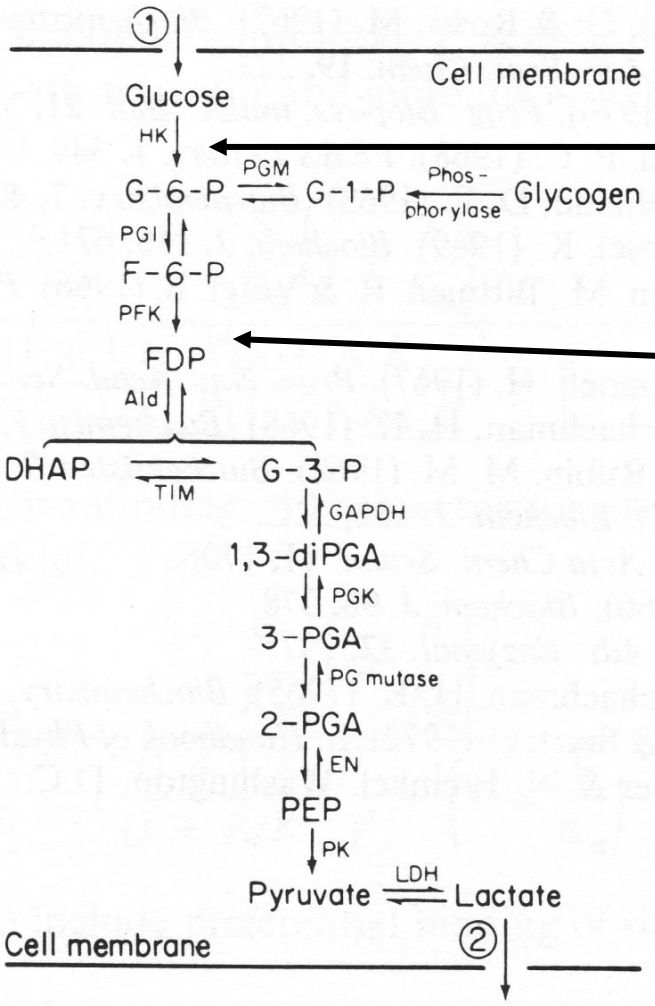
ANSC/NUTR 601
PHYSIOLOGICAL CHEMISTRY OF LIVESTOCK SPECIES
Carbohydrate Metabolism

