- C. High levels of p53 activating phosphorylation
- D. A and B are correct
- E. A and C are correct

Answer: E

Explanation: p21 is inhibitory protein. P53 needs to be activated to increase p21 transcription (it is a transcription factor and pro-apoptotic factor)

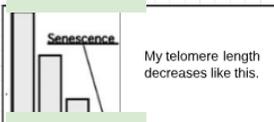
## Lecture 17

**Question:** Match the box to the description.



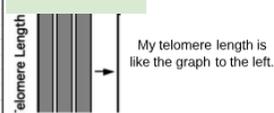
## Who am I?

### QUESTION 1



A Somatic cell

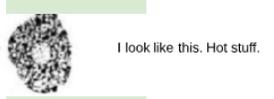
### QUESTION 2



B STEM/ Embryonic Cell

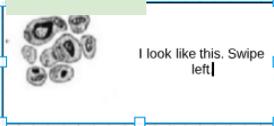
C Cancer Cell

### QUESTION 3



D Cell undergoing necrosis

### QUESTION 4



E Cell Undergoing apoptosis

### Answer:

- Q1 → A
- Q2 → B, C
- Q3 → D
- Q4 → E

### Explanation:

Somatic cell goes with the first box because telomere length decreases, leading to cellular senescence. STEM/ Embryonic cells and cancer cells goes to the second box because their telomeres don't shorten. A cell undergoing necrosis would look like the picture because that represents cell lysis (leading to inflammation), random DNA fragmentation, broken membranes, cellular swelling). A cell undergoing apoptosis would look like the picture. Those are apoptotic bodies. (ladder-like DNA)

**Question:** Gene therapy is a gene therapy method used in China to treat cancer. It acts to overexpress the p53 gene. What will this do to the cells?

- A. Induce apoptosis
- B. Enhance synthesis of inhibitory proteins like p21
- C. Induce expression of mitogens
- D. A and B are correct

E. A and C are correct

**Answer:** D

**Explanation:** p53 is a transcription factor that helps expression of inhibitory proteins, and it also induces apoptosis.

**Question:** you are a scientist and you have isolated a cell that has no telomerase activity. Which of the following cells can this be?

- A. Neuron
- B. Breast cancer cell
- C. Differentiated liver cell
- D. A and B are correct
- E. A and C are correct

**Answer:** E

**Explanation:** Neurons, cardiomyocytes, etc do not differentiate and do not have telomerase activity. Differentiated cells do not have telomerase activity.

**Question:** Human papillomavirus (HPV) encodes a protein that binds to p53 and inactivates it. Which of the following statements can describe a result of this inactivation?

- A. Inactive telomerase
- B. Replication of damaged chromosomes
- C. Ability to respond to DNA damage by radiation
- D. Upregulation of cyclin activity

**Answer:** B

**Explanation:**

P53 has a role in the G1/S checkpoint, and an inactive p53 will result in replication despite damage. P53 also is a TF for p21, which explains D and responds to DNA damage which explains C. P53 helps explain cellular senescence but doesn't directly affect telomerase (?)

**Question:** You are a doctor and a patient comes in for frostbite. Due to your extensive training, you know that the dark spot on your patient is caused by cellular necrosis and not cellular apoptosis. Which of the following medications will you prescribe to help the symptoms of this?

- A. An anti-inflammatory, to treat inflammation caused by cell lysis
- B. A protein that binds to caspase 8
- C. A drug to treat edema caused by cellular swelling
- D. A and B
- E. A and C

**Answer:** E

**Explanation:** B is apoptosis. A and C (cell lysis and swelling) have to do with necrosis and not apoptosis.

## Lecture 18

**Question:** The protein that transports SMAC/ Diablo out of the mitochondria is blocked. How will this affect the cell?

- A. Apoptosis will occur.
- B. Apoptosis cannot occur.
- C. Apoptosis will partially occur.

**Answer:** B

**Explanation:** Apoptosis cannot occur because SMAC/ Diablo are pro-apoptotic factors that inhibit the Inhibitor of Apoptosis Proteins (IAPs)

**Question:** You are principal investigator of a lab and an undergraduate comes to you with two isolated cells that he wonders is going through necrosis or apoptosis. Cell A has a fragmented DNA ladder and has more phosphatidylserine in the membrane. Cell B is condensed and the membranes remain intact.

- A. Cell A is going through apoptosis and Cell B is going through necrosis.
- B. Cell A is going through necrosis and Cell B is going through apoptosis.
- C. Cell A and Cell B are both going through necrosis.
- D. Cell A and Cell B are both going through apoptosis.

**Answer:** D

**Explanation:**

Both Cell A and Cell B have characteristics of apoptosis.

**Question:** Bcl-x is a anti-apoptotic factor (similar to CED-9 in *C. Elegans*). Knowing this information, which of the following statements can describe its mechanism of action?

- A. Bcl-x prevents the release of SMAC/ Diablo
- B. Bcl-x closes the pores of the mitochondria, preventing the release of Cytochrome C
- C. Bcl-x prevents the release of IAPs (Inhibitor of Apoptosis Proteins)
- D. A and B are correct
- E. A and C are correct

**Answer:** D

**Explanation:** SMAC/ Diablo and Cytochrome C favor apoptosis so Bcl-x must inhibit it.

**Question:** In *C. Elegans*, CED-9 fails to cleave CED-4 from CED-9. How will this affect apoptosis?

- A. The active part of CED-4 will still go on to activate CED-3 to trigger apoptosis.
- B. The active part of CED-4 will only partially activate CED-3 and partially trigger apoptosis.
- C. CED-4 will not be active but IAPs in *C. Elegans* will trigger apoptosis.
- D. CED-4 will not be active and cannot trigger apoptosis.

**Answer:** D

**Explanation:** This gets into the concept of zymogens; CED-4/CED-9 will not be active until parts of it are cleaved.

**Question:** The HIV (human immunodeficiency virus) progresses because of the induced apoptosis of T-helper lymphocytes, which are integral cells to the immune system). Which of the following statements can describe its mechanism?

- A. HIV enzymes deactivate Bcl-2.
- B. HIV enzymes bind to CED-4 which prevent its cleavage from CED-9.
- C. HIV enzymes prevent the release of Cytochrome C.

D. HIV enzymes stimulate the production of Inhibitor of Apoptosis Proteins (IAPs).

**Answer:** A

**Explanation:** Deactivating Bcl-2, an anti-apoptotic factor, will induce apoptosis. Choices B, C, and D will inhibit apoptosis.

## Lecture 19

**Question:** First I was afraid, I was petrified. Will I die? Yes or no.

- 1) My TNF receptors are not working.
- 2) I have excess BAD protein.
- 3) My Bax channels are permanently closed.
- 4) My neurotrophins cannot bind to my trophic factor receptor.
- 5) My zymogens of the caspases are uncleavable.

