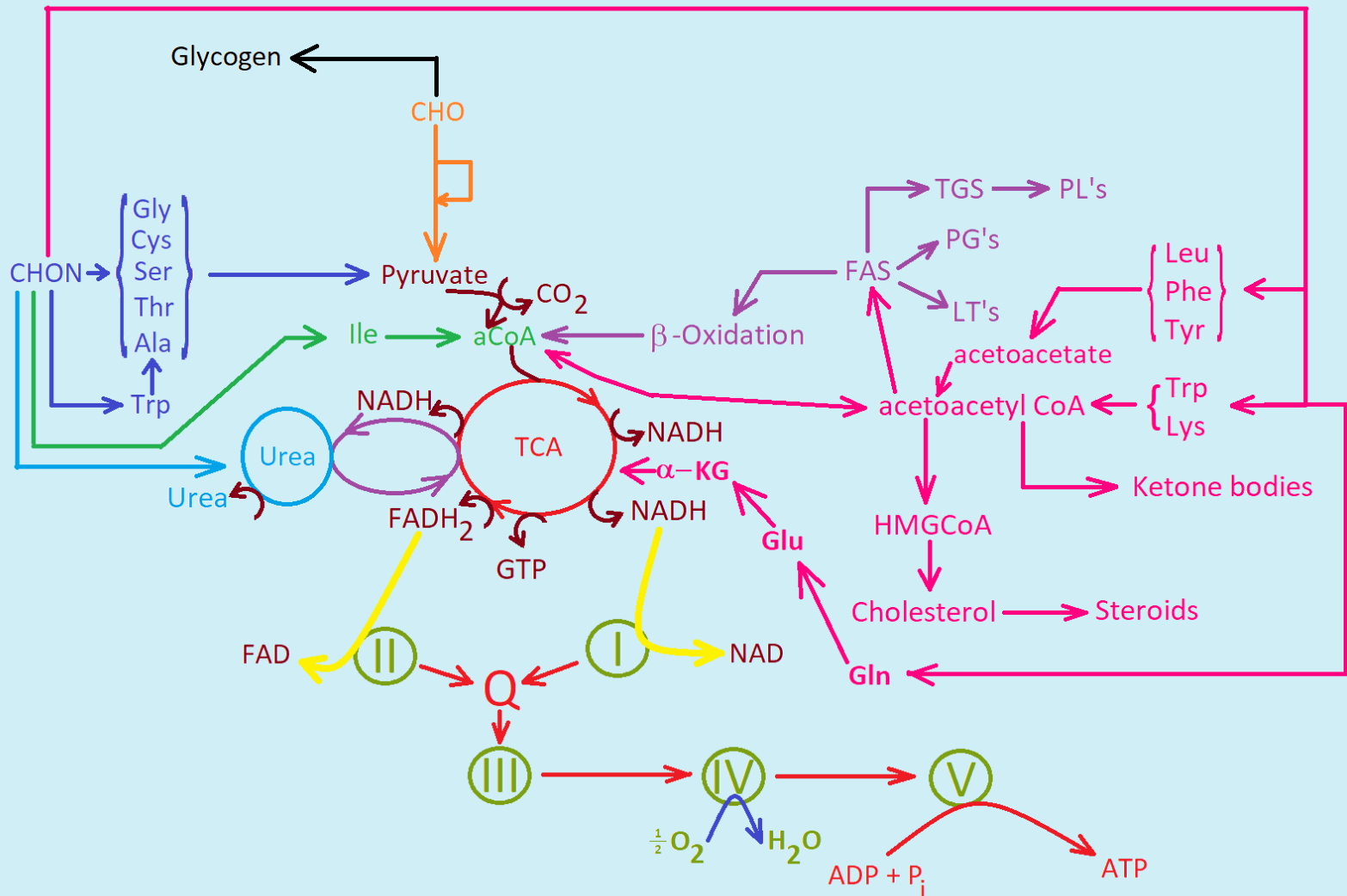


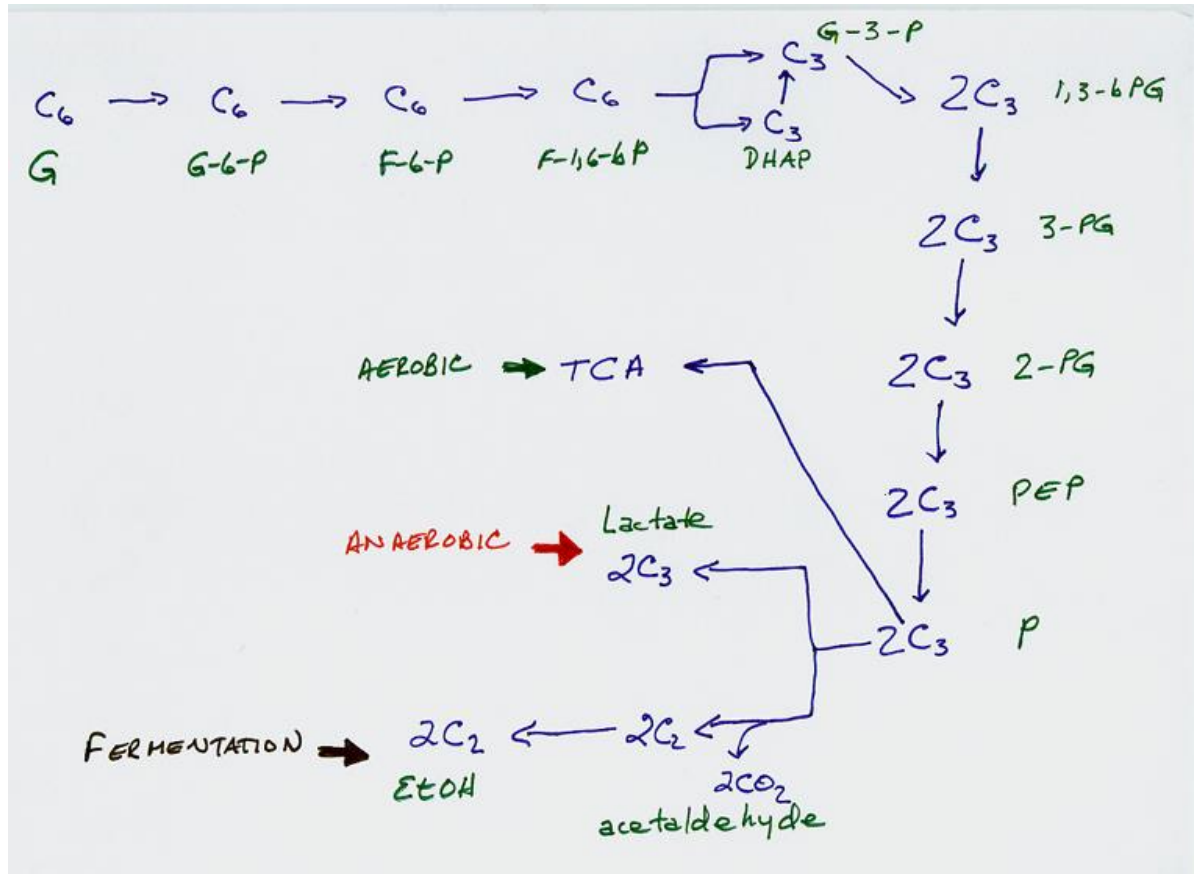
# Introduction to Intermediary Metabolism with Photosynthesis



# Carbohydrate Metabolism

## Part I

- As a general rule, whenever you hear "kinase", think ATP and magnesium ions.
- Hexokinase is a generic enzyme, capable of "working" on most hexoses.
- PFK is one of the major regulatory enzymes in the EMP pathway.

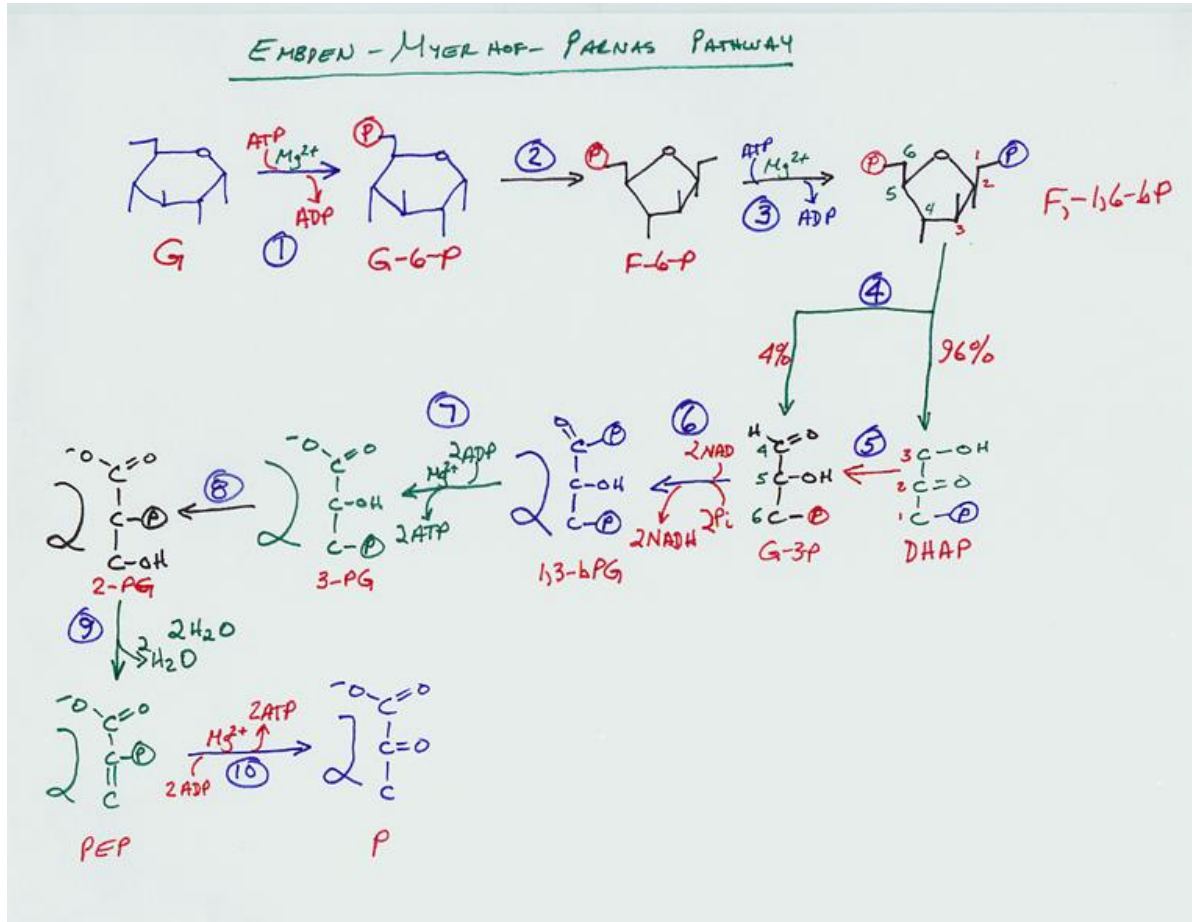


- Carbohydrate metabolism seems to get most of the time and attention throughout all of metabolism
- This pathway begins with a six-carbon sugar, glucose, and ends with 2 three-carbon intermediates (pyruvate) as glucose is oxidized.

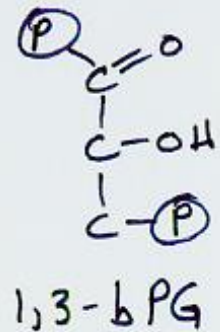
## Embden-Meyerhof-Parnas Overview

# Embden-Meyerhof-Parnas

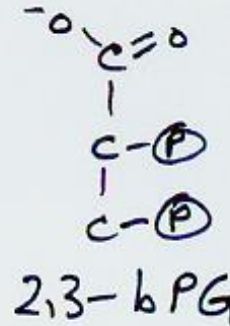
- for glucose to be adequately catabolized, it has to be "trapped" in cells



1. Hexokinase
2. Phosphoglucisomerase
3. PFK
4. Aldolase
5. Triose phosphate isomerase
6. G-3-PDH
7. Phosphoglycerate kinase
8. Phosphoglyceromutase
9. Enolase
10. Pyruvate kinase



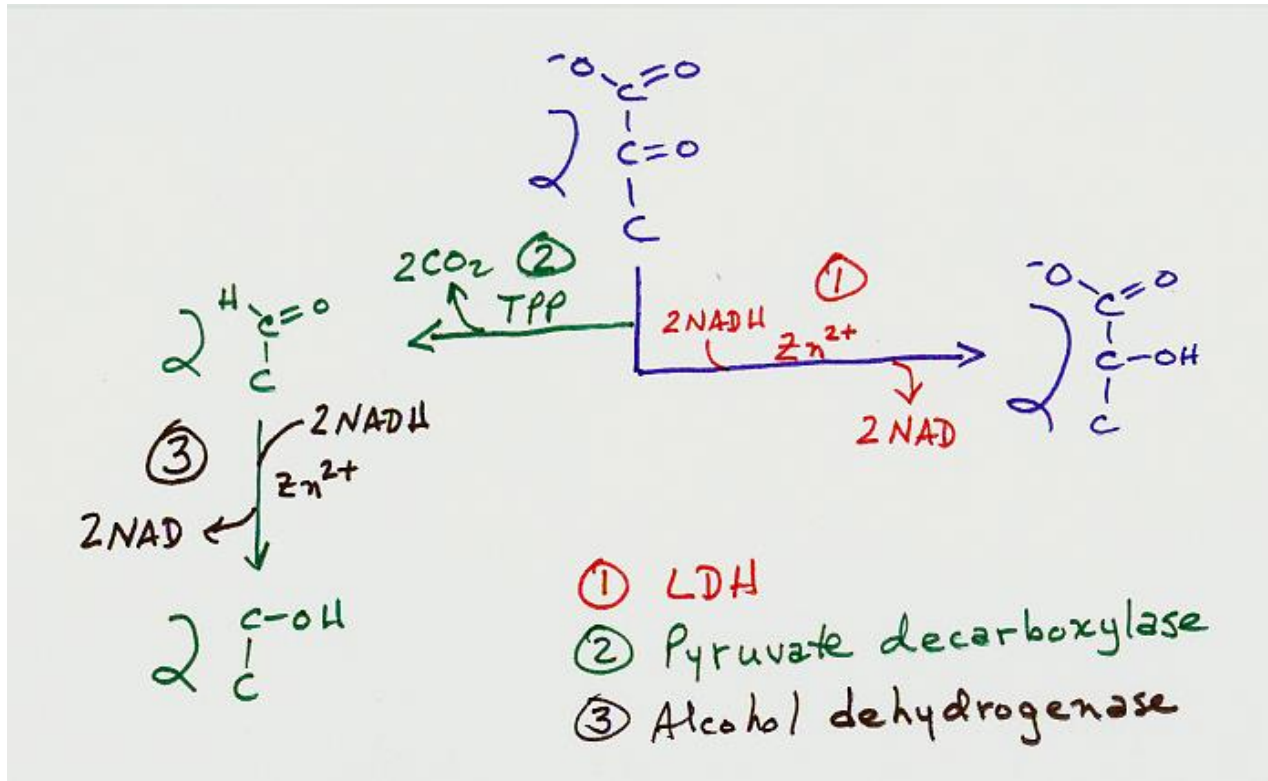
phospho-  
glycero  
mutase



## 2,3-bPG

- 2,3-bPG reduces the affinity of Hb for O<sub>2</sub> – is a primary compensatory factor in going to higher altitudes -- Is a side-rxn of EMP
- E.g., if live in San Francisco, to adapt (short term) to life at Lake Tahoe, body increases [2,3-bPG] so more oxygen is released to the cells in the body

# Anaerobic/Fermentative Metabolism



- The stoichiometry – **aerobic AND anaerobic** -- (review your Chem 121) is that per molecule of glucose (1 six-carbon sugar), TWO molecules of three-carbon sugars are formed, i.e., one times six is six, as are two times three.

# EMP – Stimulators and Inhibitors

## Inhibitors

## Stimulators (“Activators”)

- PFK: ADP and AMP
- Low energy turns on EMP
- PFK: Citrate and ATP
- G-3-PDH:  $\text{AsO}_4^{3-}$
- Enolase: fluoride ion
- Pyruvate kinase: ATP
- High energy turns off EMP – arsenate inhibits because it looks like phosphate

# ATP Summary – Used and Gained -- **AEROBIC**

## ATP Used

- Hexokinase: -1
- PFK: -1
- **Total USED = 2 ATP**

## ATP Gained

- G-3-PDH: +6 ( $\Leftrightarrow$ )
- Phosphoglycerate kinase: +2
- Pyruvate kinase: +2
- **Total GAINED: 10 ATP**

Overall: **8 ATP produced**

# ATP Summary – Used and Gained – ANAEROBIC (FERMENTATIVE, too)

## ATP Used

- Hexokinase: -1
- PFK: -1
- LDH (alcohol DH) : -6 ( $\Leftrightarrow$ )
- Total ATP USED: -8 ATP

## ATP Gained

- G-3-PDH: +6 ( $\Leftrightarrow$ )
- Phosphoglycerate kinase: +2
- Pyruvate kinase: +2
- Total GAINED: 10 ATP

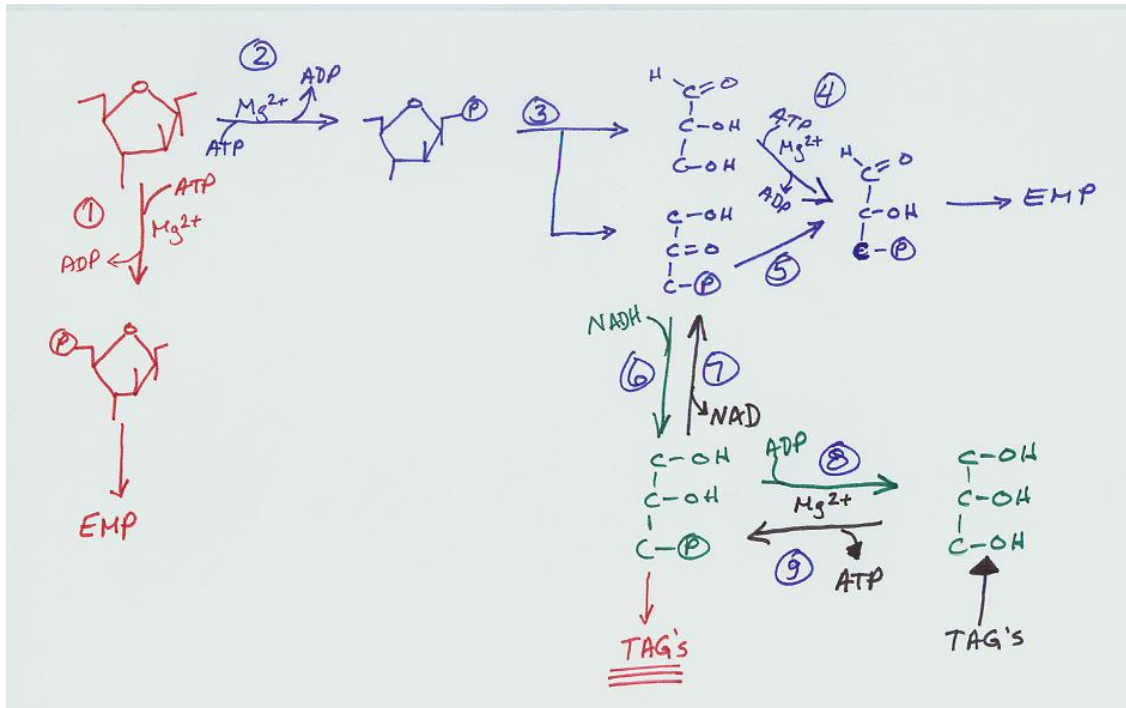
In anaerobic glycolysis (fermentation),

# ATP go down from 8 to 2 because the NADH and NAD cycle



# Fructose Metabolism

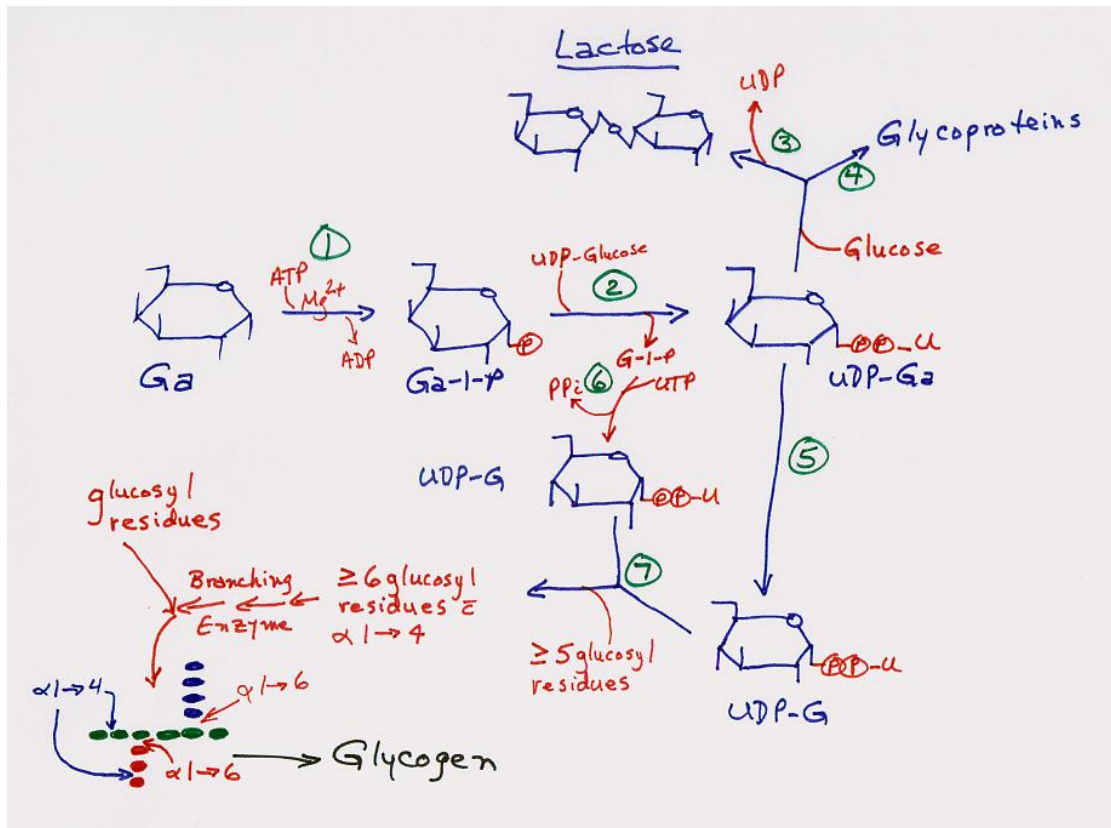
- Every now and again, a diabetic patient's parent, sibling or other relative reads about how diabetes is a disease of glucose metabolism.
- On occasion, they read a little more and discover that fructose is a carbohydrate, but not glucose.
- They then come in to see you as the health care person who knows something about diabetes and ask you, "Since fructose isn't glucose, can I substitute all of my relative's carbohydrate needs with fructose?"
- Your answer is, of course, no.
- So, why is it that your answer is no?
- The catabolism of fructose and how it intertwines with triglyceride (TGS) synthesis follows.



1. Hexokinase (adipose tissue)
2. Fructokinase (liver)
3. F-1-P aldolase
4. Triose kinase
5. Triose phosphate isomerase (TPI)
6. DHAP DH
7. Glyceraldehyde phosphate DH
8. Phosphatase
9. Glyceraldehyde kinase

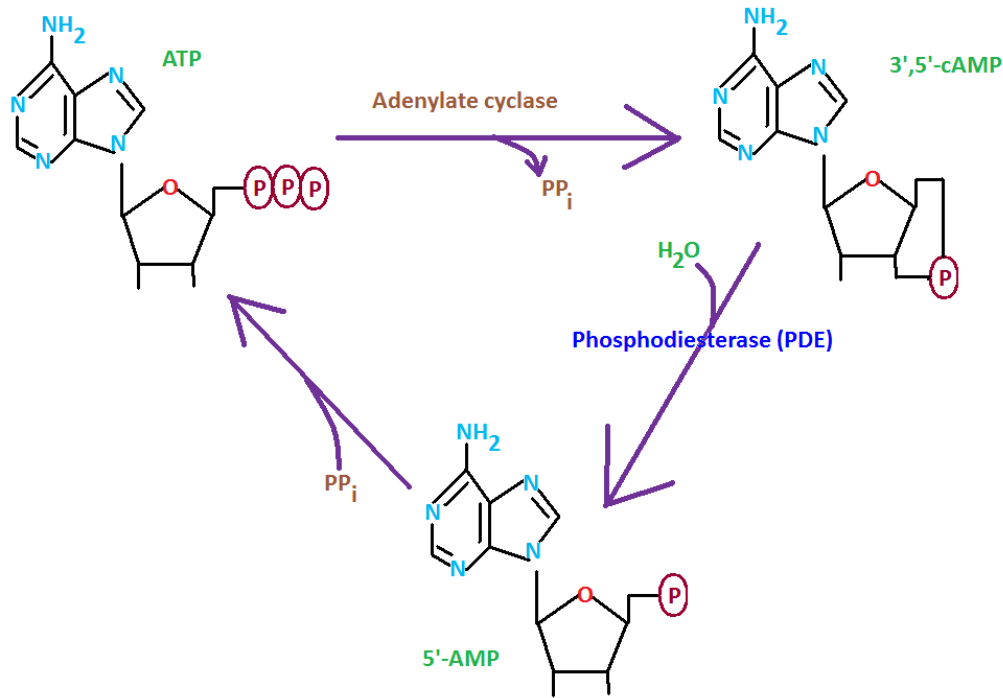
# Galactose Metabolism

- Galactose is an important constituent of lactose,
- Galactose may be used in the synthesis of glycoproteins
- Galactose may be used in the synthesis of glycogen



- Galactokinase
- Galactose-1-phosphate uridyl transferase
- Lactose synthetase (BOTH catalytic unit and specificity protein)
- Lactose synthetase (catalytic unit only)
- UDP-galactose-4-epimerase
- UDP-glucose pyrophosphorylase
- Glycogen synthetase

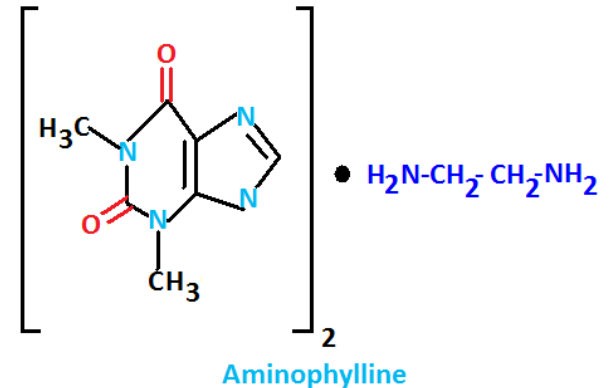
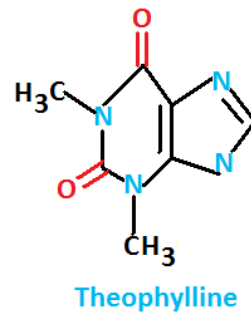
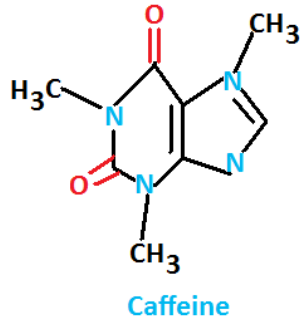
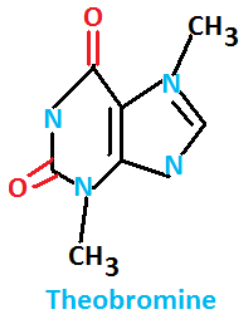
# Glycogen Metabolism Regulated by cAMP



- Phosphodiesterase (PDE) inhibited by:
  - Theophylline
  - Caffeine
  - Theobromine
- These compounds are called xanthines.
- When PDE is inactivated, cAMP levels build up, making it easier for patients to breathe (controversy).