I. Glycolysis

A. Pathway

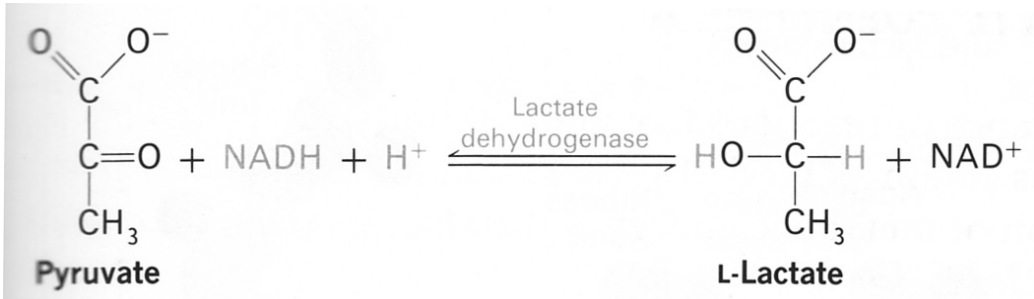


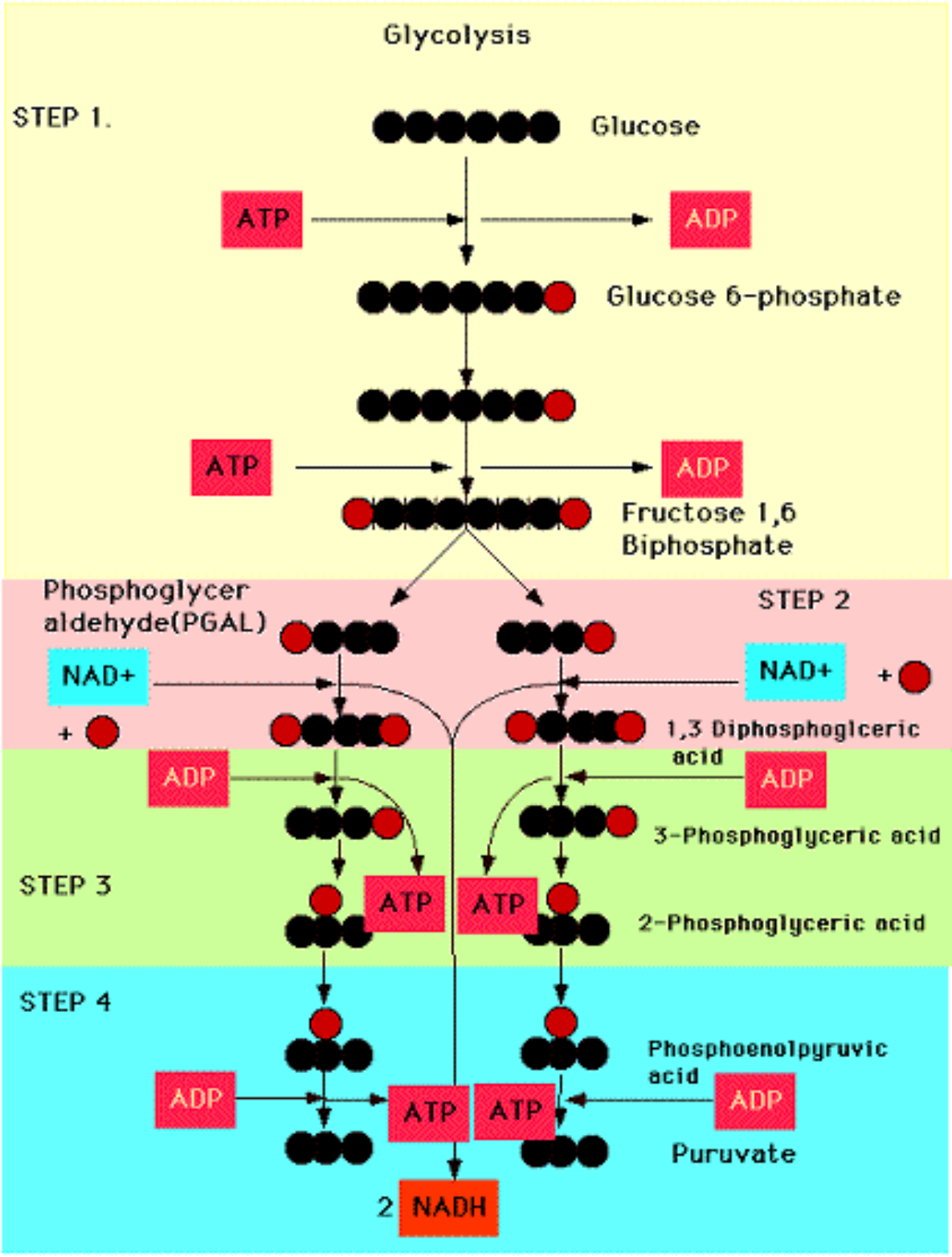
Regulation of glycolysis

Hexokinase:
Activated by glucose.
Inhibited by G6P.

6-Phosphofructokinase:
Inhibited by ATP, especially in the presence of citrate.
Activated by AMP.

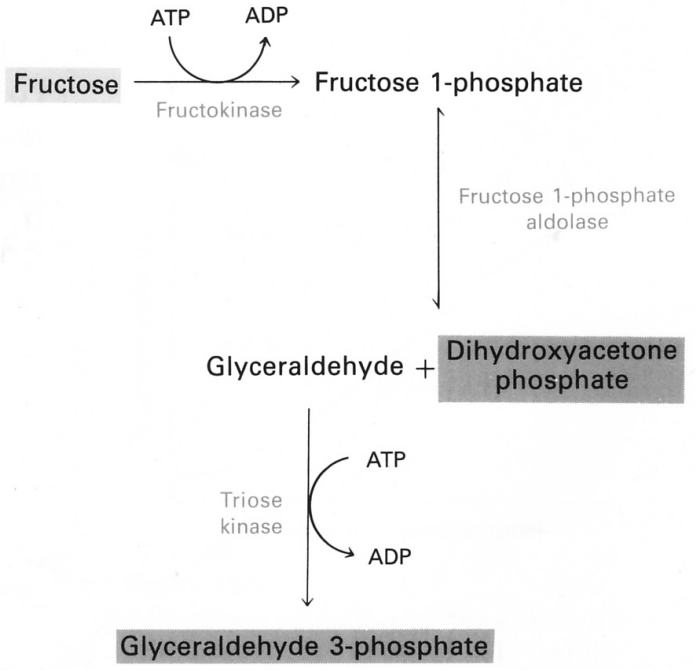
B. Pyruvate and lactate




Glycolysis
STEP 1.
Glucose
ATP
ADP
Glucose 6-phosphate
ATP
ADP
Fructose 1,6
Biphosphate
Phosphoglycer
aldehyde(PGAL)
STEP 2
NAD+
NAD+
+
+
1,3 Diphosphoglceric
acid
ADP
ADP
3-Phosphoglyceric acid
STEP 3
ATP
ATP
2-Phosphoglyceric acid
STEP 4
Phosphoenolpyruvic
acid
ADP
ATP
ATP
ADP
Puruvate
2 NADH

