Module 5
Carbohydrates

Muscles and their meridian relationship.

Meridian	Muscles
Bladder	Tibialis ant, Tibialis post, Peroneus long/ brevis, Peroneus tertius
Kidney	Psoas, Iliacus, Upper trap
Gall bladder	Popliteus
Liver	PMS, Rhomoids
Large Intestine	TFL, Hamstrings, QL
Lung	Deltoid, Serratus ant, Coracobrachialis
CV	Supraspinatus, Diaphragm
GV	Teres major
Triple warmer	Teres minor, Infraspinatus
Circulation / sex	Glut max, Glut med/min, Piriformis, Adductors, Sartorius, Gracilis
Stomach	PMC, Neck flexors, Biceps, Brachialis, Pronator teres, Pronator quadratus
Spleen	Lat dorsi, Mid trap, Lower trap, Triceps
Small intestine	Quads, Abdominals
Heart	Subscapularis

Nutrition / Muscle relationship.

Vitamin A - Latissimus dorsi, Pectoralis major clavicular, Pectoralis minor, Piriformis, Popliteus, Psoas, Quadratus lumborum, Rhomboids,Sacrospinalis, Tibialis anterior.
B. Complex - Pectoralis major clavicular, Pectoralis minor, Peroneals, Quadriceps, Subscapularis, Upper trapezius, Supinator.
Vitamin B1
Vitamin B2 - Neck extensors
Vitamin B3 - Gracilis, Neck flexors, Pectoralis minor
Vitamin B5 - Sartorius
Vitamin B6 - Opponens digiti minimi
Folic acid
Vitamin B12
Biotin
Vitamin C - Coracobrachialis, Deltoid, Diaphragm, Quadratus lumborum, Sacrospinalis, Sartorius, Serratus anterior, Middle trapezius, Lower trapezius
Vitamin D - Quadriceps, Tensor fascia lata, ICV
Vitamin E - Abdominals, Adductors, Gluteus maximus, Gluteus medius, Hamstrings, Quadratus lumborum, Sacrospinalis, Subscapularis
Vitamin K
Co-enzyme Q10
SAMe

Muscle / Meridian / Nutrition relationship.

Muscle	Meridian	Nutrition
Abdominals	SI	Vit E
Adductors	Cx	Vit E
Biceps	St	HCl, Chlorophyll
Brachio Radialis	St	HCl
Coracobrachialis	Lung	Vit C
Deltoid	Lung	Vit C, RNA
Diaphragm	CV	Vit C
Gastrocnemius	Cx	Adrenal
Gluteus max	Cx	Vit E
Gluteus med	Cx	Vit E
Gracilis	Cx	Vit B3, Adrenal
Hamstrings	LI	Vit E, HCl, Ca
ICV		Chlorophyll, Ca, Vit D, HCl
Infraspinatus	TW	Thymus
Latissimus dorsi	Sp	Vit A, EFAs, Zn
Neck extensors	St	Vit B2, B3, B6, Iodine
Neck flexors	St	Vit B3, B6
Opponens digiti min	St	Vit B6
Pectoralis major clav	St	Vit B, B12, HCl
Pectoralis major sternal	Liv	Vit A, Bile salts
Pectoralis minor		RNA, Vit A, B, B3, Zn
Peroneals	Bl	Vit B, Ca
Piriformis	Cx	Vit A
Popliteus	Gb	Vit A
Psoas / Iliacus	Kid	Vit A, E
Quadratus lumborum	LI	Vit A, C, E
Rhomboids	Liv	Vit A
Sacrospinalis	Bl	Vit A, C, E, P, Ca
Sartorius	Cx	Vit B5, B6, C, Adrenal, Zn, Ginseng
Serratus anterior	Lung	Vit C
Soleus	Cx	Vit C
SCMastoid	St	Vit B3, B6, Iodine
Subclavius		Mg
Subscapularis	Ht	Vit B, C, E
Supinator	St	Vit B, G, HCl
Supraspinatus	CV	RNA
Tensorfacialata	LI	Vit D, Probiotics, Iron
Teres major	GV	Alkaline minerals, K, P
Teres minor	TW	Iodine
Tibilais anterior	Cx /Bl	Adrenal
Tibialis posterior	Bl	Vit A
Trapezius upper	Kid	Vit A, B, EFAs, Ca
Trapezii mid & lower	Sp	Vit C, Ca
Triceps	Sp	Vit A, HCl

At rest humans burn 10% more calories in the late afternoon than they do late at night or early morning.

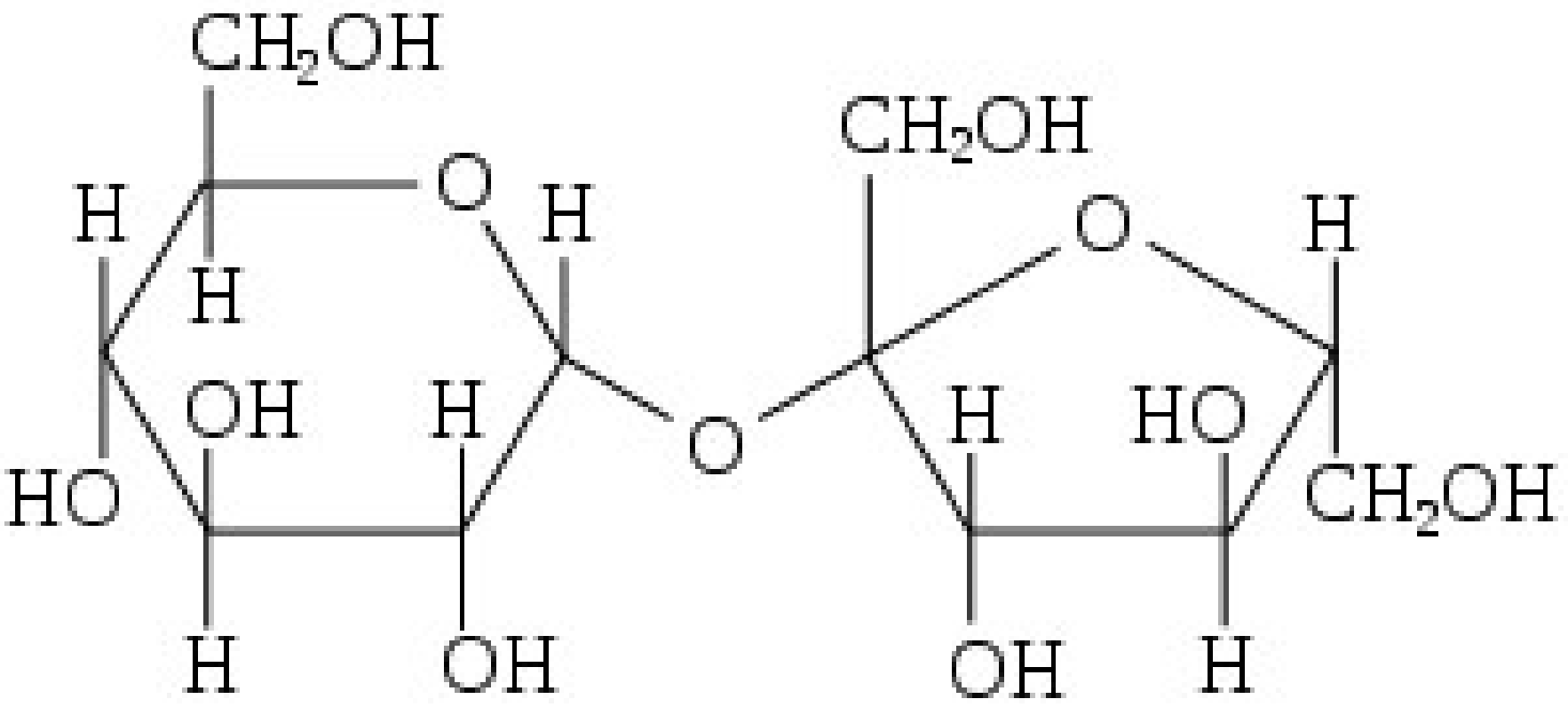
Current Biology. 11/11/ 2018 published in Time Magazine

Carbohydrates

1. Are a source of energy
2. Link with amino acids to form glycoproteins
3. Link with fatty acids to form glycolipids

Saccharides loosely refer to a number of carbohydrates, such as monosaccharides, disaccharides, or oligosaccharides.
Monosaccharides are also called "simple sugars," the most important being glucose.

Most **monosaccharides** have a formula that conforms to $C_nH_{2n}O_n$ with n between 3 and 7 (deoxyribose being an exception).
Glucose has the molecular formula $C_6H_{12}O$.
Glucose is made by photosynthesis in the green leaves of plants.



Sucrose: a disaccharide of glucose (left) and fructose (right), important molecules in the body.

Fructose, or fruit sugar, occurs naturally in fruits, some root vegetables, cane sugar and honey and is the sweetest of the sugars. It is one of the components of sucrose or table sugar. It is used as a high-fructose syrup, which is manufactured from hydrolyzed corn starch.*

*Kretchmer, Norman; Claire B. Hollenbeck (1991). "Sugars and Sweeteners". CRC Press, Inc.

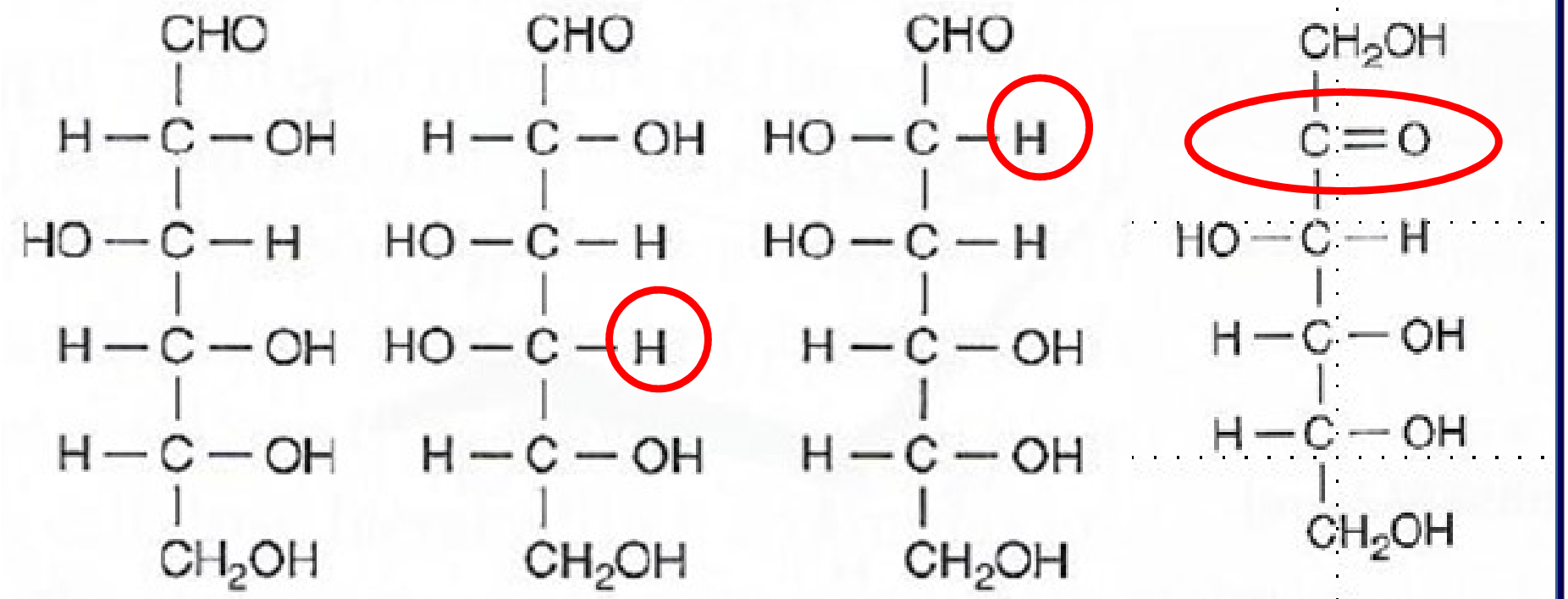
Galactose generally does not occur in the free state but is a constituent with glucose of the disaccharide lactose or milk sugar. It is less sweet than glucose. It is a component of the antigens found on the surface of red blood cells that determine blood groups.*

