Wiegle Exam 3 Biol 220

Biology 220: Exam #3
(Animal Physiology 1)
November 22, 2004

Total Points (100) 79

CALCULATORS ALLOWED

Read the questions carefully.

Most questions require only a brief and concise answer. **DO NOT** write on back of the exam.

At the end of the exam check to see that you have answered every question

If you have a question during the test, raise your hand for assistance.

Helpful facts: $\log_{10} 100 = 2$
$\log_{10} 10 = 1$
$\log_{10} 1 = 0$
$\log_{10} 0.1 = -1$
$\log_{10} 0.01 = -2$

[redacted] 25 [redacted]

1. a) Unlike the caterpillar of manduca, an earthworm is very vulnerable to dehydration. What is the major structural adaptation that accounts for the difference in sensitivity to dehydration of these two species (6 points)?

The reason why the two are different is because the manduca has a waxy cuticle that prevents water loss through the epidermis whereas the earthworm has a very permeable epidermis which water can diffuse or evaporate through easily.

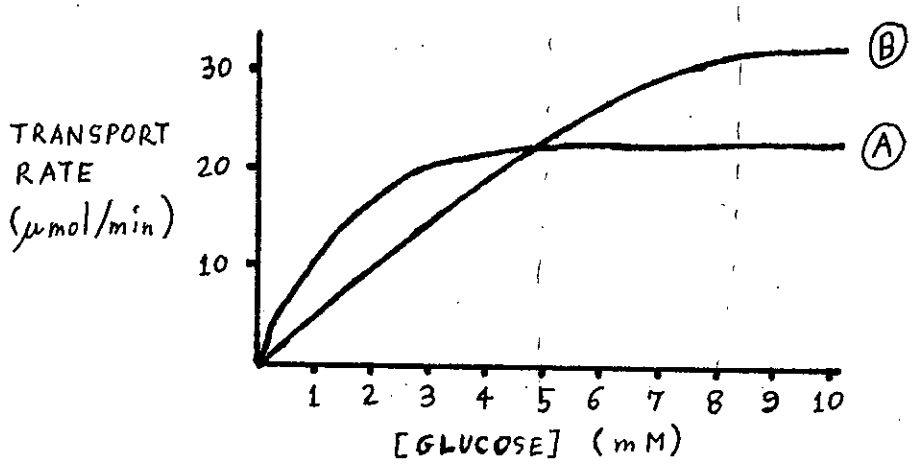
6

b) Why couldn't the earthworm adopt the same strategy as manduca to prevent dehydration (6 points)?

The reason why the earthworm could not adopt a cuticle to prevent dehydration is because the worm needs to get all of its air through that same permeable epidermis since the earthworm does not have lungs or a spiracle / tracheal system like the manduca.

6

2. Beta cells in the Islets of Langerhans of the pancreas secrete insulin at an increased rate after a meal to prevent an excessive rise in the blood glucose concentration. To do this job properly, beta cells must sense changes in the rate at which glucose is transported across their cell membrane. Assuming that the blood glucose concentration increases from about 5 to 8 mM after a meal, which of the following two glucose transporters would you expect to find in the membrane of beta cells, and why (15 points)?



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The membrane of the beta cells would most likely contain the (B) transporters since the cells have to secrete insulin and thus the cells are dependent on changes in glucose concentration in the blood. The (B) transporter has a wide range so that if the person is fasting and the glucose concentration is low then the cell won't secrete an abundance of insulin. The (A) transporter can't change its transport between the concentration 5 - 8 mM range.

[redacted] AD

3. In the disease called congestive heart failure (CHF) the hydrostatic pressure in the system of veins returning blood from the body to the heart increases significantly. This increased-pressure is transmitted backward to the capillaries, raising the hydrostatic pressure at the outflow end of the capillaries by 7-10 mm Hg. Explain how this situation can cause severe accumulation of edema and ascites in a patient with CHF. Assume that the patient's liver function remains normal (15 points).

15 good

If the patients liver functions normally then there is a correct amount of albumin within the plasma that brings in water at a constant pressure of 22mm Hg by osmosis. Normally the hydrostatic pressure is about 32 mm Hg outward near the heart and 15 mm Hg further down, but this disease increases the pressure at the out flow end by 7-10 mm Hg raising the outflow to a range of 22 -25 mm Hg. This means that all of the fluid pumped out cannot be taken back in since the osmotic pressure created by albumin is not strong enough. All the excess fluid left will be carried to lymphatic ducts creating a severe accumulation of edema and ascites.

4. a) From time to time in nature a species of animal arises in which an endocrine system is permanently activated by a "gain of function" mutation in a hormone receptor (i.e. the receptor is always transmitting the hormone's signal even in the absence of the hormone). Why are there no species of butterfly in which there is a gain of function mutation in the receptor for juvenile hormone (7 points)?