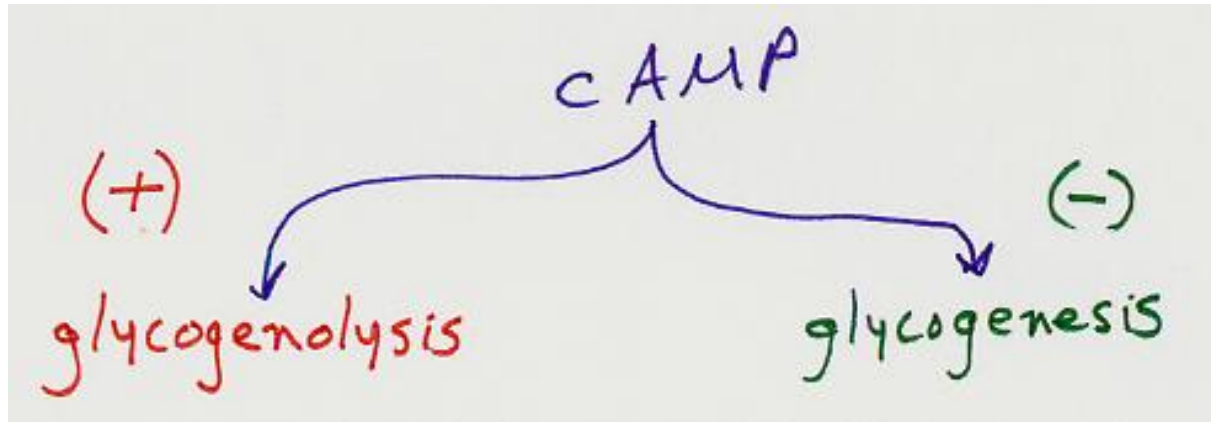
- Elevated levels of cAMP drive protein synthesis, enzyme cascades and changes in membrane permeability

# Adenylate Cyclase Inhibitors



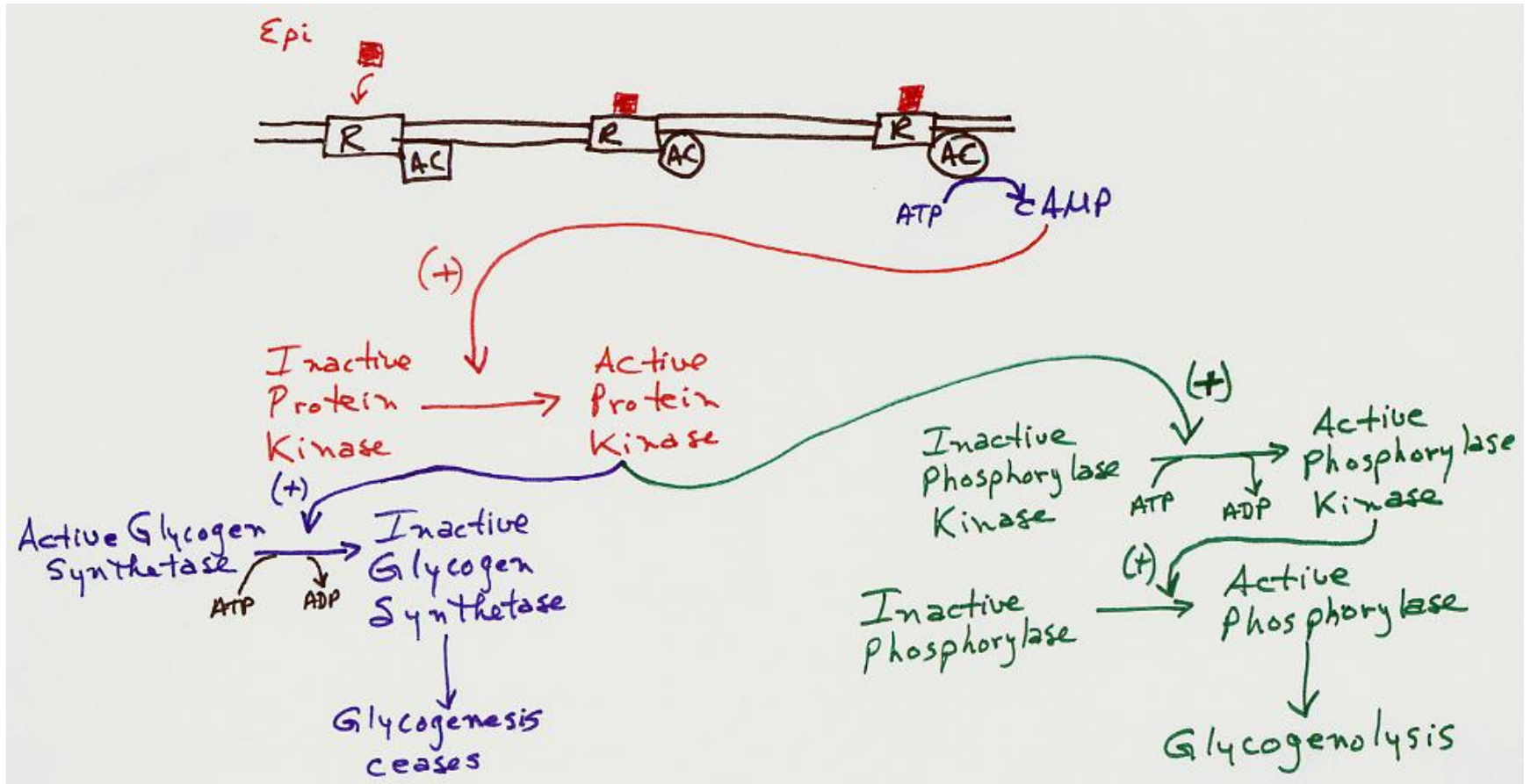
- Phosphodiesterase (PDE) inhibited by:
  - Theophylline
  - Caffeine
  - Theobromine
- These compounds are called xanthines.
- When PDE is inactivated, cAMP levels build up, making it easier for patients to breathe. Is this true or false???

# In General: Glycogen – olysis and genesis

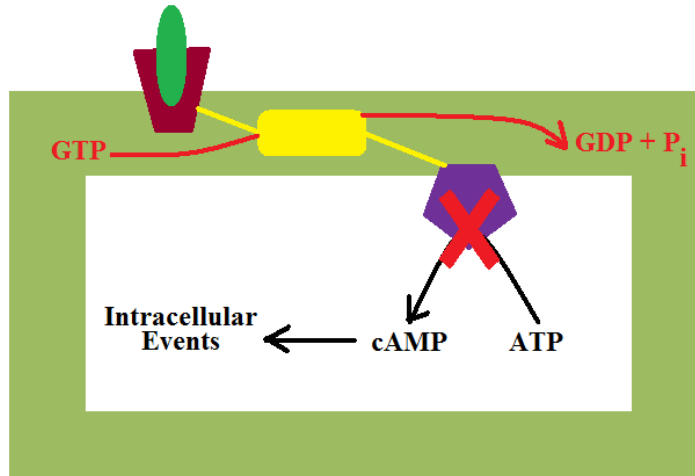
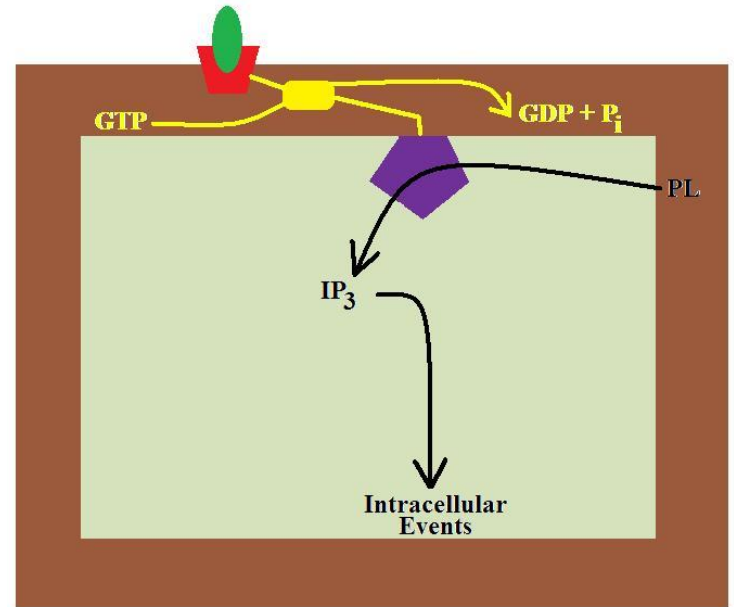
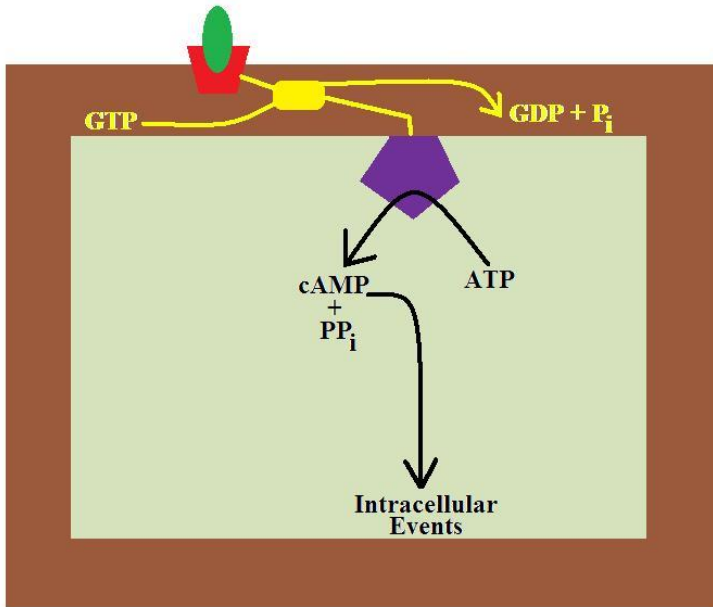


- Glycogenolysis (destruction of glycogen)
- Glycogenesis (production of glycogen)
- As the body doesn't like to be confused during times of stress, cAMP inhibits glycogenesis and activates glycogenolysis.
- One example of this occurs when epinephrine binds with the appropriate receptor on the cell membrane of a target cell.

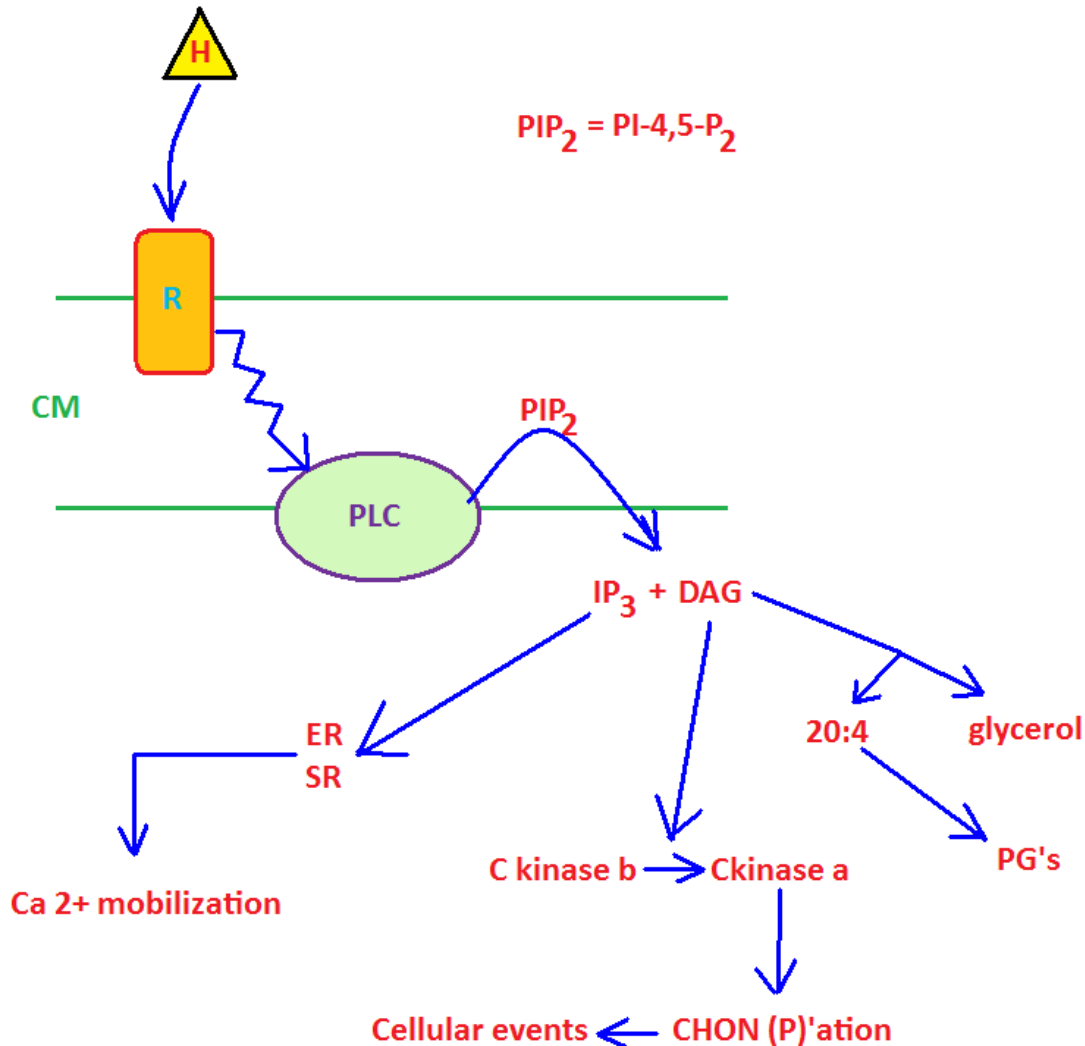
# In Detail



# G Proteins



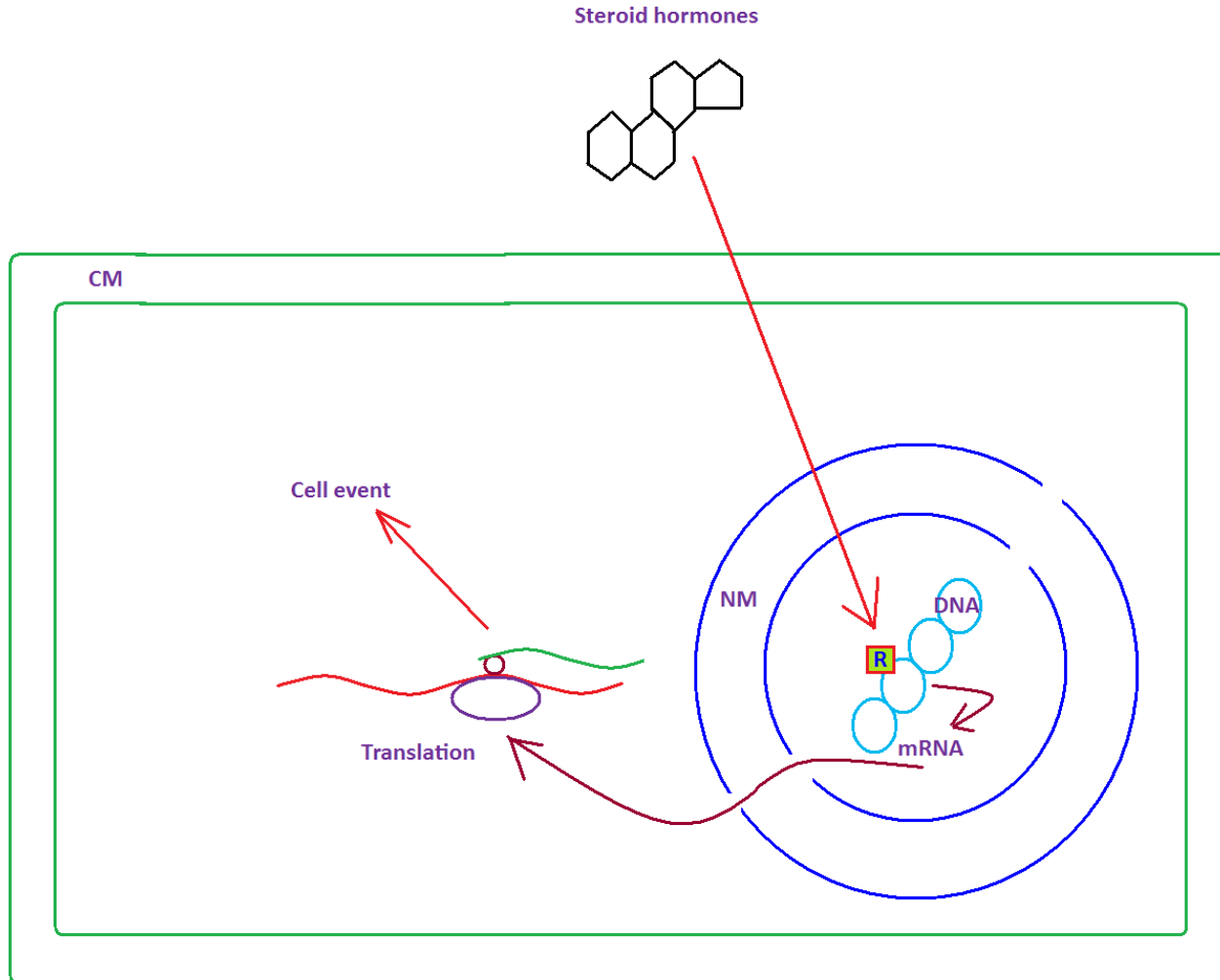
# Second Messenger: $IP_3$



$IP_3$  drives changes in:  
Ca<sup>2+</sup> concentration  
Ca<sup>2+</sup> mobilization  
GABA, AVP, ANG,  
TSH utilize  $IP_3$



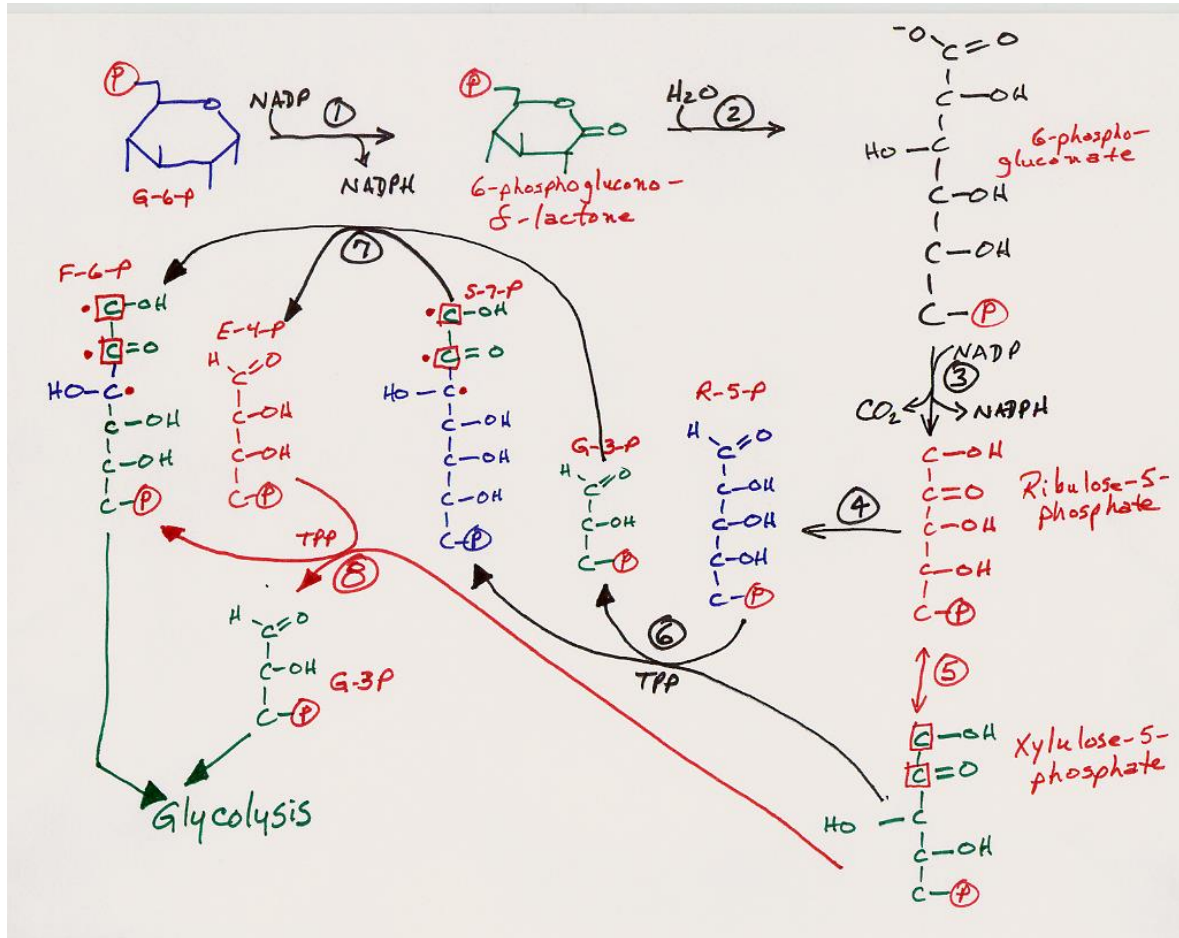
# Steroid Hormones



- Steroids drive translation
- Functionally: increased activity
- Structurally: Bulking up

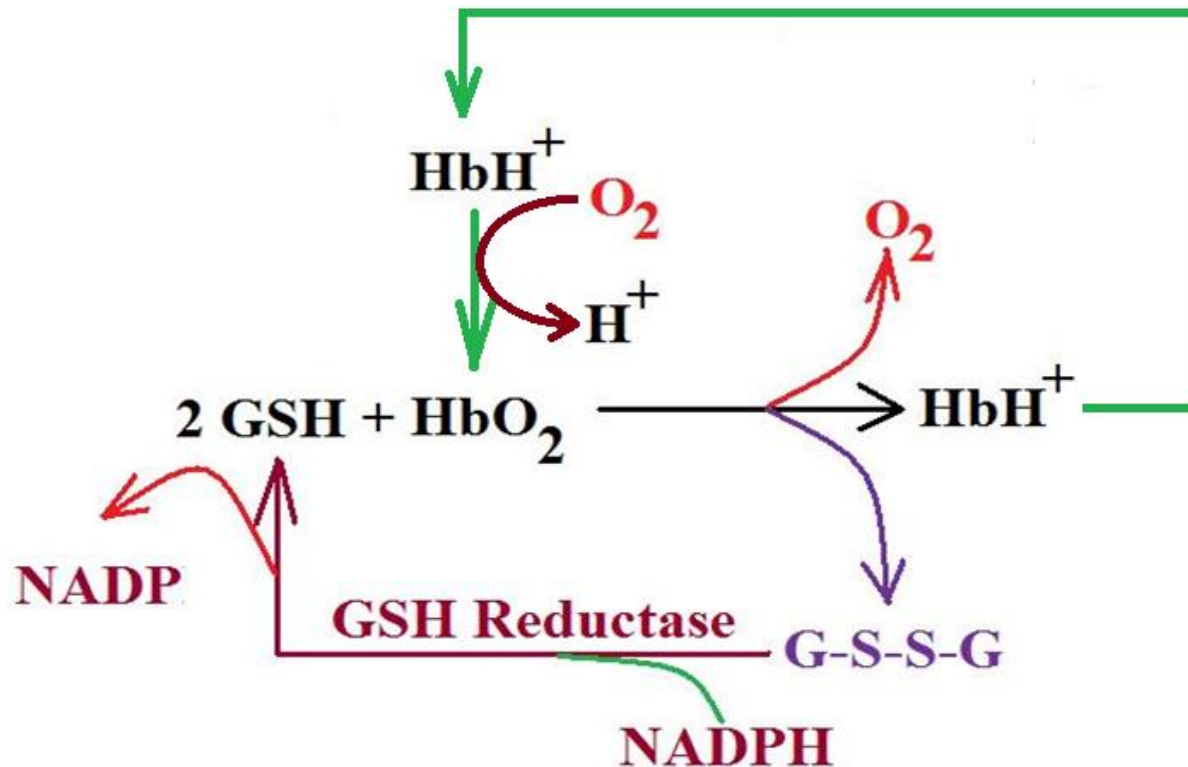
NO Second Messengers!!

# Hexose Monophosphate Shunt (Pentose Phosphate Pathway)



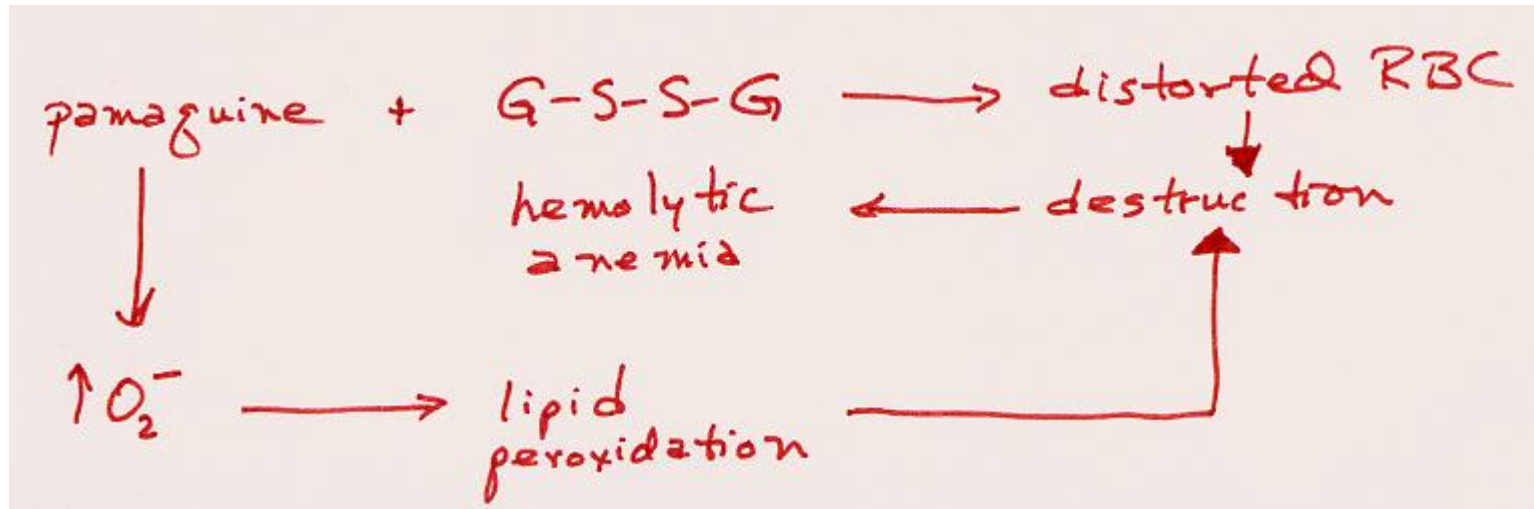
1. G-6-PDH
2. Lactonase
3. 6-P-gluconate DH
4. Phosphopentose isomerase
5. Phosphopentose epimerase
6. Transketolase -- (2 C transfer)
7. Transaldolase -- (3 C transfer)
8. Transketolase -- (2 C transfer)

# Hexose Monophosphate Shunt (Pentose Phosphate Pathway): Clinical/Significance

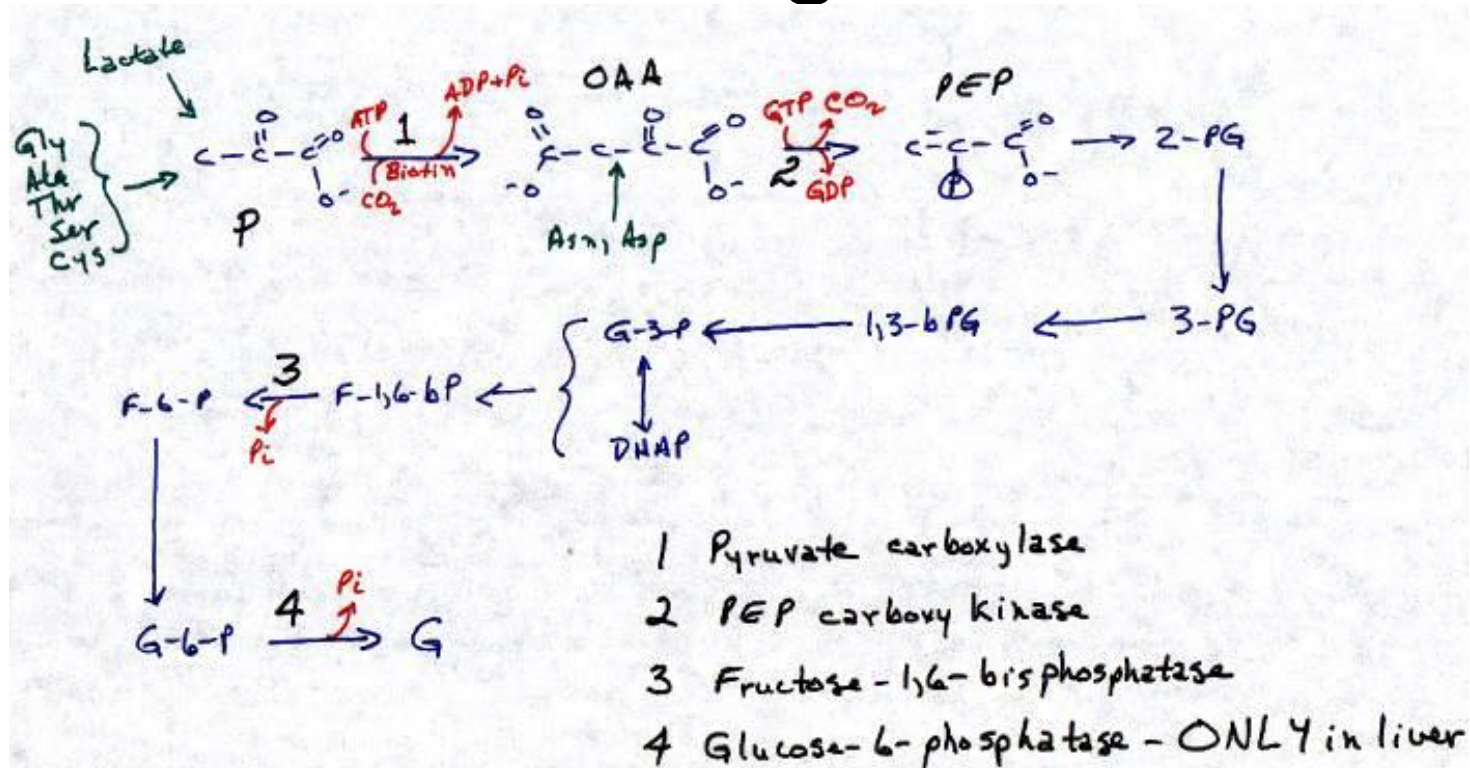


- 2 NADPH – reductive power
- Ribose – nucleoside/nucleotide synthesis
- People with G-6-PDH deficiency don't make enough NADPH to reduce G-S-S-G and causes health problems

# Hexose Monophosphate Shunt (Pentose Phosphate Pathway): Clinical Significance



# Gluconeogenesis

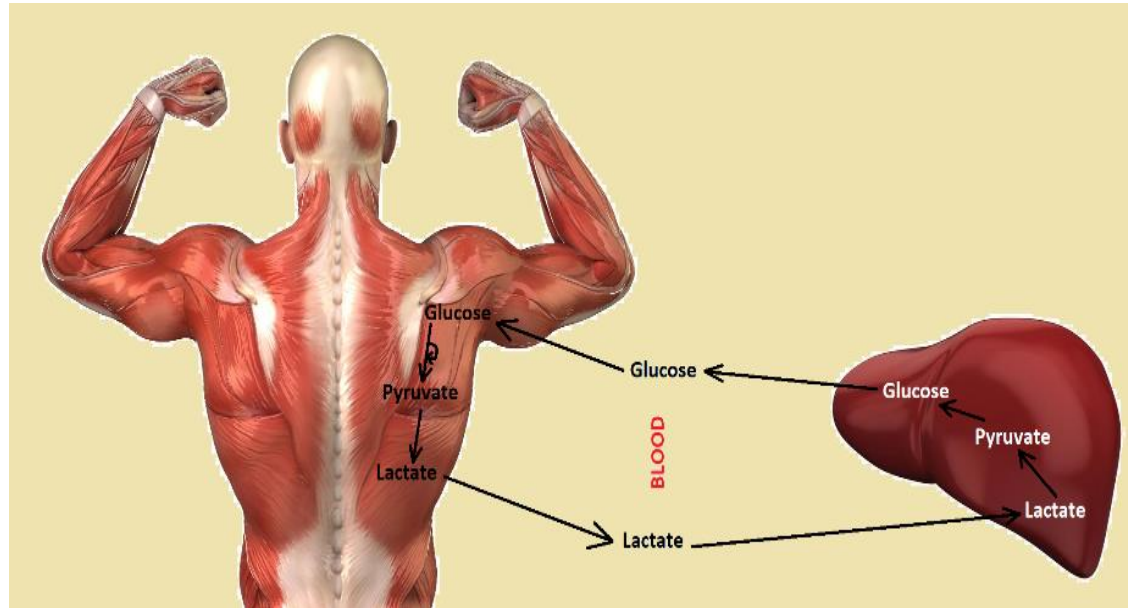


- Gluconeogenesis is NOT the absolute reverse of glycolysis.
- Some enzymes are the same -- 4 are NOT
- When the body produces new glucose, it utilizes various substrates as necessary.
- These include the carbon skeletons of amino acids and anaerobic end-products of catabolism.
- The carbon skeletons of Gly, Ala, Thr, Ser and Cys feed into gluconeogenesis via pyruvate, as does lactate.
- The carbon skeletons of Asn and Asp feed in to OAA.