**Raven, Peter H. & George B. Johnson (1995). Carol J. Mills, ed. Understanding Biology (3rd ed.). WM C. Brown. p. 203*

Glucose, dextrose or grape sugar, occurs naturally in fruits and plant juices and is the primary product of photosynthesis. Most ingested carbohydrates are converted into glucose during digestion and it is the form of sugar that is transported around the bodies of animals in the bloodstream.*

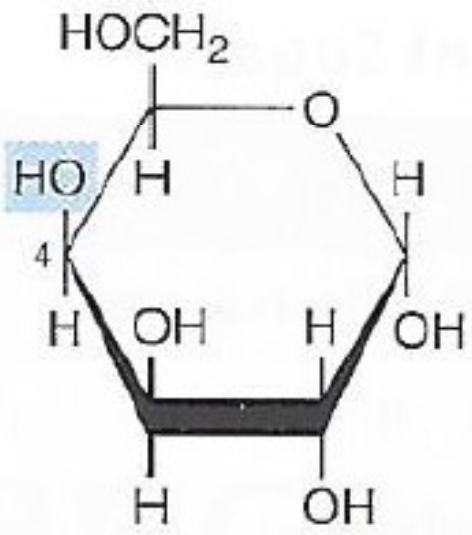
**Schenck, Fred W. (2006). "Glucose and Glucose-Containing Syrups". Ullmann's Encyclopedia of Industrial Chemistry. Wiley-VCH, Weinheim*

Simple Hexose Sugars

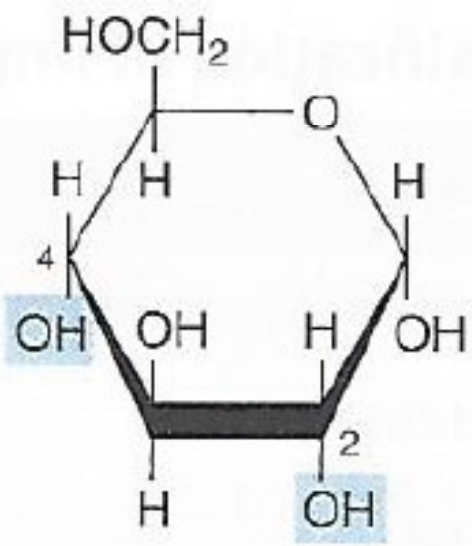


D. Glucose D. Galactose D. Mannose D Fructose

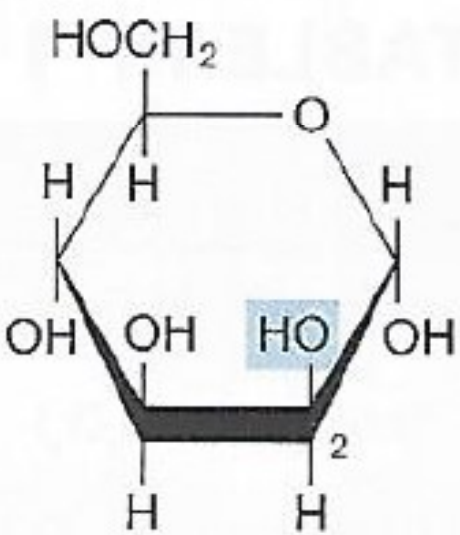
All dietary carbohydrates are converted in the body to glucose.



