BIOSCI 1820
FINAL EXAM
APR 2001

KEY

I. Recitation Section:

A. Describe one of the two ways that urea cycle disorders can be treated:

1. Diet (try to limit protein intake)
2. Drugs (phenylbutyric acid on benzoate -> α acid scavengers)

B. Describe one of the many molecular defects that can give rise to familial hypercholesterolemia:

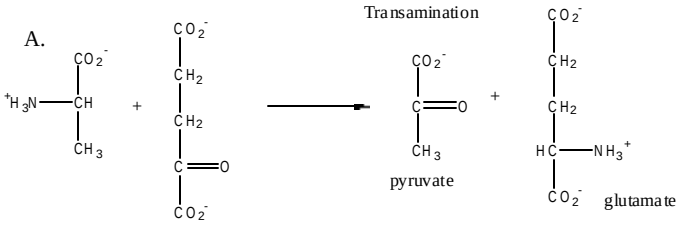
LDL^R mutants (5 classes):	-no expression
-ER -> Golgi	-cannot bind LDL
-Recycling (endosome)	-cannot discharge LDL in endosomes

C. In the classic experiment by Jagendorf and Uribe in which it was shown that a proton gradient drives the formation of ATP, how was the proton gradient established across the membranes of isolated chloroplasts?

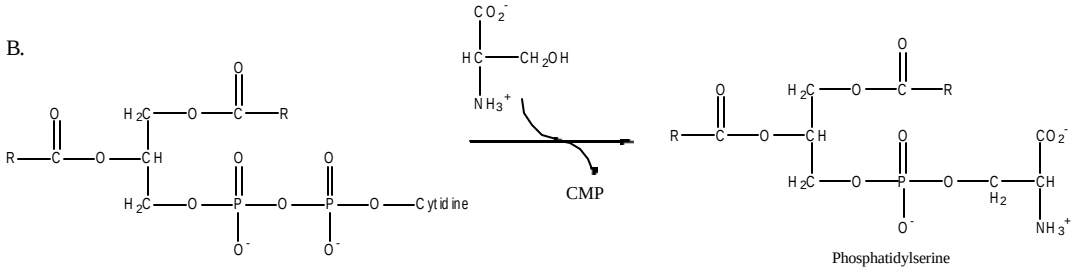
Chloroplasts were incubated in an acidic buffer & then rapidly diluted into neutral soln in order to establish a H^+ gradient.

II. Draw the structures of the products of the following reactions:

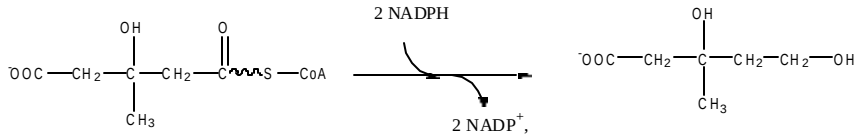
A.



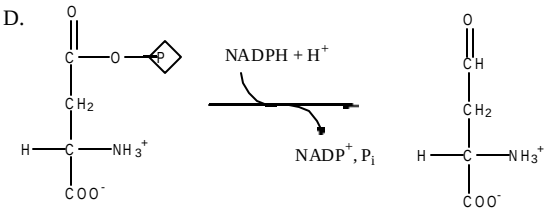
B.



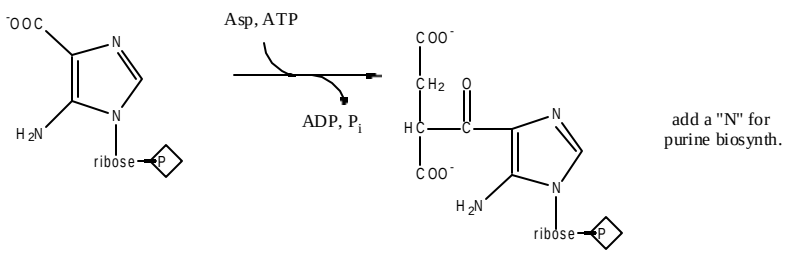
C.



D.



E.