C. Entry of fructose into glycolysis

1. Fructose – Phosphorylated by fructokinase to fructose-1-phosphate, then split to DHAP and glyceraldehyde.



II. Krebs (tricarboxylic acid or citric acid) cycle

A. Conversion of pyruvate to acetyl CoA

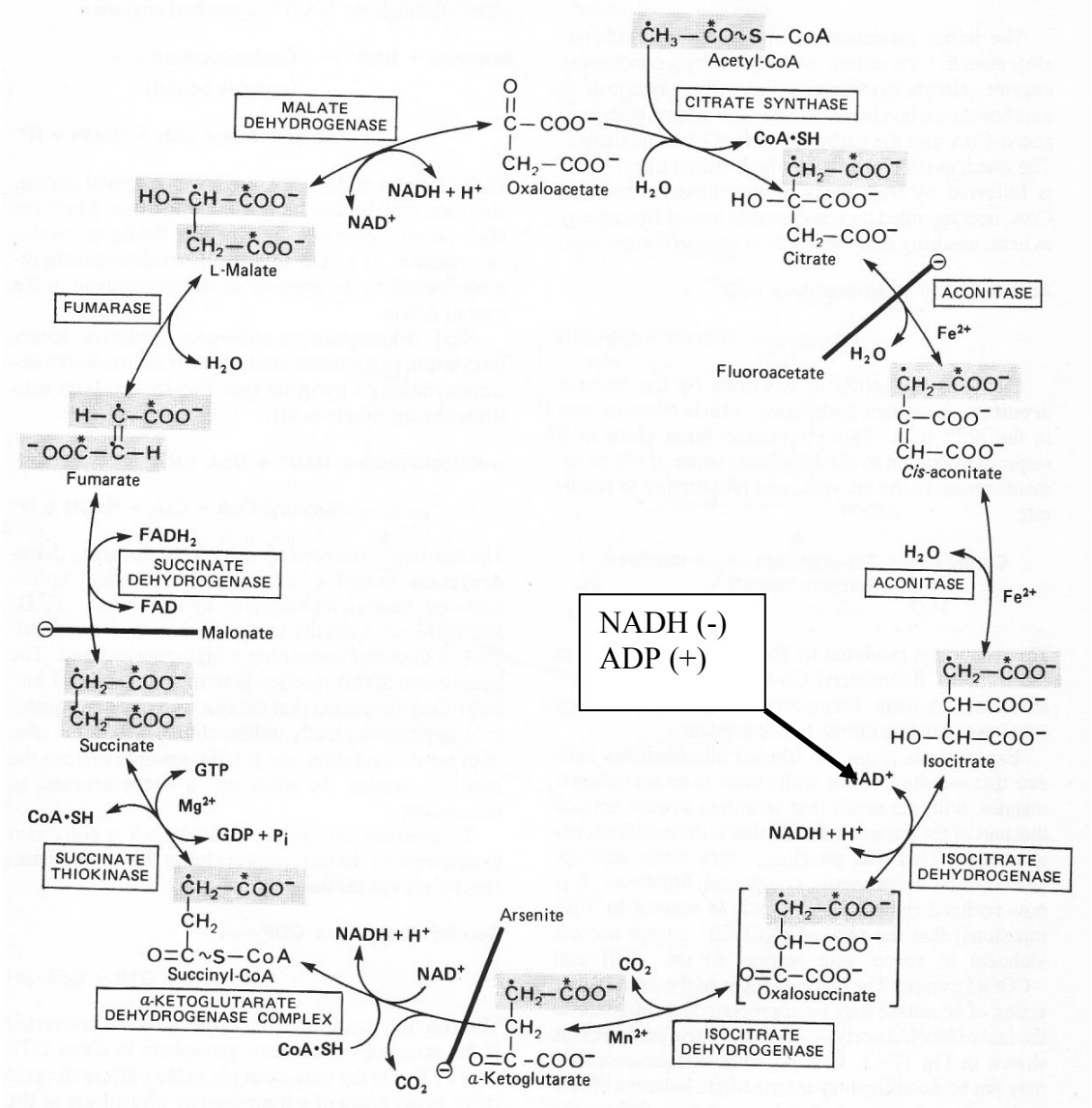
1. Pyruvate is decarboxylated by pyruvate dehydrogenase at the inner mitochondrial membrane.
2. Coenzyme A is attached by a thioester bond to acetate to form acetyl-CoA

B. Conversion of pyruvate to oxaloacetate

1. Pyruvate crosses the inner mitochondrial membrane.
2. Pyruvate is carboxylated to oxaloacetate in the mitochondrial matrix.

C. The cycle

1. Oxaloacetate condenses with acetyl-CoA to initiate the TCA cycle.
2. A net of two carbons is lost during one complete cycle.
3. Primary regulation is at isocitrate dehydrogenase.