- A. Yes the cell will die.
- B. No the cell will not die.

Answers:

- 1) B. OH NO NOT I, I WILL SURVIVE! AS LONG AS I CANNOT BIND I KNOW I WILL SURVIVE! If my TNF receptors are not working, then my cells cannot bind to the Tumor Necrosis Factor which activates the FADD/ TRADD/ which activates a series of caspases that will lead to death.
- 2) A. RIP. BAD protein will lead to binding to BCL-2 which will allow Bax to cause the release of cytochrome C through influx of ions. (Cytochrome C will then lead to an apoptotic pathway).
- 3) B. OH NO NOT I, I WILL SURVIVE! AS LONG AS I KNOW HOW TO CLOSE, I KNOW I WILL SURVIVE! With Bax closed, there is no ion gradient that will lead to the release of cytochrome C.
- 4) A. Darn, unfortunately I will pass. Neurotrophins are anti-apoptotic, in that it leads to release of BAD from BCL-2. If there are no neurotrophins (or other trophic factors), Bad will stay bound to Bcl-2.
- 5) B. OH NOT I, I WILL SURVIVE! AS LONG AS I AM UNCLEAVABLE, I KNOW I WILL SURVIVE!

**Question:** Cachexia, aka "wasting syndrome," is a disease that affects the extrinsic pathway of apoptosis. One symptom is the degradation of skeletal muscle cells. Knowing this information, which of the following statements best describes a possible mechanism of cachexia?

- A. It inactivates the "Death Domain"
- B. It inactivates the release of trophic factors
- C. It inhibits Bcl-2 protein from inhibiting apoptosis
- D. It increases the release of TNF (Tumor Necrosis Factor)

**Answer:** D

**Explanation:** B and C have to do with the intrinsic pathway. A inhibits apoptosis.

**Question:** you are a scientist trying to grow induced pluripotent cell. You give the cell the correct factors (O, S, K, M). How will you know if the cell has successfully undergone the transformation?

- A. You test it in a rat and if the rat dies, you have failed

- B. You give it antibiotics, and the ones who survive have successfully transformed
- C. You give it glucose and if it has taken in glucose, then it has successfully transformed
- D. You give it glucose and if it grows to another stem cell then it has successfully transformed

**Answer:** B

**Explanation:** Embryonic stem cells and iPS cells contains an antibiotic resistant gene. If it has transformed, it will survive antibiotics

**Question:** You are principal investigator of a lab and you notice than an undergraduate has left a cell out on a petri dish. The undergraduate forgot which type of cell it is, but knows that it is either an embryonic stem cell, iPS cell, or adult stem cell. You try to test it and regrow the cell and you see that after 40 platings, the cell can no longer grow. What type of cell is it?

- A. Embryonic stem cell
- B. iPS cell
- C. Adult stem cell
- D. The cell has died after 40 platings, so we cannot tell

**Answer:** C

**Explanation:** Adult stem cells have senescence

Tumor necrosis factor (TNF) is associated with the inflammatory response of autoimmune diseases like rheumatoid arthritis. Those with these diseases take TNF-inhibitors, which prevent this inflammation.

Which of the following statements are associated with taking TNF-inhibitors?

- A. Procaspase 8 being cleaved
- B. Formation of the death receptors
- C. Blockage of TNF receptors to prevent binding to TNF
- D. Stimulation of apoptosis

**Answer:** C

**Explanation:** A, B, and D will happen if TNF is stimulated (it is part of the extrinsic apoptotic pathway)

## Lecture 20

**Question:** Which of the following is can NOT potentially stop a tumor from spreading?

- A) Don't give it growth factors
- B) Somehow inhibit its telomerase activity
- C) Increase the tumor's susceptibility to contact inhibition
- D) Stop the tumor from creating its own blood vessels
- E) Keeping the tumor confined in the lumen

**Answer:** A

**Explanation:** The main point of this question is to realize the five phenotypes that distinguish cancer from normal cells

- 1) Lack of susceptibility to growth factors
- 2) Cellular senescence
- 3) Lack of contact inhibition
- 4) Angiogenesis

5) Metastasis

Thus, since a tumor is NOT affected by growth factors, A is the correct answer.

**Question:** Bevacizumab is a drug that is used to stop the growth of cancerous tumors. Knowing this information, which of the following statements most likely explains its mechanism of action?

- A. The drug binds to VEGF receptors, which prevent its blood vessel development
- B. The drug prevents angiogenesis so the tumor does not grow large
- C. The drug stimulates blood vessel development to provide oxygen to the cells
- D. A and B are correct
- E. A and C are correct

**Answer:** D

**Explanation:** Bevacizumab is a monoclonal antibody that binds to VEGF receptors to prevent angiogenesis, which is what statements A and B are saying. Choice C is saying the opposite.

**Question:** You are a cytologist interpreting the results of someone who just took a Fine Needle Aspiration Biopsy. You want to determine if the cancer cells have undergone metastasis. Which of the following statements describe the cytology of cancer cells which have undergone metastasis?

- A. Detection of an increase in actin filaments
- B. Increase in cells in the bloodstream
- C. An intact plasma membrane
- D. A and B are correct
- E. All of the above are correct

**Answer:** D

**Explanation:** A shows the invadopodia consisting of actin filaments. B shows the cancer cells that invade the bloodstream. In metastasis, the plasma membrane is broken.

**Question:** True or False. Cancer-causing mutations are caused by gain of function mutations since cancer cells are hyperactive and need more function to grow.

**Answer:** False, it can be a GOF of proto-oncogene or LOF of tumor suppressor gene.

**Question:** You are a scientist interpreting the results of the efficacy of a drug, but unfortunately the page you are interpreting is smeared! You can read that the Gain of Function of (blank) leads to cancer. What can the blank be?

- A. Tumor suppressor gene
- B. Proto-oncogene
- C. P53, guardian of genome
- D. A and B
- E. A and C

**Answer:** B

**Explanation:** GOF of proto-oncogenes lead to cancer, LOF of tumor suppressor genes like p53 will also lead to cancer.

## Lecture 21

**Question:** There is a loss of function mutation in the Signal Recognition Protein (SRP), and it cannot hydrolyze GTP. Which of the following statements is true?

- A. The ribosome cannot dissociate from the translocon
- B. The signal sequence cannot be cleaved
- C. The SRP cannot stop translation
- D. SRP cannot be recycled for another cycle

**Answer:** D

**Explanation:** SRP leaves the SRP receptor when GTP is hydrolyzed.

**Question:** Which statement below best explains the difference between transporting water-soluble proteins and membrane proteins?