# Gluconeogenesis

- Remember, though, that the purpose of phosphorylation of glucose in the first place is to trap it in the cell.
- Since that is a trapping mechanism, there has to be a way to remove the phosphate so that the newly formed glucose can get "dumped" into the blood.
- ONLY the liver cells contain G-6-phosphatase (4) that cleaves off the phosphate so that the glucose may be routed through the body for use as needed by its cells.
- Which tissue does NOT require insulin for glucose uptake?

These pathways are all fine and dandy, but of what significance are anaerobic glycolysis and gluconeogenesis?



- The significance is the **Cori cycle**, named after Dr. and Dr. Cori who discovered it.
- As muscle tissue anaerobically catabolizes glucose for whatever energy needs, lactate is produced.
- Lactate is small enough to freely diffuse across the muscle cell membranes into the blood.
- Once in the blood, it travels to the liver where it diffuses into liver cells.
- The lactate is used in gluconeogenesis to synthesize more glucose in the liver, which is then sent back to the muscles for utilization until aerobic catabolism catches up or until the muscle needs no more glucose.
- The Cori cycle buys time and changes the metabolic burden to the liver.

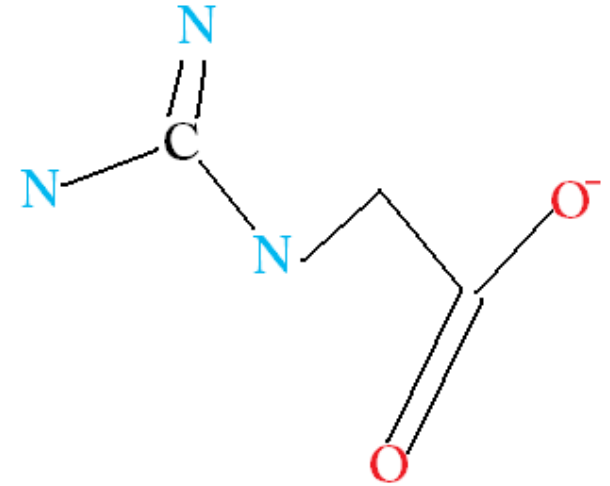
# Aerobic Energy Sources from Intermediary Metabolism

- There are three systems that provide energy to cells:
  - System 1: Phosphagen System
  - System 2: Creatine Phosphate System
  - System 3: The Krebs' Cycle (TCA; Citric Acid Cycle)
- The first system is the phosphagen system. In this system, the source of the energy is ATP. During muscular contraction, ATP is hydrolyzed to ADP,  $P_i$  and energy. When this happens, there is only enough energy for 5-6 seconds.
- So how do our cells get additional energy?
- Our cells get it via a compound called phosphocreatine (PCr or CrP; System 2). The concentration of PCr is about 2-3 times greater than the concentration of ATP. When PCr is available, it is hydrolyzed to Cr and  $P_i$  and energy. The  $P_i$  is used to re-phosphorylate ADP to make more ATP.
- This gives us about 15 seconds of maximal contractions and is used for short bursts.
- As long as the system (cell and/or tissues and/or body) remains in an aerobic state and fuel is present, the TCA (System 3) will continue to provide energy to the cells.

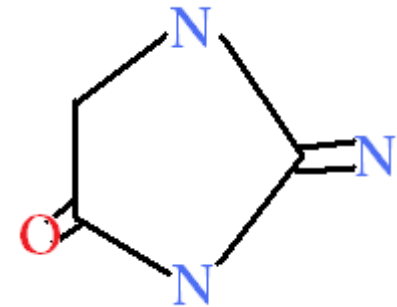


# Aerobic Energy System 2

1. Creatine: is a nitrogenous organic acid that occurs naturally in vertebrates and helps to supply energy to muscle and nerve cells. In humans and animals, approximately half of stored creatine originates from food (mainly from fresh meat). Ninety-five percent of creatine is later stored in the skeletal muscles.

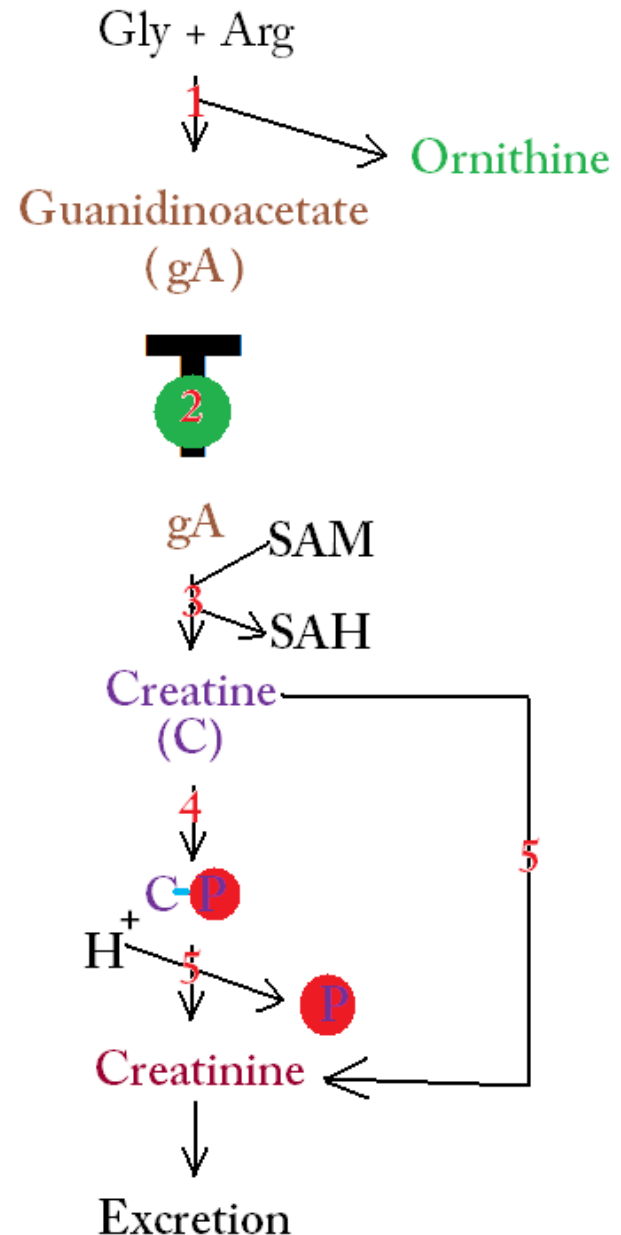


2. Creatinine: is a break-down product of creatine phosphate in muscle, and is usually produced at a fairly constant rate by the body (depending on muscle mass). Creatinine is chiefly filtered by the kidney, though a small amount is actively secreted. There is little-to-no tubular reabsorption of creatinine. If the filtering of the kidney is deficient, blood levels rise. Men tend to have higher levels of creatinine because they have more skeletal muscle than women. Vegetarians tend to have lower creatinine levels, because vegetables contain no creatine.



# Creatine/Creatinine

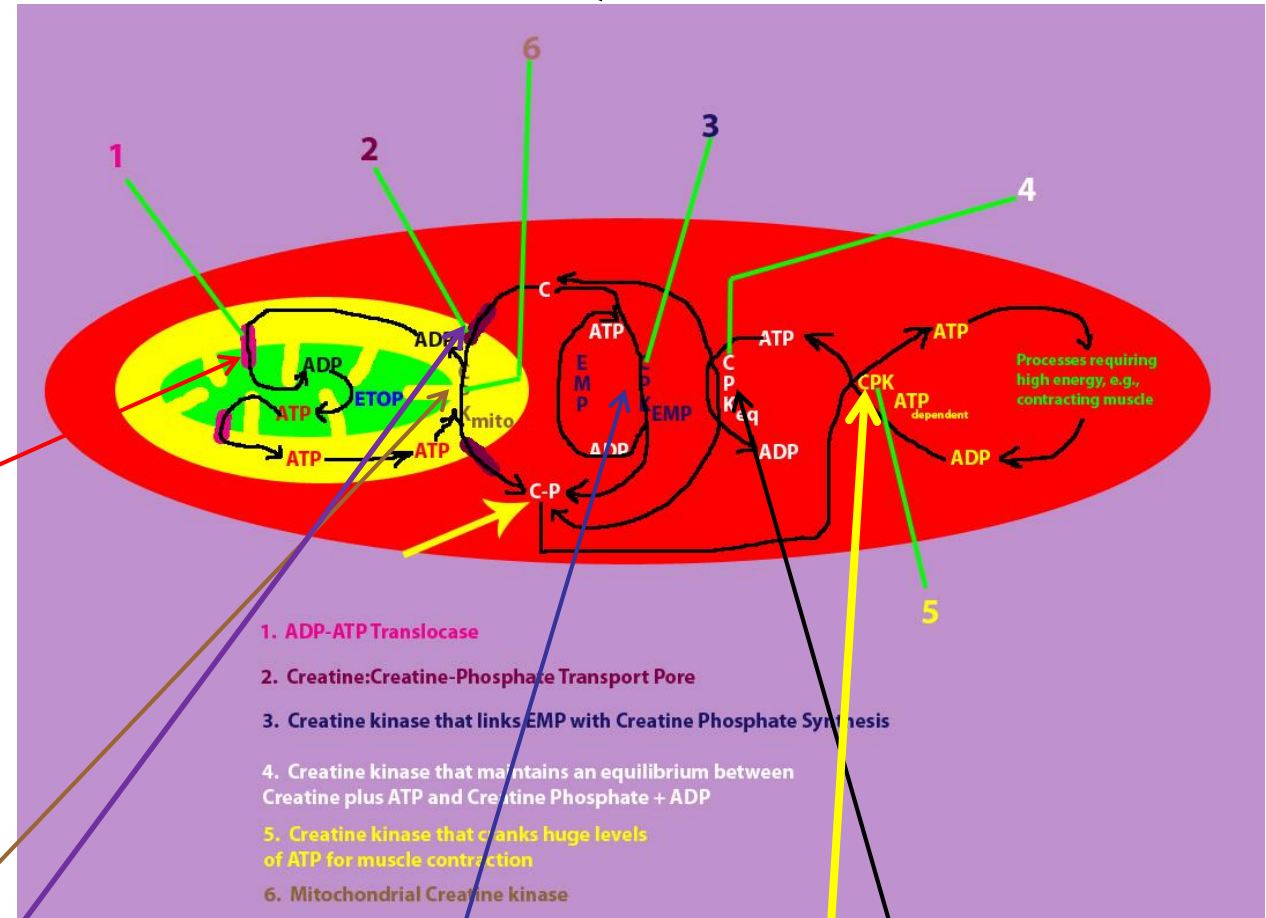
1. Mitochondrial Arginine-glycine: amidinotransferase (E.C. 2.1.4.1)
2. gA Transporter – (from mito to cytosol)
3. Cytosolic S-adenosylmethionine :guanidinoacetate-N-methyltransferase (SAM:gA NMT)
4. Cytosolic Creatine kinase
5. Cytosolic non-enzymatic cyclization



# PCr Shuttle – Quick View

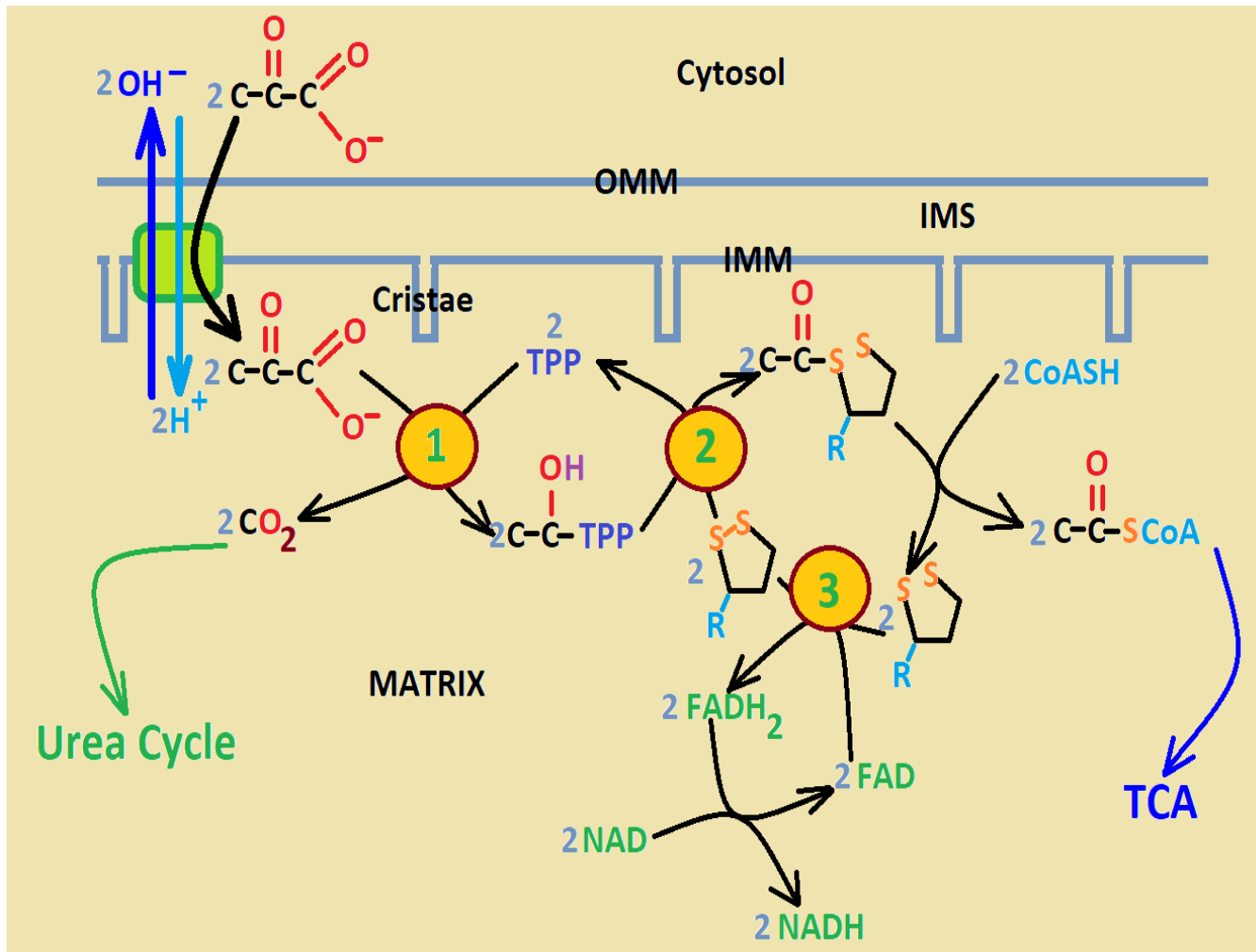
To give you an idea of what is involved in PCr utilization and synthesis, let's examine the PCr shuttle in cardiac and skeletal muscle. This shuttle increases incredibly the movement/transport of high-energy phosphate (ATP) from the matrix of the mitochondrion to the cytosol of the cell.

In the first step (1), an ADP-ATP translocase re-phosphorylates ADP to form ATP in the mitochondrial matrix. This occurs via electron transport/oxidative phosphorylation (the "ETOP" in the graphic). When the ATP is "dumped" into the intermembrane space, it is reacted with creatine via a mitochondrial creatine kinase (6) to form PCr. The PCr is then transported via a creatine-creatine phosphate (C:PCr) transport pore (2) into the cytosol to "dump" into a cytosolic PCr store.



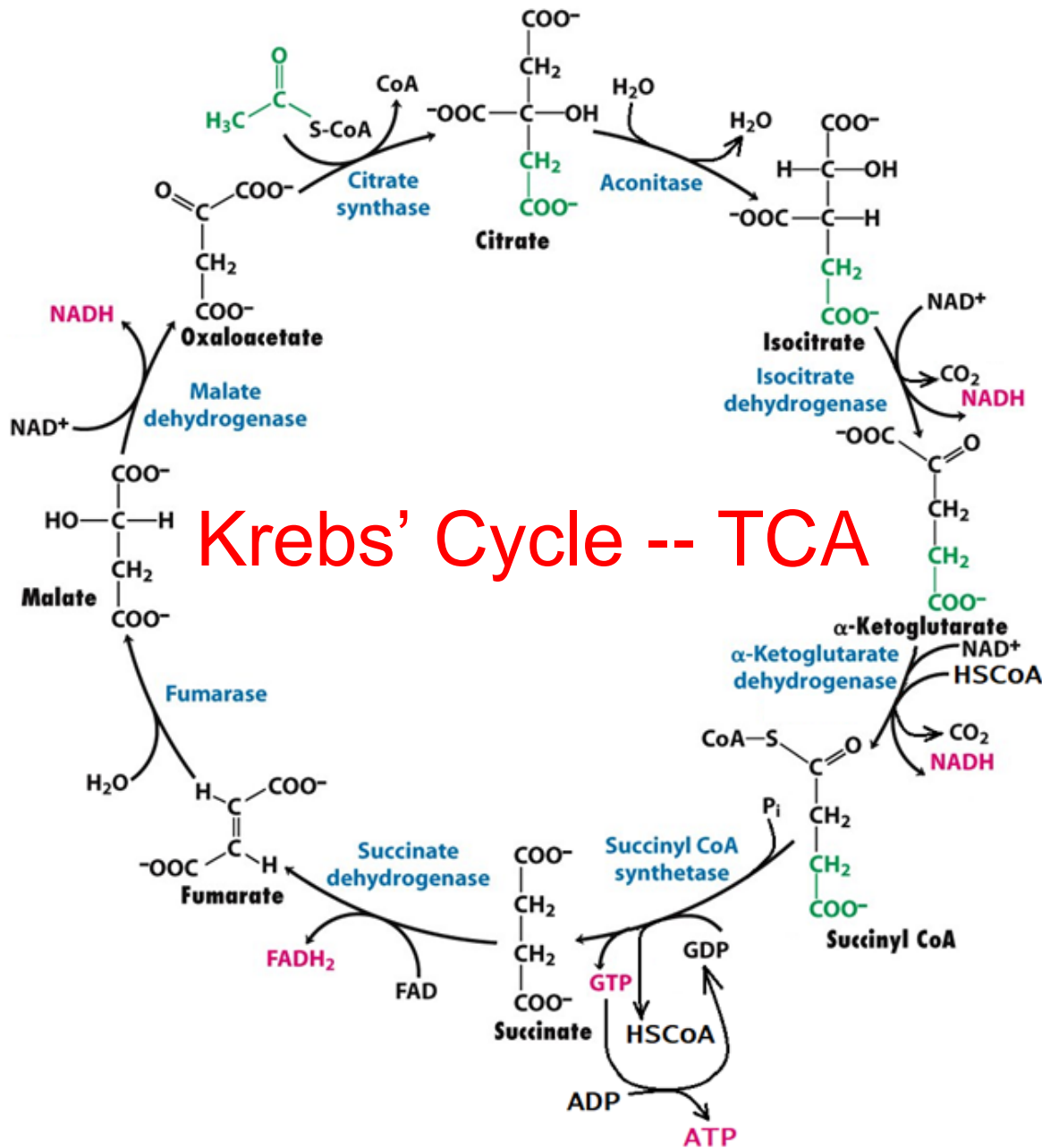
The cytosolic PCr store is derived from an EMF-linked (glycolysis-linked) creatine kinase (CK) (3) and another CK that maintains the equilibrium between C and PCr (or ATP and ADP, if you prefer)(4). The utilization of the PCr from the cytosolic store occurs via an ATP dependent CK that cranks out hugely elevated levels of ATP (5). The ATP is used by processes requiring high energy, e.g., contracting muscle.

# Pyruvate Dehydrogenase Complex – AEROBIC Energy System 3



- Pyruvate is first decarboxylated by PDH (Step 1) in the presence of thiamine pyrophosphate (TPP) to form carbon dioxide (2 moles, remember) and an acetyl-TPP intermediate.
- The acetyl is transferred from TPP to lipoic acid by DHLTA (Step 2) and, then thiolated with the addition of HSCoA (Coenzyme A) and release of reduced lipoic acid and acetyl CoA (aCoA).
- Lastly, the reduced lipoic acid is oxidized at Step 3 by dihydrolipoyl dehydrogenase.

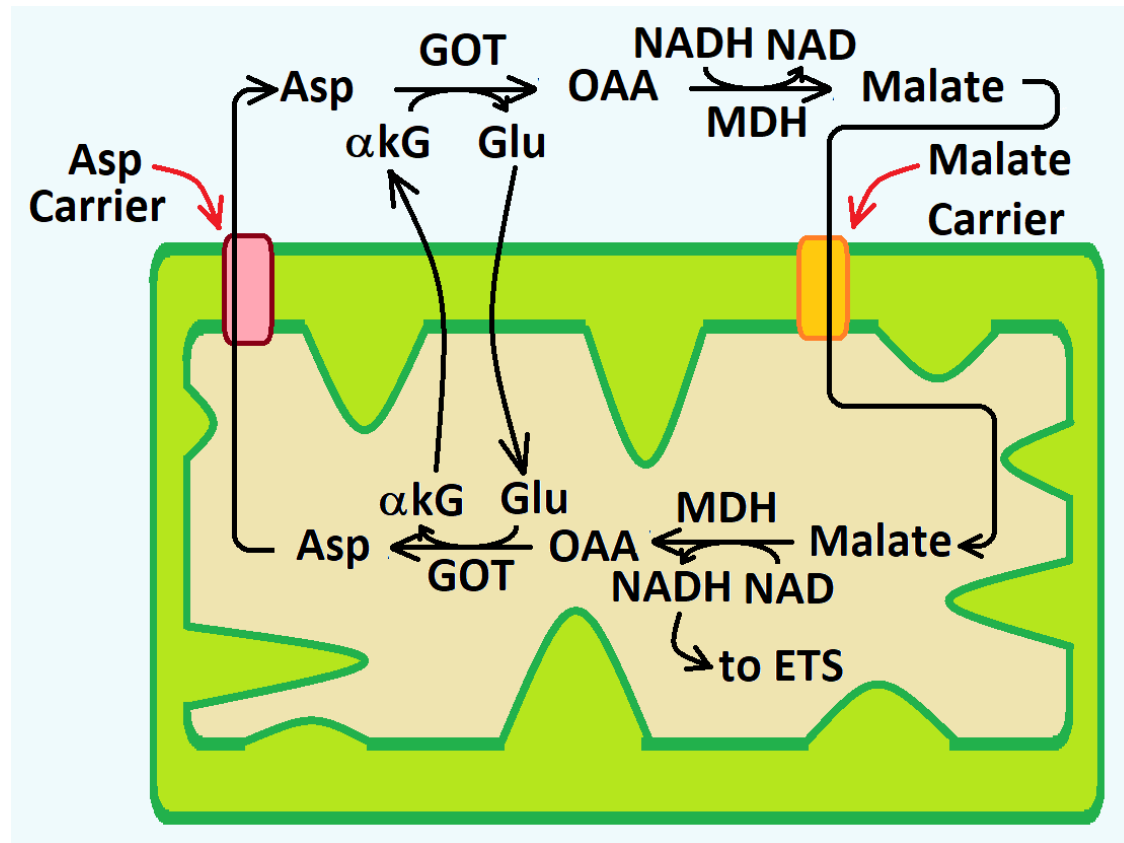
Pyruvate translocase is at the upper left and is represented by the darker green outlined rounded rectangle (filled with lime green). Note the symport transport with the pyruvate and proton and the antiport transport between the pyruvate and the hydroxide ion across the inner mitochondrial membrane (through the pyruvate translocase) and the relatively free diffusion across the outer mitochondrial membrane.