III. Gluconeogenesis

A. Essential enzymes

1. Pyruvate carboxylase (converts pyruvate to oxaloacetate)
2. Phosphoenolpyruvate carboxykinase (PEPCK) (converts oxaloacetate to PEP)
3. Fructose 1,6-diphosphatase (converts fructose 1,6-diphosphate to F-6-P).
4. Glucose-6-phosphates (converts G-6-P to free glucose)

B. Overall pathway

Pyruvate → oxaloacetate → PEP → → Fructose 1,6-diphosphate → F-6-P → G-6-P → **Glucose**

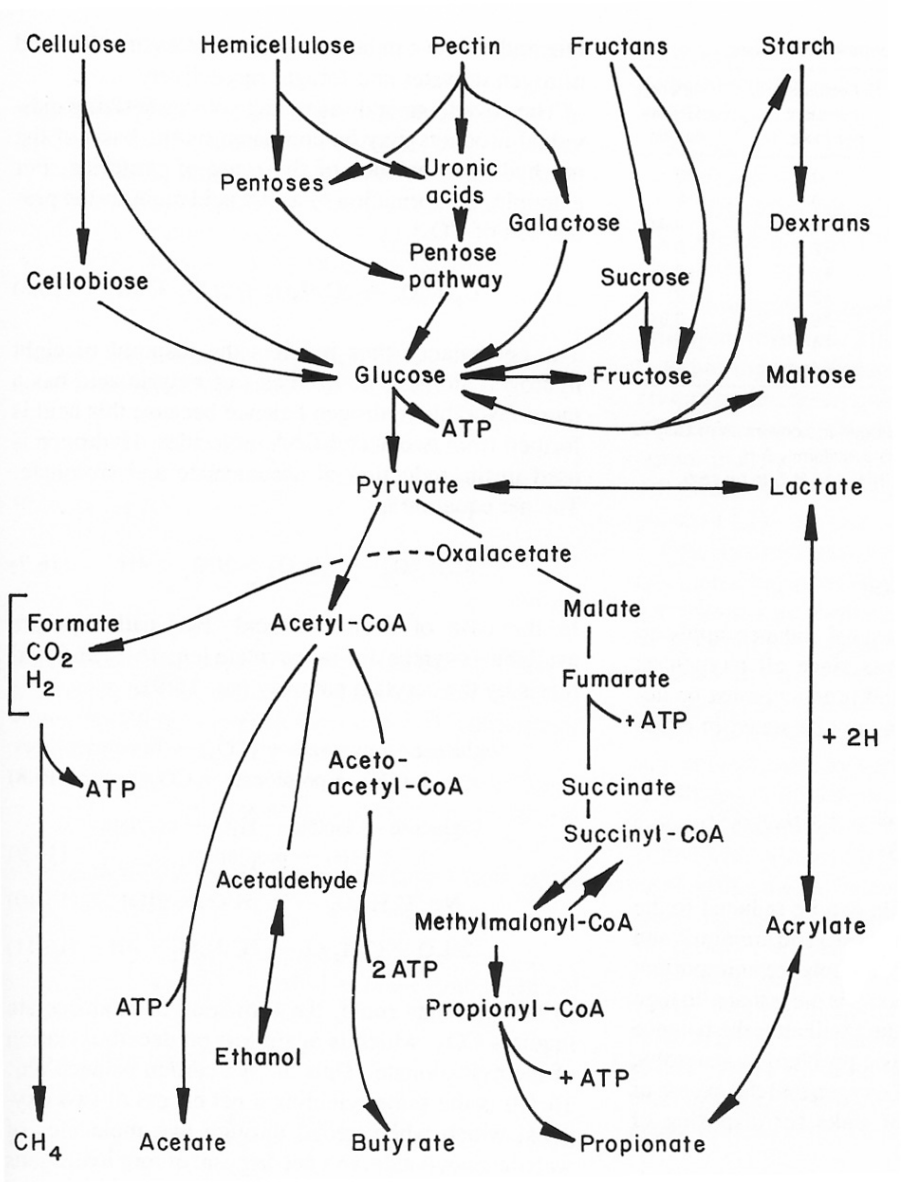
C. Organs responsible for gluconeogenesis