- A. Membrane proteins do not need SRPs and water-soluble proteins do
- B. Membrane proteins have a domain that can exit out a translocon but water-soluble proteins do not
- C. Membrane proteins enter the translocon with the N-terminus first but water-soluble proteins do not
- D. Membrane proteins are made by smooth ER but water-soluble proteins are made by rough ER
- E. None of the above are true

**Answer:** B

**Explanation:** Membrane proteins have a transmembrane domain that can exit out the translocon and be transplanted the bilayer.

**Question:** You are a scientist watching a super high-tech play-by-play of protein transport. The narrator of the play-by-play failed to mention what type of protein is being transported. Being the smart scientist you are, you know that...

- A. If it is fully folded, the protein will end up in the mitochondria
- B. If it is not fully translated, the protein could be a membrane protein
- C. If it has a transmembrane domain, the protein could be a membrane protein
- D. B and C are correct
- E. All of the following are correct

**Answer:** E

**Explanation:** If it is fully folded it cannot enter the translocon. Post-translational proteins are held unfolded by chaperones.

B and C are correct because it is cotranslational translocation.

**Question:** A translocon not only helps with transport of other proteins, but it is also a protein itself. Which of the following statements are true about translocons?

- A. Translocons require a signal recognition particle
- B. Translocons have a transmembrane domain
- C. Translocons are resident proteins
- D. A and C are correct
- E. All of the above are correct

**Answer:** E

**Explanation:** Translocons are transmembrane resident proteins (of the ER or organelle)

\*\* double check since translocons are resident proteins \*\*

**Question:** Which of the following is NOT needed for transport of proteins to the mitochondrion?

- A) Tim and Tom
- B) SRP binding
- C) Chaperone proteins
- D) Mitochondrial signaling sequence

**Answer:** B

Tim and Tom are Translocon of inner and outer membrane. Since the mitochondria has two membranes, it needs a protein to transport the peptide in.

Chaperone proteins are necessary to keep the proteins unfolded.

The mitochondrial signaling sequence allows the peptide to know where to go.

## Lecture 22

**Question:** Which of the following statements will describe a case that allows a protein to stay in the ER?

- A. If the Golgi was as basic as the ER
- B. If there was a LOF mutation in COPII proteins
- C. If there was a LOF mutation in COPI proteins
- D. A and B are correct
- E. B and C are correct

Answer: E

Explanation:

If the golgi was as basic as the ER, the protein will be secreted

No COPII proteins, no transport from ER to Golgi.

No COPI proteins, retrograde transport will cease but also anterograde will slowly cease as well

**Question:** The ninth step of vesicle mediated transport (picture above) is fusion with acceptor membrane. Which of the following statements will describe a case that DOES NOT allow vesicle fusion?

- A. Inability to hydrolyze GTP
- B. Use of GTP- $\gamma$ -S instead of GTP
- C. Loss of function in SNARE proteins
- D. A and B are correct
- E. All of the above are correct

Answer: E

Explanation: All of the above are correct because vesicles will accumulate if we cannot hydrolyze GTP to shed the coats, and SNARE proteins are necessary in docking the vesicle at the acceptor membrane.

**Question:** There is a mutation in a KDEL receptor. Instead of recognizing a KDEL sequence, it recognizes a sequence that sends the protein to the membrane. In this scenario, where will we find a protein destined for the membrane?

- A. The membrane
- B. The Golgi

- C. The ER
- D. The lysosome

**Answer:** C

**Explanation:** KDEL sequences bring ER resident proteins back to the ER. Since it recognizes a membrane protein instead, it will bring membrane proteins back to the ER

**Question:** There is a problem with the mannose-6-phosphate receptor in the trans-golgi. Instead of recognizing the M6P, it recognizes the KDEL sequence. Where would a ER resident protein be found in this case?

- A. The membrane
- B. The lysosome
- C. The Golgi
- D. The ER

**Answer:** D

**Explanation:** The ER, because the KDEL sequence gets recognized in the cis-golgi (TRICKY)

I am a protein in the ER and I want to get to the Golgi. Which of the following will NOT help me get into the cis-Golgi?

- A) COP II vesicle
- B) SNARE Proteins
- C) Protein receptors in the ER membrane
- D) COP I vesicles
- E) GTP

Answer: D

COPII vesicles are for anterograde transport from ER to Golgi.

SNARE proteins help the vesicle dock at the cis

Protein receptors in the ER membrane will allow me to stick to the receptors in the vesicles and bud out.

GTP will help COPII proteins to uncoat so snare proteins, etc. can get on.

COP I vesicles are NOT needed.

## Lecture 23

**Question:** Lumacraftor is a drug that is used to treat the symptoms of cystic fibrosis, a genetic disorder that affects protein trafficking. Without Lumacraftor, a mutation in the F508 gene will prevent the transport of a chloride protein channel to the cell membrane of exocrine glands (like sweat glands) to regulate the formation of sweat and mucus. This means that with Lumacraftor...

- A. Adaptor proteins will not coat the vesicle for transport
- B. The protein will be transported to the basolateral side
- C. There will be an increase in transport of chloride protein channels to the apical side
- D. There will be a greater prevalence of protein mutations

**Answer:** C

**Explanation:** That's what lumacraftor does. A talks of another mutation; B is incorrect because protein channels are supposed to be apical and not basolateral; D means no protein mutations

**Question:** Protein trafficking diseases like nephrogenic diabetes insipidus are characterized by the accumulation of mutant proteins at the endoplasmic reticulum. Which of the following statements are possible medications to prevent the onset of these diseases?

- A. Increase activity of chaperone proteins to stimulate proper folding of proteins
- B. Increase activity of adaptor proteins to stimulate transport of proper proteins
- C. Increase activity of exocytosis so mutant proteins can exit the cell into the body
- D. A combination of A and B
- E. A combination of A and C

**Answer:** D

**Explanation:** A and B are the mechanism of Lumacraftor (for cystic fibrosis). Mutant proteins into the body (choice C) will not help the onset of diseases.

**Question:** An epithelial cell has a mutation in their adaptor proteins and it cannot transport their protein channels on the apical side. Which of the following methods can alleviate this problem?

- A. Fix the mutation in the adaptor proteins
- B. Use adaptor proteins from a neuron
- C. Increase receptor activity to stimulate exocytosis
- D. A and B are correct
- E. A and C are correct

**Answer:** D

**Explanation:** Fixing the mutation will obviously help. Since adaptor proteins are conserved through different types of cells, B will also help. C doesn't have to do with trafficking to the apical membrane.

**True/ False:** Cholesterol uptake via pH gradient holds the same principles as KDEL retrieval via pH gradient.

**Answer:** False

**Explanation:** Cholesterol binds at high pH while KDEL binds at low pH.

**Question:** A patient presents with atherosclerosis. Cholesterol keeps on clotting in the blood. After many tests (that are yet to exist) the doctor confirms that the etiology is linked to cholesterol uptake in the cell. Which of the following leads to a problem in LDL uptake?