α-D-Galactose

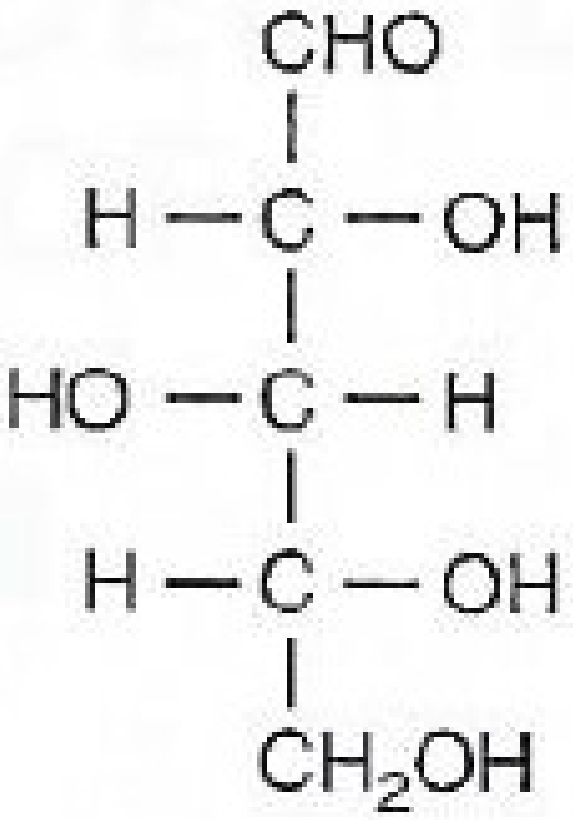


α-D-Glucose

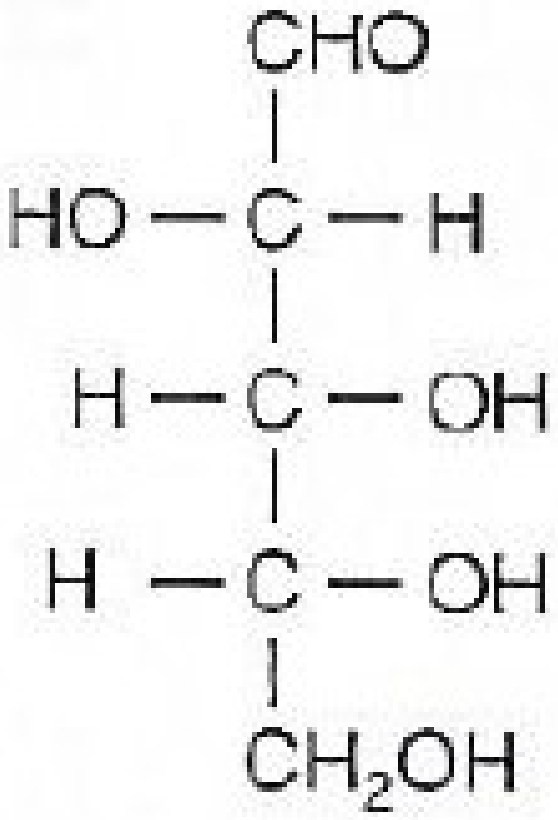


α-D-Mannose

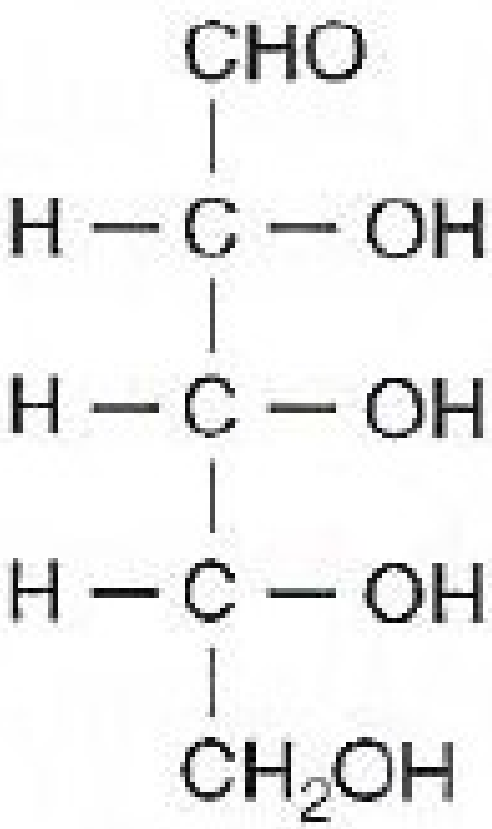
Simple Pentose Sugars



D-Xylose

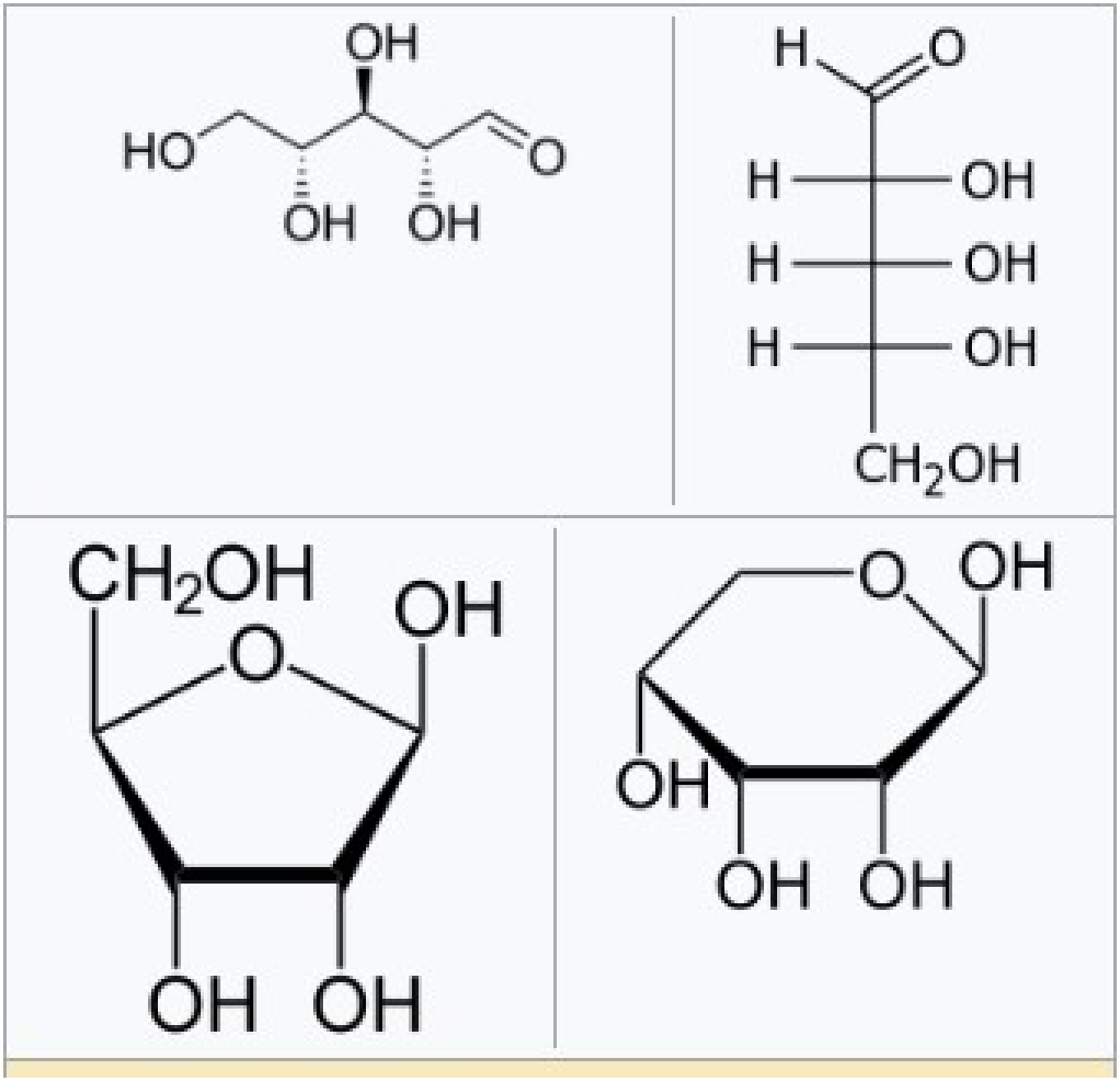


D-Arabinose



D-Ribose

Ribose



Ribose forms part of the backbone of RNA. It is related to deoxyribose, which is found in DNA. Phosphorylated derivatives of ribose such as ATP and NADH play central roles in metabolism. cAMP and cGMP, formed from ATP and GTP, serve as secondary messengers in some signalling pathways.*

**The Merck Index: An Encyclopedia of Chemicals, Drugs, and Biologicals (11th ed.), Merck, 1989, ISBN091191028X, 8205*

EFSA confirm safety of D-ribose in tea and sports drinks

By Will Chu

19-Dec-2018 - Last updated on 17-Dec-2018 at 15:00 GMT

POST A COMMEN

D-ribose lowdown

D-ribose is a naturally occurring five-carbon sugar and plays a role in adenosine triphosphate (ATP) production that goes in to fuel processes like muscle contraction.

When the ATP stores are exhausted, glucose is converted to ribose and then finally ATP restoring energy levels.

Ribose supplements aid in this process and have featured as a sports nutrition product capable of aiding in energy recovery and reduce muscle soreness.

Bioenergy produces its D-ribose by fermentation of a strain of *Bacillus subtilis*, which is found in the human gastrointestinal tract.

Atherosclerosis. 2014 Dec;237(2):725-33. doi: 10.1016/j.atherosclerosis.2014.10.101. Epub 2014 Nov 1.

Ribose-cysteine increases glutathione-based antioxidant status and reduces LDL in human lipoprotein(a) mice.

Kader T[1], Porteous CM[1], Williams MJ[2], Gieseg SP[3], McCormick SP[4].

Author information

Abstract

OBJECTIVE: D-ribose-L-cysteine (ribose-cysteine) is a cysteine analogue designed to increase the synthesis of glutathione (GSH). GSH is a cofactor for glutathione peroxidase (GPx), the redox enzyme that catalyses the reduction of lipid peroxides. A low GPx activity and increased oxidised lipids are associated with the development of cardiovascular disease (CVD). Here we aimed to investigate the effect of ribose-cysteine supplementation on GSH, GPx, lipid oxidation products and plasma lipids in vivo.

METHODS: Human lipoprotein(a) [Lp(a)] transgenic mice were treated with 4 mg/day ribose-cysteine (0.16 g/kg body weight) for 8 weeks. Livers and blood were harvested from treated and untreated controls (n = 9 per group) and GSH concentrations, GPx activity, thiobarbituric acid reactive substances (TBARS), 8-isoprostanes and plasma lipid concentrations were measured.

RESULTS: Ribose-cysteine increased GSH concentrations in the liver and plasma ($P < 0.05$). GPx activity was increased in both liver (1.7 fold, $P < 0.01$) and erythrocytes (3.5 fold, $P < 0.05$). TBARS concentrations in the liver, plasma and aortae were significantly reduced with ribose-cysteine ($P < 0.01$, $P < 0.0005$ and $P < 0.01$, respectively) as were the concentrations of 8-isoprostanes in the liver and aortae ($P < 0.0005$, $P < 0.01$, respectively). Ribose-cysteine treated mice showed significant decreases in LDL, Lp(a) and apoB concentrations ($P < 0.05$, $P < 0.01$ and $P < 0.05$, respectively), an effect which was associated with upregulation of the LDL receptor (LDLR).

CONCLUSIONS: As ribose-cysteine lowers LDL, Lp(a) and oxidised lipid concentrations, it might be an ideal intervention to increase protection against the development of atherosclerosis.

TABLE 14–3 Hexoses of Physiologic Importance

Sugar	Source	Biochemical Importance	Clinical Significance
D-Glucose	Fruit juices, hydrolysis of starch, cane or beet sugar, maltose and lactose	The main metabolic fuel for tissues; "blood sugar"	Excreted in the urine (glucosuria) in poorly controlled diabetes mellitus as a result of hyperglycemia
D-Fructose	Fruit juices, honey, hydrolysis of cane or beet sugar and inulin, enzymic isomerization of glucose syrups for food manufacture	Readily metabolized either via glucose or directly	Hereditary fructose intolerance leads to fructose accumulation and hypoglycemia
D-Galactose	Hydrolysis of lactose	Readily metabolized to glucose; synthesized in the mammary gland for synthesis of lactose in milk. A constituent of glycolipids and glycoproteins	Hereditary galactosemia as a result of failure to metabolize galactose leads to cataracts
D-Mannose	Hydrolysis of plant mannan gums	Constituent of glycoproteins	

Harper's Illustrated Biochemistry 29th Edition Pub Lange. Page 135

The blood level of glucose fluctuates in disease states. Dietary carbohydrate is stored in the liver and muscle tissue as glycogen. Glycogen is a polymer of glucose units. Once the liver and muscles are full of glycogen, the excess is converted into fat and stored in adipose tissue.*

*Role of Nutrition in Health and Disease by W.E. Cornatzer, Pub Thomas. Page 105

The stored glycogen is a reservoir to keep the blood sugar at a constant level. In fasting there is enough liver glycogen to last one day. On the second day of fasting the conversion of amino acids to glucose commences (gluconeogenesis).*

*Role of Nutrition in Health and Disease by W.E. Cornatzer, Pub Thomas. Page 105

The liver is the organ that regulates blood glucose by the enzyme glucose-6-phosphatase. Muscle tissue does not contain glucose-6-phosphatase which is necessay for the converting of muscle glycogen to blood glucose. It is active however not only in the liver but in the kidneys, GUT and brain.*

*Role of Nutrition in Health and Disease by W.E. Cornatzer, Pub Thomas. Page 106

The brain and CNS derives most of its energy from the oxidation of glucose. The brain accounts for 20% of the total O2 requirement in a resting individual.*

The brain is the most energy hungry organ in the body.

*Role of Nutrition in Health and Disease by W.E. Cornatzer, Pub Thomas. Page 106

Glucose is required for red cell for fat synthesis. Glucose is essential for the production of glycerol. Glucose is the major energy source for skeletal and cardiac muscle which can also oxidise fatty acids for energy. ATP is the high energy phosphate compound derived from the oxidation of glucose.

*Role of Nutrition in Health and Disease by W.E. Cornatzer, Pub Thomas. Page 106

Each filament of muscle tissue contains actin and myosin proteins that will contract are two mitochondria providing the ATP energy for contraction.

*Role of Nutrition in Health and Disease by W.E. Cornatzer, Pub Thomas. Page 106

Monosaccharides are single polyhydroxy aldehyde or ketone. Glucose is an aldo-hexose. The enzymatic hydrolysis of starch and glycogen yield glucose. The disaccharides sucrose and lactose yield one glucose each plus frucose and galactose respectively.

*Role of Nutrition in Health and Disease by W.E. Cornatzer, Pub Thomas. Page 108

Maltose yields two glucose molecules.
Dietary glucose is absorbed across the intestinal tract by a specific transport system.

*Role of Nutrition in Health and Disease by W.E. Cornatzer, Pub Thomas. Page 109

Hyperglyceamia is caused by Diabetes mellitus, Acromegaly, Cushings syndrome, Pheochroocytoma, hyper function of anterior pituitary, hyperfunction of adrenal cortex and pancreatic disease.

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Hypoglyceamia is caused by pancreatic islet beta cell tumour (adenoma or carcinoma), liver necrosis, cirrhosis, hepatitis, anterior pituitary hypofunction, adrenocortical hypofunction, Von Gierke's glycogen storage disease (deficiency of glucose-6-phosphatase) -

*Role of Nutrition in Health and Disease by W.E. Cornatzer, Pub Thomas. Page 109

Forbes' glycogen storage disease (deficiency of amyl-1,6-glucosidase), Hers' glycogen storgae disease (deficiency of hepatic phosphorylase), glycogen storage disease (deficiency of glycogen synthetase), poor nutrition.* --

*Role of Nutrition in Health and Disease by W.E. Cornatzer, Pub Thomas. Page 109

Following ethanol ingestion, erythroblastosis fetalis, amino acid leucine hypersensitivity, infantile giantism, Reyes syndrome (virus or aspirin sensitivity) and following strenuous exercise.*

*Role of Nutrition in Health and Disease by W.E. Cornatzer, Pub Thomas. Page 109

Glycosuria

Results when the renal threshold (150-170mg/dl) for glucose is exceeded or with tubular transport defect. It occurs in diabetes mellitus, renal glycouria, acromegaly, giantism, Cushings syndrome, ACTH hyperfunction, pancreatitis, phenchromacytoma-

*Role of Nutrition in Health and Disease by W.E. Cornatzer, Pub Thomas. Page 109

Inflammatory renal disease, pregnancy, Fanconi's syndrome (proximal tubular defects) and administration of corticosteroids, allergy, morphine and oral contraceptives.