A. Liver – produces glucose for the rest of the body

B. Kidney cortex – produces glucose for its own use

IV. Fermentation

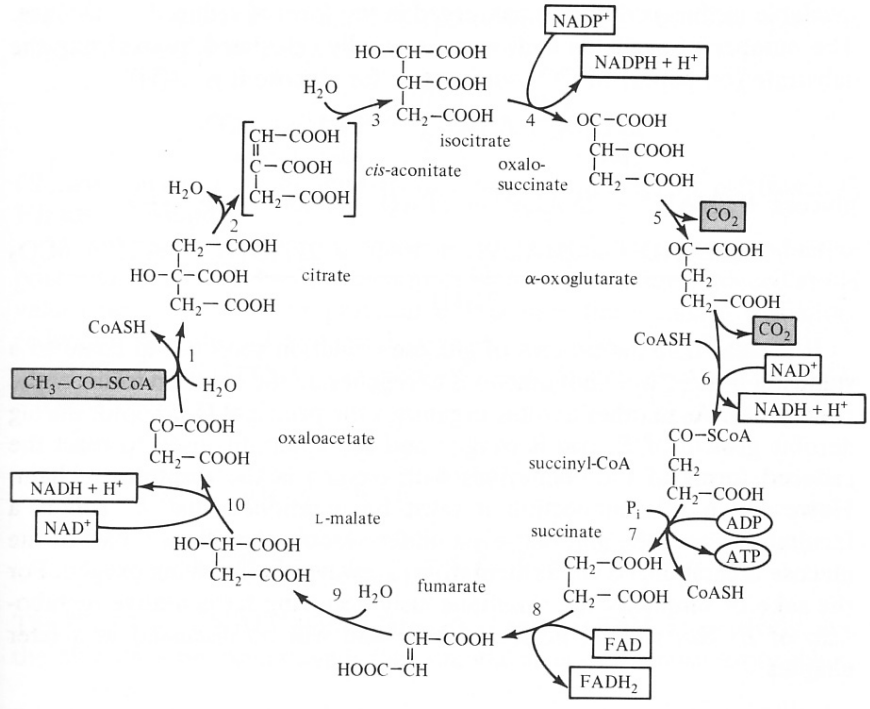
A. Products of fermentation



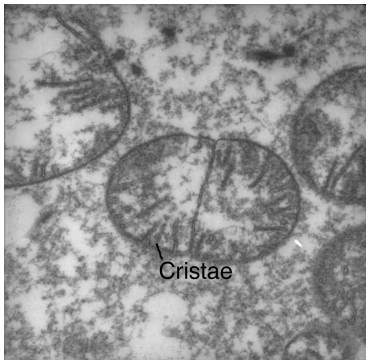
All carbohydrates funnel into pyruvate. Six-carbon intermediates are converted to pyruvate, which then is used to make AcCoA or oxaloacetate.

All VFA are produced from pyruvate. VFA production provides ATP for bacteria.

B. TCA cycle in bacteria

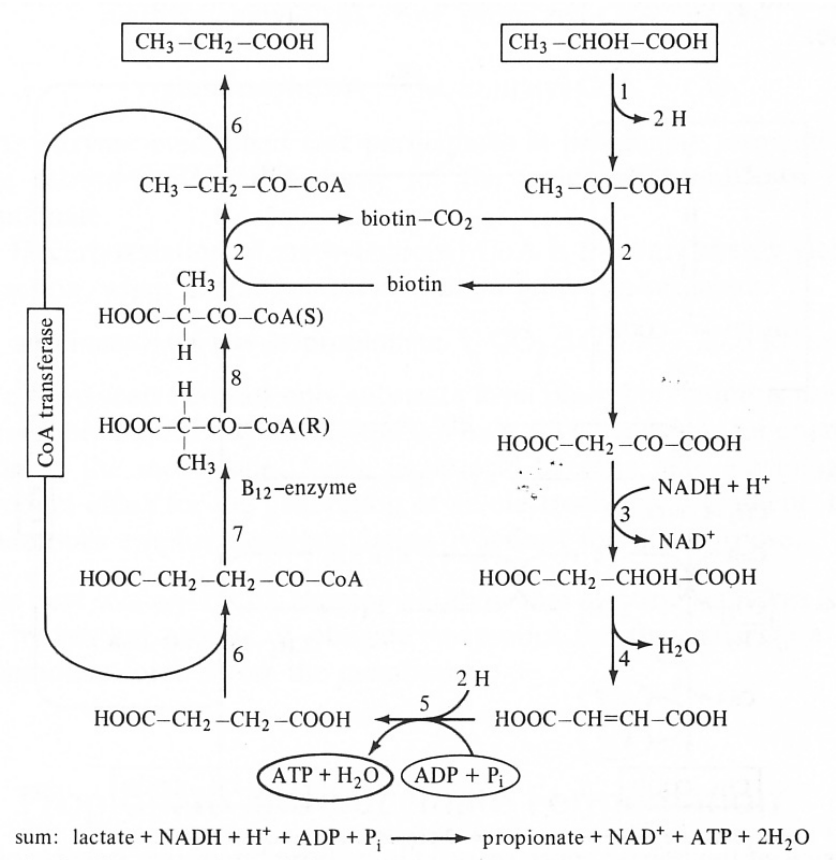


The TCA cycle is identical in bacteria and mitochondria of eukaryotes.



Eukaryotic mitochondrion

C. Propionate formation in bacteria



sum: lactate + $NADH + H^+$ + ADP + P_i ⟶ propionate + NAD^+ + ATP + $2H_2O$

Propionate formation is a reversal of the entry of propionate into the TCA cycle.
The primary substrate for propionate is *lactate*, which is converted to pyruvate → OAA → malate → fumarate → succinate → succinyl-CoA → methylmalonly-CoA → propionyl-CoA → propionate.
(To make glucose, bovine liver mitochondria partially reverse the pathway: Propionate → OAA Then: OAA → PEP → glucose

