

NAME: _____

DIRECTIONS: Please make sure that you have all 8 pages of this exam. There are **SEVEN** questions. Answer all parts of all questions. Read through the entire question before you answer. Note that each question indicates an approximate time you should allot to it--do not overextend your welcome at any particular question! The point values of each question are also noted.

Note that questions 5-7 will take the majority of your time, so plan accordingly.

PLEASE WRITE CLEARLY.

***** GOOD LUCK *****

1. (5 points, 3 minutes) Reversible phosphorylation is a control mechanism used throughout metabolism. What are the general names of the enzymes involved in reversible phosphorylation and what general reactions do they catalyze?

kinases catalyze the addition of a phosphate group to a target

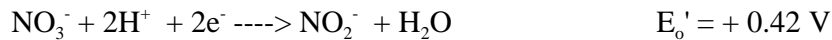
phosphatases catalyze the removal of a phosphate group from a target

2. (7 points, 5 minutes) Throughout our discussion of metabolism we have discussed coupled reactions. Why is this such an important phenomenon?

Many of the reactions in metabolism are thermodynamically unfavorable, that is, they have positive ΔG s. By coupling these reactions to thermodynamically favorable ones (with negative ΔG s) the overall reaction can become favorable because the ΔG s are additive.

3. (8 points, 7 minutes) Nitrite ion (NO_2^-) is toxic to many microorganisms and is therefore often used as a preservative in processed foods. However, members of the genus *Nitrobacter* oxidize nitrite to nitrate (NO_3^-), using the energy released upon the transfer of oxygen to drive ATP synthesis. (That is, with the exception of nitrite replacing glucose as the source of electrons, these microorganisms synthesize ATP in the usual manner.)

Given the E_o' values below calculate the maximum yield of ATP under standard conditions that can be obtained per mole of nitrite ion oxidized. Your answer need not be an integer. Show all your work.



Recall: $G^{o'} = -nF E_o'$ where $F = 23.06 \text{ kcal}/[(\text{mol})(\text{V})]$.
 $G_{\text{ATP synthesis}}^{o'} = + 7.3 \text{ kcal/mol}$

For the net reaction:



we must reverse the direction of the top reaction above which causes a change in the sign of the standard potential:

$$E_o' = - 0.42 \text{ V}$$

Adding this to the $E_o' = + 0.82 \text{ V}$ for the bottom reaction, we get a $E_o' = +0.4\text{V}$

Using the equation $G^{o'} = -nF E_o'$, and knowing that $n=2$ because there are 2 electrons involved in this redox couple, we then get $G^{o'} = -18.448 \text{ kcal/mol}$.

We can see by the POSITIVE E_o' and the NEGATIVE $G^{o'}$ that this is a favorable reaction.

Since the $G_{\text{ATP synthesis}}^{o'} = + 7.3 \text{ kcal/mol}$ we can obtain

$18.448/7.3 = 2.5 \text{ ATP per mole of nitrite ion oxidized.}$

4. (10 points, 10 minutes) Suppose you discover a mutant yeast whose glycolytic pathway is shorter because of the presence of a new enzyme that catalyzes the reaction



Although this mutant enzyme shortens glycolysis by one step, how does it affect anaerobic ATP production? Aerobic ATP production?

The mutant enzyme allows the phosphoglycerate kinase step to be bypassed because glyceraldehyde 3-phosphate is oxidized directly to 3-phosphoglycerate without the formation of 1,3-bisphosphoglycerate. Hence, the mutant organism eliminates one of the two ATP-producing steps in glycolysis.

Since two ATP molecules are needed in the preparatory phase of glycolysis (steps 1 and 3), no net ATP is produced by the mutant organism under anaerobic conditions. If the mutant organism operates aerobically, however, the effect of the missing ATP-producing step is minimal because the major portion of the energy in glucose is captured in subsequent steps during its complete oxidation to CO_2 and H_2O through the citric acid cycle and oxidative phosphorylation. Thus, if all else were the same, the mutant would generate approximately 28 ATP under aerobic conditions instead of the 30 ATP for a wild-type yeast.