- A) Hydrophobic LDL is binds to apolipoprotein B so it can be taken into the cytosol
- B) The pH inside the cell progressively gets *higher*
- C) LDL is uncoated from its receptor sometime inside the cell
- D) LDL is transported by endosomes instead of vesicles inside the cell

Answer: B

Explanation:

LDL binds to its receptor at high pH and dissociates at low pH (unlike KDEL). Its dissociation causes release in the cell

## Lecture 24

**Question:** In a dividing cell, which of the following proteins will be most important?

- A. Microfilaments
- B. Intermediate filaments
- C. Microtubules
- D. Kinesin

**Answer:** B

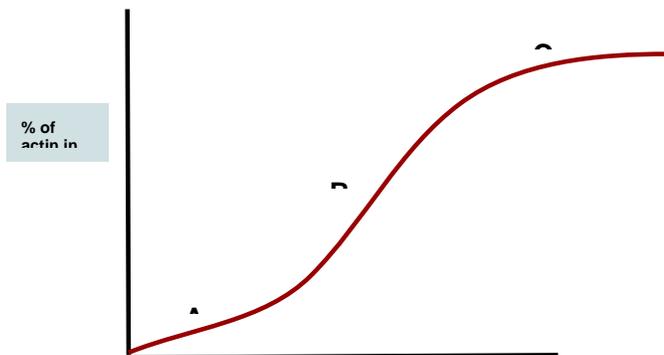
**Explanation:** Intermediate filaments are used to form the mitotic spindle

**Question:** You are studying a microfilament and it has lost the ability to hydrolyze ATP. Which of the following statements best describes what will happen to the microfilament?

- A. It will shorten
- B. It will lengthen
- C. It will stay the same length
- D. It will fall apart completely

**Answer:** B

**Explanation:** Actin-ATP have high affinity to actin-ATP. Thus it will grow. (To grow, the microfilament must add on to the + side faster than it hydrolyzes ATP on the - end).



The following questions refer to the graph above.

**Question:** The following statement(s) describe(s) Part C of the graph:

- A. This part of the graph shows nucleation
- B. This part of the graph shows treadmilling
- C. This part of the graph shows the microfilament at a constant length
- D. A and C are correct
- E. B and C are correct

**Answer:** E

**Explanation:** Part C shows the steady state/ treadmilling, where the rate of actin added equals the rate of actin removed.

**Question:** The following statement(s) describe(s) Part B of the graph:

- A. This part of the graph shows nucleation

- B. This part of the graph only happens when the amount of actin filaments are above critical concentration
- C. This part of the graph shows the filament growing
- D. A and C are correct
- E. B and C are correct

**Answer:** E

**Explanation:** Part B of the graph shows the filament growing (it is the elongation phase) and only happens with enough actin concentration.

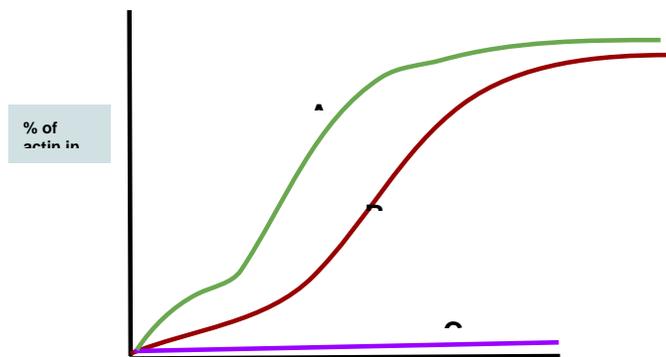
**Question:** The following statement(s) describe(s) Part A of the graph:

- A. This part of the graph shows nucleation
- B. This part of the graph is forms the critical mass to add more actin units
- C. This part of the graph shows the filament at a constant length
- D. B and C are correct
- E. A and C are correct

**Answer:** D

**Explanation:** This is nucleation, and choice B is the definition of nucleation

## Lecture 25



The following questions refer to the figure above. The critical concentration of this particular filament is denoted by  $C_c$ .

**Question:** Curve C refers to the situation when...

- A. The concentration of monomers is less than  $C_c$ .
- B. The concentration of monomers is more than  $C_c$  and can be estimated by  $C_c+1$
- C. The concentration of monomers is more than  $C_c$  and can be estimated by  $100C_c$
- D. The concentration of monomers is equal to  $C_c$
- E. Two or more of the above can be correct.

**Answer:** A

**Explanation:** Nothing can happen if the monomer concentration is less than critical concentration

**Question:** Curve B refers to the situation when...

- A. The concentration of monomers is less than  $C_c$ .
- B. The concentration of monomers is more than  $C_c$  and can be estimated by  $C_c+1$
- C. The concentration of monomers is more than  $C_c$  and can be estimated by  $100C_c$
- D. The concentration of monomers is equal to  $C_c$
- E. Two or more of the above can be correct.

**Answer:** E

**Explanation:** Curve B is normal, and the concentration of monomers can be equal to  $C_c$  or slightly more ( $C_c + 1$ )

**Question:** Curve A BEST refers to the situation when...

- A. The concentration of monomers is less than  $C_c$ .
- B. The concentration of monomers is more than  $C_c$  and can be estimated by  $C_c+1$
- C. The concentration of monomers is more than  $C_c$  and can be estimated by  $100C_c$
- D. The concentration of monomers is equal to  $C_c$
- E. Two or more of the above can be correct.

**Answer:** C

**Explanation:** Since there is a faster nucleation phase, the monomer concentration must be a LOT higher than  $C_c$

**Question:** Latrunculin is a medication that prevents the polymerization of microfilaments. Knowing this information, which of the following statements can describe a possible mechanism of latrunculin?

- A. Latrunculin prevents the ATPase activity of actin-ATP.
- B. Latrunculin binds to actin and prevents it from interacting with other actin
- C. Latrunculin extends the nucleation phase of the microfilament.
- D. A and C are possible answers
- E. B and C are possible answers

**Answer:** E

**Explanation:** B prevents actin from polymerizing; A will allow actin to keep growing because it cannot convert ATP  $\rightarrow$  ADP (and actin-ATP have high affinity for each other). Extending the nucleation phase will prevent growth.

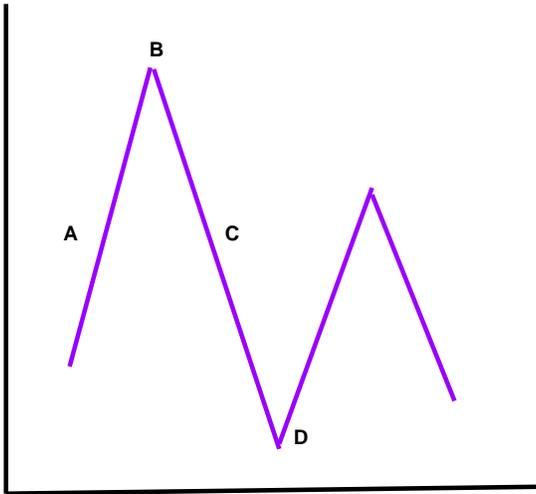
**Question:** The  $C_c^+$  of a filament is 5 mM, and the  $C_c^-$  of a filament is 7 mM. Currently the concentration of monomers is 6 mM. Which of the following statements describe the current situation?