4 for this JH receptor

The reason why there is no species of butterfly to have a gain of function/mutation is because if the fact that if a catepillar detects any amount of JH (real or artificial) the insect will not molt into a pupa and begin metamorphosis to become a butterfly. The catepillar can't have any JH in order to become a butterfly so the reason why there are no gain of mutations in butterflies for this JH mutation is because if there were a gain of mutation in the juvenile insect it would never be able to become a butterfly. adult ⇒ reproductive

b) Assume you create a gain of function mutation of the juvenile hormone receptor by exposing butterfly eggs to X-rays. How will the ultimate size reached by caterpillars with the mutation compare with the ultimate size reached by caterpillars without the mutation (explain your reasoning)? Assume both caterpillars are well fed (7 points).

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The catepillars with the mutation will always be much ~~smaller~~ than the normal catepillars because the JH receptor in the mutated insect will never cease thus the ecdysone - JH interaction will always result in keeping the catepillar in juvenile form. Whereas the normal insect will have a decreasing amount of JH signals throughout its life and w - the ecdysone - JH interaction will allow the insect the molt into larger and larger instars until it finally becomes a pupa / since the the JH signal has decreased to nothing.

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[redacted] AD

5. a) TSH is a polypeptide hormone that stimulates thyroid cells to secrete thyroxine by means of the same second messenger pathway that epinephrine uses to stimulate liver cells to release glucose from glycogen into the blood. Why is epinephrine unable to stimulate the thyroid gland to secrete thyroxine (5 points)? The reason why epinephrine is unable to stimulate the secretion of thyroxine is because the cascade of reactions that occur in liver cells when stimulated by epinephrine will not occur in the thyroid when epinephrine tries to stimulate a TSH receptor. Just because both epinephrine and TSH trigger amplification events doesn't mean that every reaction and/or step in the amplification processes is identical thus epinephrine won't produce T_4 like TSH. No receptor

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b) Isobutylmethylxanthine (IBMX) is a drug that raises intracellular cyclic AMP (cAMP) levels by inhibiting the enzyme that normally breaks down cAMP. If you gave an IBMX infusion to a person, what would happen to the blood thyroxine concentration (before any compensatory changes have had a chance to occur), and why (5 points)? The cAMP molecule acts like a second messenger in the T_4 production process so when TSH stimulates the thyroid cAMP is produced to then go on and produce T_4 in an amplification like manner. So if IBMX raises cAMP levels and is given to a person then you would see much more T_4 being produced by the same amount of TSH that initially stimulated the thyroid.

5

c) Based on your answer to part (b), what would the IBMX infusion eventually do to the person's blood TSH concentration, and why (5 points)?

It would eventually lower the TSH concentration significantly since TSH production is under negative feedback control. Thus the high levels of T_4 would signal the anterior pituitary to slow down TSH production.

5

6. a) What effect do you predict tetrodotoxin, a blocker of voltage-gated sodium channels, would have on the resting potential of a human neuron, and why (6 points)? Not involved in RMP

It would keep the human neurons in their resting potential at all times even when stimulated by a signal because the neuron could not be able to have an action potential since the Na+ gated channel was blocked and since an AP needs an influx of Ap to drive the process.

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b) What effect do you predict a drug that blocks 90% of the activity of voltage-gated potassium channels would have on the duration of an action potential in a human neuron, and why (6 points)?

A human neuron that had this drug used on it would only only 10% of the voltage gated K^+ channels to work so the repolarization during an action potential would take much longer since the K^+ can only flow out of 10% of its K^+ channels. Thus the duration of the entire Action potential would be much longer than normal.

6

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[redacted] AD

7. The neurons of a particular slug have the following distribution of ions across their plasma membranes:

	Concentration inside	Concentration outside
K^+	150 meq/l	15 meql/
Na^+	1.2 meq/l	120 meq/l

In the resting state, the conductance of the membrane for potassium ions is 90 mho, and the conductance for sodium is 10 mho.

What is the resting membrane potential of the cell (show your work and include sign and units, 10 points)?

$E_{K+} = -\frac{60}{1} \log\left[\frac{150}{15}\right] = -60\,mV$ ✓ $E_{Na^+} = -\frac{60}{1} \log\left[\frac{1.2}{120}\right] = 120\,mV$ ✓

$E_m = \frac{90}{100}(-60\,mV) + \frac{10}{100}(120\,mV) = -42\,mV$ ✓

10/10

During an action potential the sodium conductance transiently increases to a maximum of 90 mho, and the potassium conductance is unchanged. What is the membrane potential at the peak of the action potential (show your work and include sign and units, 7 points)?

$E_{K+} = -60\,mV$ $E_{Na^+} = 120\,mV$ but now at peak of AP Na conductance is 90 mho

$E_m = \frac{90}{180}(-60) + \frac{90}{180}(120\,mV) = +30\,mV$ ✓

7/7

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