5. (25 points, 13 minutes) Compare and contrast the characteristics and functions of coenzyme Q (ubiquinone) and cytochrome c. Feel free to use diagrams, although explanations in text should accompany them. There is more space on the next page if you need it.

Here are the main points I'm looking for. Each point touched upon is worth 2 points (notice that they add up to more than 25 points, thus not all need to be mentioned, but some are more important than others and I'm specifically looking for those, 3 points are for overall ideas and clarity):

both are mobile electron carriers

Q is inside the membrane

cyt c is a peripheral membrane protein

Q carries 2 electrons

Q gets these electrons from FADH₂ and NADH

cyt c carries 1 electron

Q cycle allows electrons to go from Q to cyt c

Q accepts electrons from complex I and II and carries them to complex III

cyt c accepts electrons from complex III and carries them to complex IV

cyt c has a heme prosthetic group

this is all in the mitochondria inner membrane

this is all part of the electron transport chain

electrostatic interaction of cyt c with targets

6. (15 points, 12 minutes) Since both glycolysis and gluconeogenesis are irreversible, there is no thermodynamic barrier to their simultaneous operation.

a. We know that gluconeogenesis is not simply a reversal of glycolysis. Why is this so? In your explanation be sure to mention at least two of the important steps and the enzymes involved.

Gluconeogenesis and glycolysis are not simply reversals of each other. First, there are three main steps in glycolysis that are essentially irreversible. These are the conversion of glucose to glucose 6-phosphate catalyzed by hexokinase, the conversion of fructose 6-phosphate to fructose 1,6-bisphosphate by phosphofructokinase, and the conversion of phosphoenolpyruvate to pyruvate by pyruvate kinase. Because these steps are irreversible, gluconeogenesis uses different reactions to bypass these steps in its production of glucose from precursors such as pyruvate or lactate. For example, glucose 6-phosphatase catalyzes the removal of the phosphate from glucose 6-phosphate and fructose 1,6-bisphosphatase catalyzes the removal of a phosphate from fructose 1,6-bisphosphate.

b. What would the result be if both pathways were operating simultaneously and at the same rate?

Gluconeogenesis uses 6 high energy phosphate bonds (4 ATP and 2 GTP) while glycolysis produces only 2 ATP. Thus, if both pathways were operating simultaneously and at the same rate, we would have a net loss of 4 high energy phosphate bonds.

c. What is the general term that describes how the situation described in part b is avoided?

reciprocal regulation

7. (30 points, 20 minutes) While working in the lab your experiment takes an unexpected turn leading to a very loud BOOM, thus causing you to have a major "flight or fight" response. As a result, epinephrine is released and travels to your muscles while glucagon travels to your liver. Explain the effects of these two hormones on glycogen metabolism in the two tissues. Be sure to mention the end products and the advantage to having these two specific routes in addition to discussing the mechanism of the receptors, pathways and enzymes involved. What is going on locally and globally? Feel free to use diagrams, although explanations in text should accompany them. There is more space on the next page if you need it.

Here are the main points I'm looking for synthesized into a clear discussion:

both hormones trigger cAMP cascade

discussion of cAMP cascade

activation of PKA

phosphorylase a is activated

phosphorylase kinase is activated

glycogen synthase is deactivated

glycogen breakdown is turned on

glycogen synthesis is turned off

PP1 is inhibited

PP1 is inhibited by phosphorylation of G subunit

PP1 inhibition keeps phosphorylase kinase active

PP1 inhibition keeps phosphorylase a active

PP1 inhibition keeps synthase off

muscle - glycogen breakdown

muscle - formation of glucose 6-phosphate enters glycolysis makes ATP

liver - glycogen breakdown, formation of glucose

liver - releases glucose to blood

liver has glucose 6-phosphatase, muscles don't

PP1 inhibitor activated by phosphorylation

Use this space for question 7.

*****END OF EXAM*****