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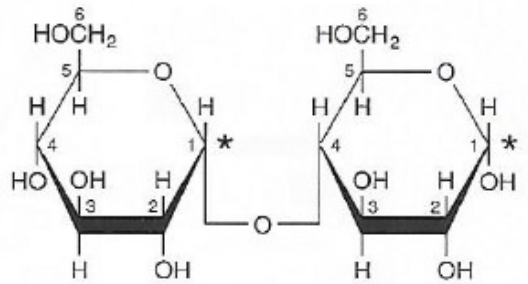
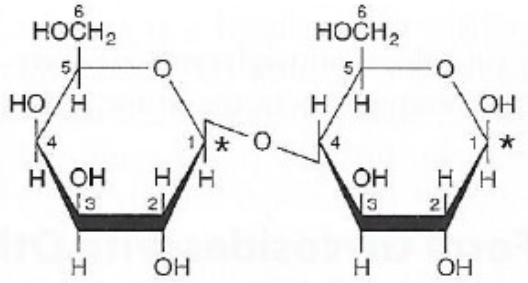
Maltose



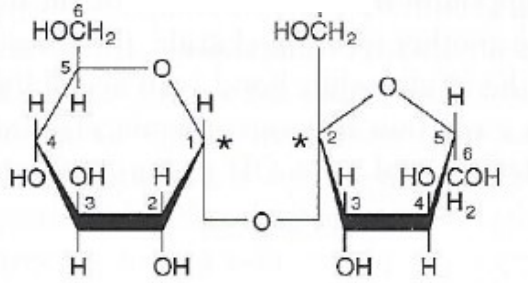
O-α-D-glucopyranosyl-(1 → 4)-α-D-glucopyranose

Lactose



O-β-D-Galactopyranosyl-(1 → 4)-β-D-glucopyranose

Sucrose



O-α-D-Glucopyranosyl-(1 → 2)-β-D-fructofuranoside

Disaccharides of physiological importance

Disaccharides of physiological importance

Sucrose – glucose + fructose

Lactose – galactose + glucose

Maltose – glucose + glucose

Isomaltose – glucose + glucose

Lactulose – galactose + fructose

Trehalose – glucose + glucose

Starch is in two forms

Amylose which is a polymer of glucose in alpha linkages makes up 15-20%.

Amylopectin which is highly branched made up of chains of glucose made up of chains of glucose with one chain branched with another. 80-85%.

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Glycogen is the polysaccharide stored in the liver and muscles and small amounts in the brain, kidney and other cells. It is a more highly branched structure than amylopectin.*

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Inulin is a starch found in tubers and roots of Jerusalem artichokes, dandelions, dahlias and chicory. It is composed of fructose units in beta linkage.

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Cellulose is a linear unbranched polymer of glucose units in beta linkage. All the digestive enzymes that hydrolyse starch are specific for the alpha-glucose linkage thus no enzymes in humans that can digest cellulose.

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Dietary glucose is absorbed across the intestinal cell by a specific transport protein and enters the portal blood to go to the liver. In all cells there is in the plasma membrane, protein involved in transporting glucose into the cell. The liver regulates blood glucose.*

*Role of Nutrition in Health and Disease by W.E. Cornatzer, Pub Thomas. Page 121

There are 3 enzymes that phosphorylate glucose.

1. **Glucokinase** requires insulin only found in the liver.
2. **Hexakinase** requires ATP.

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3. **Glucose-6-phosphatase** phosphorylates glucose and can hydrolyse glucose-6-phosphate to produce glucose which can diffuse from the liver cell into extracellular spaces including blood making it the major enzyme regulating blood glucose.

*Role of Nutrition in Health and Disease by W.E. Cornatzer, Pub Thomas. Page 121

Glycogenesis.

In muscle cell insulin is necessary for glucose to enter the plasma membrane. The glucose is phosphorylated by hexakinase converting it to glucose-6-phosphate requiring Mg++ and ATP.

*Role of Nutrition in Health and Disease by W.E. Cornatzer, Pub Thomas. Page 121

It is then converted to glucose-1-phosphate by phosphoglucomutase requiring Mg++. This enzyme action is reversible. Glucose-1-phosphate is then converted through several steps to glycogen by glycogen synthase.

*Role of Nutrition in Health and Disease by W.E. Cornatzer, Pub Thomas. Page 121

Glycogenolysis

With exercise Adrenalin / noradrenalin bind to the surface of the muscle wall at a specific beta receptor protein. These hromones activate adenylate cyclase to produce cAMP which ultimately converts glycogen to glucose-1-phosphate

*Role of Nutrition in Health and Disease by W.E. Cornatzer, Pub Thomas. Page 124

Glycogen storage disease type 1a – deficiency of glucose-6-phosphatase.

Type 1b – transport function of G-6-P is absent.

Type 1c – transport system for orthophosphate, pyrophosphate and carbamoyl phosphate is absent.

*Role of Nutrition in Health and Disease by W.E. Cornatzer, Pub Thomas. Page 125

Insulin is necessary for glucose to enter the membrane (plasma) of all cells except the liver, brain and intestine. The number of receptor sites for insulin to bind to varies with different cells. Adipocytes have many receptor protein sites for insulin and thus bind and hold insulin.*

*Role of Nutrition in Health and Disease by W.E. Cornatzer, Pub Thomas. Page 134

Insulin induces translocation of glucose transport proteins in muscle and fat.
Stimulates lipid synthesis
Stimulates glucokinase.
Stimulates pyruvate dehydrogenase
Stimulates glycogen synthase

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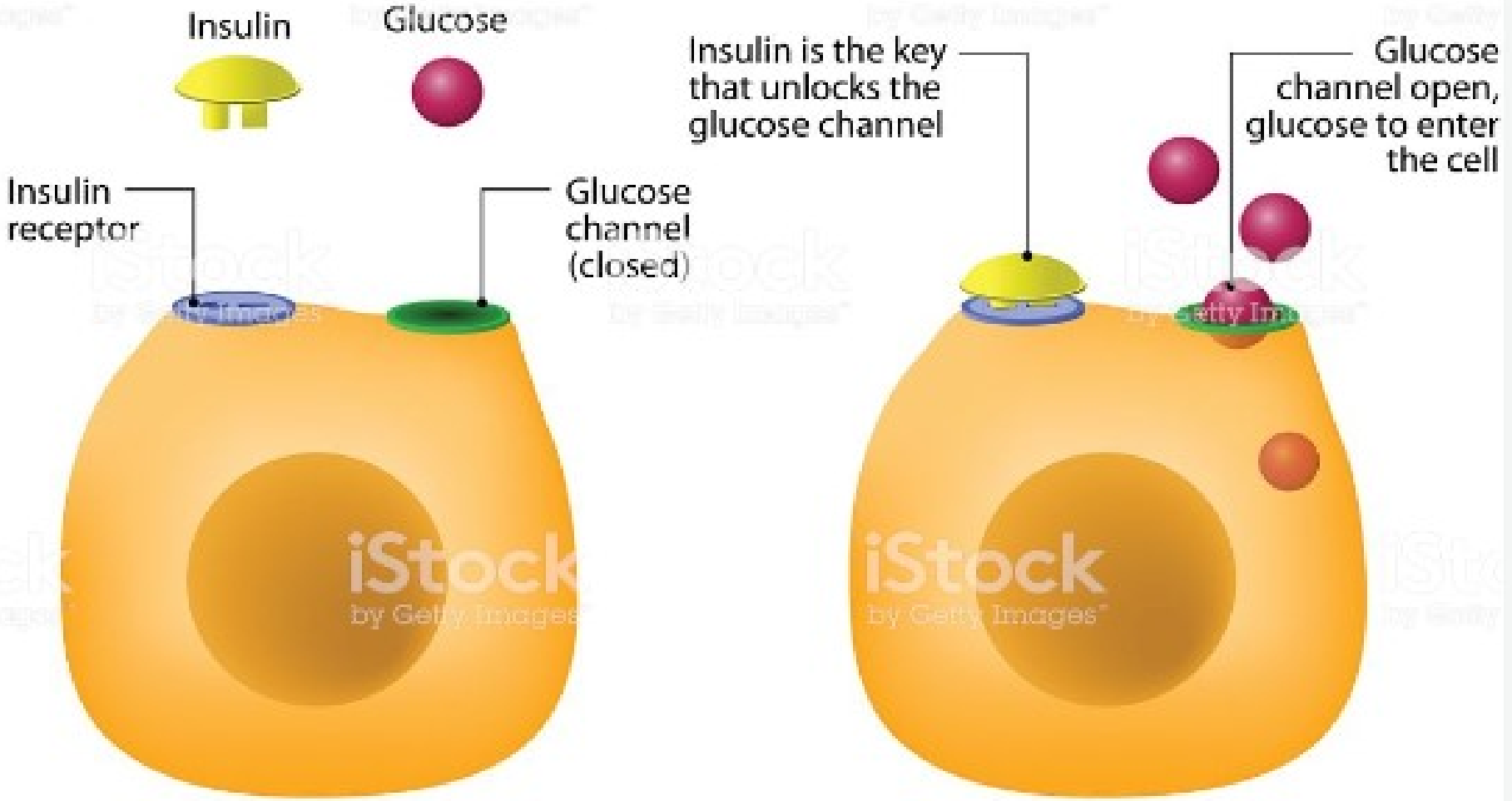
HOW DOES INSULIN WORK?



Glucagon is a hyperglyceamic-glycogenolytic hormone secreted by the A cells of the pancreas and raises blood glucose opposite to insulin. It binds to the liver cells and produces glucose-1-phosphate (glycogenolysis).

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Thyroid T4 and T3 The enzyme alpha –glycerolphosphate dehydrogenase reduces dihydroxyacetone phosphate during glycolysis and is stimulated by T4 and T4. T4 and T3 also stimulate glucose-6-phosphatase, glucose-6-phosphatase dehydrogenase in the liver.*

*Role of Nutrition in Health and Disease by W.E. Cornatzer, Pub Thomas. Page 137

Cortisol stimulates gluconeogenesis by the enzymatic activity of

Pyruvate carboxykinase

Pyruvate carboxylase

All transaminases

Fructose-1,6-diphosphatase

Glucose-6-phosphatase

Glycogen synthetase

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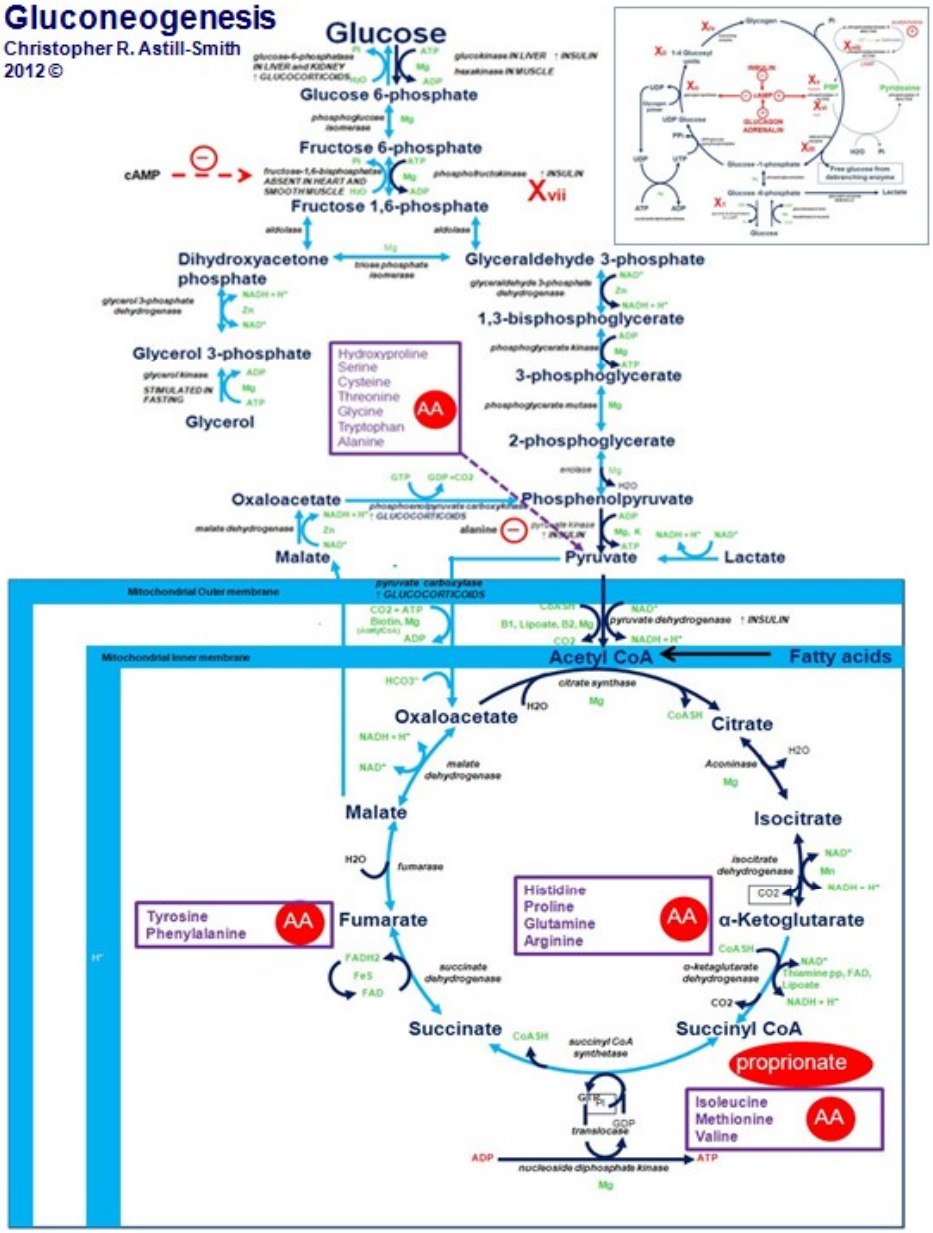
Gluconeogenesis