## TCA: ATP Summary – All MADE

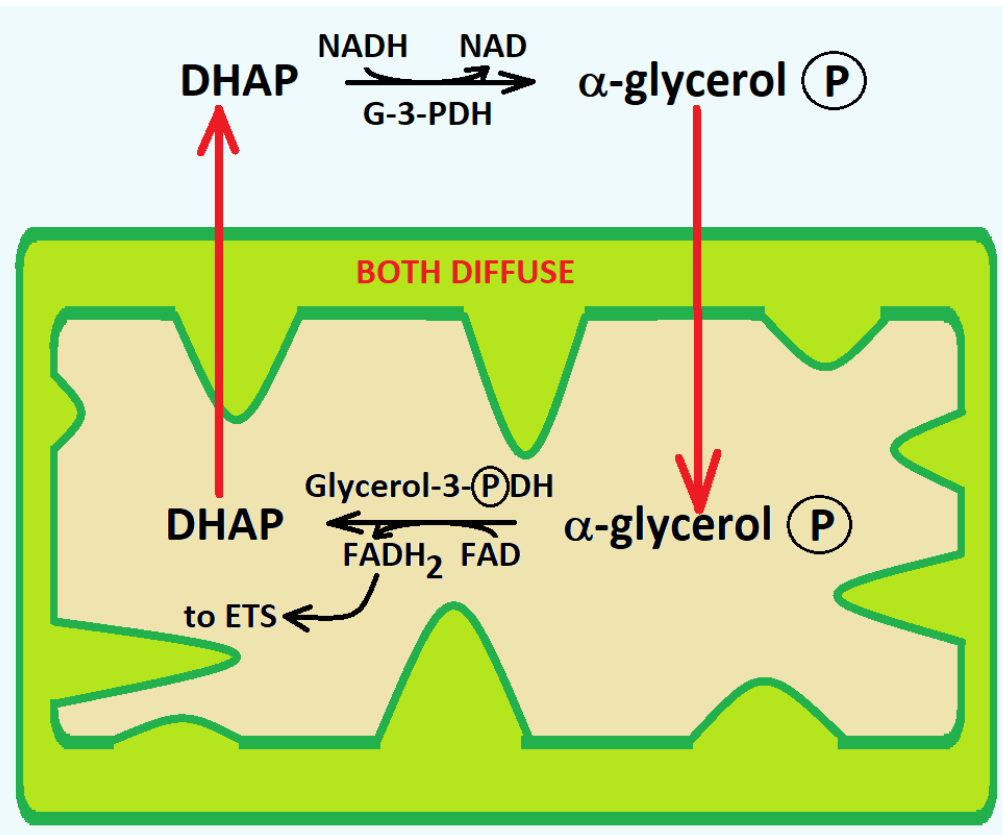
- PDH: +6 (⇌)
- iCDH: +6 (⇌)
- αKGDH: +6 (⇌)
- sCoA synthetase: +2
  - SDH: +4 (⇌)
  - MDH: +6 (⇌)
  - TOTAL: +30
- Glycolysis: +8
- **WAY TOTAL:**
- **38 ATP under aerobic conditions**

# ATP Synthesis by Tissue: Clarification



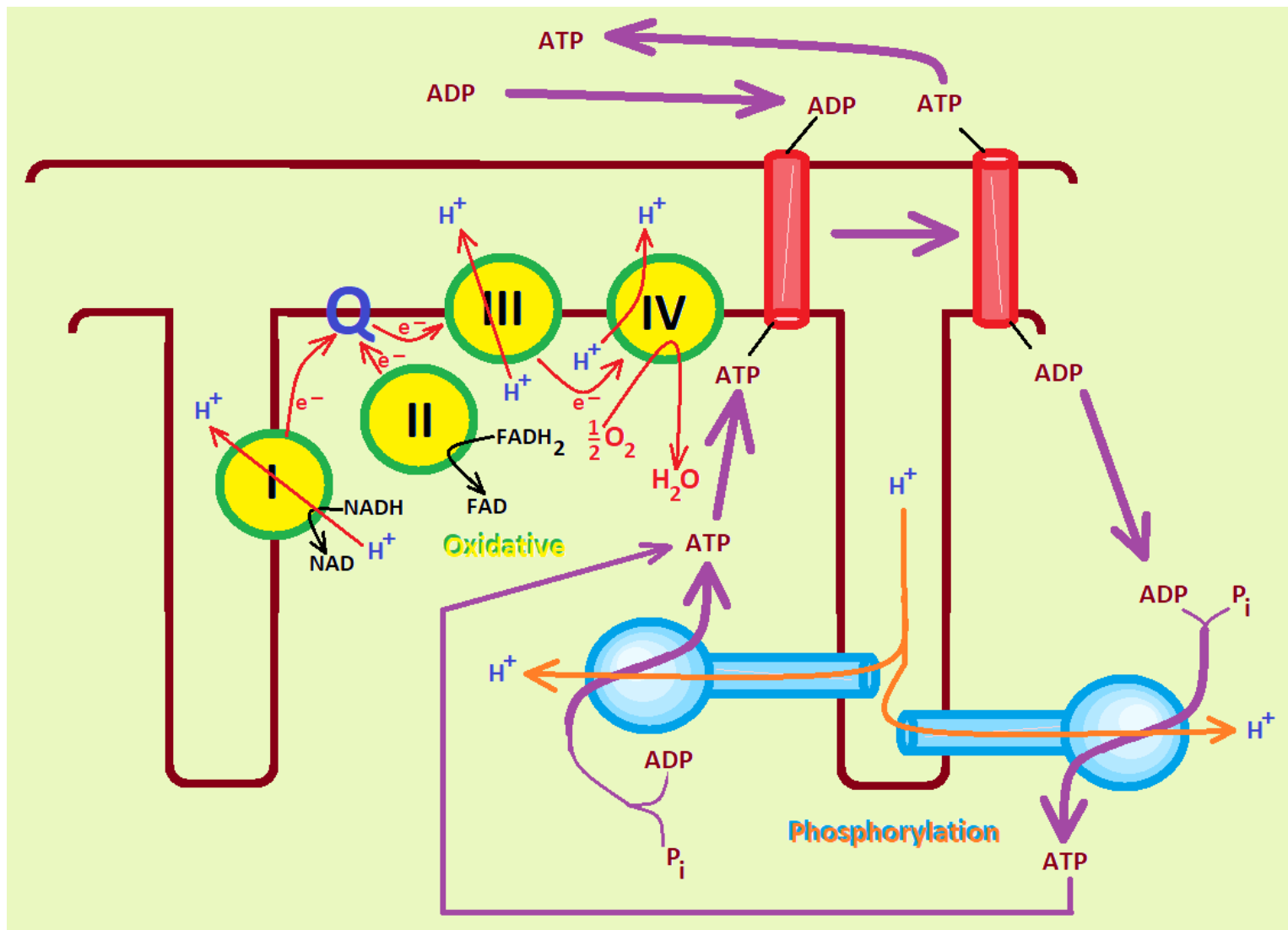
- NAD/NADH will NOT cross mito membranes – how get in there?
- Aspartate-Malate Shuttle!
- **In HEART and LIVER = 38 ATP from aerobic glycolysis and TCA**
- Note: OAA won't cross mito membrane, either

# ATP Synthesis by Tissue: Clarification



- Any other way? YES!!!!
- Glycerol phosphate shuttle in SKELETAL MUSCLE
- Has an effect on ATP production because NADH = 3 ATP and FADH<sub>2</sub> = 2 ATP
- Hence, 36 ATP produced in aerobic glycolysis and TCA in MUSCLE

# ET-OP

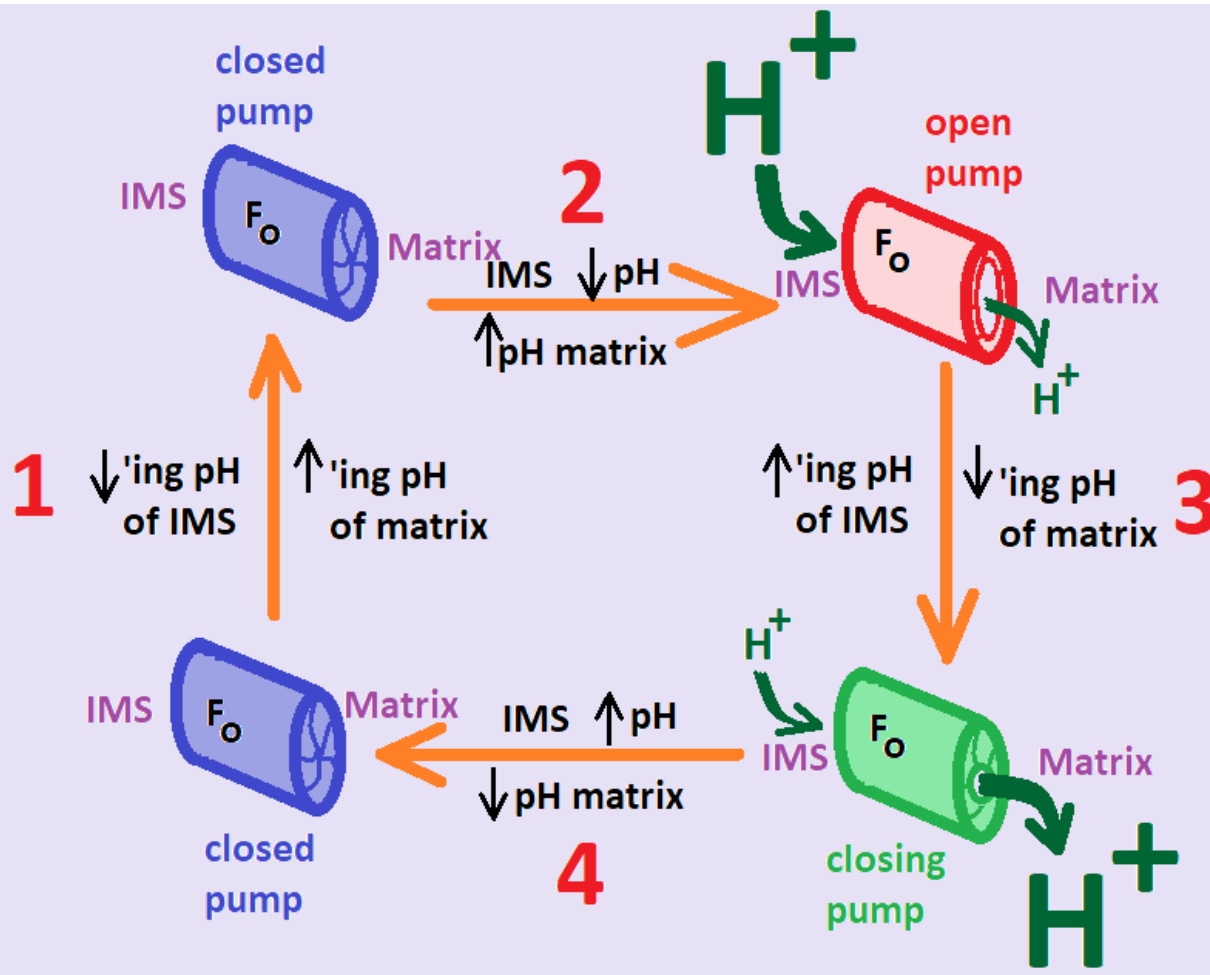


- In order to fully understand why
  - one mole of NADH is worth 3 ATP molecules and
  - why one mole of  $FADH_2$  is worth 2 molecules,
- we need to discuss how each is
  - oxidized by electron transport (ET)
  - to form ATP through oxidative phosphorylation (OP; the combination of the two is called ET-OP)

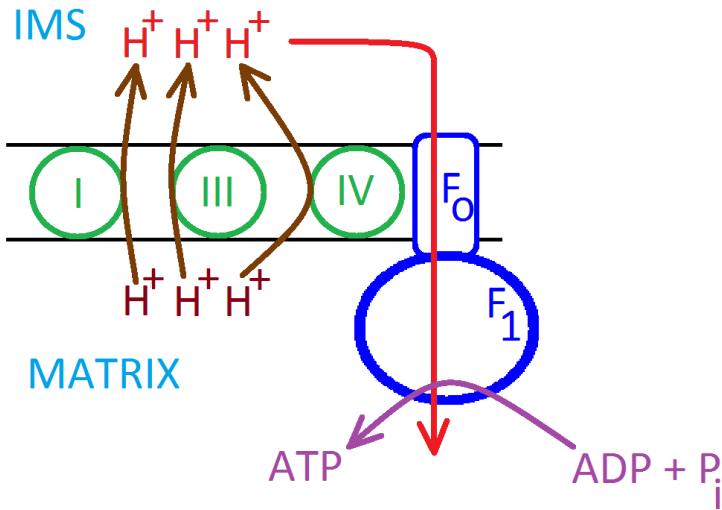


# Proton Pump Functioning

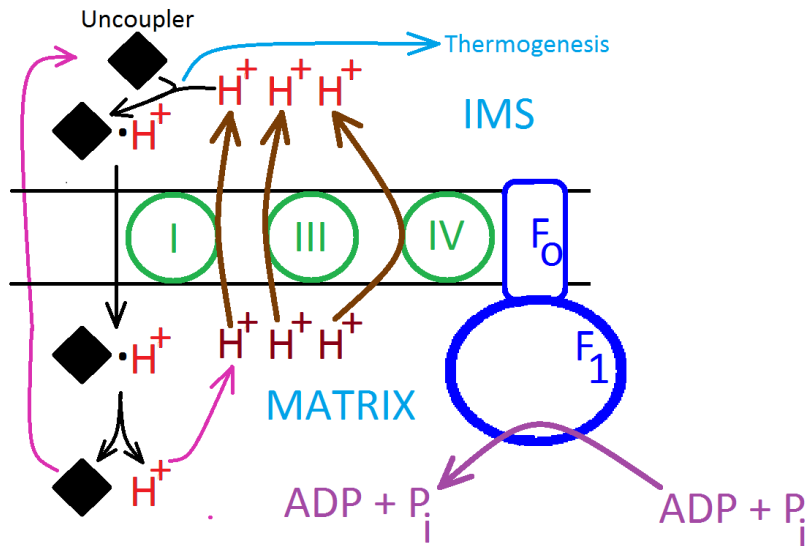
1.  $H^+$  moving out of matrix at I, III, IV
2. pH gradient established –  $F_o$  opens and  $H^+$  run through to drive ATP'ase
3.  $[H^+]$  in IMS decreasing to point of maximal flow into matrix
4.  $H^+$  being “recycled” and re-starting cycle –  $F_o$  fully closed



# Application of Proton Pump in ETOP -- WAT



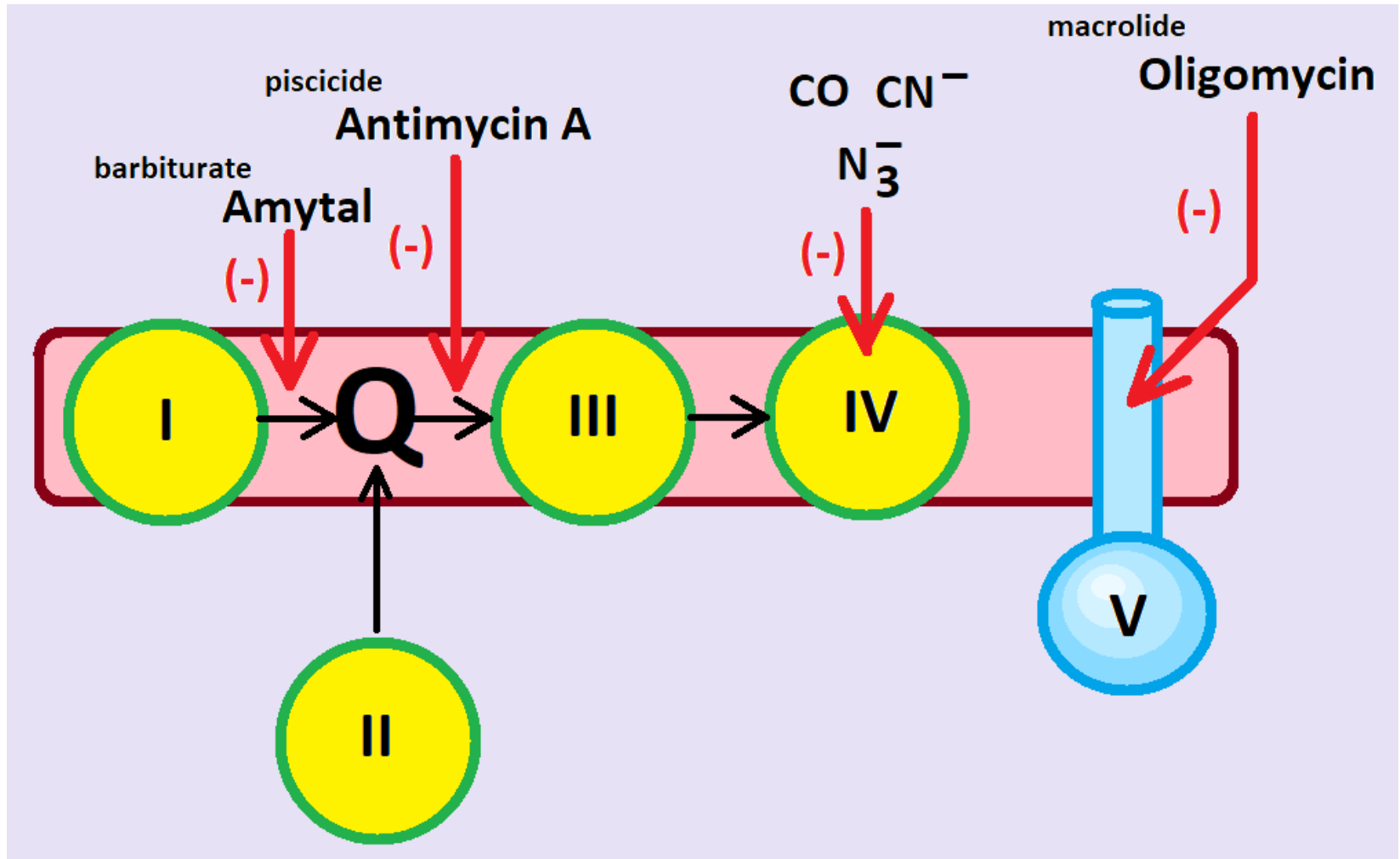
- White Adipose Tissue
- Not as many mito



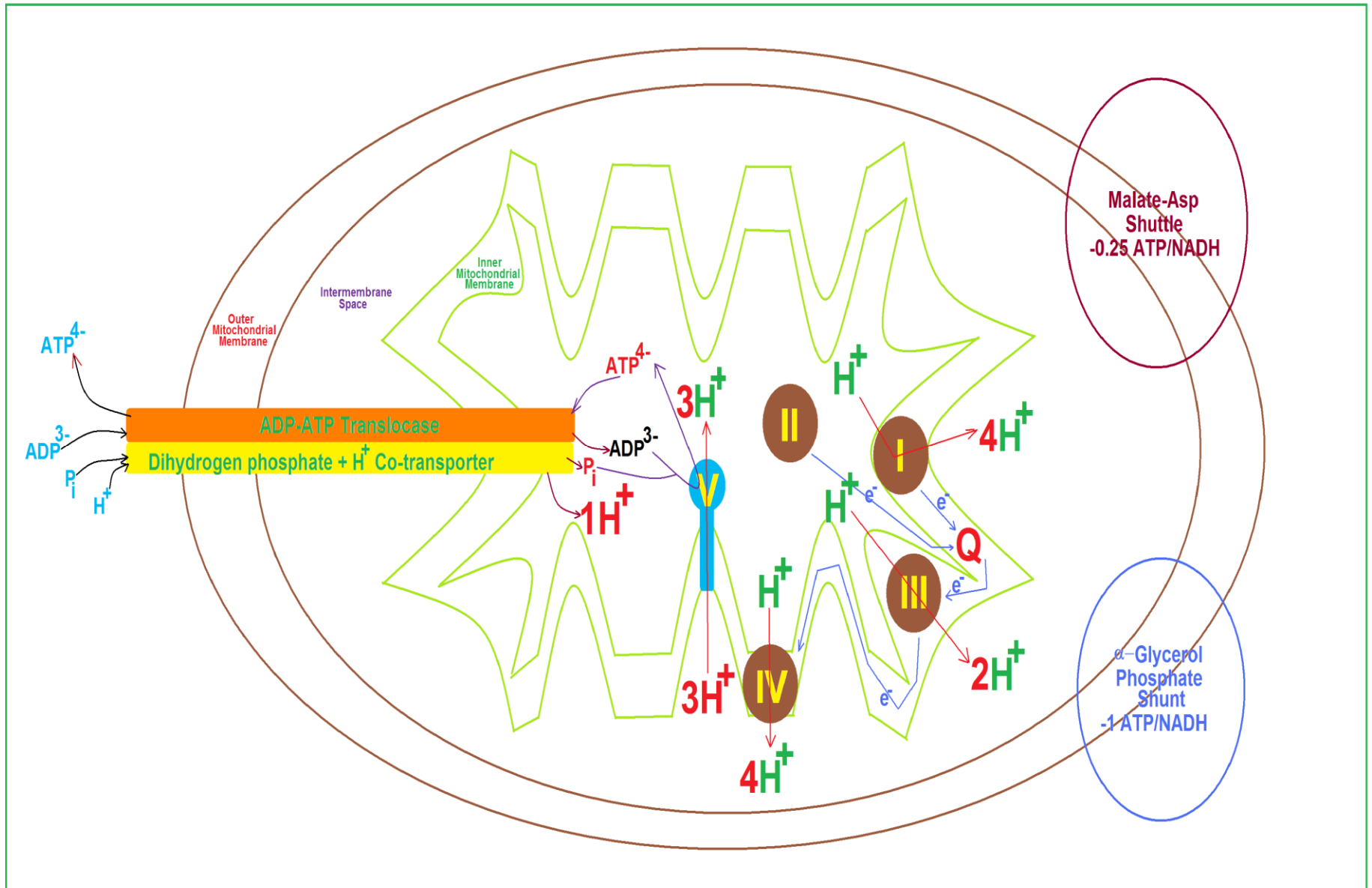
- Brown Adipose Tissue
- Lots of Mito
- Thermogenesis
- T<sub>4</sub> and T<sub>3</sub> regulate
- Fatty Acids act as uncouplers
- NE regulates FA release in BAT

# Application of Proton Pump in ETOP -- BAT

# ET-OP Inhibition



# ATP Fine Tune



# NADH: through ETS

- ADP-ATP Translocase coupled with Dihydrogen phosphate/Proton Co-Transporter
- Complex 1 requires 4 protons to pass on electron to Complex III
- Complex III requires 2 protons to pass on electron to Complex IV
- Complex IV requires 4 protons to reduce molecular oxygen to water
- 10 protons exported per NADH to make an ATP

# FADH<sub>2</sub>: through ETS

- ADP-ATP Translocase coupled with Dihydrogen phosphate/Proton Co-Transporter
- Complex III requires 2 protons to pass on electron to Complex IV
- Complex IV requires 4 protons to reduce molecular oxygen to water
- 6 protons exported per FADH<sub>2</sub> to make an ATP

# Complex V and Co-Transporter

- 3 protons required to turn on  $F_0$
- 1 proton co-transported with  $P_i$
- 4 protons imported to synthesize 1 ATP molecule

## Currently Accepted Stoichiometry

$$\frac{\# \text{ ATP}}{1 \text{ NADH}} = \frac{\frac{10 \text{ protons}}{\text{NADH}}}{\frac{4 \text{ protons}}{\text{ATP}}} = 2.5 \frac{\text{ATP}}{\text{NADH}} \qquad \frac{\# \text{ ATP}}{1 \text{ FADH}_2} = \frac{\frac{6 \text{ protons}}{\text{FADH}_2}}{\frac{4 \text{ protons}}{\text{ATP}}} = 1.5 \frac{\text{ATP}}{\text{FADH}_2}$$

# ATP Fine Tune – Part 2

## **Type I Muscle Fibers**

- Red Fibers
- Slow Twitch
- For Endurance
- Small Diameter
- Aerobic
- Lots of Mitochondria
- Malate-Aspartate Shuttle
- Liver, Heart, Kidney
- Back Muscles, too

## **Type II Muscle Fibers**

- White Fibers
- Fast Twitch
- For Explosive Bursts of Power
- Large Diameter
- Anaerobic
- Fewer Mitochondria
- $\alpha$ -Glycerol Phosphate Shunt
- Skeletal Muscle, Brain
- Digits and Extraocular Muscles
- Store Glycogen

# Amino Acid Metabolism

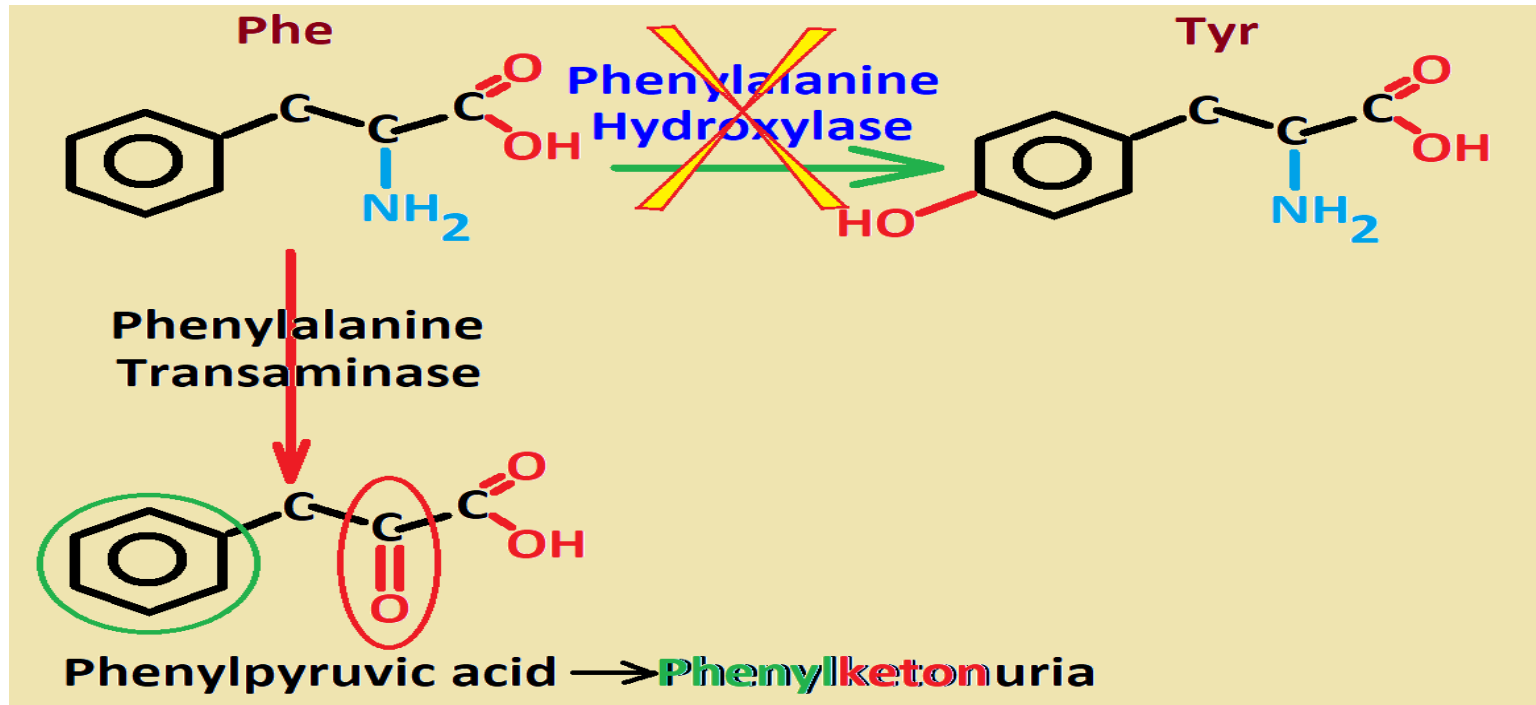
## Part II

### A Narrow View of [Semi-]Essential Amino Acid Metabolism – PVT TIM HALL

- Positively charged Amino Acids: Histidine (his), Arginine (arg), Lysine (lys)
- Aromatic Amino Acids: Phenylalanine (phe), Tryptophane (trp)
- BCAA: Valine (val), Isoleucine (ile or ileu), Leucine (leu)
- Neutral Amino Acids: Methionine (met), Threonine (thr)
- \*semi-essential
- We'll examine as groups where possible