IV. Glycogen metabolism

A. Liver

1. Contains up to 6% glycogen.

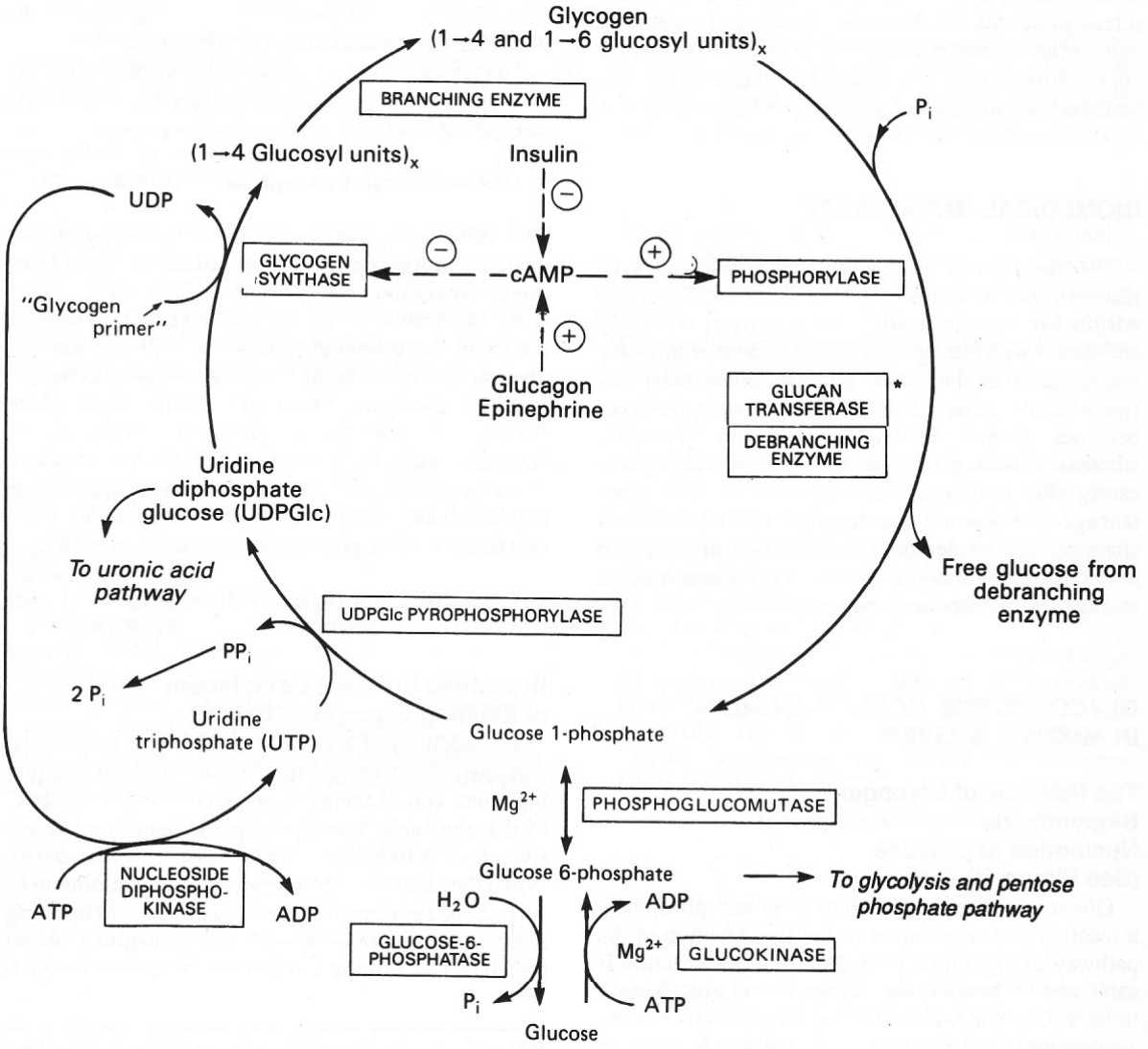
2. Provides glucose for systemic metabolism.

B. Muscle

1. Rarely exceeds 1% (very consistent).

2. Because of muscle mass, muscle contains three to four times as much glycogen as liver.

C. Overview of glycogen turnover

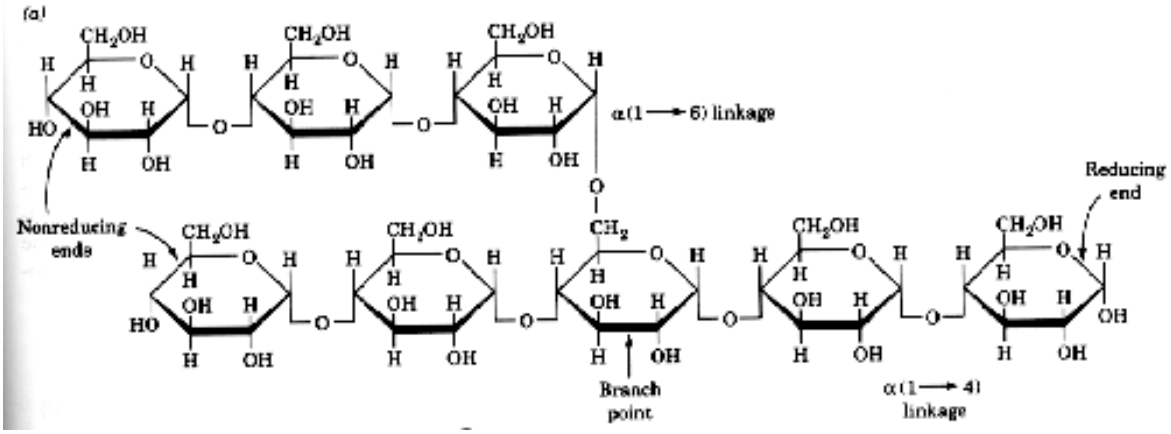


D. Glycogen branching

1. Structure of glycogen

a. Backbone consists of α-1,4 glycosidic linkages.

b. Branchpoints consist of α-1,6 glycosidic linkages.