Christopher R. Astill-Smith
2012 ©



ENERGY


Ultimately all our
energy comes
from the sun

DEFICITS IN ENERGY PRODUCTION

Loss of Energy, Pain and Difficulty in memory recall are the most common symptoms complained of by patients attending any health care practitioner.

There is a common link between all these symptoms.

80% energy produced goes to heat to keep us warm.

Of the remainder One third is involved with the active process of the cellular Sodium / Potassium pumps.

Another third is involved with enzymatic activity.

The final third of energy production is for contractile and non-contractile tissues such as cilia.

The daily 1500–2000 Calories recommended for a human adult are taken as a combination of **oxygen and food molecules**, the latter mostly carbohydrates and fats, of which glucose ($C_6H_{12}O_6$) and stearic acid ($C_{57}H_{110}O_6$) are convenient examples.

The food molecules are oxidised to carbon dioxide and water in the mitochondria

$C_6H_{12}O_6 + 6O_2 \rightarrow 6CO_2 + 6H_2O$

$C_{57}H_{110}O_6 + 81.5O_2 \rightarrow 57CO_2 + 55H_2O$

and some of the energy is used to convert ADP into ATP

$ADP + HPO_4^{2-} \rightarrow ATP + H_2O$

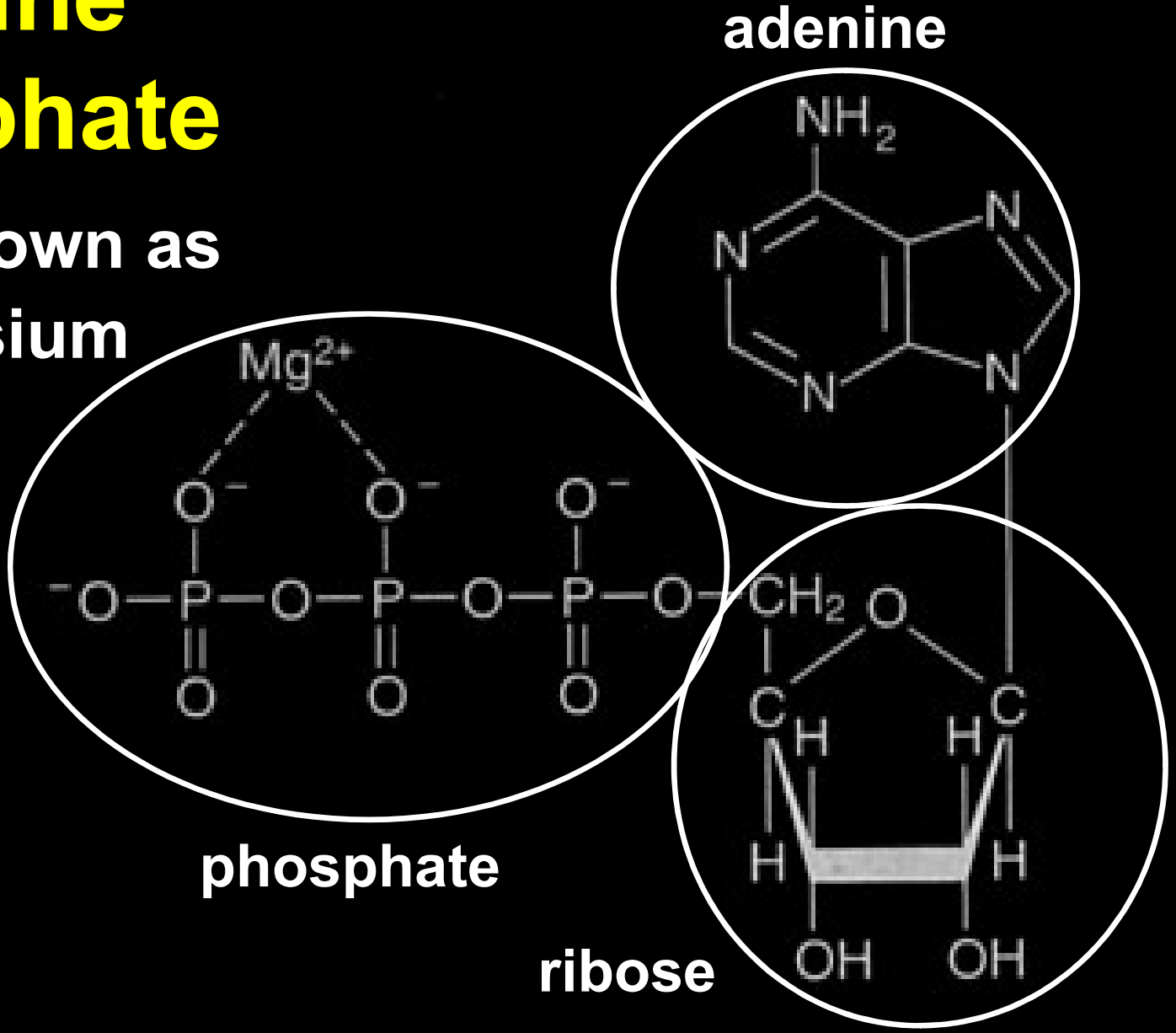
80% of the chemical energy in the carbohydrate or fat is converted into heat.

ATP is used as "energy currency", and some of the chemical energy it contains when split and reacted with water, is used for other metabolism.

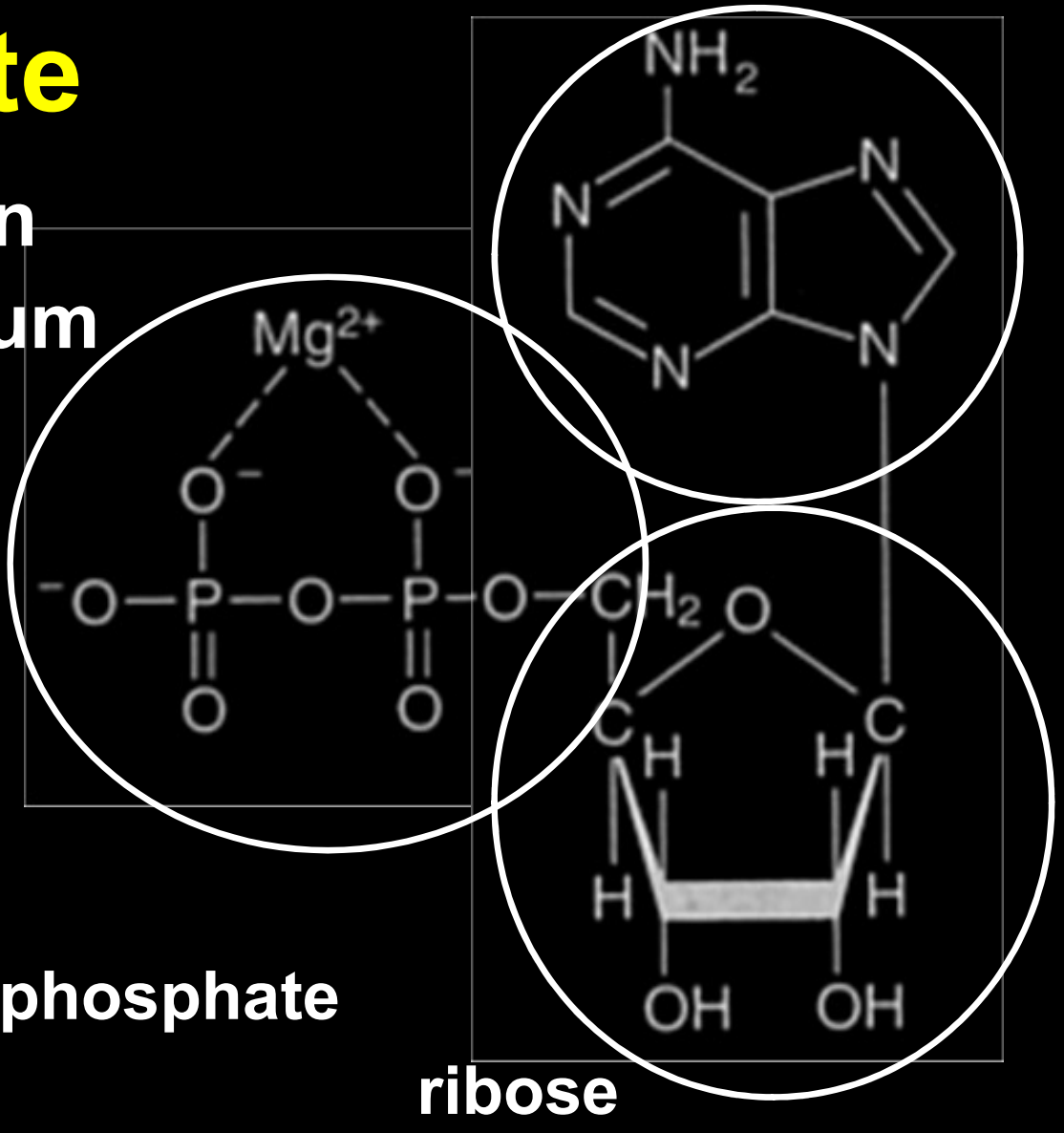
(At each stage of a metabolic pathway, 80% chemical energy is converted into heat).

Only a tiny fraction of the original chemical energy is used for work.