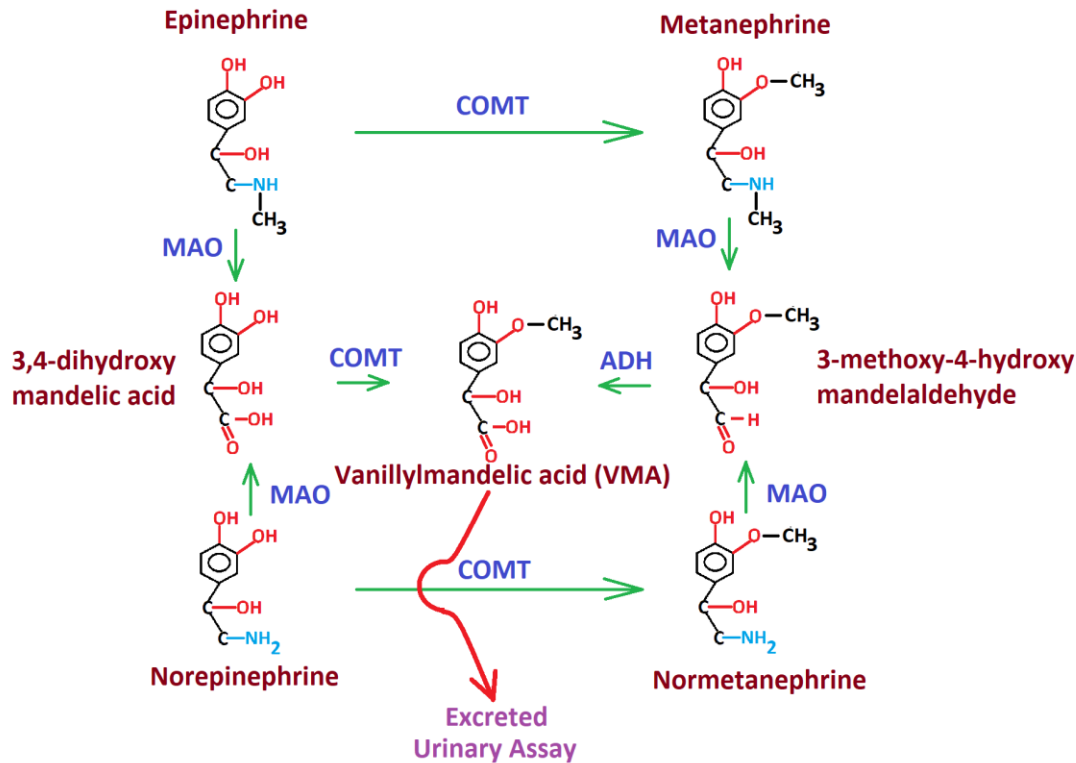
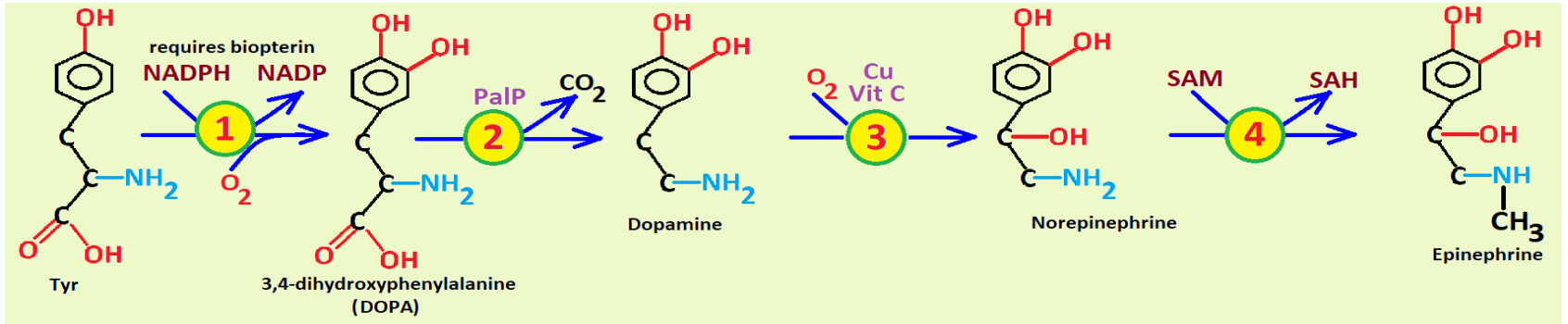


# Inborn Error of Metabolism -- PKU



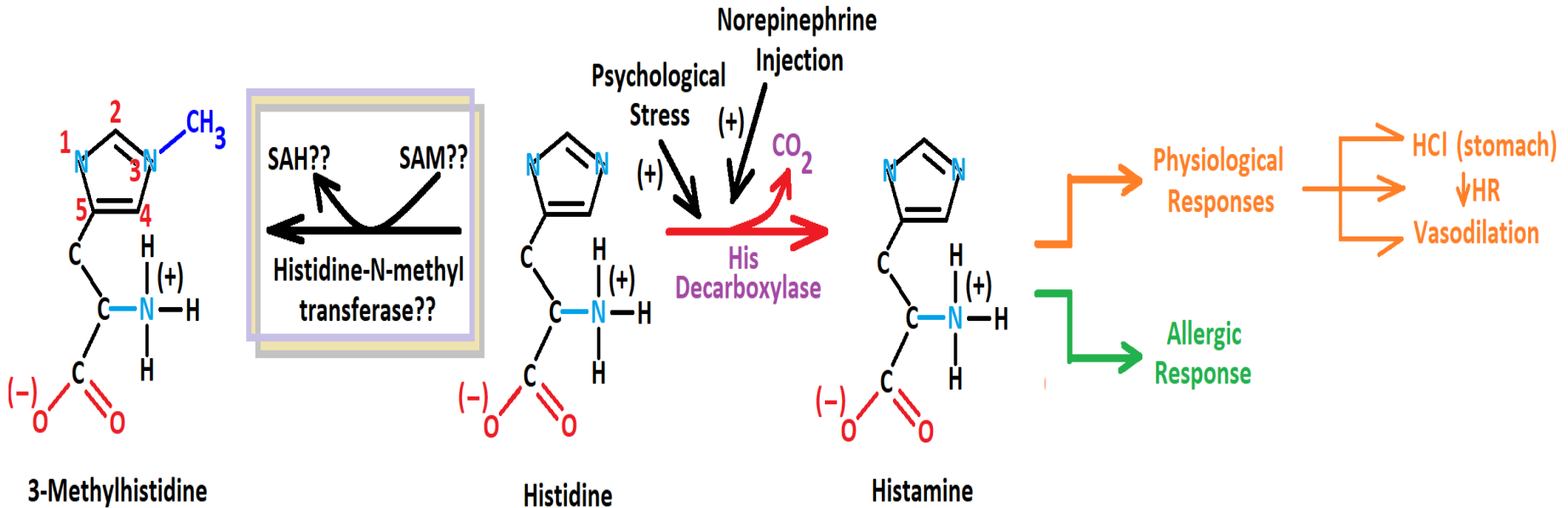
- Phenylketonuria
- A cause of mental retardation
- Tend to be blonde, blue-eyed and fair skin – WHY???
- PIGMENT!
- Which amino acid becomes essential, then?

# Catecholamines: Anabolism



# Catecholamines: Catabolism

# Histidine → Histamine



## 3-methylhistidine

Catabolite of muscle contraction

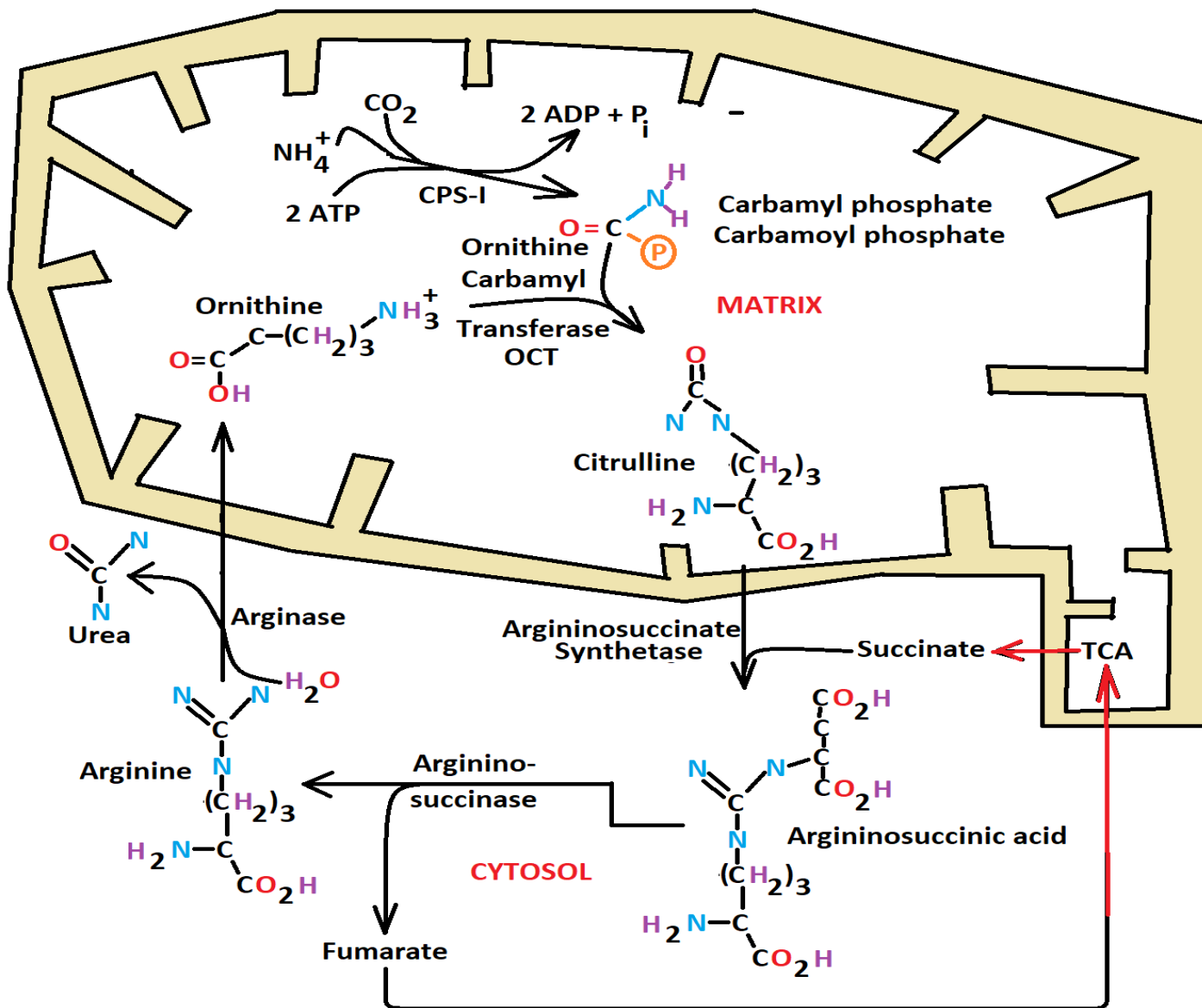
NOT further metabolized but excreted unchanged

Useful in determining muscle protein turnover ACCURATELY

# Significance of Lysine

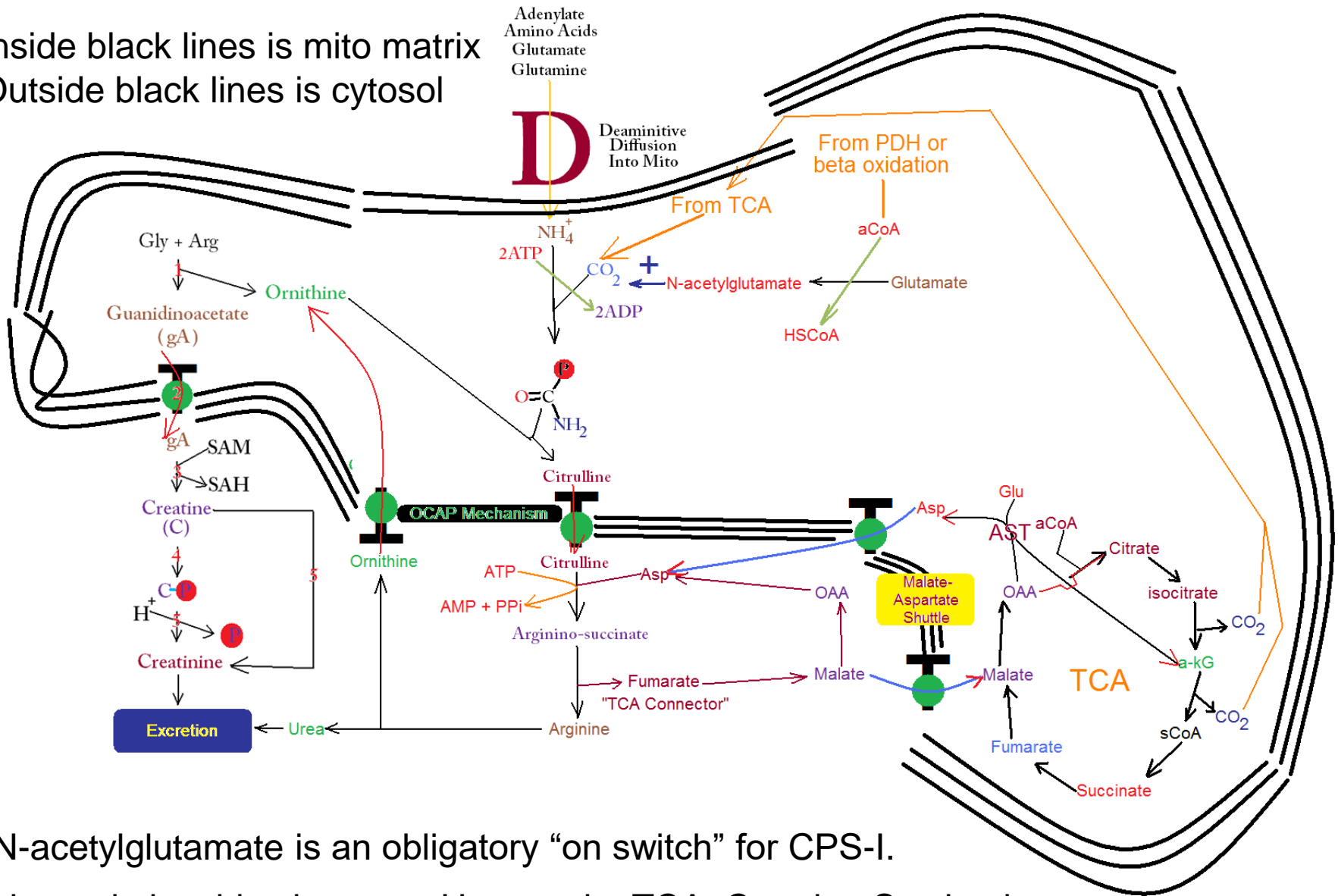
- Provides crosslinking in collagen
- Provides crosslinking in elastin
- When crosslinking is inhibited
  - in severe Cu deficiency,
  - after ingesting sweet pea toxin [ $\beta$ -aminopropionitrile]
- This causes **lathyrism**
- There is increased solubility of the collagen
- Increased rigidity of elastin
- Both cause death – generally due to aortic rupture from a lack of elasticity at the aortic root – an “aortic blow out” or aneurism

# Arginine and The Urea Cycle



# Creatine, Urea, TCA and Malate-Aspartate Shuttle Interconnections

Inside black lines is mito matrix  
Outside black lines is cytosol



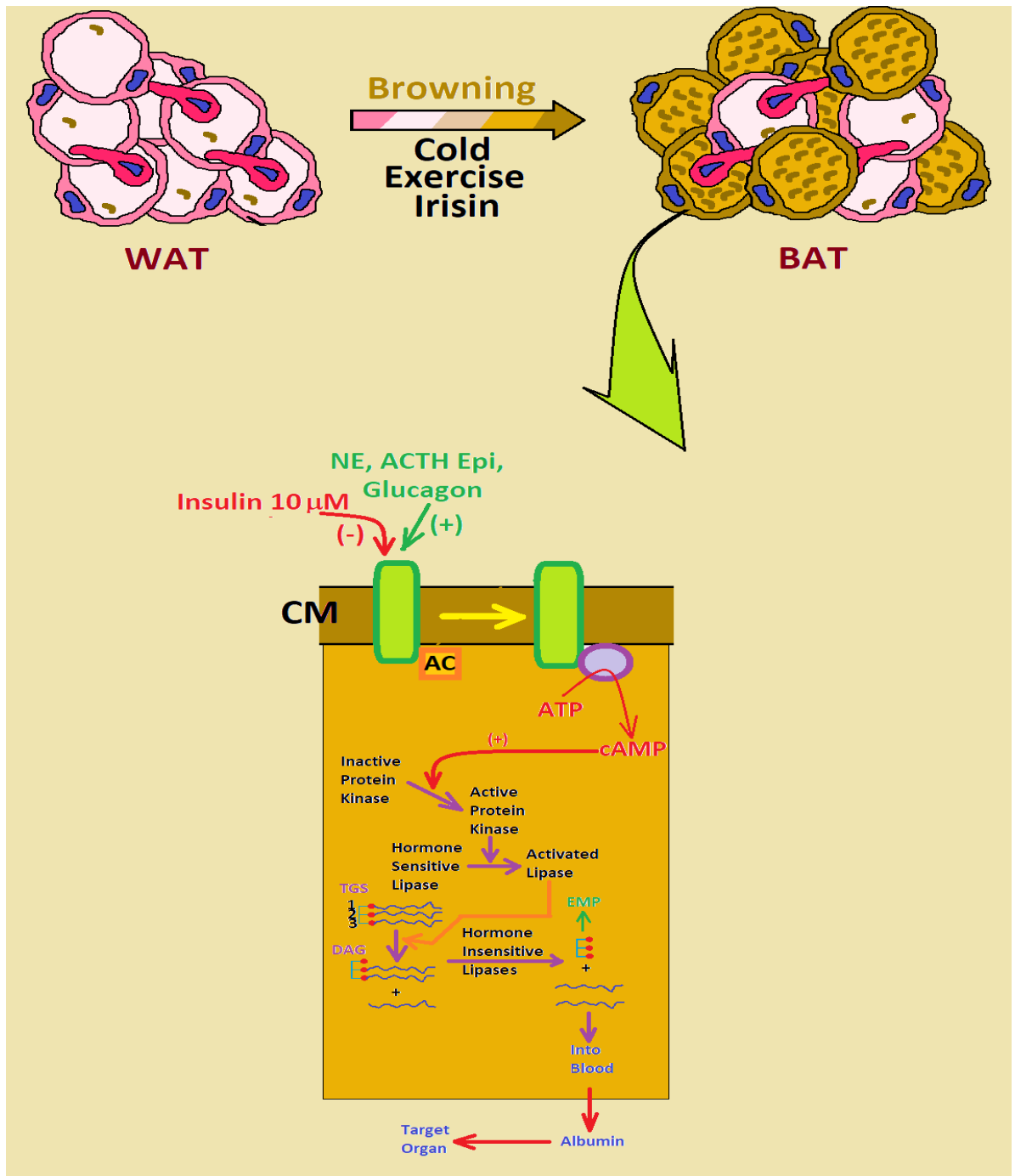
N-acetylglutamate is an obligatory "on switch" for CPS-I.

Note relationships between Urea cycle, TCA, Creatine Synthesis and Asp-Malate Shuttle.

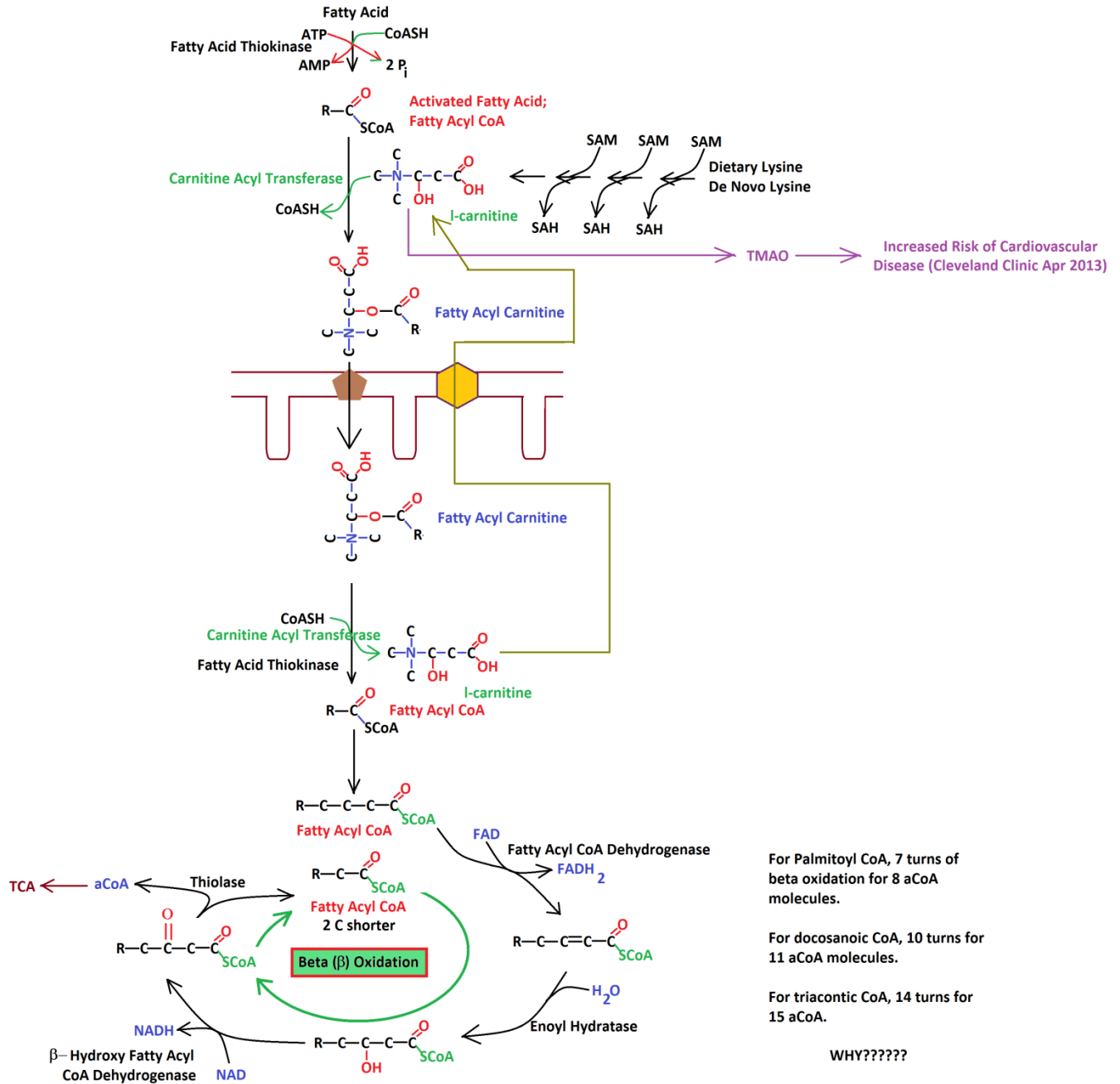
# Lipid Metabolism

## Part III

Glucagon effect minimal  
in humans



# β-Oxidation of Fatty Acids



For Palmitoyl CoA, 7 turns of beta oxidation for 8 aCoA molecules.

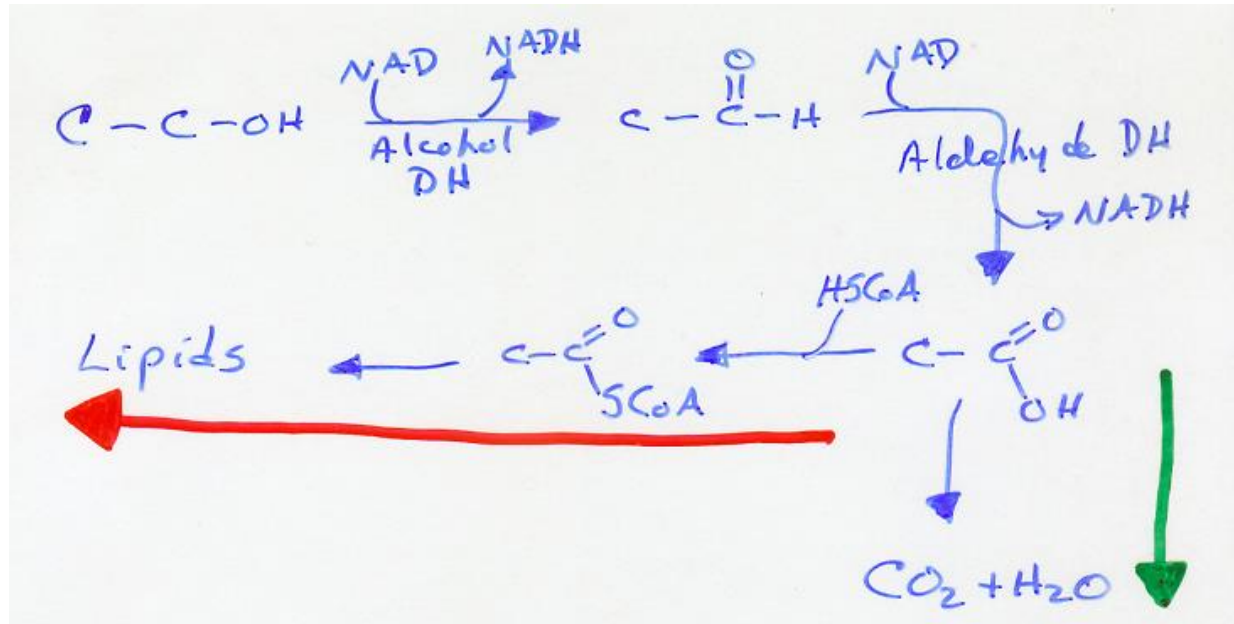
For docosanoic CoA, 10 turns for 11 aCoA molecules.

For triacontic CoA, 14 turns for 15 aCoA.

WHY???????

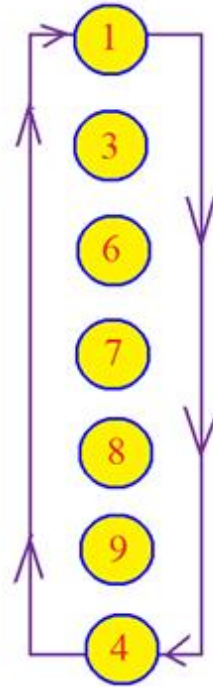
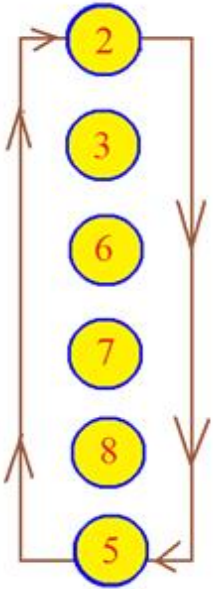
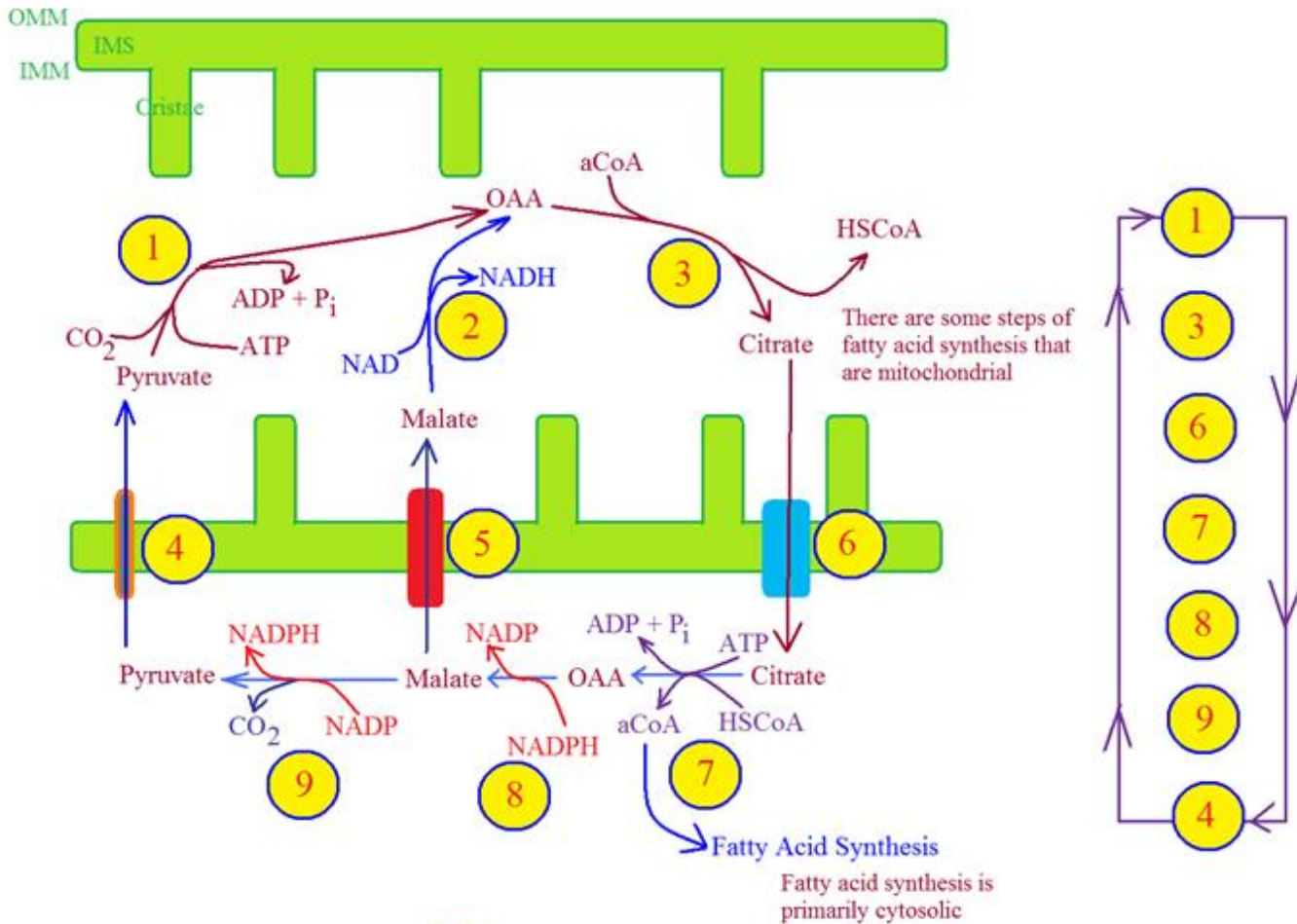


# Ethyl Alcohol Metabolism



- Makes mito an incredibly REDUCED environment
- With lots of  $\text{NADH}$ , this inhibits ALL  $\text{NAD}$ -requiring enzymes
- Therefore,  $\text{EtOH}$  is typically used in fat synthesis rather than in oxidation