- A. This is called steady state.
- B. This is called treadmilling.
- C. The rate of addition is equal to the rate of removal.
- D. A and C are correct
- E. B and C are correct

**Answer:** B

**Explanation:** Choice C is the definition of Choice A (steady state). The situation above doesn't describe steady state, it describes treadmilling.

## Lecture 26



**Question:** Which of the following statements describe Part B of the graph above?

- A. There is a GTP cap
- B. There is a factor like MAP present
- C. There is low concentration of ATP
- D. The filament is growing quickly
- E. None of the above are correct

**Answer:** E

**Explanation:** If there is a GTP cap or a 'rescue' factor present like MAP, it will grow (Part A of the graph). ATP has to do with microfilaments and not microtubules.

**Question:** Which of the following statements describe Part D of the graph above?

- A. The microtubule is gaining a GTP cap
- B. There is a factor like MAP present
- C. There is high concentration of ATP
- D. A and B can be correct
- E. None of the above are correct

**Answer:** D

**Explanation:** Part D shows rescue, and the microtubule will gain a GTP cap and start growing again. It is possible that a stabilization factor is present. ATP doesn't have to do with growth of microtubules.

**Question:** Which of the following statements describe part D of the graph above?

**Question:** A microtubule is currently growing. Which of the following statement(s) describe the current situation?

- A. There is a GTP cap
- B. There is a catastrophe factor present, like kinesin
- C. There will be a lower concentration of alpha/beta tubulin dimer.
- D. A and B are correct
- E. A and C are correct

**Answer:** E

**Explanation:** Catastrophe factors will cause catastrophes (rapid depolymerization). There is a lower concentration of the dimer because it is being added to the microtubule.

**Question:** Colchicine is a drug that prevents the polymerization of microtubules. Which of the following statement(s) describe the possible mechanism of this drug?

- A. Colchicine binds to actin filaments to prevent it from interacting with each other
- B. Colchicine binds to tubulin dimers to prevent it from interacting with each other
- C. Colchicine prevents the GTP cap in microtubules from falling off
- D. Colchicine inhibits catastrophe factors, like kinesin 13

**Answer:** B

**Explanation:** Actin doesn't have to do with microtubules; Choices C and D will cause the polymerization of microtubules.

**Question:** A piece of cellular cargo is being transported to the nucleus. Which of the following statements correctly describe the situation?

- A. The cargo is being carried by the tails of the motor protein
- B. The cargo is being carried by a kinesin
- C. The cargo is being carried by a protein that cannot hydrolyze ATP
- D. A and B are correct
- E. A and C are correct

**Answer:** A

**Explanation:** Cargo is carried by motor protein tails. It would be carried by a dynein. If it is moving, the motor protein must hydrolyze ATP.

## Lecture 27

**Question:** At the end of the Muscle Power Stroke, SERCA (SR calcium pump) pumps calcium back into the sarcoplasmic reticulum. What happens if there is a mutation in the SERCA pumps?

- A. Tropomyosin will cover the actin binding sites
- B. Prolonged contraction
- C. Muscle fatigue
- D. Actin cannot bind to myosin

**Answer:** B

**Explanation:** Since calcium is not pushed back into the SR, there will be calcium available to bind to troponin to take tropomyosin off the actin binding sites. Thus, actin can bind to myosin. There will be prolonged contraction because the muscle power stroke ends when calcium is pumped back in the SR.

**Question:** There is a mutation in the myosin head and it has lost its ability to hydrolyze ATP. Which of the following statements describe what will happen?

- A. The myosin head will be attached to actin
- B. The myosin head will be in the cocked position
- C. The myosin head will be in a low energy position
- D. The myosin head will be able to pull the z-lines to the m-lines of the sarcomere

**Answer:** C

**Explanation:** ATP will decrease the affinity of the myosin head to actin, and thus will be in the low-energy position (and the head will not be attached to actin). It will not be in the cocked position, because that is when ATP is hydrolyzed. The myosin head will not be able to work in the Sliding Filament mechanism without being able to hydrolyze ATP.

**Question:** There is a mutation in troponin C and it is unable to recognize calcium. Which of the following statements will describe what happens?

- A. Myosin will not be able to bind to actin
- B. Tropomyosin will cover the actin binding site
- C. There will be an increase in calcium uptake
- D. A and B are correct
- E. All of the above are correct

**Answer:** D

**Explanation:** Without calcium, the actin binding site will remain covered and myosin cannot bind to actin. This is independent of calcium uptake, which is regulated by SERCA pumps.

**Question:** Muscle contraction depends on voltage-gated receptors. Which of the following are voltage-gated receptors?

- A. Acetylcholine receptor on the motor end plate
- B. Ryanodine receptor
- C. Dihydropyridine receptor
- D. T-tubules

**Answer:** C

**Explanation:** The DHAP receptor is opened with the depolarization of the membrane carried by T-tubules (T-tubules help propagate the action potential through the muscle fiber and is not a VG receptor). Ryr receptor is opened mechanically by the DHAP receptor. Acetylcholine receptor is a muscarinic receptor opened by acetylcholine.

**Question:** Put the following statements in the correct order.

1. Ryr receptor is mechanically opened by DHAP
2. Action potential travels down the T-tubule
3. Acetylcholine is released from the motor neuron and binds to receptors on the motor end plate.
4. DHAP is opened by action potential
5. Na<sup>+</sup> comes in
6. Ca<sup>2+</sup> floods out the sarcoplasmic reticulum

- A. 3, 5, 2, 4, 1, 6
- B. 1, 4, 5, 3, 2, 6
- C. 3, 2, 5, 1, 4, 6
- D. 6, 3, 2, 4, 1, 5

**Answer:** A

**Lecture 28**

**Question:** There is a mutation in a voltage gated calcium channel in a neuron. Which of the following will occur?

- A. Neurotransmitters cannot be synthesized
- B. There will be an inhibitory response
- C. There will be an accumulation of vesicles at the presynaptic membrane.
- D. Nothing will happen because Calcium is not involved in neurotransmission; only Na<sup>+</sup> is