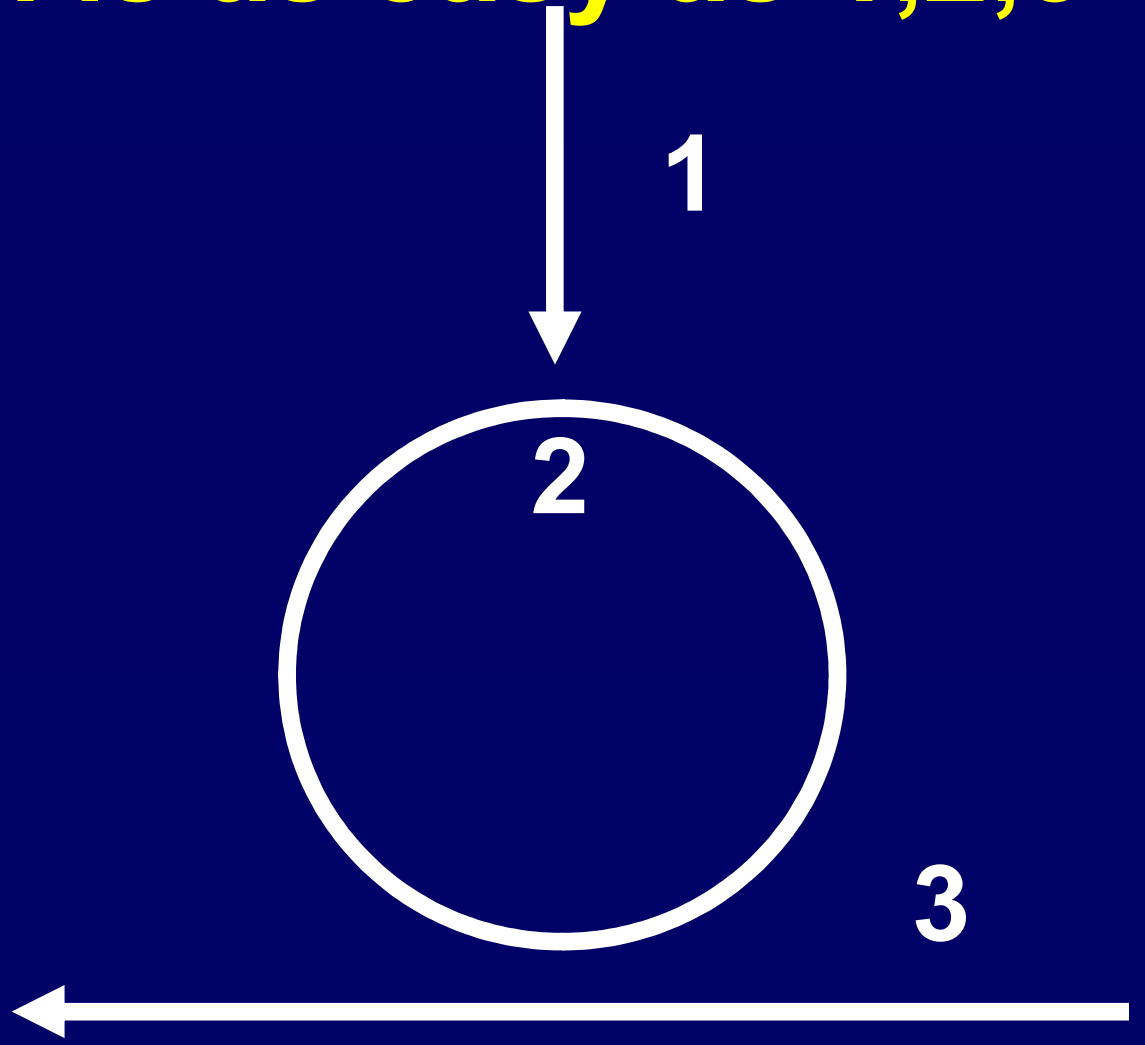
Adenosine triphosphate (ATP) shown as the magnesium complex.

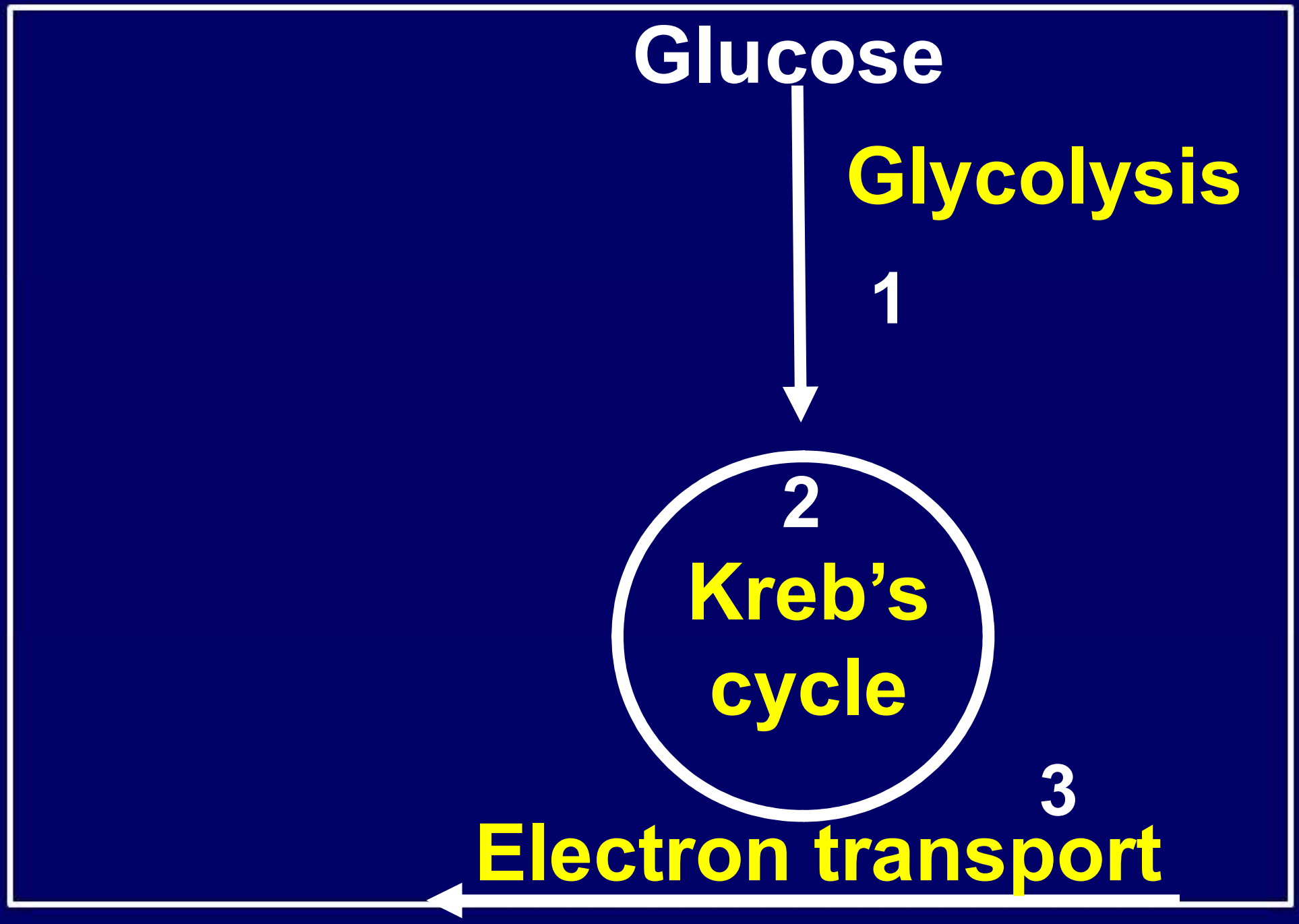


Adenosine diphosphate (ADP) shown as the magnesium complex.