# Fatty Acid Synthesis



1 Pyruvate carboxylase

2 MDH

3 Citrate synthetase

4 Pyruvate transporter

5 Malate- $\alpha$ kG Transporter

6 Citrate transporter

7 Citrate lyase

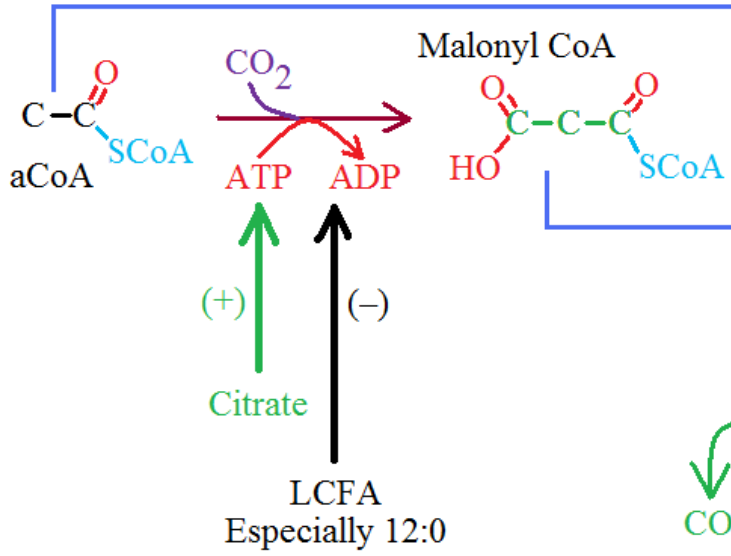
8 MDH

9 Malic Enzyme

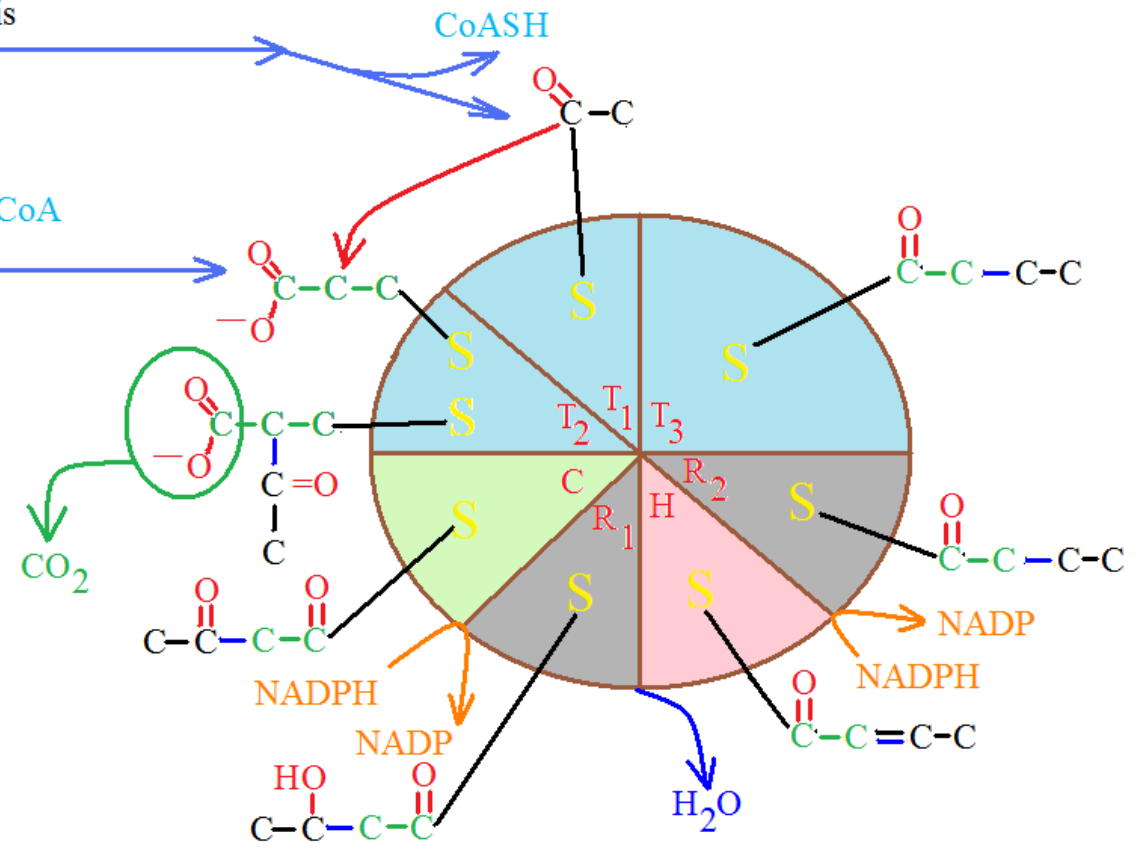
Malate dehydrogenase  
(OAA-decarboxylating;  
NADP; E.C. 1.1.1.40)

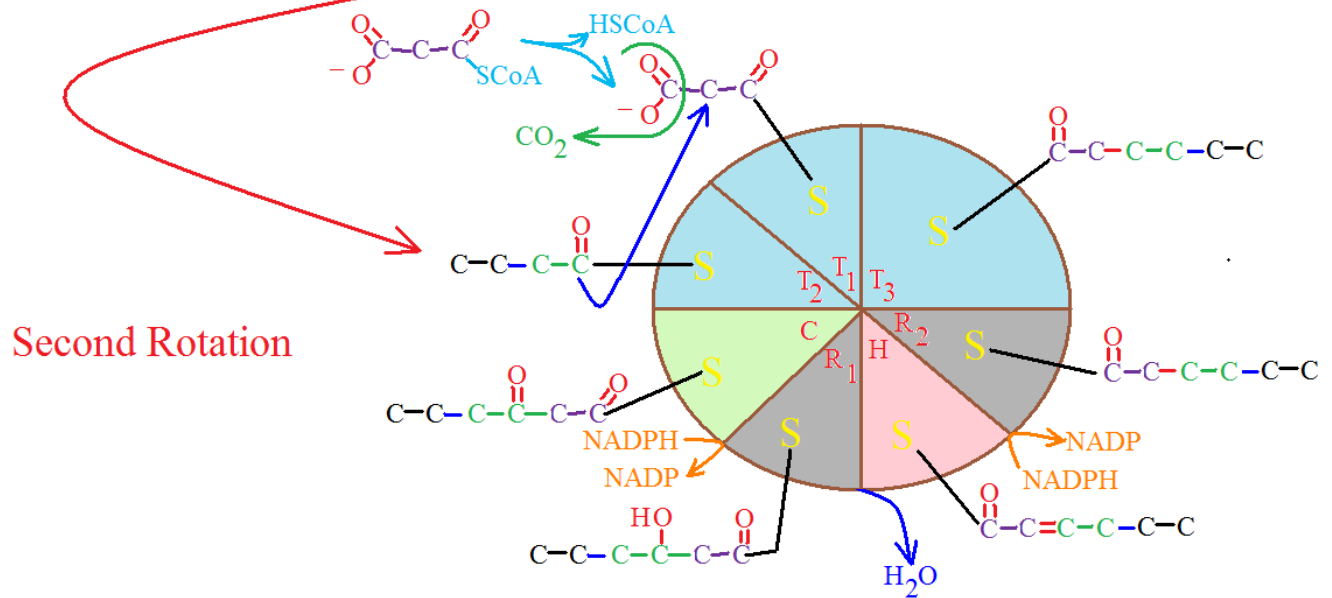
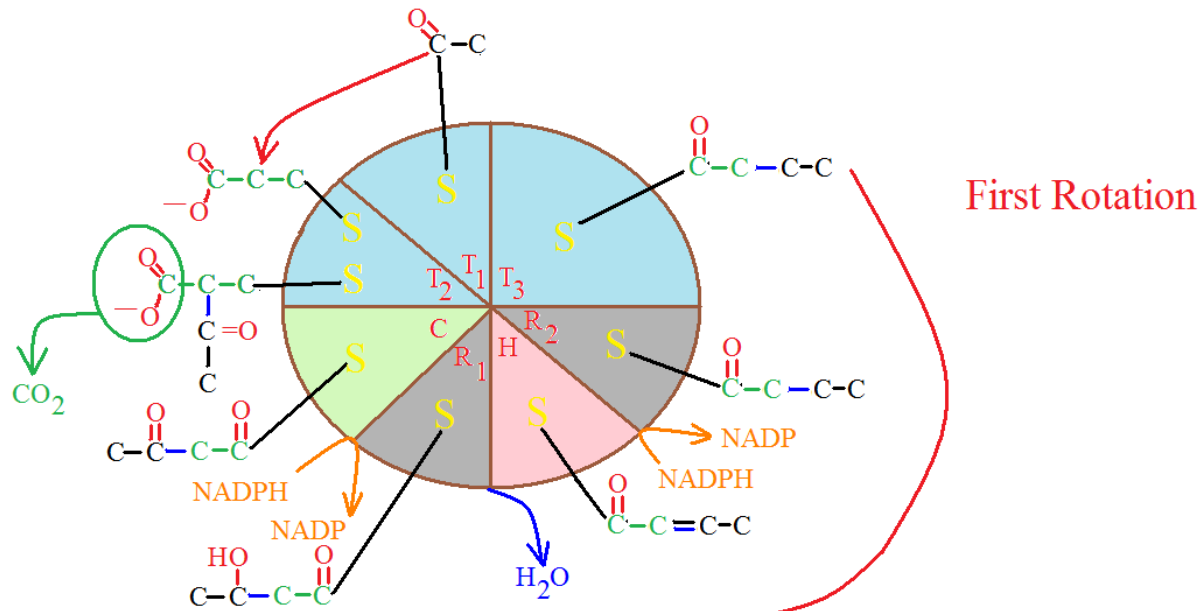
# Acetyl-CoA Carboxylase

RATE LIMITING Step in Fatty Acid Synthesis

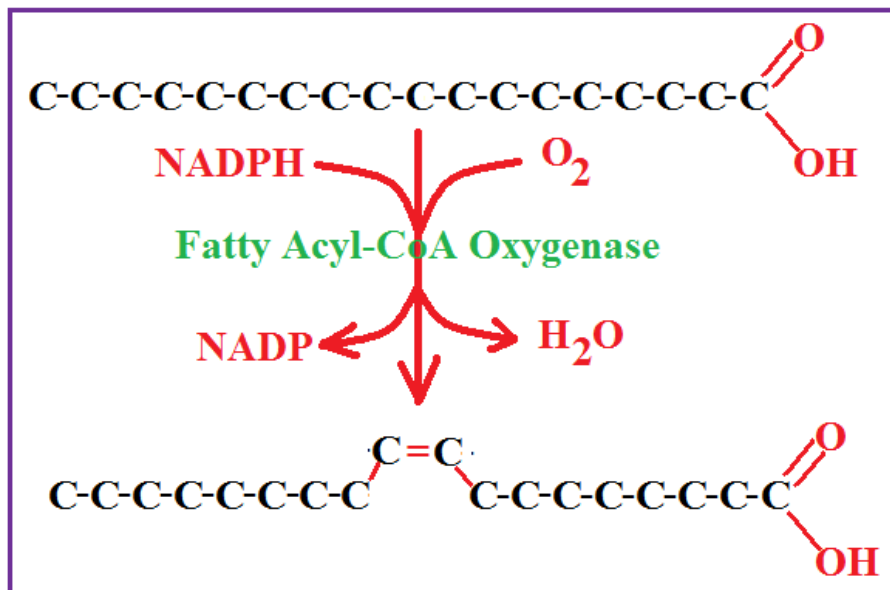


# Fatty Acid Synthetase: 1 Protein, 7 Activites





# 18:1, n-9 Biosynthesis

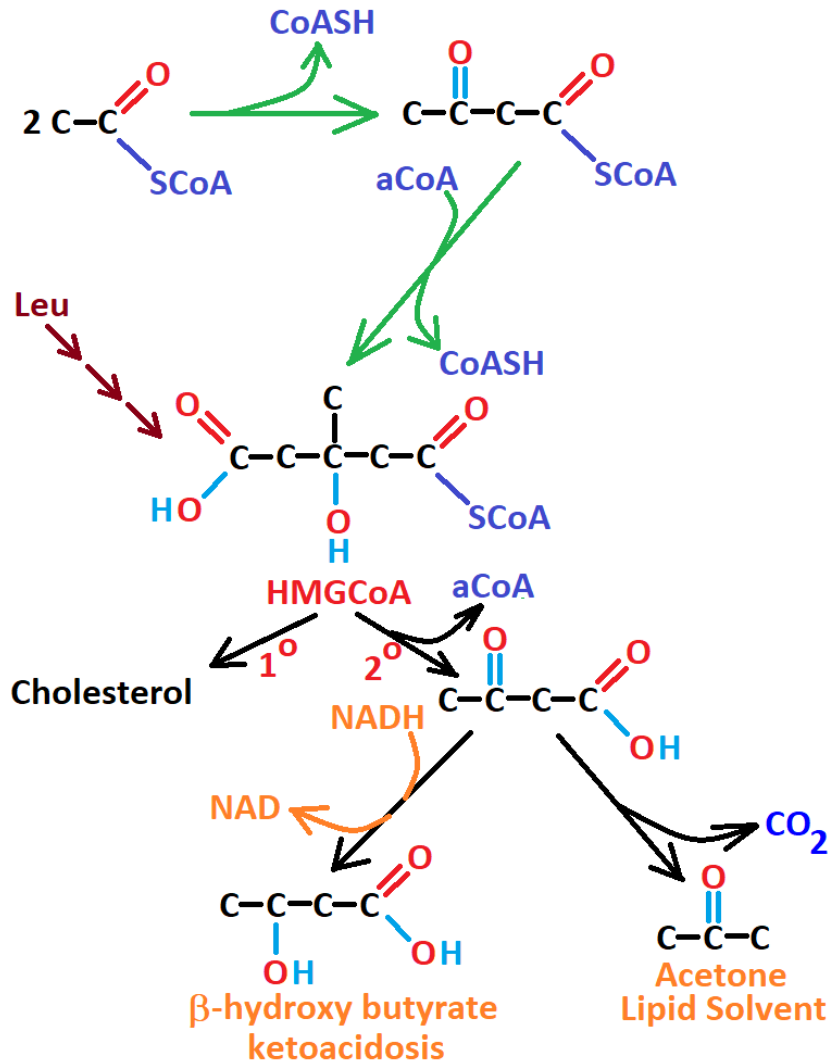


Elongation of 18:1, n-9 in vertebrates does not seem to occur commonly: mammals (excepting herbivores and non-seafood eating animals) lack the enzymes to unsaturate FA's beyond the #9 position; plants don't seem to elongate 18:1, n-3 to 20:5, n-3, although it seems that herbivores, et al, do.

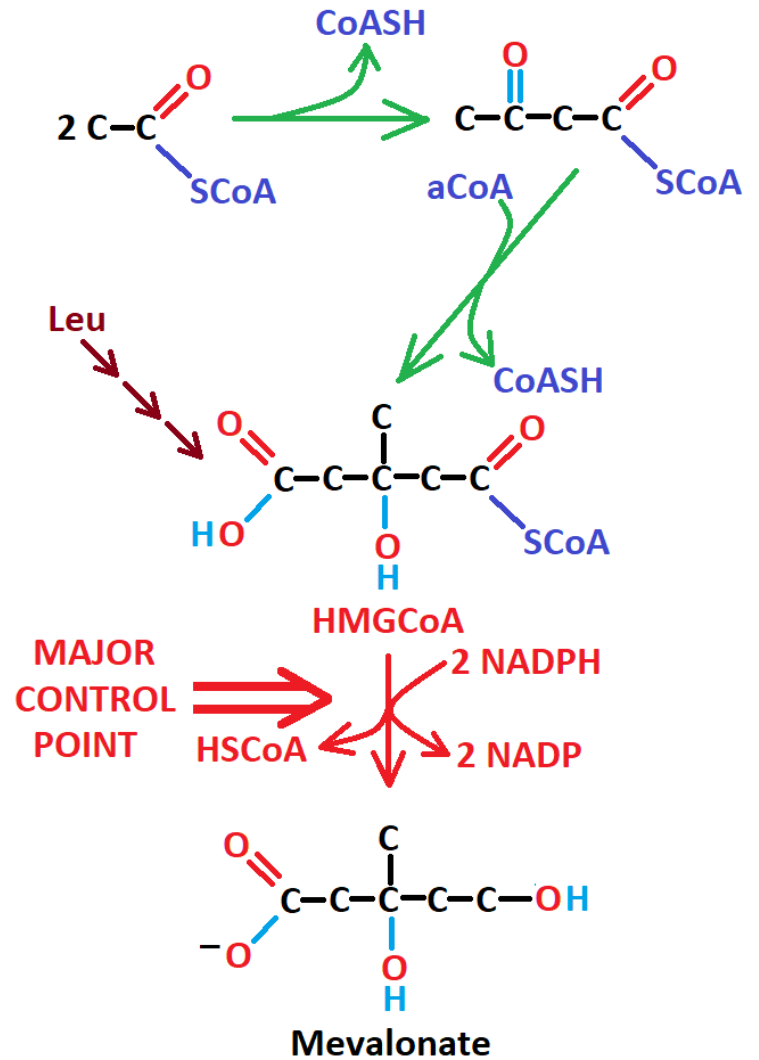
LOTS of confusing data on MUFA to PUFA (n-3) conversion

Elongation of 18:1, n-9 in plants DOES occur in the synthesis of ALA (18:3, n-3)

# Acetyl CoA

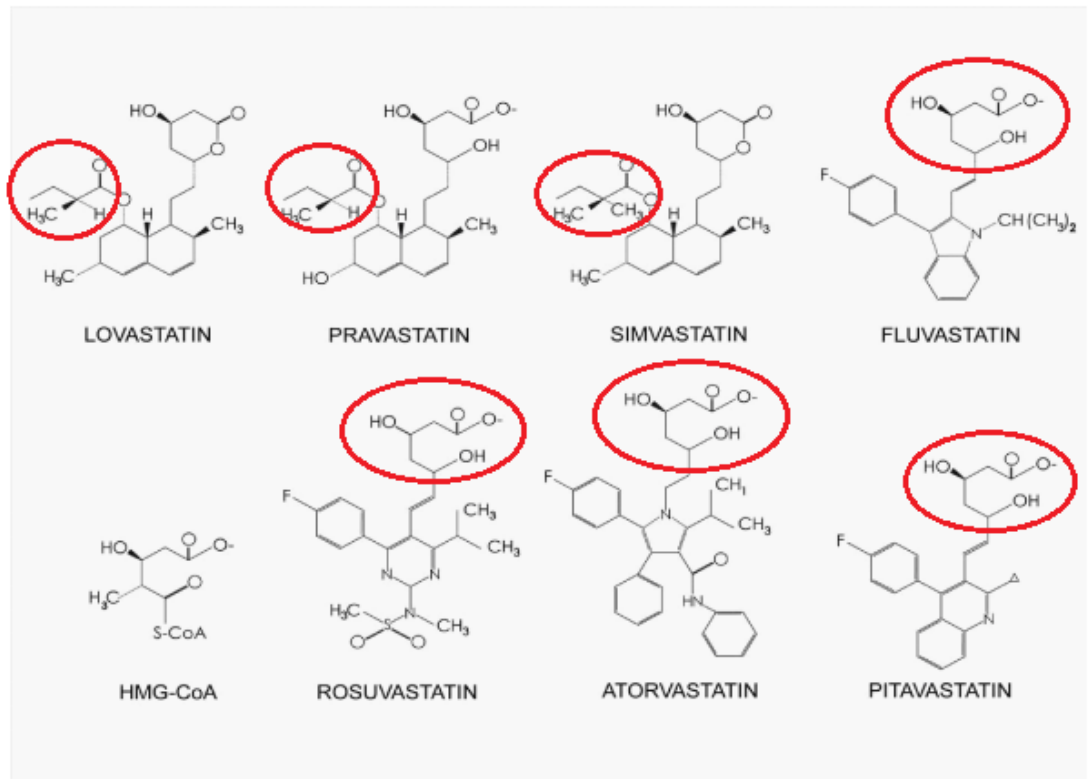
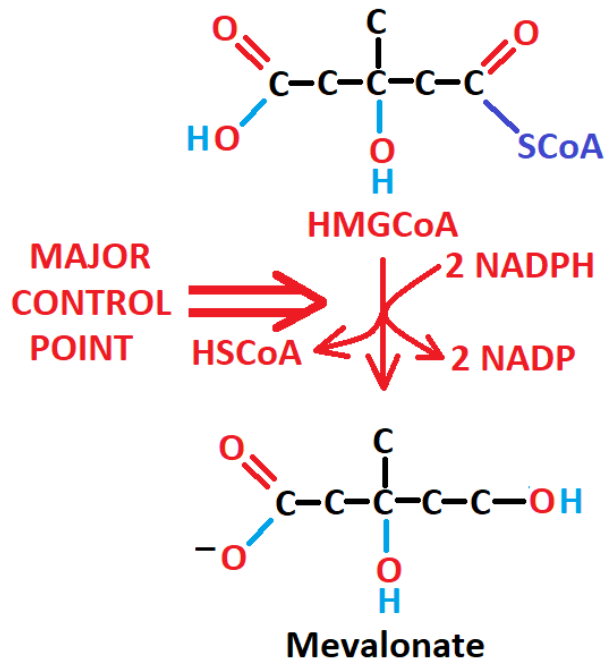
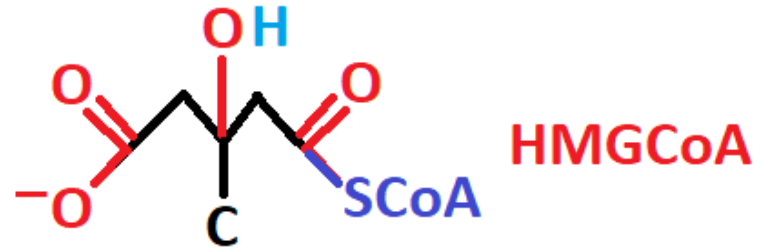


# Cholesterol Biosynthesis

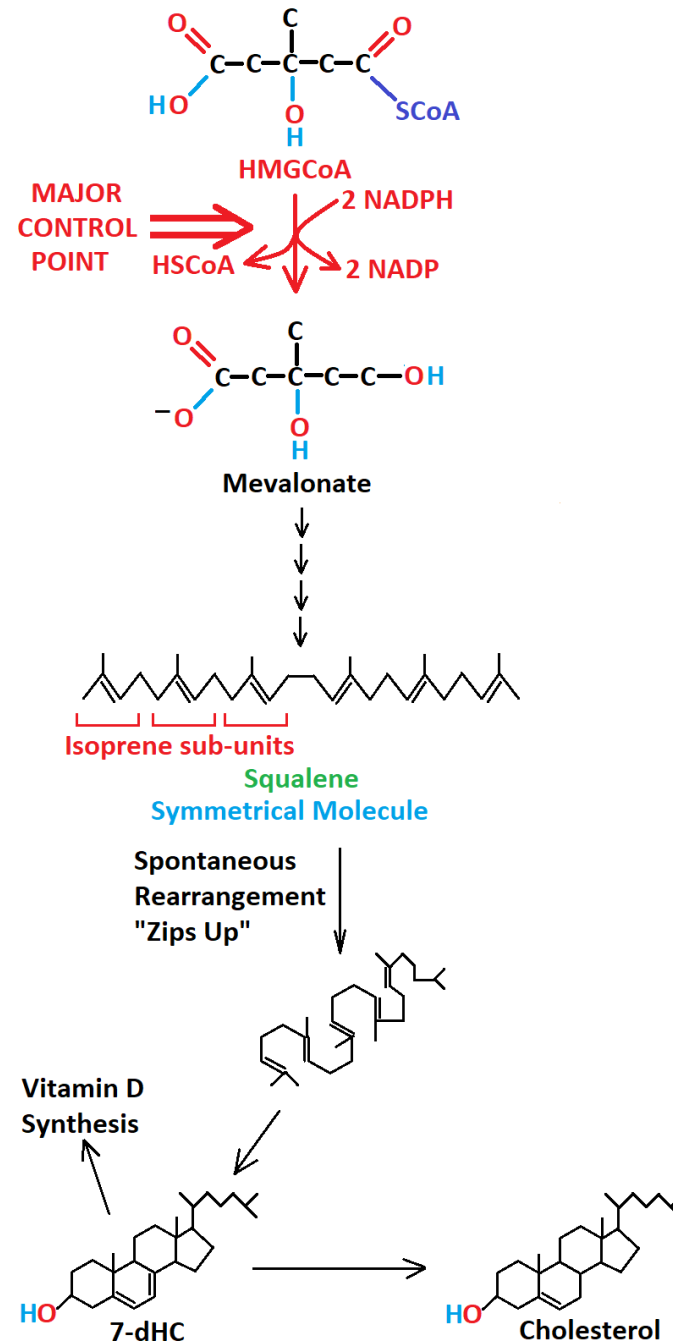


# HMGCoA Reductase and Inhibitors

## ( $\beta$ -hydroxy- $\beta$ -methyl glutaryl coenzyme A)



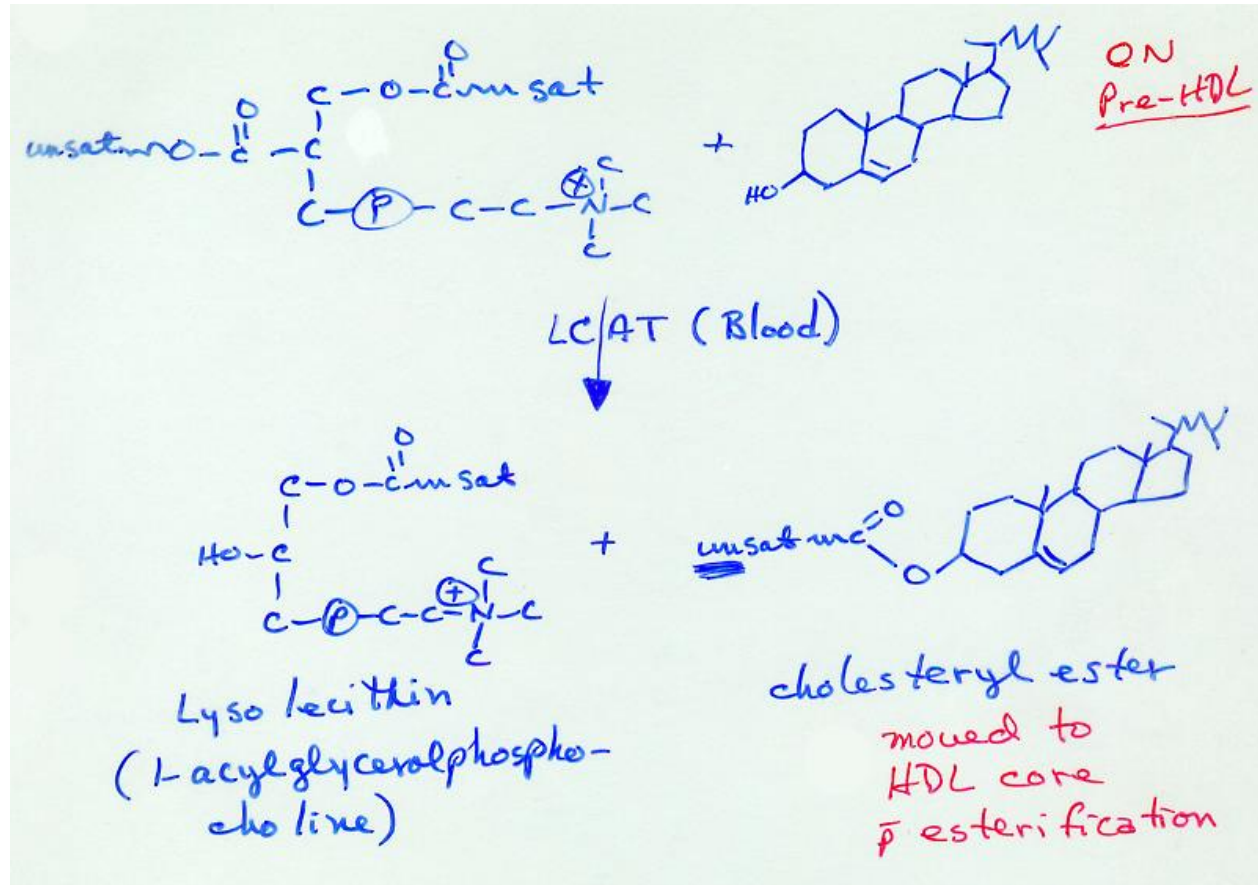
# Cholesterol Synthesis





# Lecithin-Cholesterol Acyl Transferase: "LCAT"

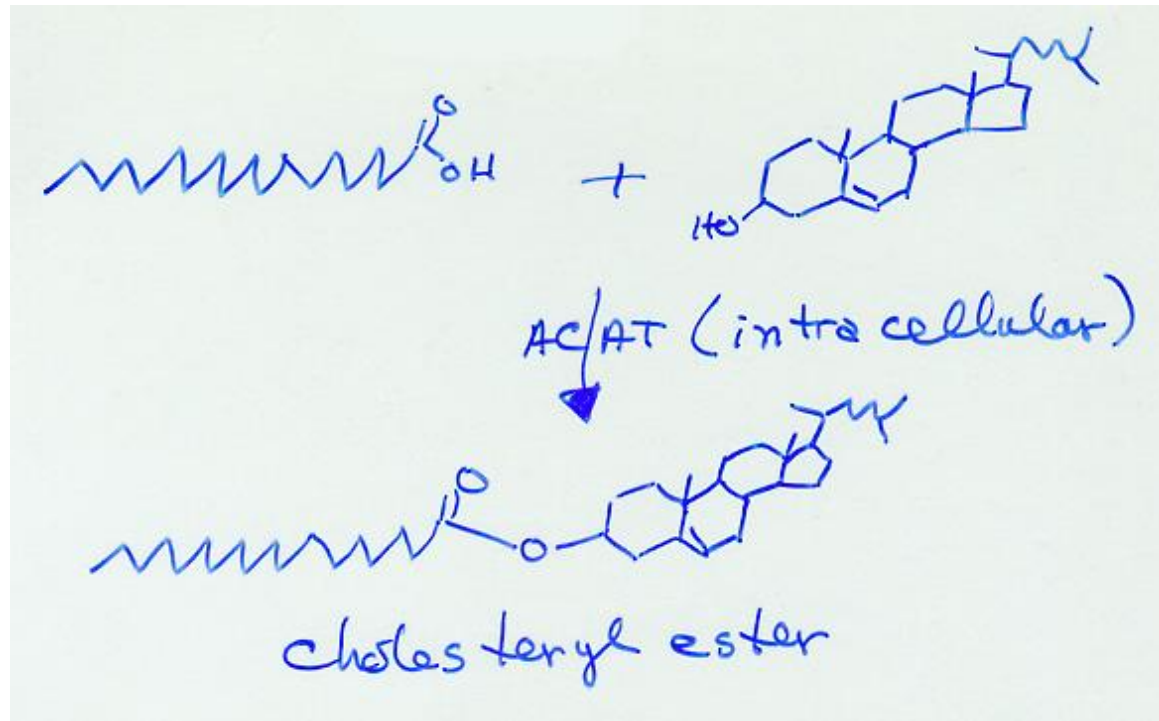
- Used in lipid TRANSPORT



cholesteryl ester biosynthesis;  
esters then transported into the core of a developing lipoprotein;  
makes HDL spherical; bound to HDL's and LDL's in the blood.