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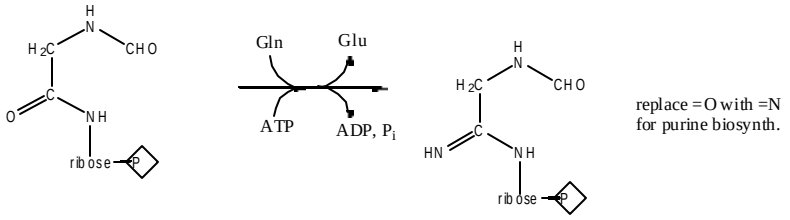
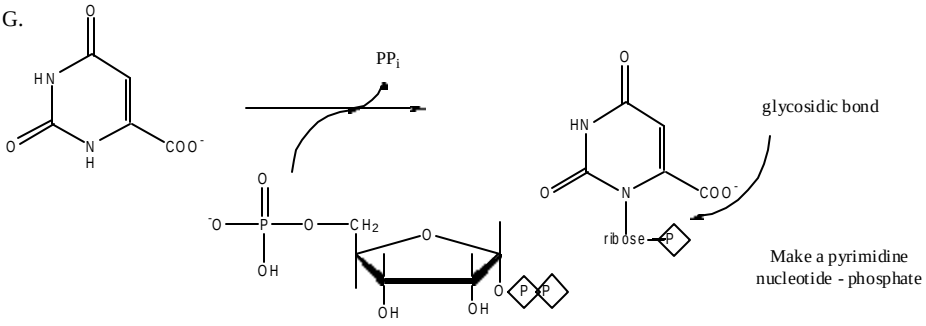
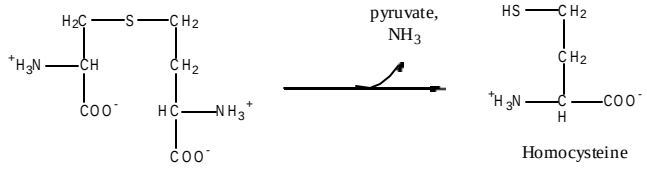


G.



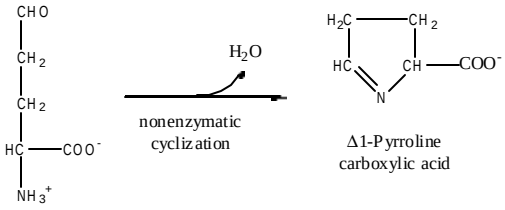
PRPP

H.



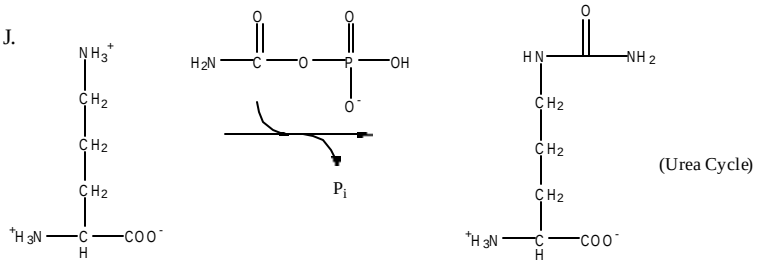
Homocysteine

I.



Δ1-Pyrroline
carboxylic acid

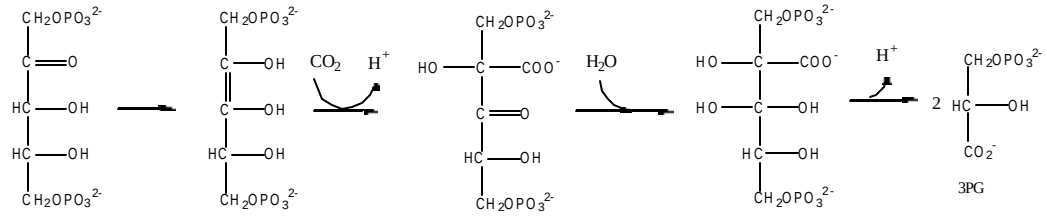
J.



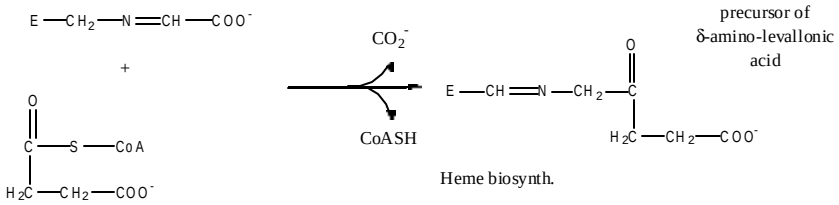
Citrulene

Rubisco

K.



L.



Heme biosynth.

III. Multiple Choice

Which of the following statements about photosynthesis is false?

A. Protons are pumped into the thylakoid lumen granal
B. Photosystem II is located in the ~~stromal~~ lamellae
C. Species such as sorghum and sugar cane are known as C-4 plants
D. Pheophytin is essentially the same as chlorophyll except that two H^+s are bound in place of Mg^{2+}
E. Rubisco is activated by increases in the Mg^{2+} concentration and pH

Which of the following metabolites is not utilized by the heart?

A. fatty acids
B. glucose
C. lactate
D. ketone bodies
E. All of the above are utilized by the heart

Lactose is a disaccharide composed of:

A. galactose-glucose
B. sucarose-glucose
C. glucose-mannose
D. fucose-galactose
E. glucose-glucose

Glucagon performs each of the following in liver except:

A. decreases glycogen synthesis
B. increases glucose release from liver cells
C. increases the activity of adenylate cyclase
D. ~~decreases~~ triacylglycerol hydrolysis
E. All of the above statements are true

Each of the following are intermediates in or products of ketone body metabolism except:

A. acetone
B. β-hydroxybutyrate
C. HMG-CoA
D. cholesterol
E. acetyl-CoA

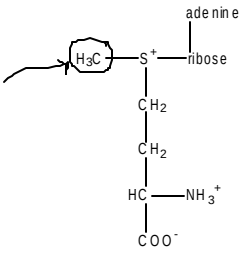
On a carbon-per-carbon basis, which of the following is the best energy source?