2. Mechanism of branching

a. 11 α-1,4 glycoside residues are added to a chain.

b. The terminal six residues are transferred to an adjacent chain in a α-1,6 glycosidic linkage.

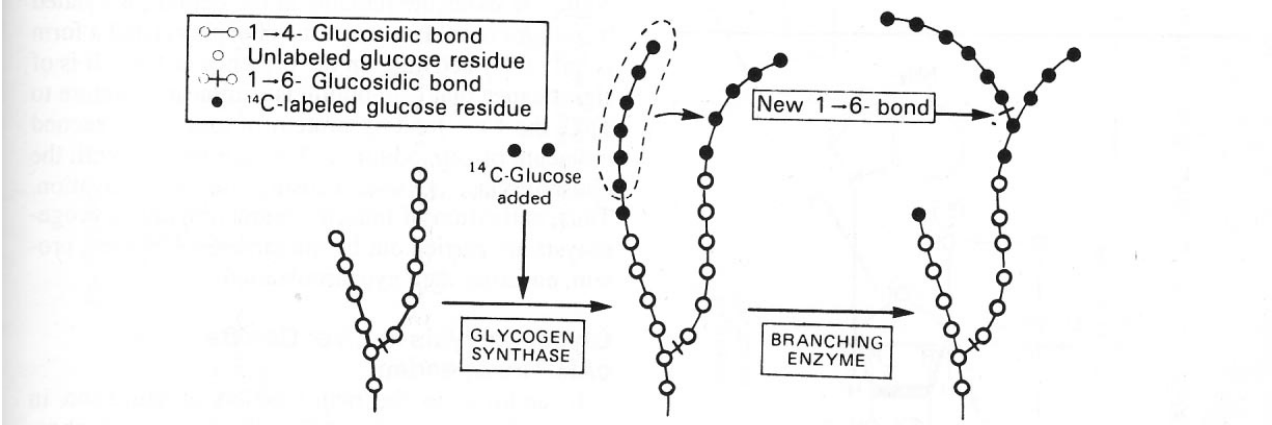
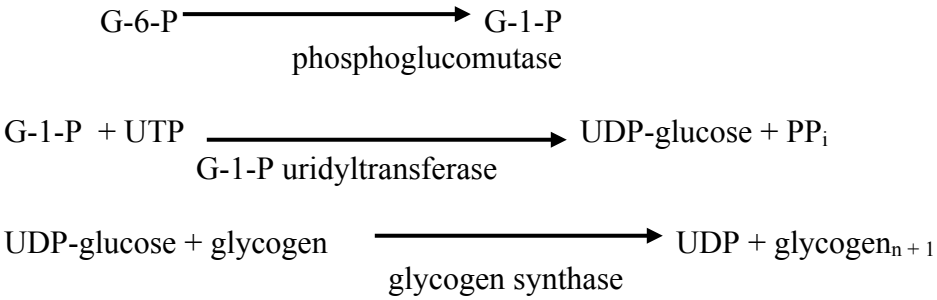


Figure 20–3. The biosynthesis of glycogen. The mechanism of branching as revealed by adding ^{14}C-labeled glucose to the diet in the living animal and examining the liver glycogen at further intervals.

Glycogen synthesis



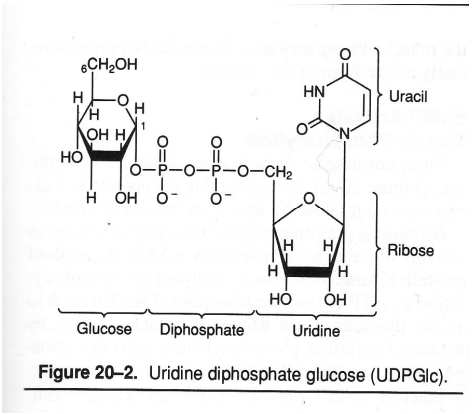
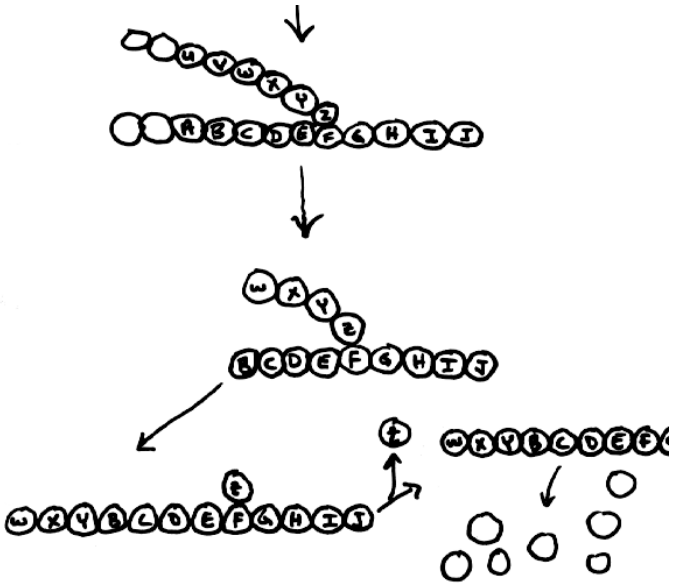


