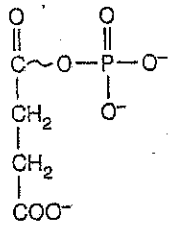
BIOSC 1820
Metabolic Pathways and Regulation
Spring, 2012
Prof. Jeffrey L. Brodsky
Quiz #3
February 22, 2012

NAME: KEY

1. The following molecule is an intermediate in the reaction catalyzed by which enzyme?

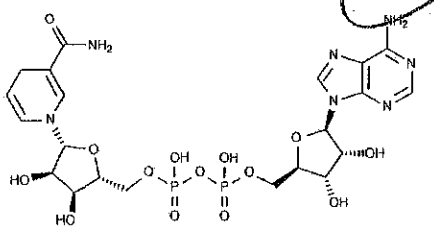


Succinyl CoA synthetase

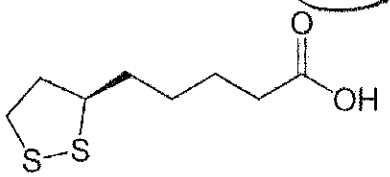
+1

2. Are the following depicted cofactors in the (A) oxidized or (B) reduced forms?

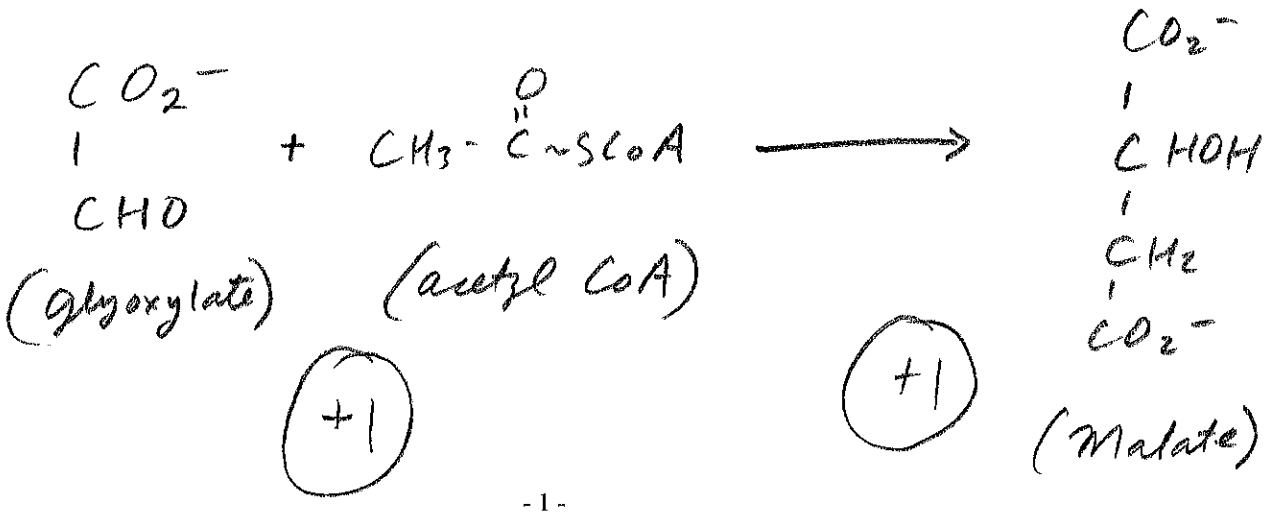
B +1



A +1



3. Draw the reaction—including the structures AND the names of the reactants and products—that is unique to the glyoxylate cycle and that is highly favorable (if there are co-factors used in this reaction, their name(s) can be abbreviated) (2 points):



4. Each of the following enzymes are activated by calcium *except*:

A. Pyruvate dehydrogenase
B. Isocitrate dehydrogenase
C. α-ketoglutarate dehydrogenase
D. Malate dehydrogenase
E. None of the above, each of these enzymes is activated by calcium

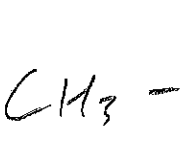
+1

5. Which of the following molecules is exported from the mitochondria and then serves as a precursor for the synthesis of hexoses and other sugars in plant cells?

A. Malate
B. Oxaloacetate
C. Fumarate
D. Acetyl CoA
E. None of the above

+1

6. In the absence of NAD, which metabolite (that was discussed in handout #5) do you think is most likely to accumulate in mitochondria? (Please draw the structure AND state the name)

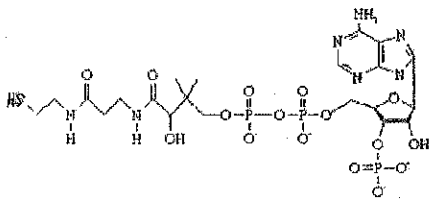


+½

Pyruvate would accumulate +½

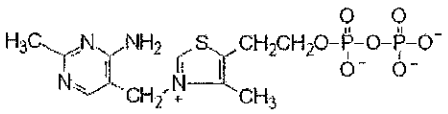
7. What are the FULL names of the following vitamins/cofactors

A.