**Answer:** C

**Explanation:** Calcium causes vesicles (filled with NTs) to fuse with the membrane

**Question:** What is NOT a way to remove a neurotransmitter from the synapse?

- A. Use acetylcholinesterase (breaks down acetylcholine)
- B. Wait for it to diffuse out the membrane
- C. Use a SSRI (selective serotonin reuptake inhibitor)
- D. All of the above are ways to remove a neurotransmitter from the synapse

**Answer:** C

**Explanation:**

These are all real-world examples of 1) break down 2) diffusion and 3) reuptake. However C INHIBITS reuptake

**Question:** All of the following are ways that small amino acid neurotransmitters (GABA, Glycine, Acetylcholine) and peptide neurotransmitters (endorphins) differ EXCEPT:

- A. Calcium is required for vesicle fusion for small neurotransmitters but calcium is NOT required for vesicle fusion in peptide neurotransmitters
- B. Small neurotransmitters are synthesized in the synaptic terminal but peptide neurotransmitters are synthesized in the cell body
- C. The vesicular membrane for small neurotransmitters are recycled but not for peptide neurotransmitters
- D. Small neurotransmitters can be found in the active zone of the synapse but peptide neurotransmitters can be found anywhere in the synapse

**Answer:** A

**Explanation:**

All are differences except A, because calcium is required in both cases

**Question:** Which of the following are FALSE in regards to steroid hormones?

- A. Steroid hormones need a carrier protein to be carried in blood
- B. Steroid hormones can diffuse through the plasma membrane
- C. Steroid hormones act to make fast changes
- D. Steroid hormones can affect gene transcription

**Answer:** C

**Explanation:** steroid hormones make slow changes (ie puberty!! :( )

**Question:** Which of the following are TRUE in regards to ionotropic versus metabotropic signaling?

- A. Ionotropic signaling results in slow responses but metabotropic signaling results in fast responses
- B. Ionotropic signaling can use the same signals as metabotropic signaling
- C. Ionotropic signaling typically use GPCRS but metabotropic signaling typically use ligand-gated channels
- D. Ionotropic signaling has tons of amplification but metabotropic signaling has no amplification

**Answer:** B

**Explanation:** All of the explanations are opposite except for B, because they can use the same signals (ie both can be stimulated by ACh, etc)

## Lecture 29

**Question:** This question refers to the HP Axis.

Iodine is needed to make thyroxine. If the thyroid gland does not have enough iodine, which of the following statements accurately represent what will happen?

- A. There will be high levels of TRH (Thyroid releasing hormone)
- B. There will be high levels of TSH (Thyroid stimulating hormone)
- C. There will be high levels of thyroxine
- D. A and B are correct
- E. A and C are correct

**Answer:** D

**Explanation:** Low iodine means low levels of thyroxine. No negative inhibition so high levels of TRH and TSH.

**Question:** There is a tumor in the thyroid gland and the thyroid makes excess thyroxine. Which of the following statements accurately represents what will happen?

- A. There will be low levels of TRH
- B. There will be high levels of TSH
- C. There will be low levels of TSH
- D. More than one of the above can be correct

**Answer:** D

**Explanation:** High thyroxine= high negative inhibition= low levels of TRH and TSH

**Question:** Insulin is a peptide hormone made by pancreatic beta cells. Which of the following are true regarding insulin?

- A. Insulin must be bound to a hydrophilic carrier protein in the blood
- B. Insulin can pass through the plasma membrane without any assistance
- C. Insulin binds to nuclear receptors
- D. Insulin can lead to relatively fast responses

**Answer:** D

**Explanation:** A-C describes hydrophobic hormones. Answer D is a characteristic of peptide hormones

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## Regulation of Cortisol Release

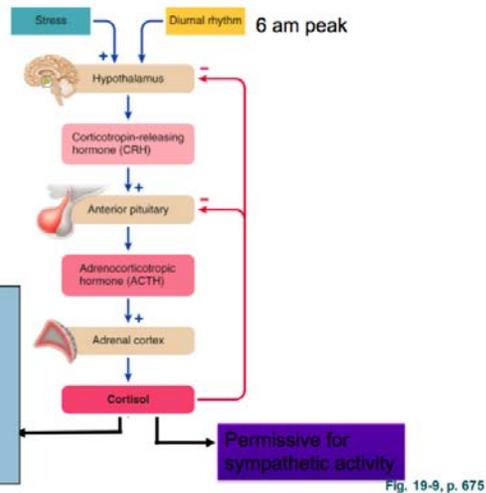


Fig. 19-9, p. 675

In class you have learned about the HP Axis. Above shows an example of the HPA Axis, the Hypothalamus- Pituitary Adrenal Axis. The following questions refer to the figure above.

**Question:** There is a tumor in the adrenal cortex, causing it to increase release of cortisol. This will...

- A. Result in low metabolic actions
- B. Result in low levels of ACTH
- C. Result in hyperactivity in the posterior pituitary
- D. Result in high levels of ACTH

**Answer:** B

**Explanation:** Because high negative inhibition

**Question:** High levels of ACTH can be a result of..

- A. High levels of CRH (corticotropin-releasing hormone)
- B. High levels of cortisol
- C. Hyperactivity of the adrenal cortex
- D. Hypoactivity of the anterior pituitary

**Answer:** A

**Explanation:** high levels of cortisol will result in inhibition, high levels of ACTH will RESULT IN hyperactivity of adrenal cortex. High levels of ACTH is a result of HYPERactivity of anterior pituitary and not hypoactivity.

## Lecture 30

**Question:** Which of the following is false about the circadian rhythm?

- A. The suprachiasmatic nucleus is necessary for circadian timing
- B. Circadian rhythms are regulated by internal cues

Commented [2]: redo this figure

- C. Since light affects the circadian rhythm, eyes are necessary to maintain it.'
- D. Circadian rhythms can be reset

**Answer:** C

**Explanation:** eyes are not necessary because the retinal ganglion cells feed directly to the SCN and not the eyes.

Circadian rhythms CAN be reset (jet lag) and are regulated endogenously. There are experiments (sham lesions in mice) done to prove that SCN is necessary.

**Question:** There is a mutation in the genes and Per and Cry are prevented from dimerizing. Which of the following most accurately describes what might happen? (assume that Per and cry cannot be degraded by the proteasome)

- A. CLOCK and BMAL1 will not be inhibited
- B. The amount of Per and cry proteins will accumulate
- C. There will be normal transcription of Per and cry mRNAs
- D. All of the above are true

**Answer:** D

**Explanation:** Per and cry must dimerize to prevent CLOCK and BMAL1 from activating expression. Thus, CLOCK and BMAL1 will not be inhibited, and there will be normal (un-inhibited) transcription of per and cry. Since there is no autoregulation, Per and cry will accumulate.