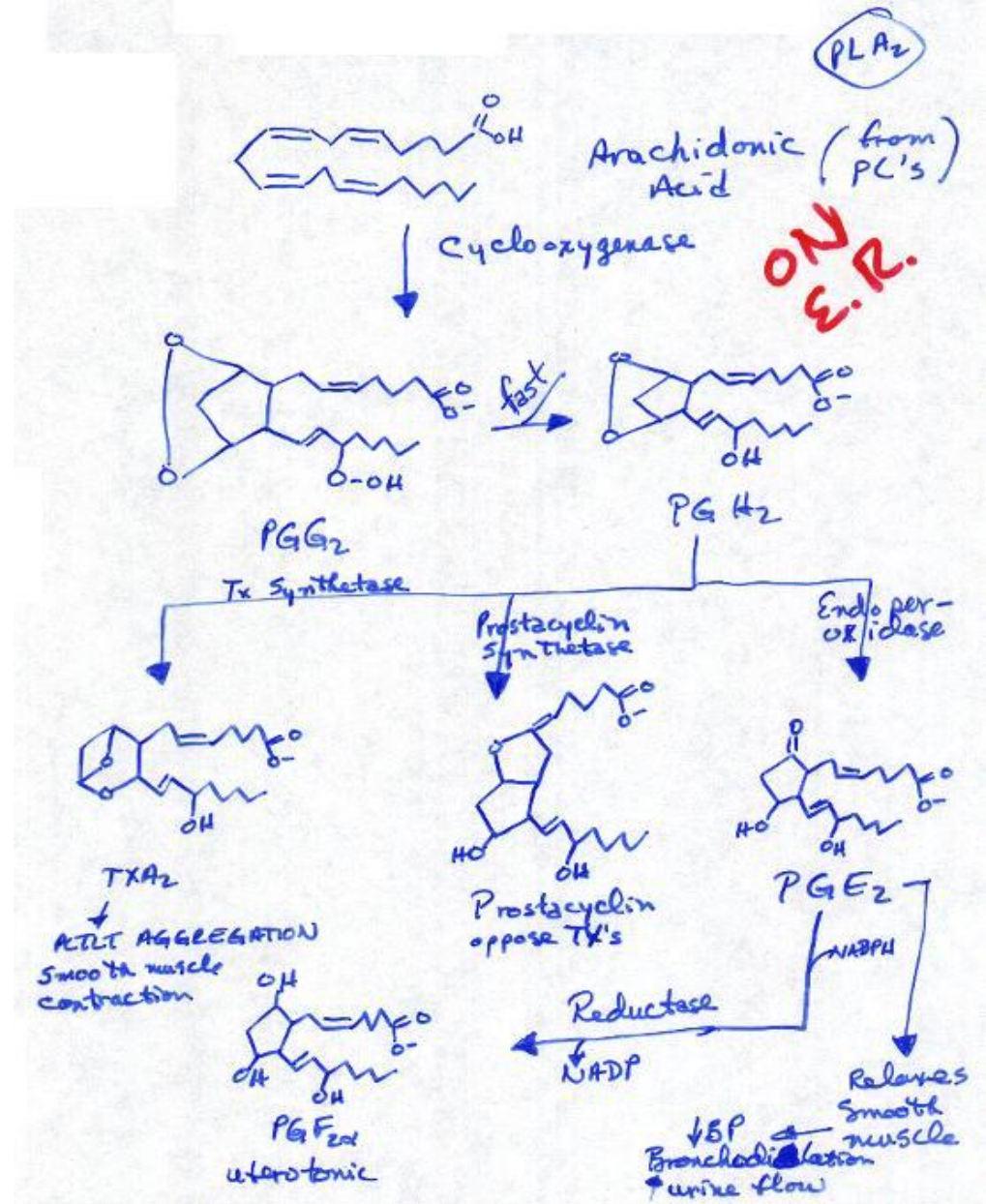
Acyl CoA: **C**holesterol **A**cy **T**ransferase: “**ACAT**”  
aka Sterol-O-Acyl Transferase (SOAT)

- Prepares cholesterol for intracellular STORAGE



- promotes accumulation of cholesterol ester in the fat droplets within cytosol;
- prevents toxic accumulation of free cholesterol in cell membranes
- significant role in foam cell generation and atherosclerosis by accumulating cholesteryl esters in macrophages and blood vessels

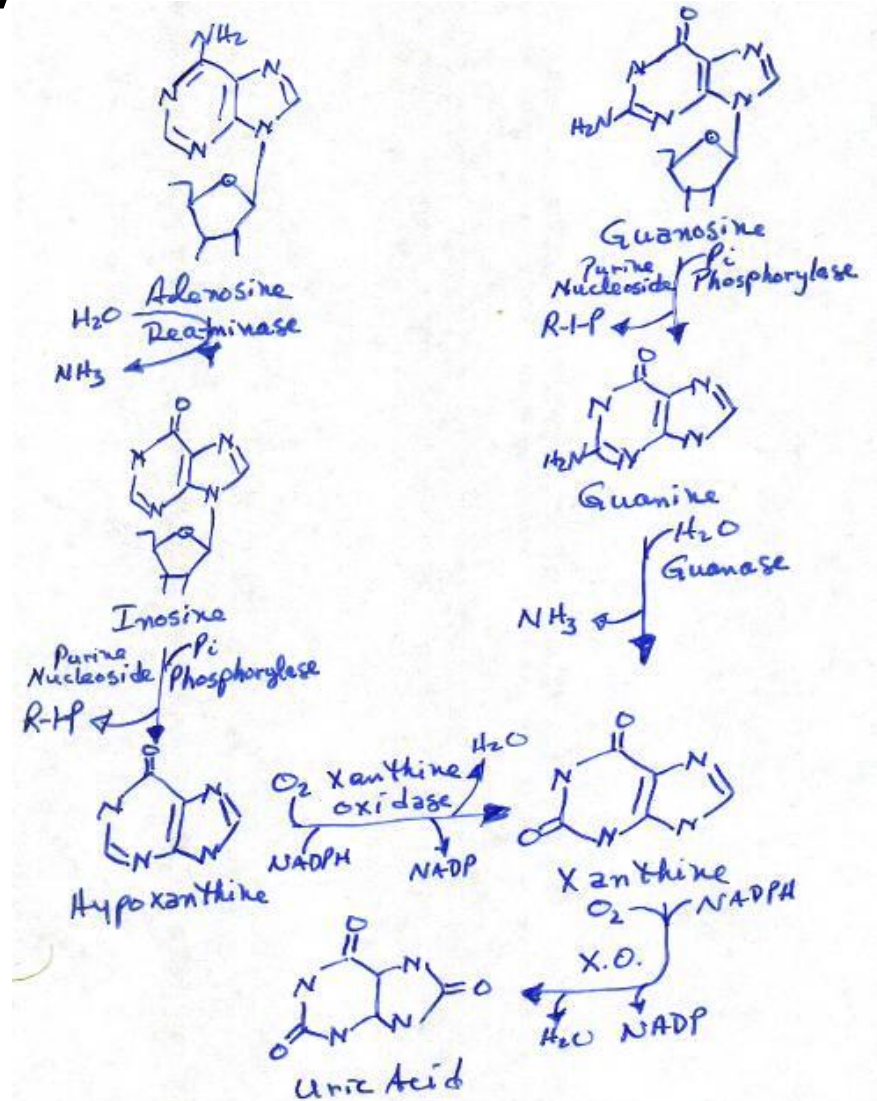
# Prostaglandins



# Nucleic Acid Metabolism

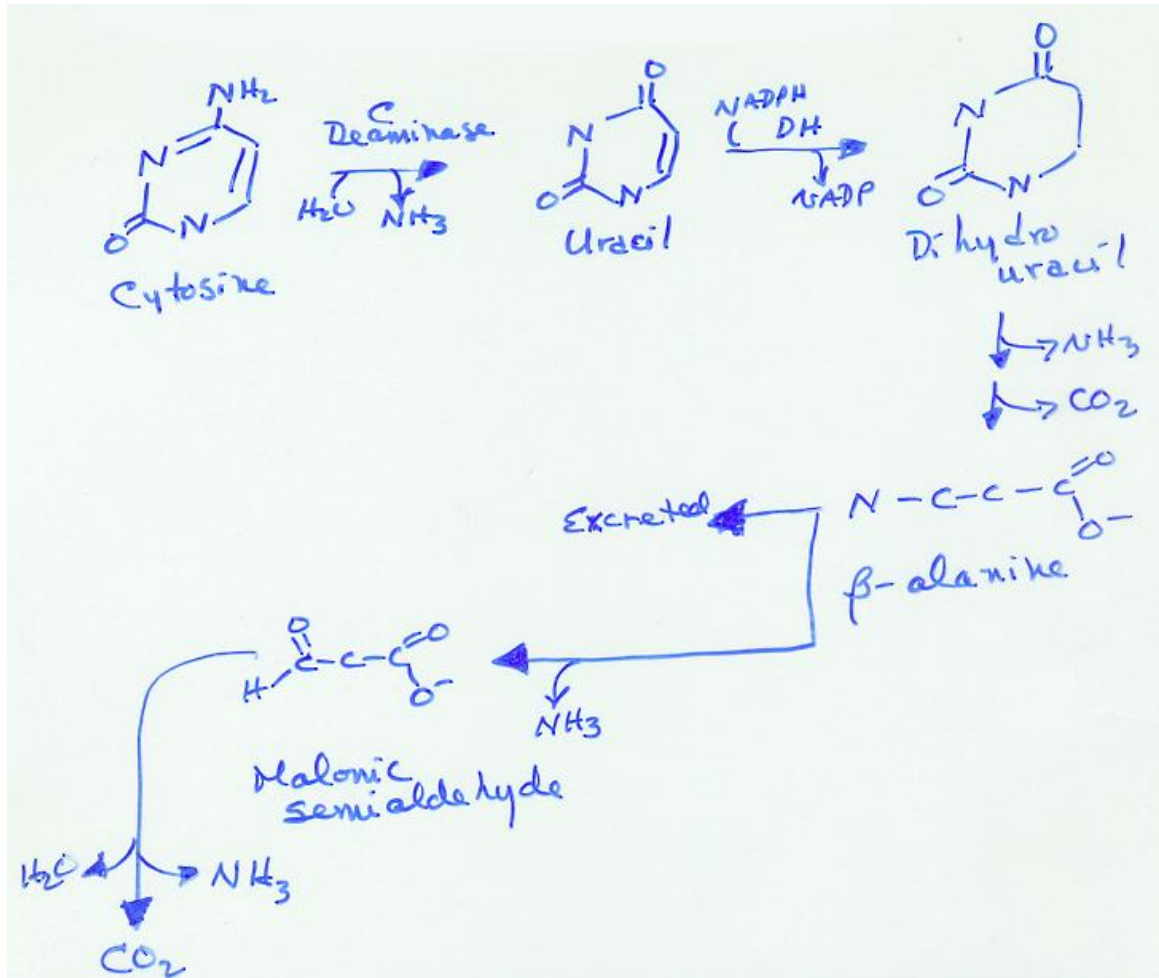
- Part IV

## Purines





# Pyrimidines

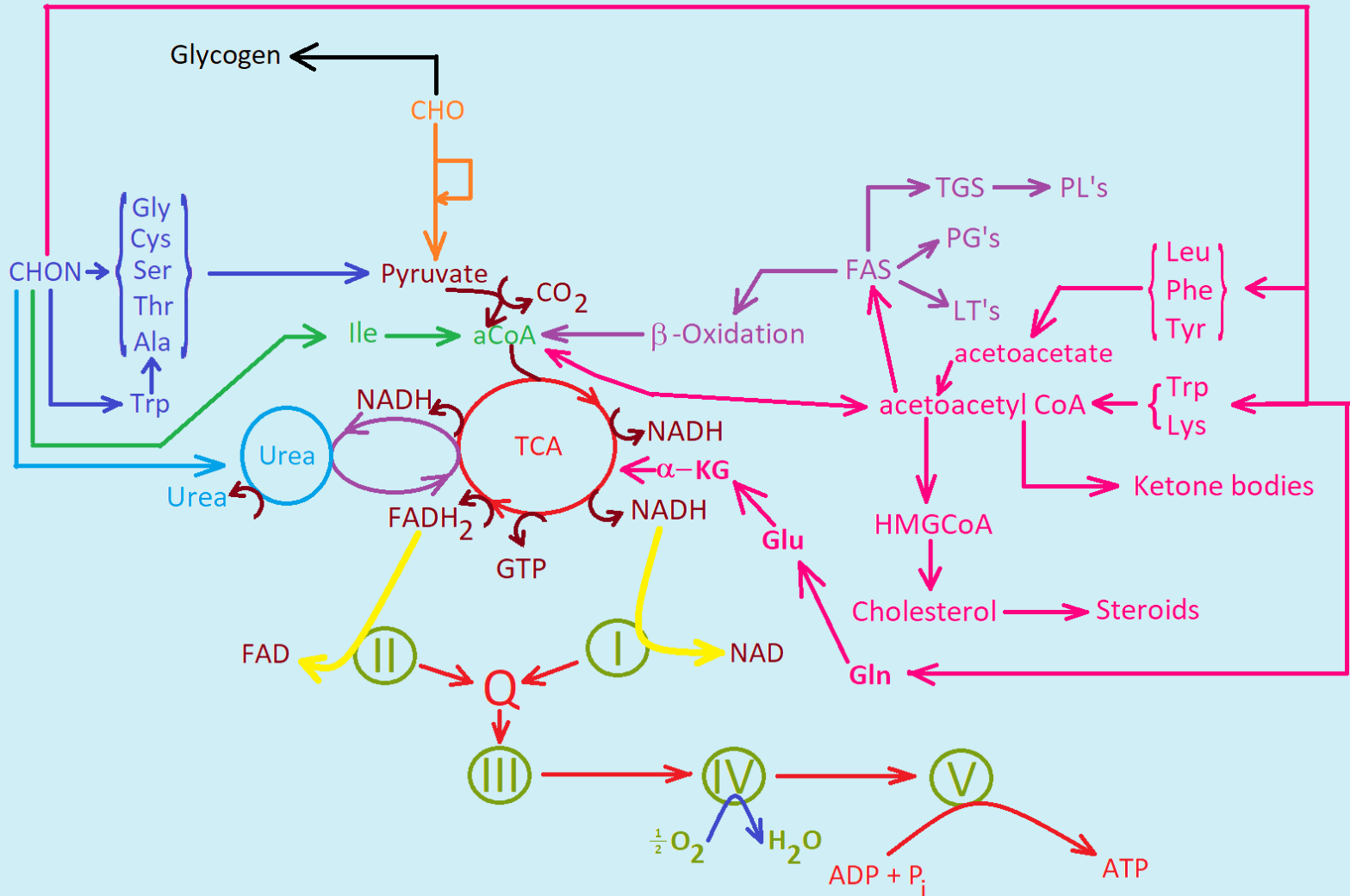


- Cytosine deaminase is highly elevated in some solid tumor cells
- Inhibition with tetahydrouridine improves therapy due to reduced drug degradation



# Integration of Metabolism

## SSDD



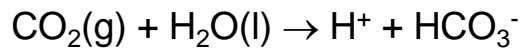
# Carbonic Acid-Bicarbonate Ion: Physiological Interactions

## Carbon dioxide: CO<sub>2</sub>

CO<sub>2</sub> is fairly soluble in water (more soluble in cold water like in cold soda; less soluble in warm water like in "flat" soda).

A saturated solution at 1 atm and 25° C is approximately 0.033M. At equilibrium only 0.17% of dissolved CO<sub>2</sub> is in the form of carbonic acid (H<sub>2</sub>CO<sub>3</sub>).

An aqueous solution of CO<sub>2</sub> is typically acidic:



Generated in PDH and TCA

CO<sub>2</sub> plays a major role in maintaining the pH of blood (and sea water).

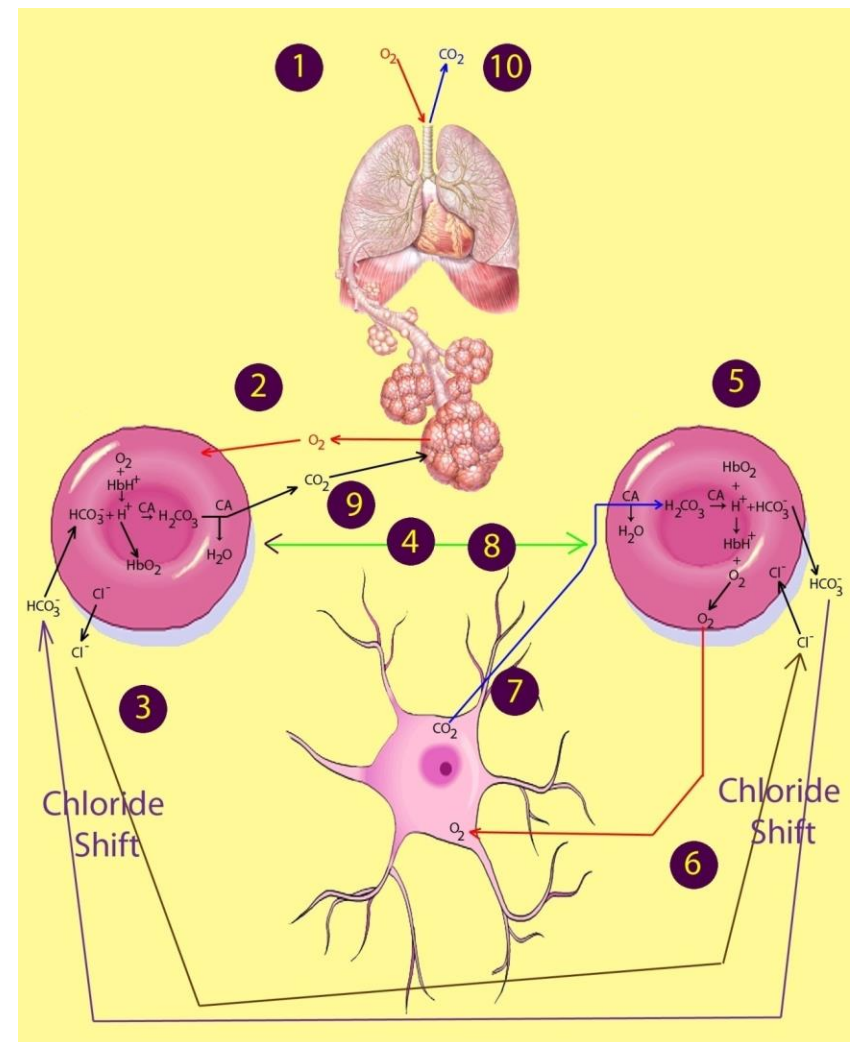
CO<sub>2</sub> is not normally transported as such, rather as HCO<sub>3</sub><sup>-</sup>.

This occurs via an enzymatic reaction catalyzed by carbonic anhydrase:



**IMPORTANT** in acid/base balance

## Red Cells and Acid-Base Balance



Cl<sup>-</sup> and CO<sub>2</sub> are intertwined



Condition causing slowed passage of CO<sub>2</sub> from blood to lung

CO<sub>2</sub> Retention

Increased H<sub>2</sub>CO<sub>3</sub> biosynthesized from the excess CO<sub>2</sub> and H<sub>2</sub>O via Carbonic Anhydrase

Excess H<sub>2</sub>CO<sub>3</sub> Dissociates to release more H<sup>+</sup>

pH of ECF drops:  
**RESPIRATORY ACIDOSIS**

Condition causing increased production of organic acids

Increased H<sup>+</sup> from dissociating organic acids

Buffers overcome by the excess protons

Excess H<sup>+</sup> accumulates

pH of ECF drops:  
**METABOLIC ACIDOSIS**

## Compensatory Mechanisms for Acidosis

reduced pH turns on breathing centers

blows off CO<sub>2</sub>

reduces H<sub>2</sub>CO<sub>3</sub> due to reduced CO<sub>2</sub> present to react with H<sub>2</sub>O

less H<sup>+</sup> from reduced H<sub>2</sub>CO<sub>3</sub> dissociation

Kidney pees out protons; reabsorbs more bicarb into blood (titration!)

pH of ECF rises:  
**COMPENSATION!**

**ACIDOSIS**  
pH go down, HCO<sub>3</sub><sup>-</sup> go down, pCO<sub>2</sub> go up

Condition causing excessive elimination of  $\text{CO}_2$  from blood to lung

$\text{CO}_2$  Blown off

Decreased  $\text{H}_2\text{CO}_3$  biosynthesized from the less  $\text{CO}_2$  and  $\text{H}_2\text{O}$  via Carbonic Anhydrase

Reduced  $\text{H}_2\text{CO}_3$  dissociates to release LESS  $\text{H}^+$ , Excess base accumulation ( $\text{HCO}_3^-$ , e.g.)

pH of ECF rises:  
RESPIRATORY ALKALOSIS

Alkali intake or excessive loss of  $\text{H}^+$

Increased base levels OR lowering of  $\text{H}^+$  levels in ECF

pH of ECF rises:  
METABOLIC ALKALOSIS

## Compensatory Mechanisms for Alkalosis

elevated pH turns off breathing centers

retain  $\text{CO}_2$

increases  $\text{H}_2\text{CO}_3$  due to increased  $\text{CO}_2$  present to react with  $\text{H}_2\text{O}$

more  $\text{H}^+$  from elevated  $\text{H}_2\text{CO}_3$  dissociation

Kidney reabsorbs protons into blood; excretes more bicarb into urine (titration!)

pH of ECF drops:  
COMPENSATION!

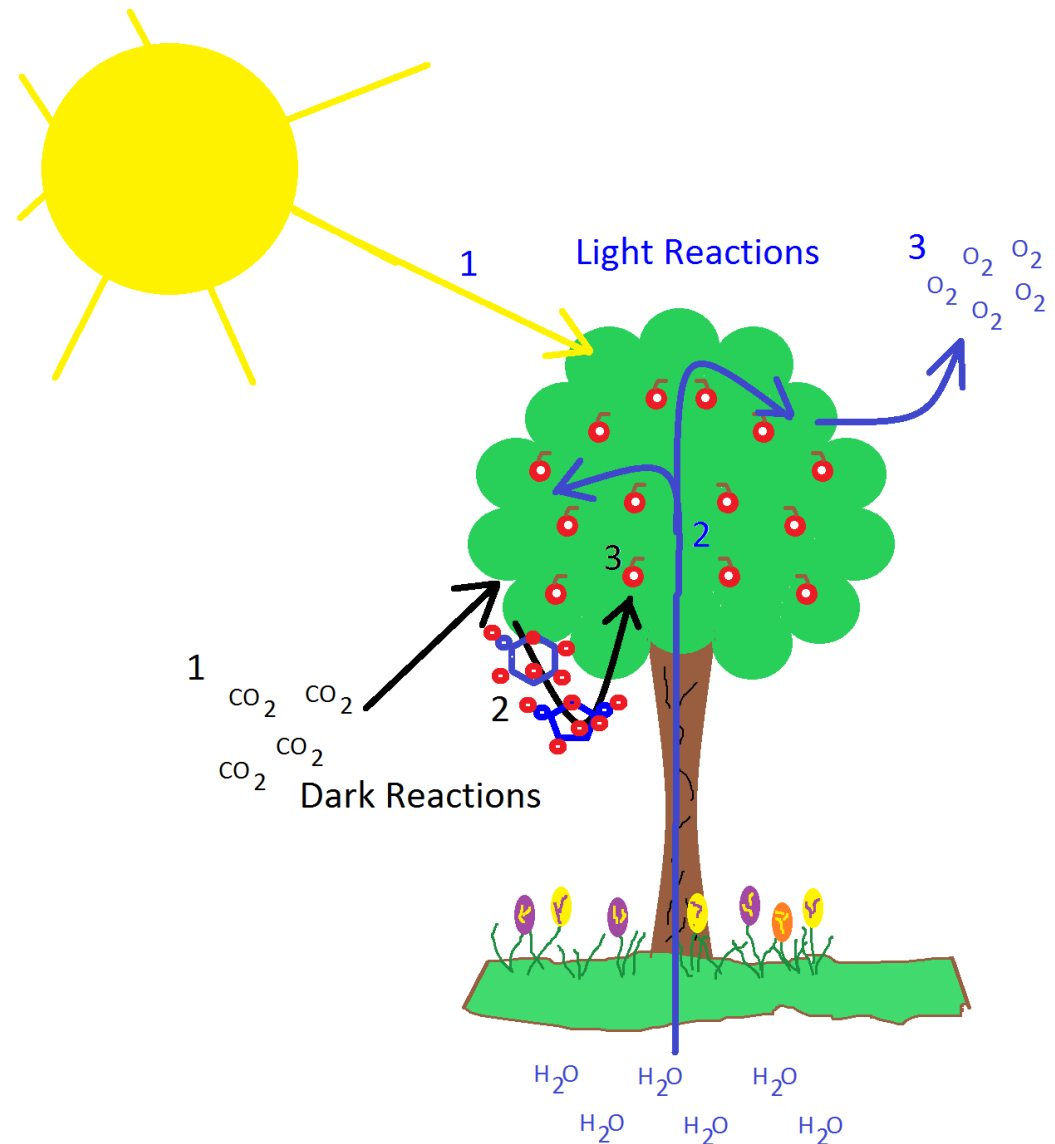
**ALKALOSIS**  
pH go up,  $\text{HCO}_3^-$  go up,  $\text{pCO}_2$  go down

# Photosynthesis



# Photosynthesis Definition and Superficial Overview

- Photosynthesis is the process used by plants to convert light energy from the sun into chemical energy that can be later released to fuel the organisms' activities.
- This energy is stored as carbohydrates, which are biosynthesized from carbon dioxide and water – hence the name photosynthesis, from Greek for "light", and synthesis, "putting together".
- In most cases, oxygen is also released as a waste product. Most plants, most algae, and cyanobacteria perform photosynthesis, and such organisms are called photoautotrophs.
- This process maintains atmospheric oxygen levels and supplies all of the organic compounds and most of the energy necessary for life on Earth.



# C<sub>3</sub> vs C<sub>4</sub> Plants

**C<sub>3</sub> vs C<sub>4</sub> plants:** Most plants, designated **C<sub>3</sub>**, fix CO<sub>2</sub> initially via RuBP Carboxylase, yielding the **3-carbon compound 3-phosphoglycerate (3-PG)**. E.g., beans, rice, wheat, potatoes, all woody trees.