Understanding energy production is as easy as 1,2,3



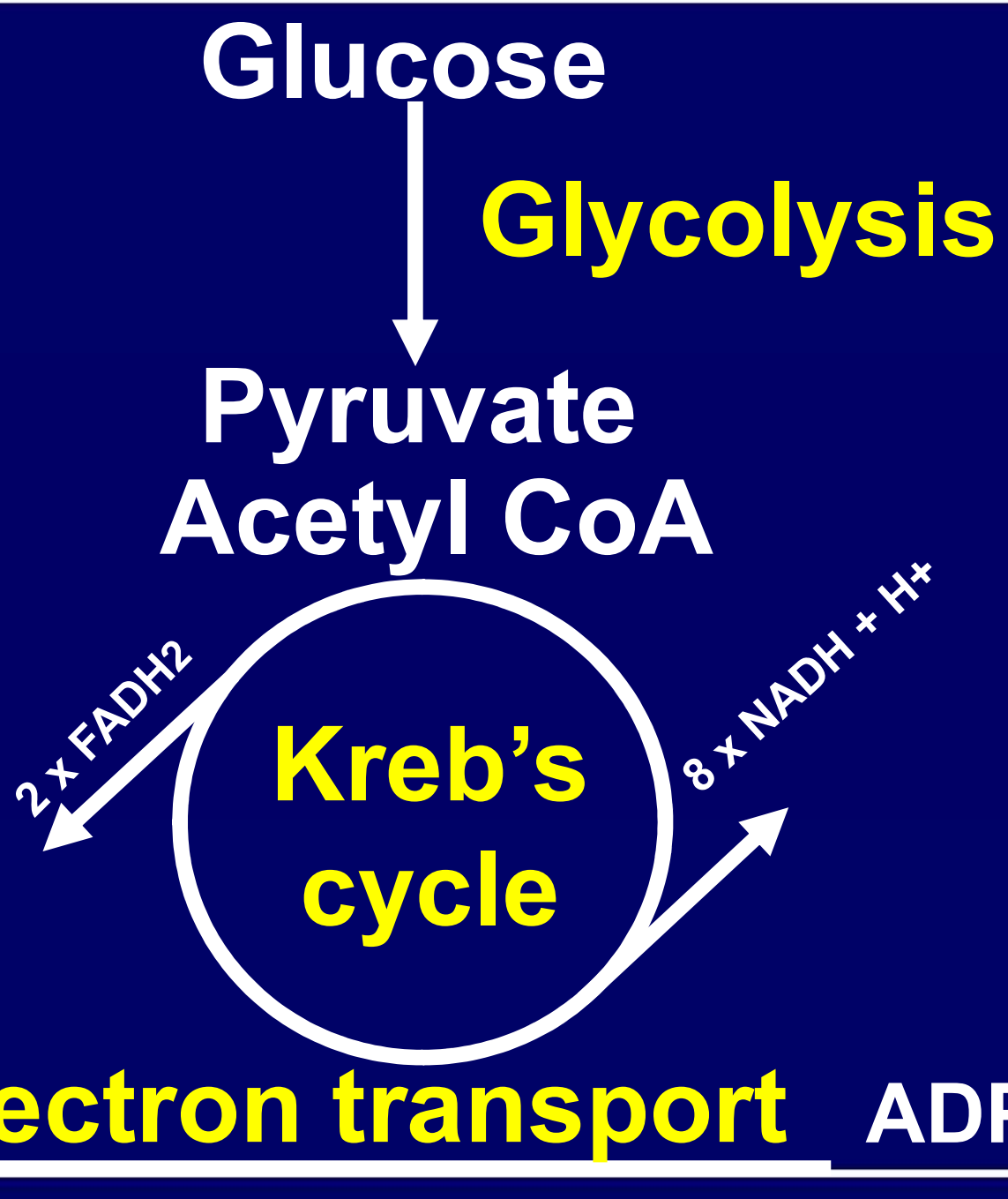

Glucose
Glycolysis
1
2
Kreb's
cycle
3
Electron transport

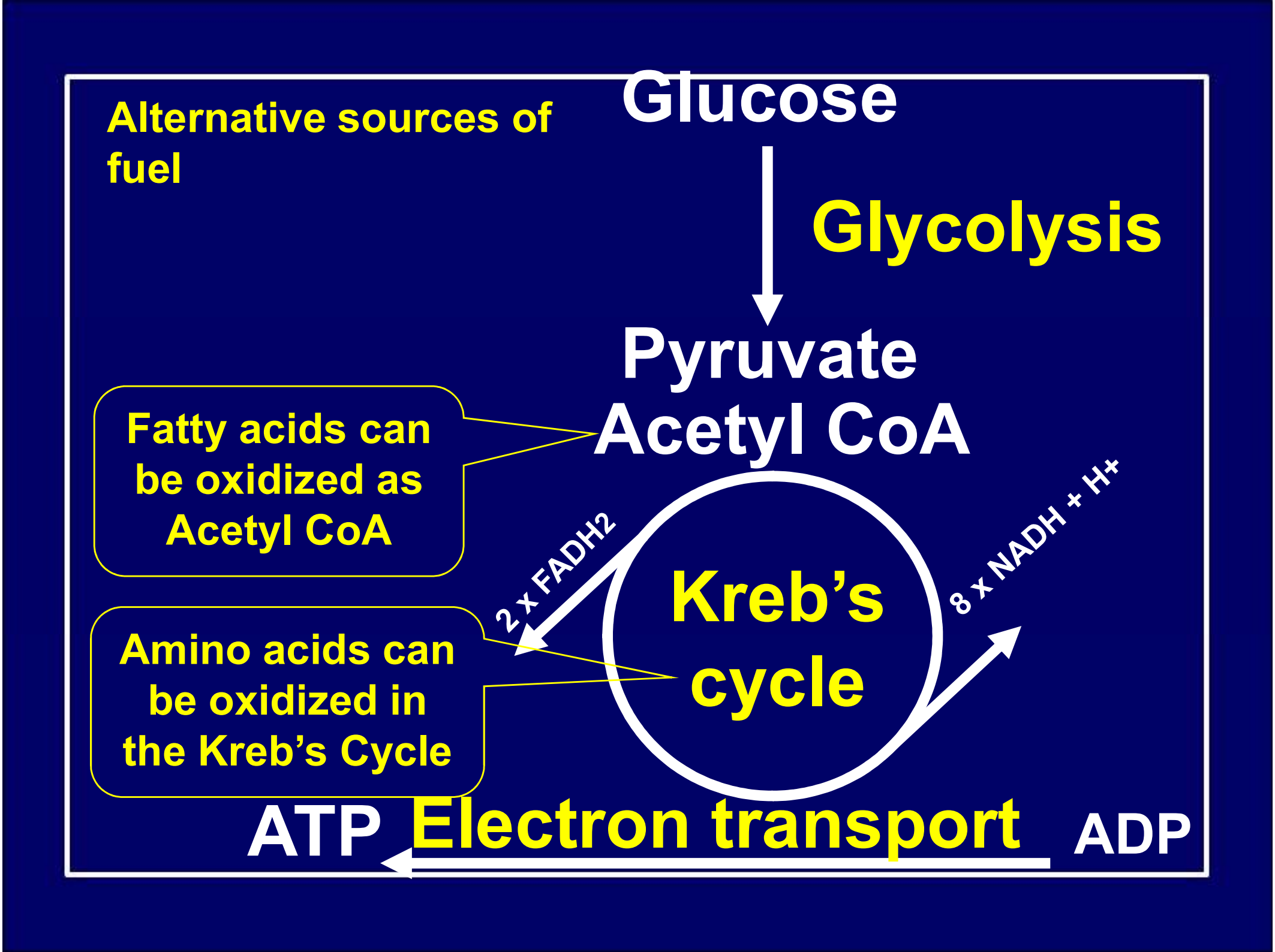
For every 1 molecule of Glucose **38 molecules of ATP** are formed.

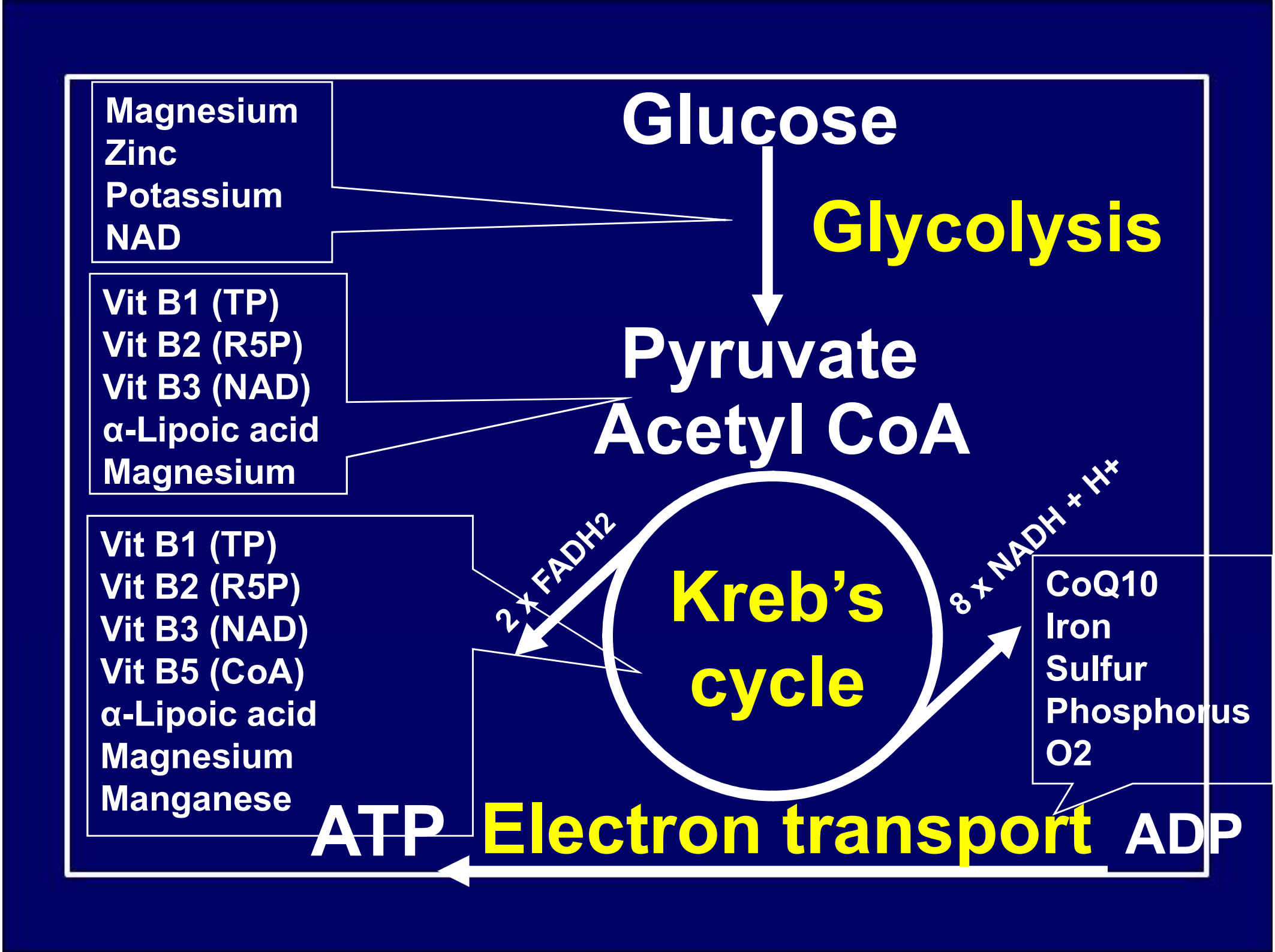
8 ATP by Glycolysis

2 ATP in the Kreb's cycle

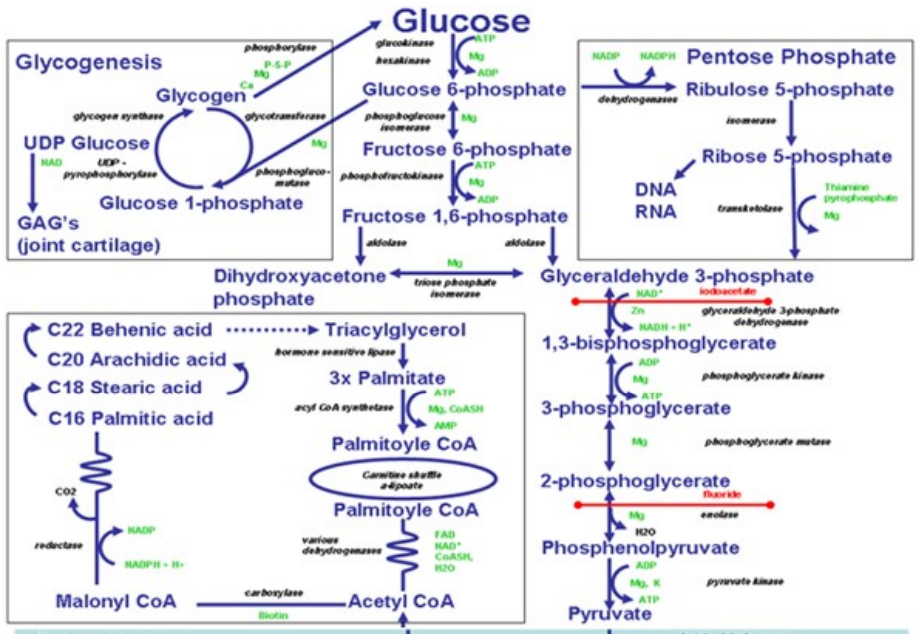
28 ATP by Electron transport



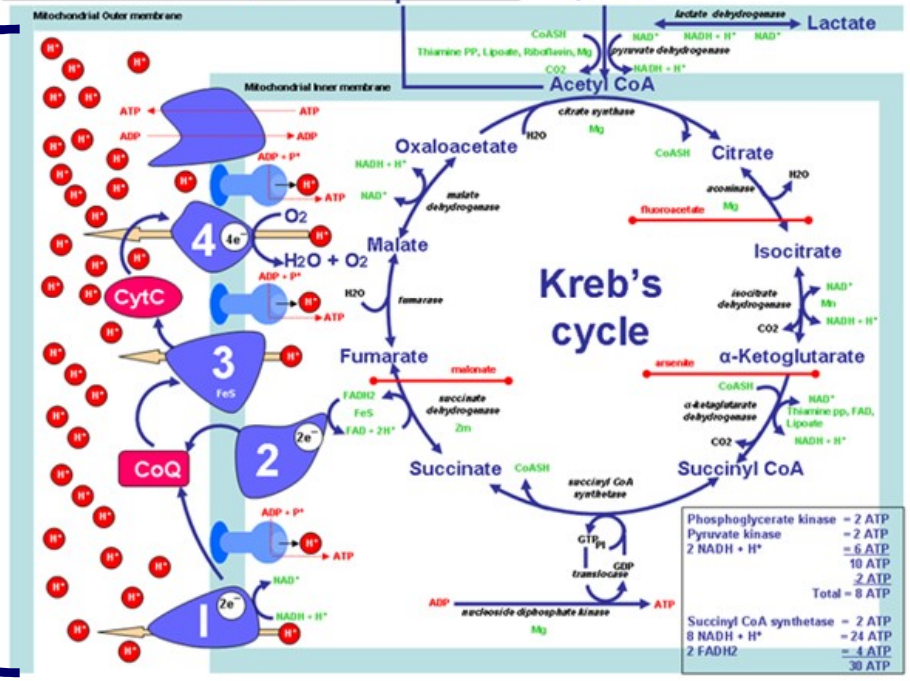

Alternative sources of fuel
Glucose
Glycolysis
Pyruvate
Acetyl CoA
Fatty acids can be oxidized as Acetyl CoA
Amino acids can be oxidized in the Kreb's Cycle
2 x FADH2
Kreb's cycle
8 x NADH + H+
ATP
Electron transport
ADP