**Question:** The per and cry cycle is an example of

- A. Positive feedback loop
- B. Negative feedback loop
- C. Positive allosteric modulation
- D. Competitive inhibition

**Answer:** B

**Explanation:** increased amounts of Per and cry will make it autoregulate and decrease its own synthesis.

**Question:** Which of the following statements is true?

- A. The circadian rhythm cannot be entrained
- B. Abdominal organs like the liver have its own rhythm so are entirely independent of the SCN
- C. An example of a zeitgeber is the internal clock of the pancreas
- D. Using an actogram, we can determine whether an animal is nocturnal or diurnal

**Answer:** D

**Explanation:**

Circadian rhythms can be entrained. Organs have their own rhythm but the SCN synchronizes the clocks. Zeitgeber is an external cue.

**Question:** If there was a mutation in CREB

- A. There cannot be light entrainment
- B. The cycle will stay the same
- C. There will be a decrease in per and cry
- D. All of the above are correct

**Answer:** D

**Explanation:** CREB increases per and cry synthesis in light entrainment, but per and cry is an intrinsic cycle that does not need external cues.

## Lecture 31

**Question:** Which of the following is true about epigenetics?

- A. Epigenetic modifications cannot be passed down from generation to generation
- B. The epigenome is an inflexible structure that never changes
- C. Epigenetic refers to changes in the DNA sequence itself
- D. Methylation of DNA makes it harder for it to be transcribed

**Answer:** D

**Explanation:** Methylation can prevent binding of transcription factors

**Question:** You are studying a gene and you notice that there are a high concentration of cytosines by a gene promoter. Will this gene be described?

- A. It is highly likely that this gene will be transcribed because cytosine has a -NH<sub>2</sub> group that provides a good 'hook' for transcription factors
- B. It is highly likely that this gene will be transcribed because cytosine can easily bind to promoters
- C. It is highly unlikely that this gene will be transcribed because there is a high probability that high concentration of cytosines will result in DNA methylation
- D. It is highly unlikely that this gene will be transcribed because 90% of cytosines are acetylated.

**Answer:** C

**Explanation:** a high concentration of Cytosines = CpG island= 70-80% of CpG islands are methylated

**Question:** You want a gene to be transcribed! Which of the following methods will allow for higher probability of gene transcription?

- A. Acetylation of histone proteins
- B. Methylation of histone proteins
- C. Phosphorylation of histone proteins
- D. Two of the above are correct
- E. All of the above are correct

**Answer:** E

**Explanation:** Don't get confused about methylation of histone proteins and methylation of cytosines. Methylation of histone proteins will increase steric hindrance and favor euchromatin. Acetylation and phosphorylation provide negative charges which repel negative DNA strands so they also favor euchromatin.

**Question:** Which of the following statements about histone proteins are false?

- A. Histone proteins are acidic, so they repel DNA since DNA is negatively charged
- B. There is a higher likelihood of transcription if there is nucleosomes are on top of a promoter
- C. Heterochromatin is formed by acetylation of histone proteins
- D. All of the above are false

**Answer:** D

**Question:** There is a mutation in the KAT protein, which facilitates acetylation of histone proteins. Which of the following describe a consequence of this mutation?

- A. Heterochromatin will be favored
- B. Euchromatin will be favored
- C. There will be a lower attraction between DNA and histone proteins
- D. It will be easier to transcribe the gene

**Answer:** A

**Explanation:** The KAT protein will acetylate histones which favor euchromatin (and decrease attraction between DNA and histone proteins and make it easier to transcribe). Thus, a mutation will favor heterochromatin.

## Lecture 32

**Question:** The agouti gene gives a mouse a yellow pigment instead of a black/ brown pigment. Which of the following about the agouti gene is true?

- A. The black spots correspond to a methylated IAP promoter
- B. The yellow spots correspond to a methylated IAP promoter
- C. Black and yellow spots are independent of methylation and are a result of mutation
- D. Black and yellow spots are independent of external factors, such as feeding

**Answer:** A

**Explanation:** If the promoter is methylated, the agouti gene will not be transcribed.

**Question:** Which of the following are TRUE about epigenetics?

- A. Epigenetics are caused by DNA mutations
- B. What a mother eats during pregnancy cannot affect the baby's epigenome
- C. The epigenome can affect behavior
- D. Since it is so variable, epigenetics is not a good target for drug therapy

**Answer:** C

**Explanation:** an example is the good mom/ bad mom experiment in lecture.

**Question:** Which of the following is FALSE regarding the Licking-groom-arched-back (LG-ABN) experiment,

- A. Decreased methylation resulted in 'better moms', i.e. arched back nursing
- B. Decreased glucocorticoid receptor resulted in 'bad moms', i.e. regular nursing
- C. The genes responsible for glucocorticoid receptor is completely independent of external stimuli
- D. Methylation of the CpG islands near the Nr3c1 gene results in decreased transcription of that gene

**Answer:** C

**Explanation:** this gene transcription can be stimulated by tactile stimulation.

**Question:** You treat a high LG-ABN ('good mother') with methionine for 7 days. Methionine increases cytosine methylation. This injection...

- A. Will prevent transcription of Nr3C1, a gene necessary to produce adequate amounts of glucocorticoid receptor

- B. Can be passed onto future offspring
- C. Can be reversed by demethylation
- D. All of the above are correct

**Answer:** D

**Question:** All of the following are examples of epigenetic changes EXCEPT

- A. DNA methylation
- B. Radiation that leads to a change in sequence
- C. Formation of heterochromatin/ euchromatin
- D. Histone acetylation

**Answer:** B

**Explanation:** Changes in sequences are NOT epigenetic change examples.

## Lecture 33

**Question:** Metformin is a drug used to treat Type II Diabetes. Which of the following ways does NOT describe a possible mechanism of action?

- A. Metformin increases insulin sensitivity by cells
- B. Metformin decreases hepatic glucose production
- C. Metformin increases glucose uptake by cells
- D. Metformin increases the glycogen breakdown into glucose

**Answer:** D

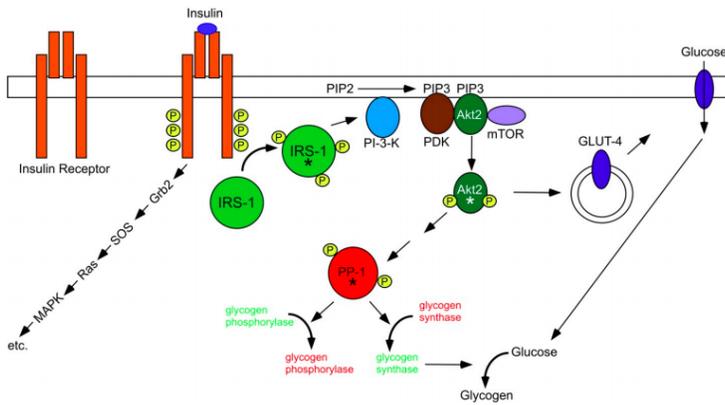
**Explanation:** One symptom of Type II Diabetes is increased glucose in blood and decreased glucose sensitivity. The drug treating this disease should not increase the amount of insulin.