Plants designated **C<sub>4</sub>** have one cell type in which phosphoenolpyruvate (PEP) is carboxylated via the enzyme PEP Carboxylase, to yield the **4-carbon compound oxaloacetate (OAA)**. The oxaloacetate is converted to other 4-carbon intermediates that are transported to cells active in photosynthesis, where **CO<sub>2</sub> is released** by decarboxylation. E.g., corn, sugarcane.

**C<sub>4</sub> plants maintain a high ratio of CO<sub>2</sub>/O<sub>2</sub>** within photosynthetic cells, thus minimizing **photorespiration**.

**Photorespiration** is a light-dependent process in some plants resulting in the oxidation of glycolic acid and release of carbon dioxide that under some environmental conditions (such as high temperature) tends to inhibit photosynthesis; sometimes called oxidative photosynthetic carbon cycle, or C<sub>2</sub> photosynthesis.

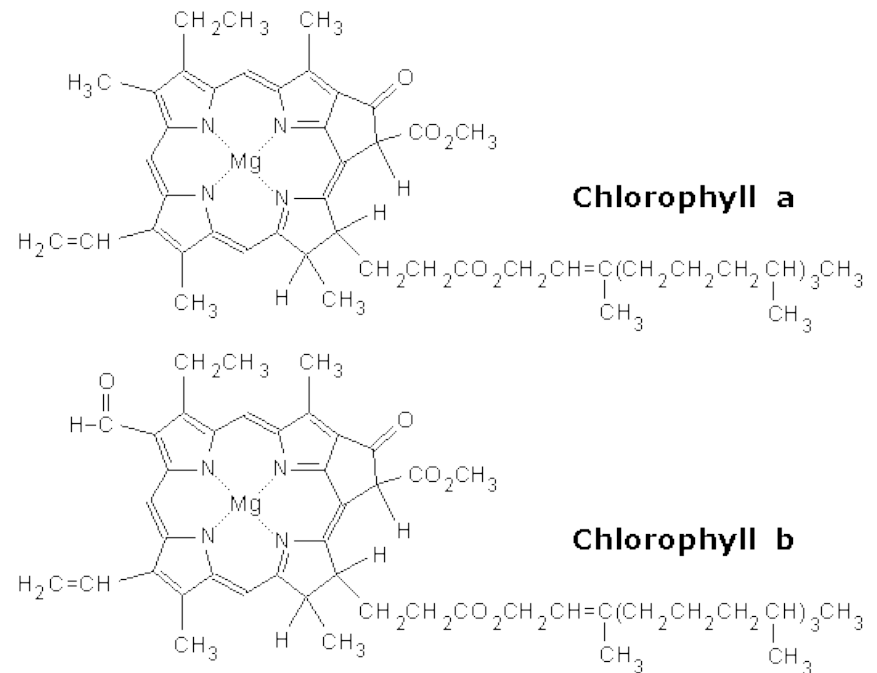
**altitude:** C<sub>4</sub> low altitudes, C<sub>3</sub> high altitudes

# RuBisCO

- Ribulose-1,5-bisphosphate carboxylase/oxygenase.
- Catalyzes the carboxylation of ribulose-1,5-bisphosphate (also known as *RuBP*).
- In the first major step of carbon fixation.
- The most abundant protein on Earth.
- Most noted achievement is its method of introducing inorganic carbon into the biosphere.
- When there is sunlight present, the enzyme is turned on.
- When there is no light present, the enzyme is turned off.

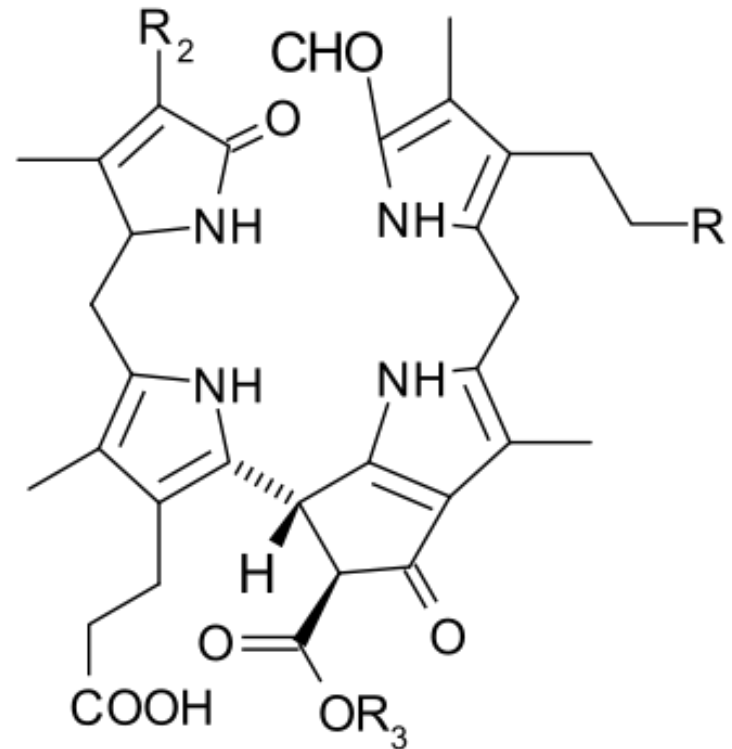
# Chlorophyll

- Chlorophyll a: absorbs energy from wavelengths of violet-blue and orange-red light.
- It reflects (transmits) green/yellow light, and contributes to the green color of most plants.
- Chlorophyll b: color is yellow, and it primarily absorbs blue light.



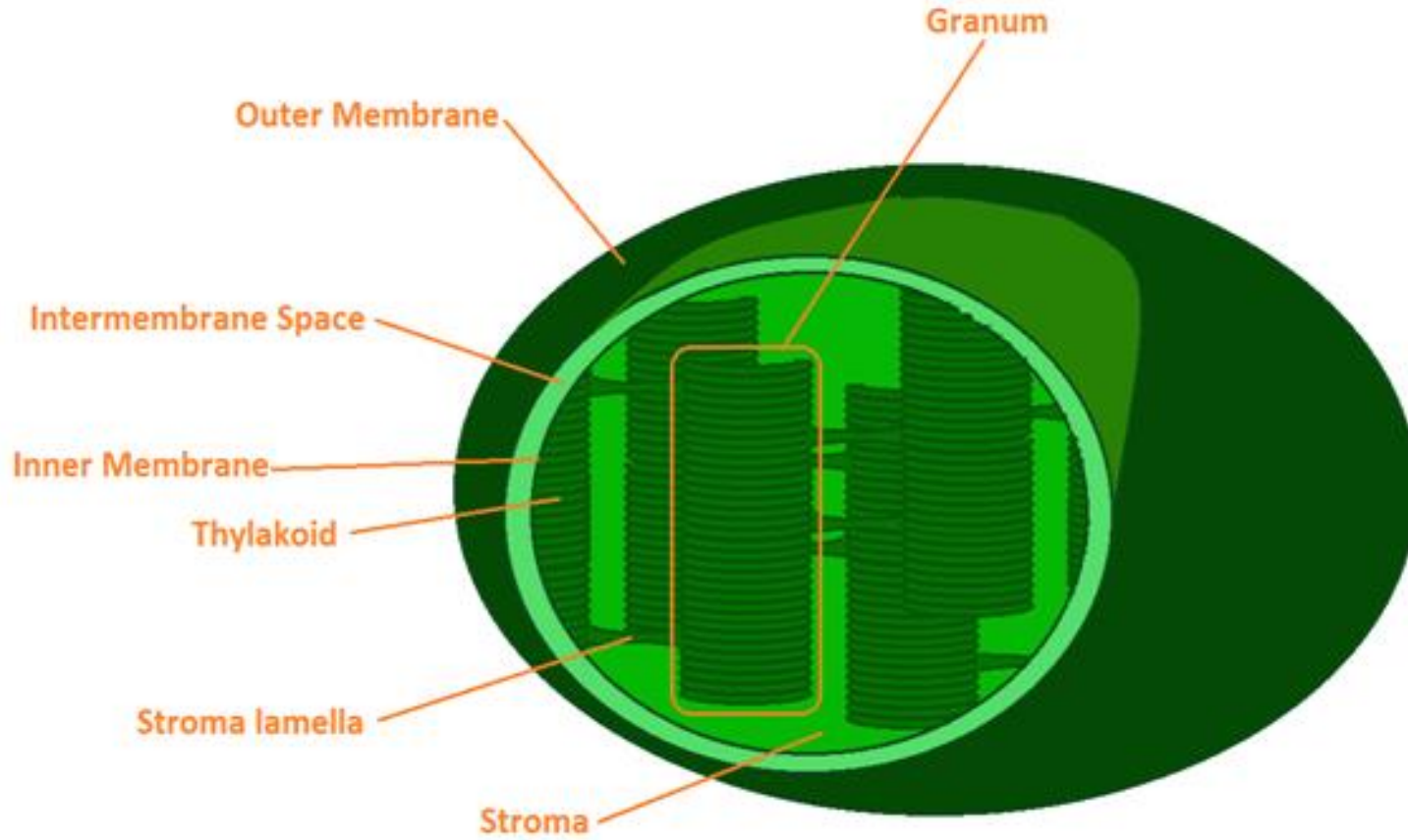
# Autumn Leaves

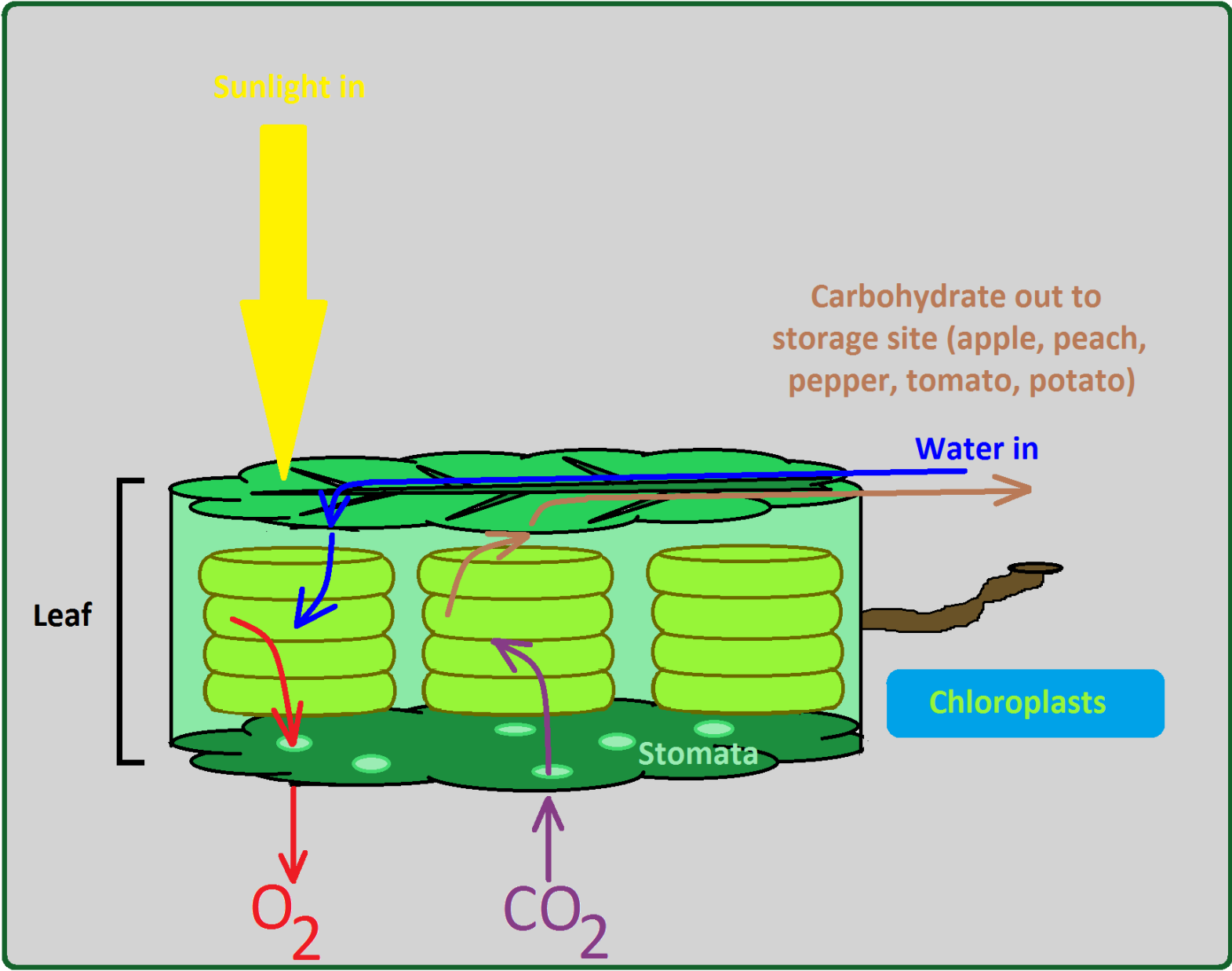
- When leaves change colors in the autumn, chlorophyll is converted to a group of tetrapyrroles known as nonfluorescent chlorophyll catabolites (NCC's).



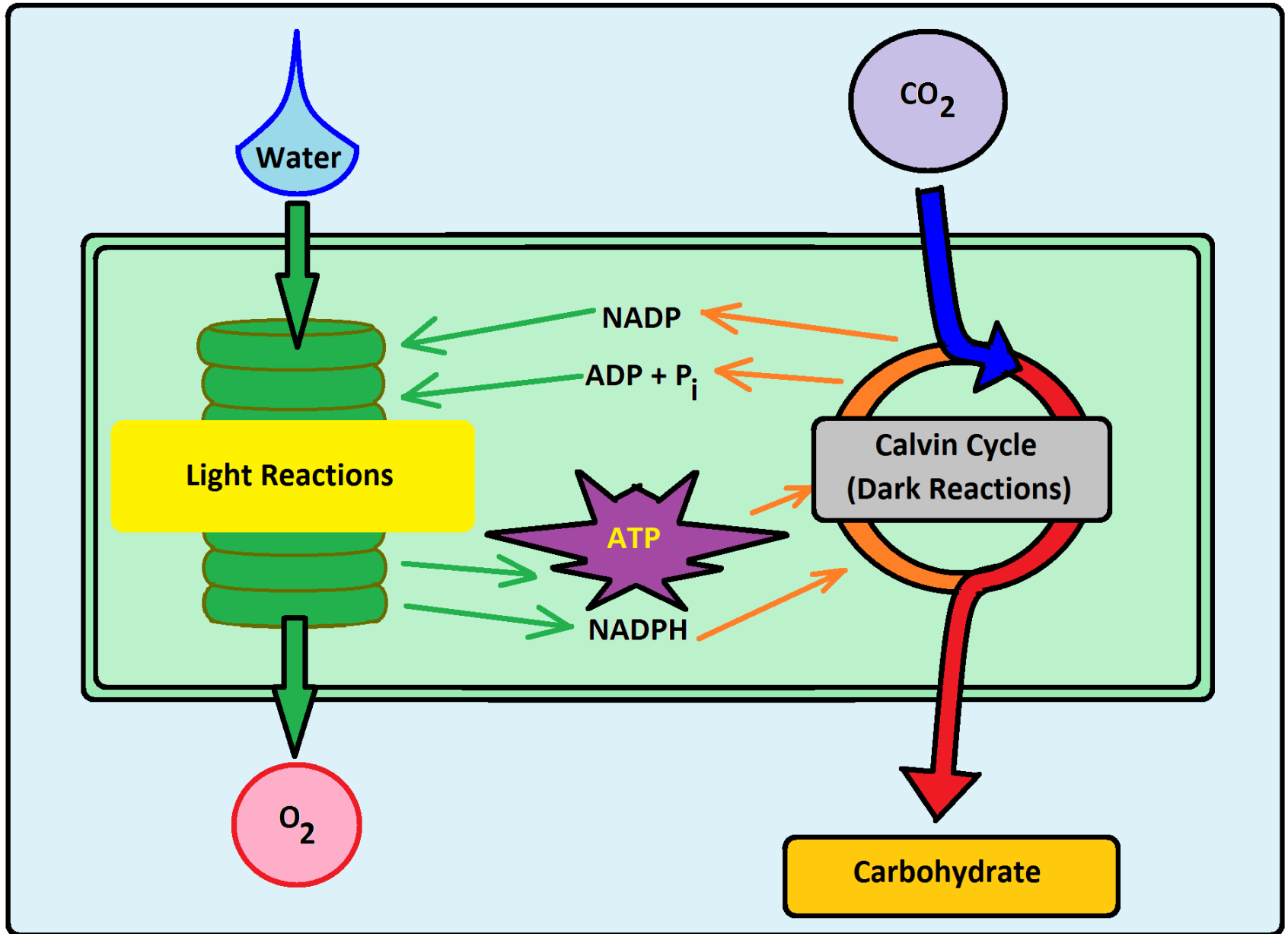


# Chloroplast Review





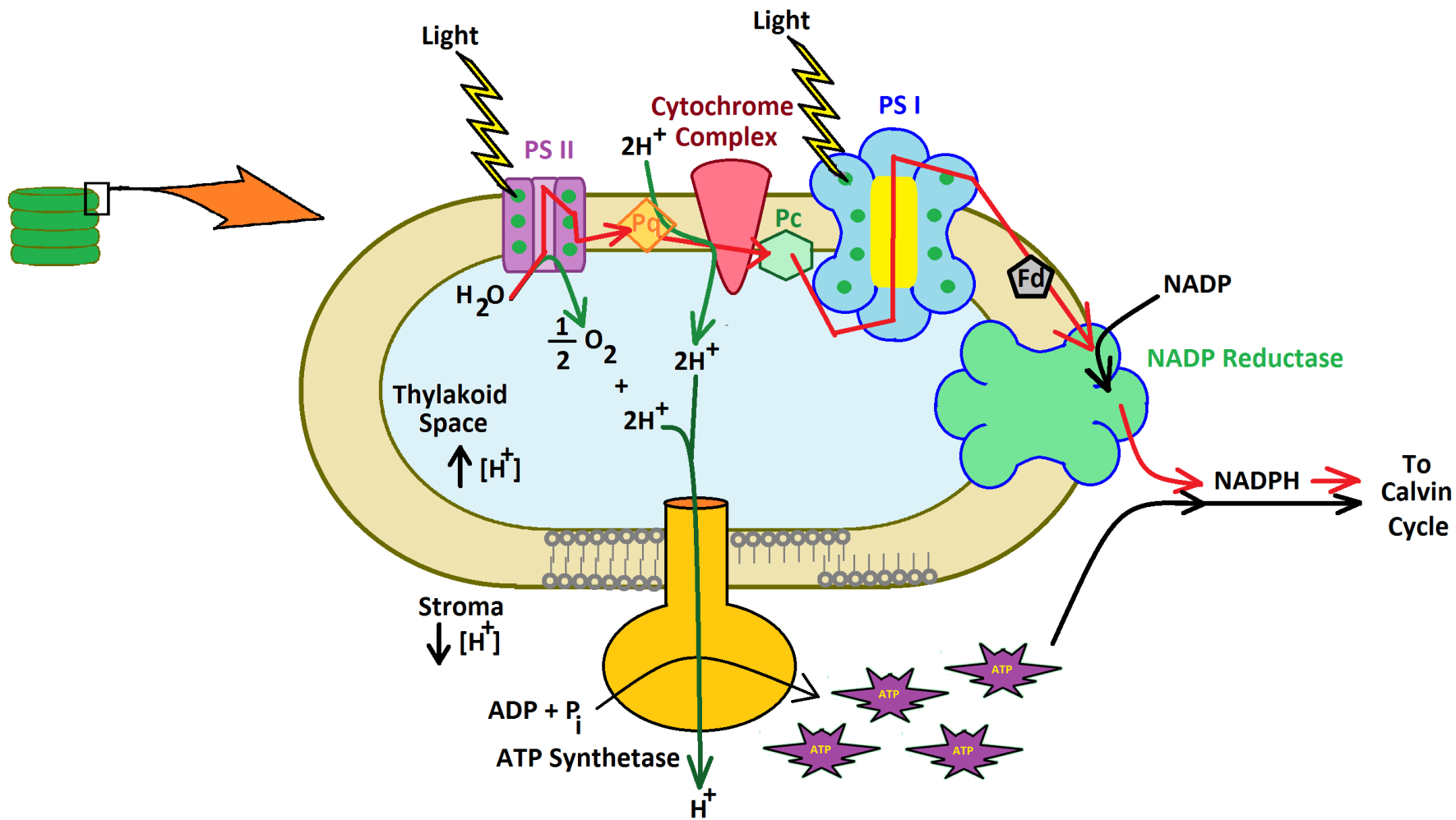
# Superficially Inside the Chloroplast

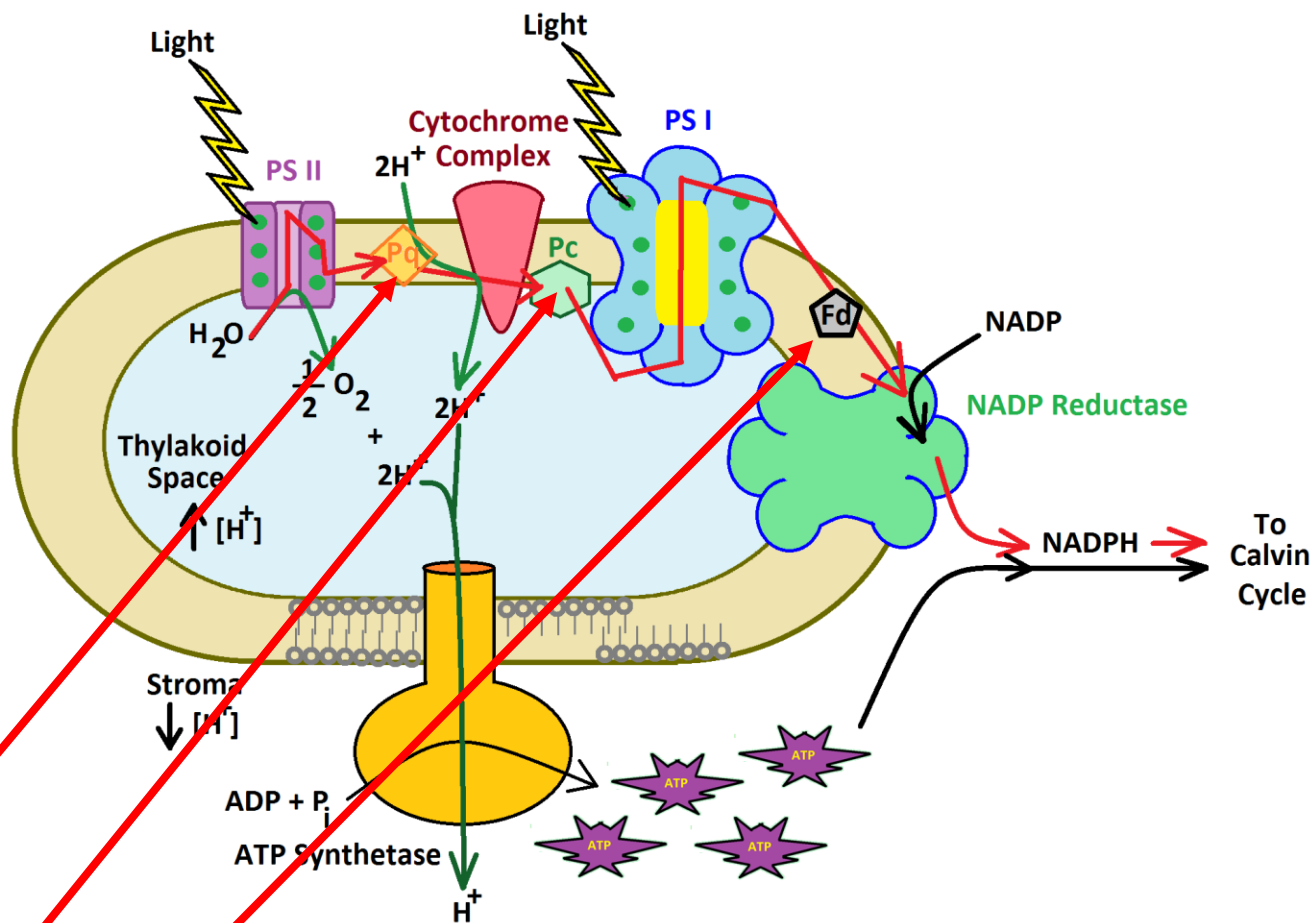


# Mito in Plant Cells?

- Plant cells have mitochondria to produce energy!
- Mitochondria allow for a more extensive breakdown of glucose, which results in more ATP for the cell.
- Chloroplasts do not supersede mitochondria.
- Chloroplasts create carbohydrates from sunlight.
- Plant cells still need mitochondria to break down the carbohydrate.

# Light Reactions – Thylakoid Membrane and Stroma



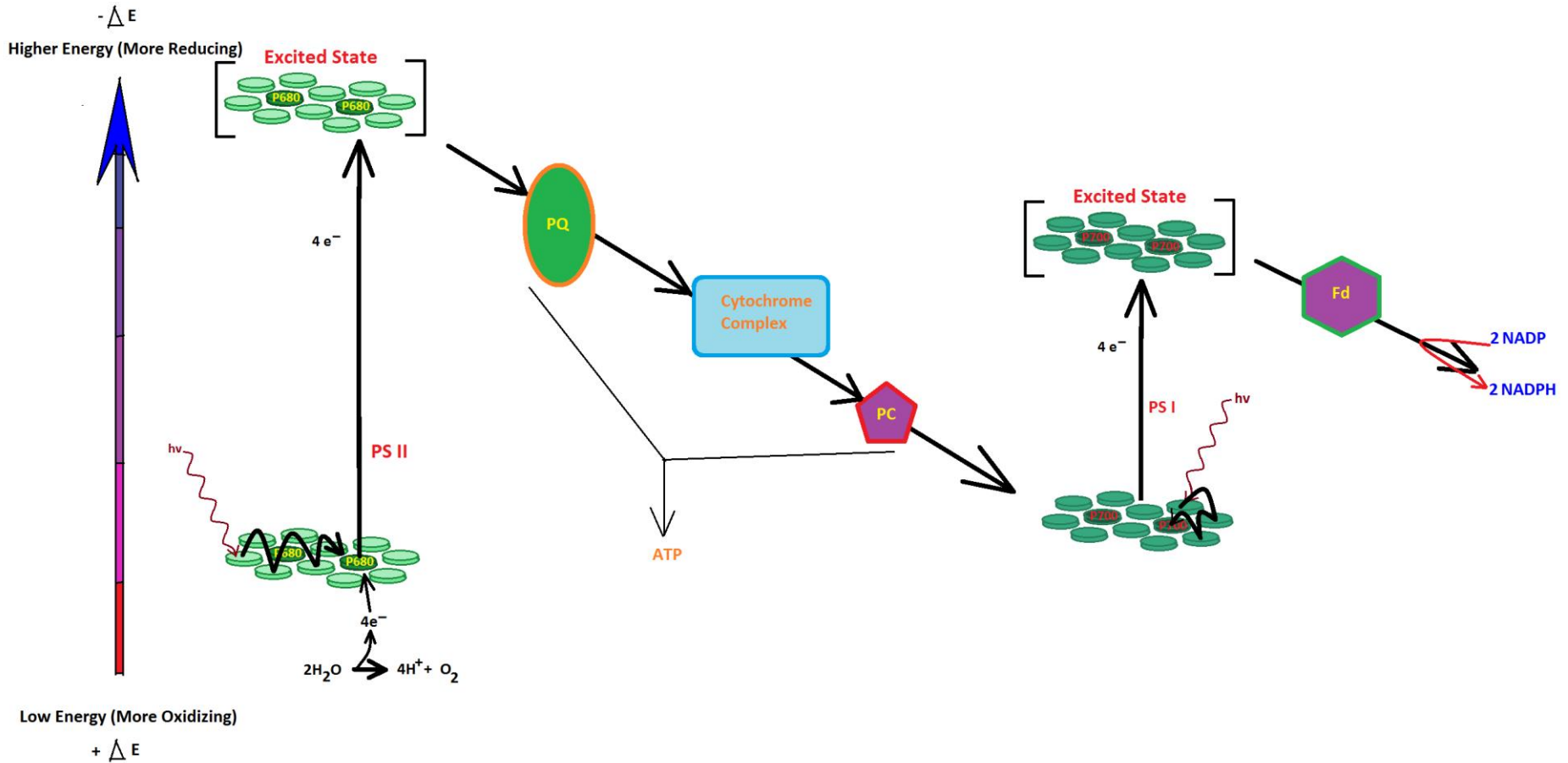


Pq = plastoquinone, is a molecule in the electron transport chain

Pc = plastocyanin, is a copper-containing compound in the electron transport chain that accepts electrons. Plastocyanin's dependence on copper is one reason that copper is a vital nutrient for plants.

Fd = ferredoxin, is a protein that's not involved in the electron transport chain, but is still involved in the light reactions. This protein moves electrons excited by a different molecule of chlorophyll than the electrons from the electron transport chain to the NADP Reductase that stores this light-derived energy in the form of NADPH.

# Z Scheme



# Calvin-Benson Cycle (Dark or Light Independent Cycle)