A. amino acids
B. glucose
C. saturated fatty acid most reduced
D. unsaturated fatty acid
E. glycogen

Tyrosine can be converted into each of the following exept:

A. thyroid hormone
B. epinephrine
C. DOPA
D. Dopamine
E. Serotonin ← Trp is precursor

The depicted molecule is used to catalyze which of the following processes:



AdoMet

A. Sulfur addition
B. Methyl group additions
C. Nitrogen donor
D. Carboxylate addition
E. None of the above

How many ATPs can be produced by the complete oxidation of the following molecule in the cell (assume that each FADH and NADH give rise to 2 and 3 ATPs respectively)?

$CH_2—CH_2—CH_2—CH_2—CH_2—COO^-$

2 rounds β oxidation: FADH x2 4
NADH x3 6
10

-2 to activate

3 AcCoA (TCA) x12 = 36

10 + 36 - 2 = 44

A. 46
B. 44
C. 29
D. 27
E. 51

The precursor for creatine is which amino acid?

A. tryptophan
B. asparagine
C. arginine
D. tyrosine
E. histidine

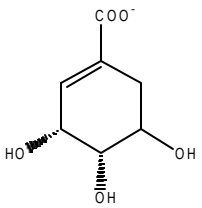
Which of the following statements about the *de novo* synthesis or degredation of pyrimidines is incorrect?

A. ATP activates their synthesis
B. CTP inhibits their synthesis
C. Carbamoyl phosphate is an intermediate in the pathway
D. The pyrimidine is assembled from the start directly onto a ribose-phosphate
E. The breakdown products of pyrimidines are generally not toxic since they are quite soluble

assembled as "free base"

The depicted moleucle is a precursor of which of the following?

A. cninamon
B. lignin
C. tryptophan
D. cayenne
E. All of the above



shikimate

IV. Regulation

A. It was recently reportat that -- besides activating adenylate cyclase -- the GTP-bound form of an α subunit of a G protein could bind to phosphodiesterase and inhibit its activity. Why does this make sense?

Inhibiting phosphodiesterase inhibits cAMP breakdown; adenylate cyclase increases cAMP levels, so the two effects are complementary.

B. A mutant form of cAMP-dependant protein kinase ("protein kinase A") that is always **active** is expressed in muscle cells. What would be the effect on lipid and glycogen metabolism in the muscle cell? Why?

If the kinase were always on, phosphorylase kinase would always be active (as would phosphorylase) & the hormone sensitive lipase would always be active; thus glycogen + lipids would be broken-down.

C. Anothe rtarget of Gα-GTP is phospholipase C. What are the two products of this enzyme and what are their respective effects in the cell? [worth two credits]

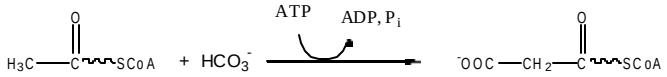
Phospholipase C cleaves PIP_2 -> DAG + IP_3
IP_3 liberates calcium from the ER.
DAG is an activator of protein kinase C.

V. The real world...

The following is an abstract that appeared in the March 30th issue of Science. Read the abstract and answer the following questions:

> Malonyl-coenzyme A (malonyl-CoA), generated by acetyl-CoA carboxylases ACC1 and ACC2, is a key metabolite in the regulation of energy homeostasis. Here, we show that $Acc2^{-/-}$ mutant mice have a normal life span, a higher fatty acid oxidation rate, and lower amounts of fat. In comparison to wild type, Acc2-deficient mice had 10- and 30-fold lower levels of malonyl-CoA in heart and muscle, respectively. The fatty acid oxidation rate in the soleus muscle of the $Acc2^{-/-}$ mice was 30% higher than that of wild-type mice and was not affected by addition of insulin; however, addition of insulin to the wild-type muscle reduced fatty acid oxidation by 45%. The mutant mice accumulated 50% less fat in their adipose tissue than did wild-type mice. These results raise the possibility that pharmacological manipulation of ACC2 may lead to loss of body fat in the context of normal caloric intake.

A. In mice, there are two forms of acetyl-CoA carboxylase (*ACC1* and *ACC2*). Write the reaction (structures of the reactants and product) catalyzed by this enzyme: [2 credits]



B. Deletion of one of the forms of the enzyme gives rise to thin mice. Does this make sense? Why or why not?

If you are less able to make malonyl-CoA (which is THE building block during FA synthesis) you will be less able to make/store FAT.