**Question:** Which of the following treatments will be effective in treating Type I Diabetes but not Type II Diabetes?

- A. An antibody that prevents the killing of beta cells in the pancreas
- B. Diet modifications
- C. Exercise
- D. A drug that improves insulin sensitivity

**Answer:** A

**Explanation:** Choice D is for Type II Diabetes. For Type II Diabetes, it is mostly insulin resistance that causes hyperglycemia.



IRS-1: Insulin Receptor Substrate-1

PP-1: Protein Phosphatase-1

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The diagram above shows the pathway to uptake insulin in the cell.

**Question:** There is a mutation in the AKT2 protein, which activates the protein to synthesize glycogen and helps transport the GLUT 4 to the membrane. Which of the following is NOT an effective method to alleviate the effects of this?

- A. Adding a helper protein that will help GLUT-4 get transported to the membrane
- B. Provide another protein that helps with the synthesis of glycogen
- C. Increasing insulin sensitivity
- D. Inserting glucose transporters in the membrane artificially

**Answer:** C

**Explanation:** A, B, and D are effects DOWNSTREAM of the mutation, which make it more effective to alleviate the effects. However, C is upstream and will not affect the mutation of the AKT2 protein.

**Question:** Which of the following statements are true about diabetes?

- A. Type I Diabetes mainly affects the older population
- B. There is no correlation between obesity and diabetes
- C. Type I Diabetes is usually associated with ketoacidosis, where the body makes ketones because it does not have enough sugar
- D. Type I Diabetes results in weight gain because the body must compensate for glucose loss

**Answer:** C

**Explanation:** Type I Diabetes is usually early onset and is associated with rapid weight loss.

**Question:** Which of the following statements are false about glucose uptake and breakdown?

- A. Glycogen can be broken down to increase glucose in the bloodstream
- B. Since glucose is a small molecule, it does not require any extra help from proteins to be transported into the cell
- C. Hormones play a role in the breakdown of glycogen
- D. Glucose uptake requires dimerization of a receptor tyrosine kinase

**Answer:** B

**Explanation:** Glucose requires a GLUT transporter

## Lecture 34

**Question:** Which of the following is correct about the gill-withdrawal reflex in aplysia?

- A. The gill-withdrawal reflex in response to a weak shock is an example of habituation because the response to the same stimulus gets smaller as the aplysia is stimulated
- B. The gill-withdrawal reflex in response to a weak shock is an example of desensitization because the response to the same stimulus gets smaller as the aplysia is stimulated
- C. The gill-withdrawal reflex in response to a weak shock is an example of homeostasis because the aplysia withdraws its gills to maintain a set point
- D. The gill-withdrawal reflex in response to a weak shock is an example of associative learning because two different stimuli are integrated together

**Answer:** A

**Explanation:** Desensitization is a stronger reflex with the same stimulus; gill-withdrawal reflex is non-associative learning.

**Question:** All of the following are ways that one can modify brain connections EXCEPT

- A. Changing the strength of individual connections (i.e. increasing strength of synapses)
- B. Changing the pattern of connections (ie making synaptic contacts between pre-existing neurons)
- C. Changing amount of neurons by synthesizing new ones
- D. Changing the operations performed at each part of the network

**Answer:** C

**Explanation:** Straight from the slides! Neurogenesis is very rare.

**Question:** You are studying in the library and someone brings out a huge bag of baby carrots and hummus. Every few seconds, you hear a \*CRUNCH\*. This crunching noise becomes more and more annoying as time goes on. What is this an example of?

- A. Habituation
- B. Sensitization
- C. Associative learning
- D. All of the above

**Answer:** B

**Explanation:** Increased stimulus, strengthened reflex

**Question:** You walk inside a classroom and hear that the air conditioning is making a humming noise. During lecture, however, you are so immersed in the material and forget about the humming noise. This is an example of

- A. Habituation
- B. Sensitization
- C. Associative learning
- D. All of the above

**Answer:** A

**Explanation:** Same stimulus, decreased reflex

**Question:** Which of the following is FALSE about the sensitization circuit?

- A. The circuit involves serotonin release in aplysia
- B. The circuit results in increased response to the same stimuli
- C. The circuit results in a weaker synapse
- D. The circuit involves a recorded change in the motor neuron rather than the sensory neuron

**Answer:** C

**Explanation:** The sensitization circuit involves serotonin release by an interneuron, which acts on the axon terminal of the sensory neuron (thus not affecting a recorded change in the sensory neuron, but rather, the motor neuron).

It does NOT result in a weaker synapse because it is an increased response to the same stimuli.

## Lecture 35/ 36

**Question:** Which of the following are TRUE about the costs and benefits of escape behavior?

- A. A higher threshold makes it easier to escape
- B. A higher intensity makes the animal less likely to succeed to escape
- C. A lower threshold makes the animal more likely to succeed to escape
- D. A higher threshold makes the animal have more 'false alarms.'

**Answer:** C

**Explanation:**

LOW INTENSITY:

- Less likely to succeed
- Lower cost to execute

HIGH INTENSITY

- Very likely to succeed
- Higher cost to execute

LOW THRESHOLD for initiative escape behavior

- Very likely to succeed
- High cost for false alarms

HIGH THRESHOLD for initiative escape behavior

- Less likely to succeed
- Lower cost for false alarms

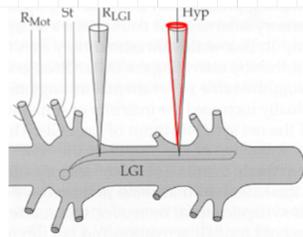
**Question:** In the crayfish experiment, what is the point of hyperpolarizing the neuron?

- A. We are studying the necessity of the LGI Neuron, so we need to activate it by hyperpolarizing it to see if it is necessary to elicit a response
- B. We are studying the necessity of the LGI Neuron, so we need to INactivate it by hyperpolarizing it to elicit a response
- C. We are studying the sufficiency of the LGI Neuron, so we need to INactivate it by hyperpolarizing it to elicit a response
- D. We are studying the sufficiency of the LGI Neuron, so we need to INactivate it by hyperpolarizing it to elicit a response

**Answer:** B

**Explanation:** See table below for a summary of experiments.

Experiment 1: SUFFICIENCY of LG neuron	Experiment 2: NECESSITY OF LG Neuron	cannot just perform an ablation experiment because fibers in passing problem
Results: stimulate LG neuron gets the same behavior	Put in 4 electrodes (3 in St, Rmot, RLgi), and one to hyperpolarize	
Conclusion: LG Is sufficient!	Hyperpolarize the cells in the LGI to inactivate it	
	Results: No action potential!	
	Conclusion: LG Is necessary	



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