Figure 20–2. Uridine diphosphate glucose (UDPGlc).

V. Glycogen degradation

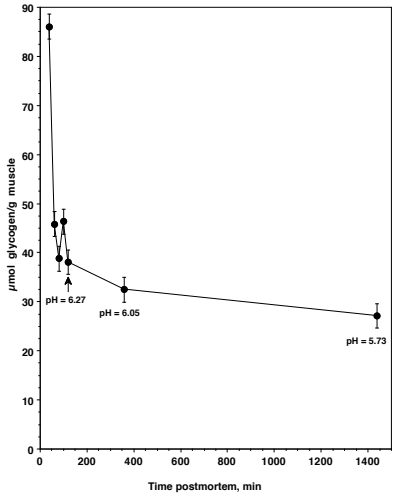
A. Glycogen phosphorylase adds phosphate groups to the 1-carbon of glucosyl residues of glycogen, producing G1P.

B. This reaction also produces free glucose at branch points.

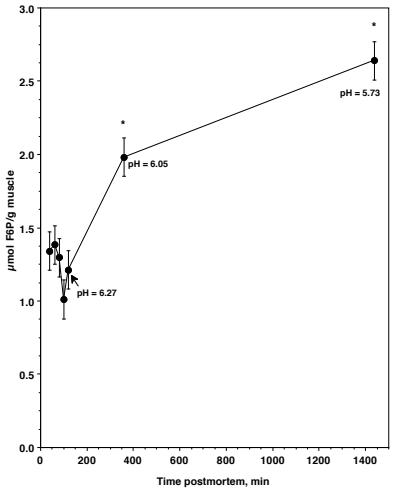


Postmortem metabolism in bovine muscle

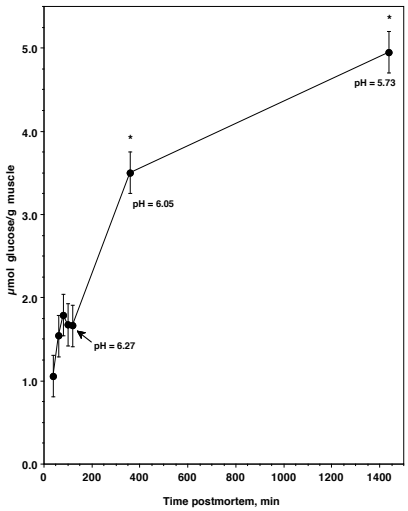
A. Muscle glycogen declined to about one-third of initial values.



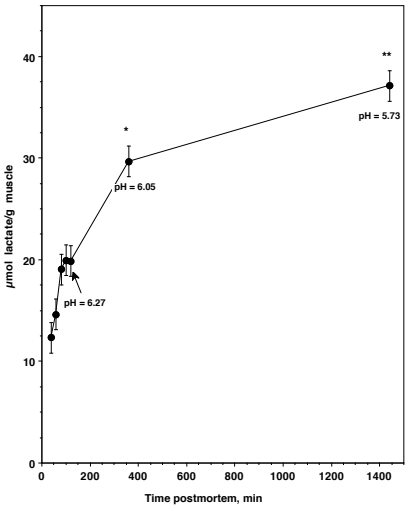
D. F6P increases, indicating inhibition at 6-PFK.



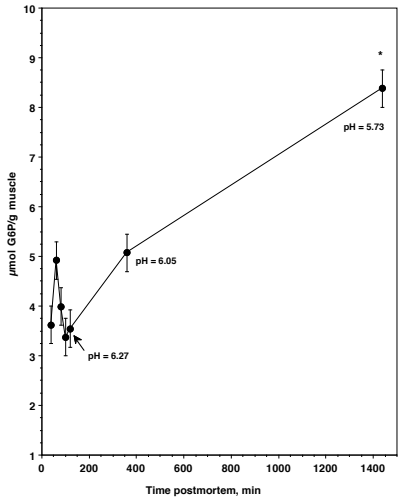
B. Glucose increases over 5-fold, caused by debranching of glycogen.



E. Lactate increases 3-fold, which causes the decline in pH.



C. G6P increases as F6P increases, which would inhibit hexokinase activity.



F. The pH declines to 6.75 by 4 h postmortem.