Coenzyme A +1

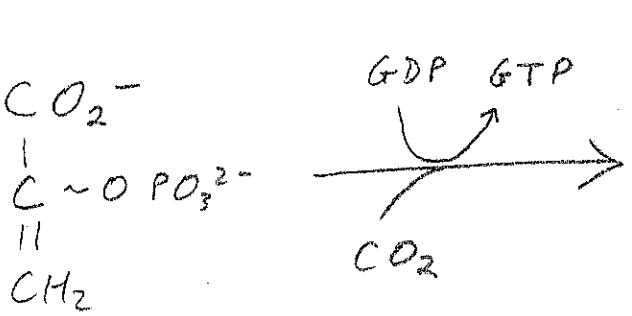
B.



thiamine pyrophosphate (+1)

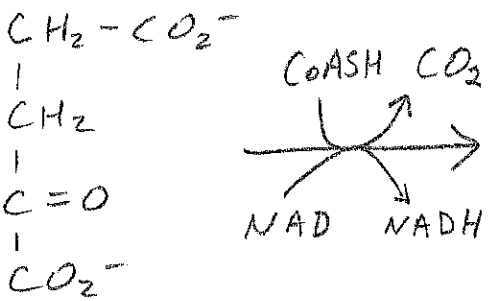
8. Draw the structures AND give the names of the products of the following reactions:

A.



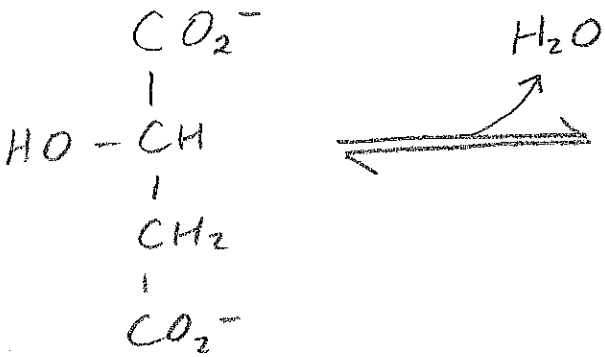
oxaloacetate (+1/2, +1/2)

B.



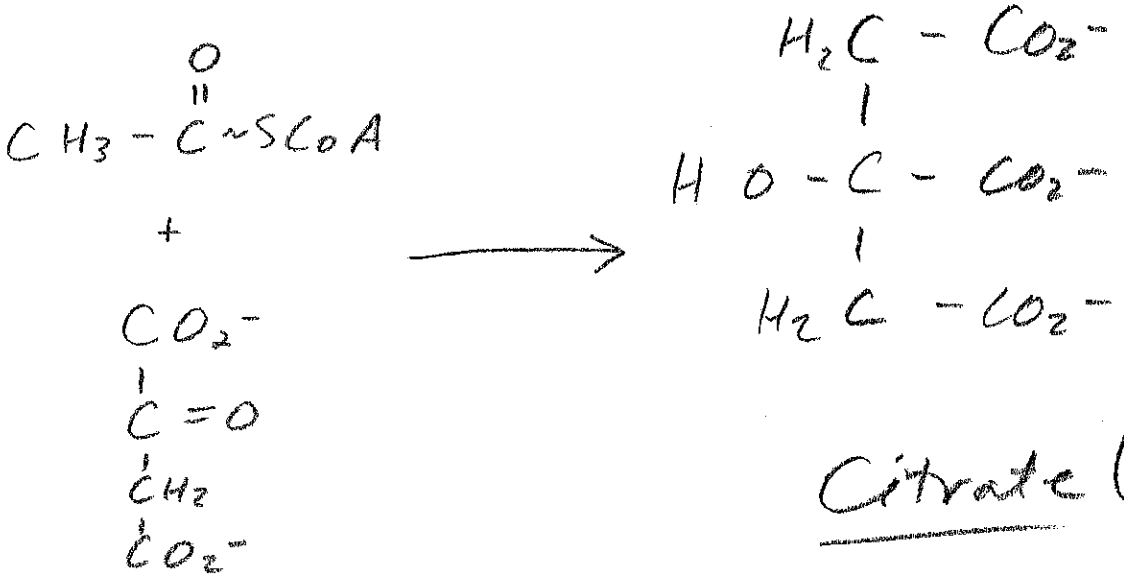
Succinyl-CoA (+1/2, +1/2)

C.



fumarate (+1/2, +1/2)

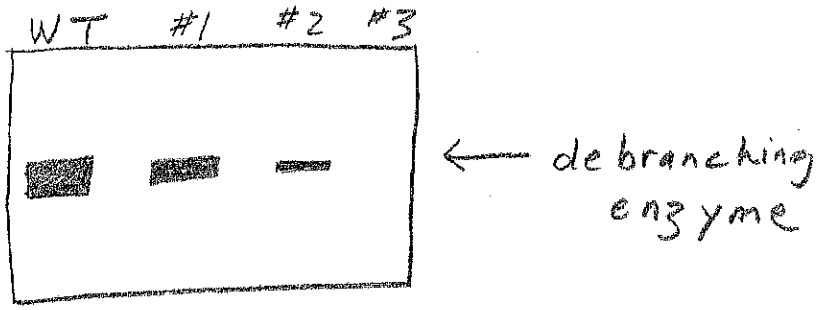
D.



+1/2

Citrate +1/2

9. Cori Disease is type IIIa Glycogen Storage Disease caused by a defect in the glycogen debranching enzyme. Two different types of mutations in the gene encoding this enzyme have been identified. You have recently analyzed the levels of this enzyme in a wild type (WT, i.e., healthy) individual and in three different patients by western blot analysis. The results are shown below:



What can you infer about the genotype of each of the three patients?

#1: heterozygote in which one allele is "null" (no protein produced) +1

#2: Compound heterozygote, in which two different mutant alleles are produced (one is probably null and one produces an unstable protein).

/15

#3: homozygote null (no protein produced)