Magnesium
Zinc
Potassium
NAD
Glucose
Glycolysis
Vit B1 (TP)
Vit B2 (R5P)
Vit B3 (NAD)
α-Lipoic acid
Magnesium
Pyruvate
Acetyl CoA
Vit B1 (TP)
Vit B2 (R5P)
Vit B3 (NAD)
Vit B5 (CoA)
α-Lipoic acid
Magnesium
Manganese
2 x FADH2
Kreb's cycle
8 x NADH + H+
CoQ10
Iron
Sulfur
Phosphorus
O2
ATP
Electron transport
ADP

Energy pathway



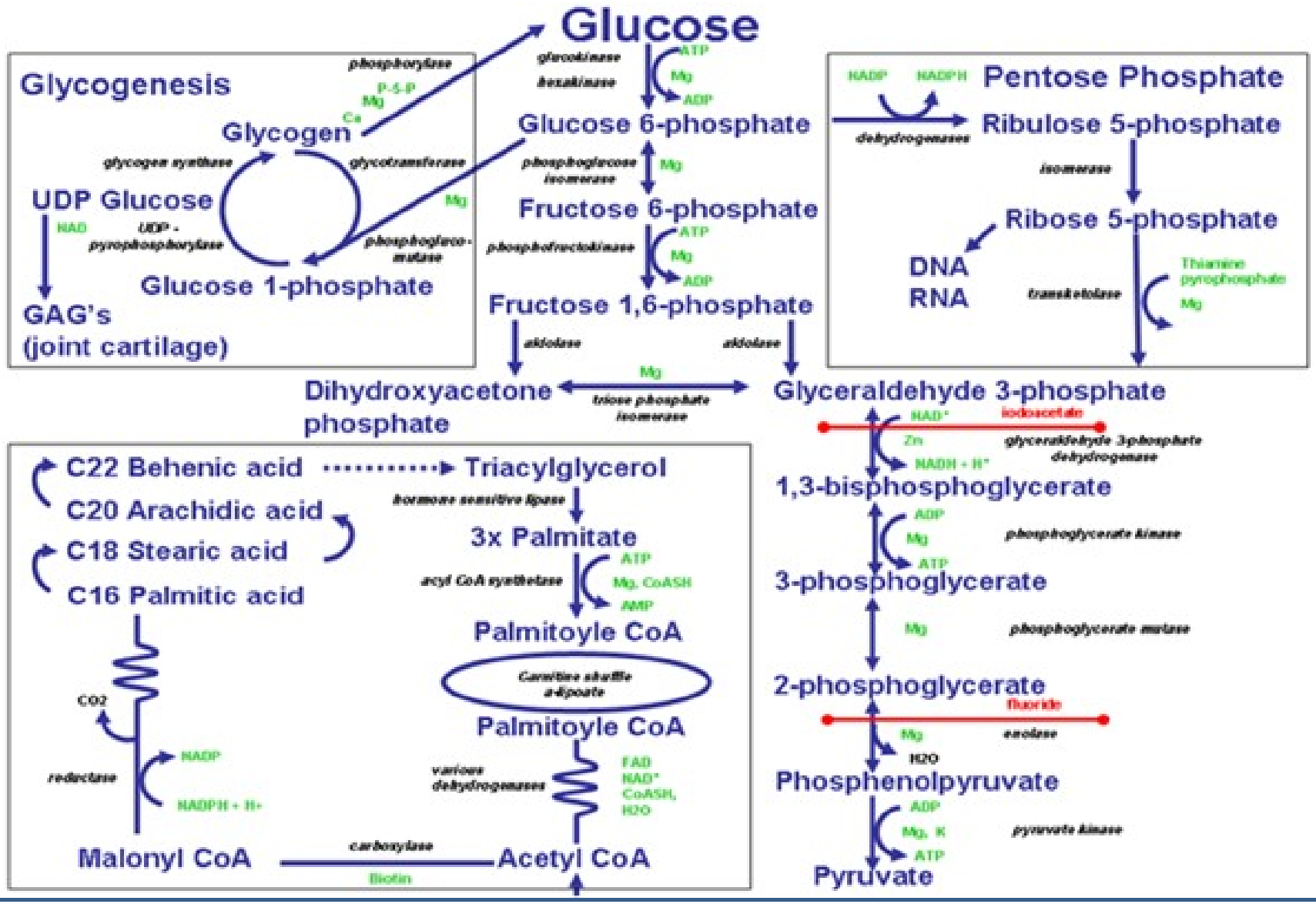
Glycolysis



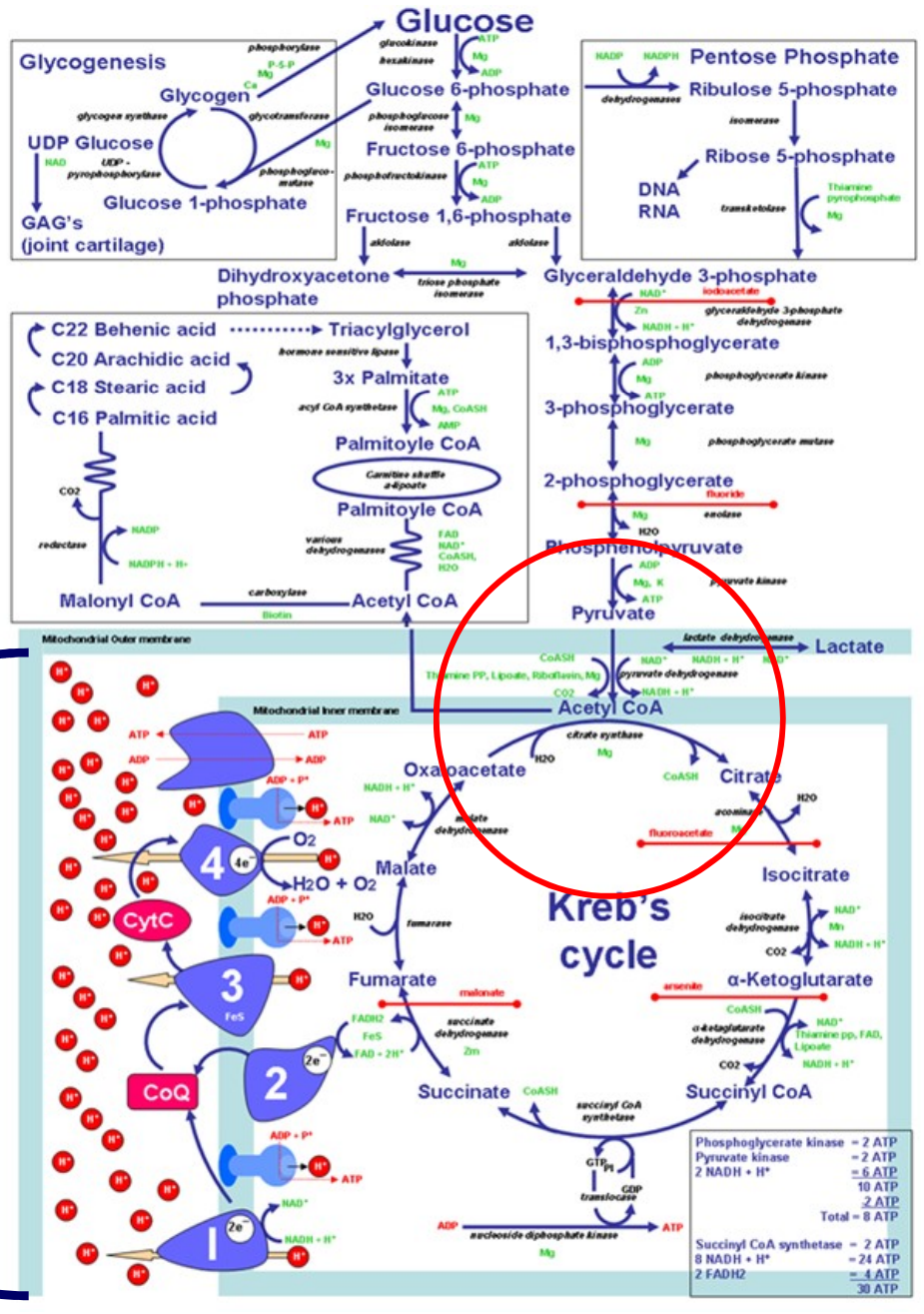
Electron transport or Oxidative phos-phorylation pathway

Citric Acid Cycle

Glycolysis


Glucose
glucokinase
hexokinase
ATP
Mg
ADP
Glycogenesis
phosphorylase
P-5-P
Mg
Ca
Glycogen
glycogen synthase
glycotransferase
UDP Glucose
NAD
UDP - pyrophosphorylase
phosphoglucomutase
Mg
Glucose 1-phosphate
GAG's
(joint cartilage)
Glucose 6-phosphate
phosphoglucose isomerase
Mg
Fructose 6-phosphate
phosphofructokinase
ATP
Mg
ADP
Fructose 1,6-phosphate
aldolase
aldolase
Dihydroxyacetone phosphate
Mg
triose phosphate isomerase
Glyceraldehyde 3-phosphate
NADP
NADPH
Pentose Phosphate
dehydrogenases
Ribulose 5-phosphate
isomerase
Ribose 5-phosphate
DNA
RNA
transketolase
Thiamine pyrophosphate
Mg
NAD+
Iodoacetate
Zn
NADH + H+
glyceraldehyde 3-phosphate dehydrogenase
1,3-bisphosphoglycerate
ADP
Mg
ATP
phosphoglycerate kinase
3-phosphoglycerate
Mg
phosphoglycerate mutase
2-phosphoglycerate
fluoride
Mg
enolase
H2O
Phosphenolpyruvate
ADP
Mg, K
ATP
pyruvate kinase
Pyruvate
C22 Behenic acid
C20 Arachidic acid
C18 Stearic acid
C16 Palmitic acid
Triacylglycerol
hormone sensitive lipase
3x Palmitate
acyl CoA synthetase
ATP
Mg, CoASH
AMP
Palmitoyle CoA
Carnitine shuttle
α-lipoate
Palmitoyle CoA
CO2
reductase
NADP
NADPH + H+
various dehydrogenases
FAD
NAD+
CoASH,
H2O
Malonyl CoA
carboxylase
Biotin
Acetyl CoA

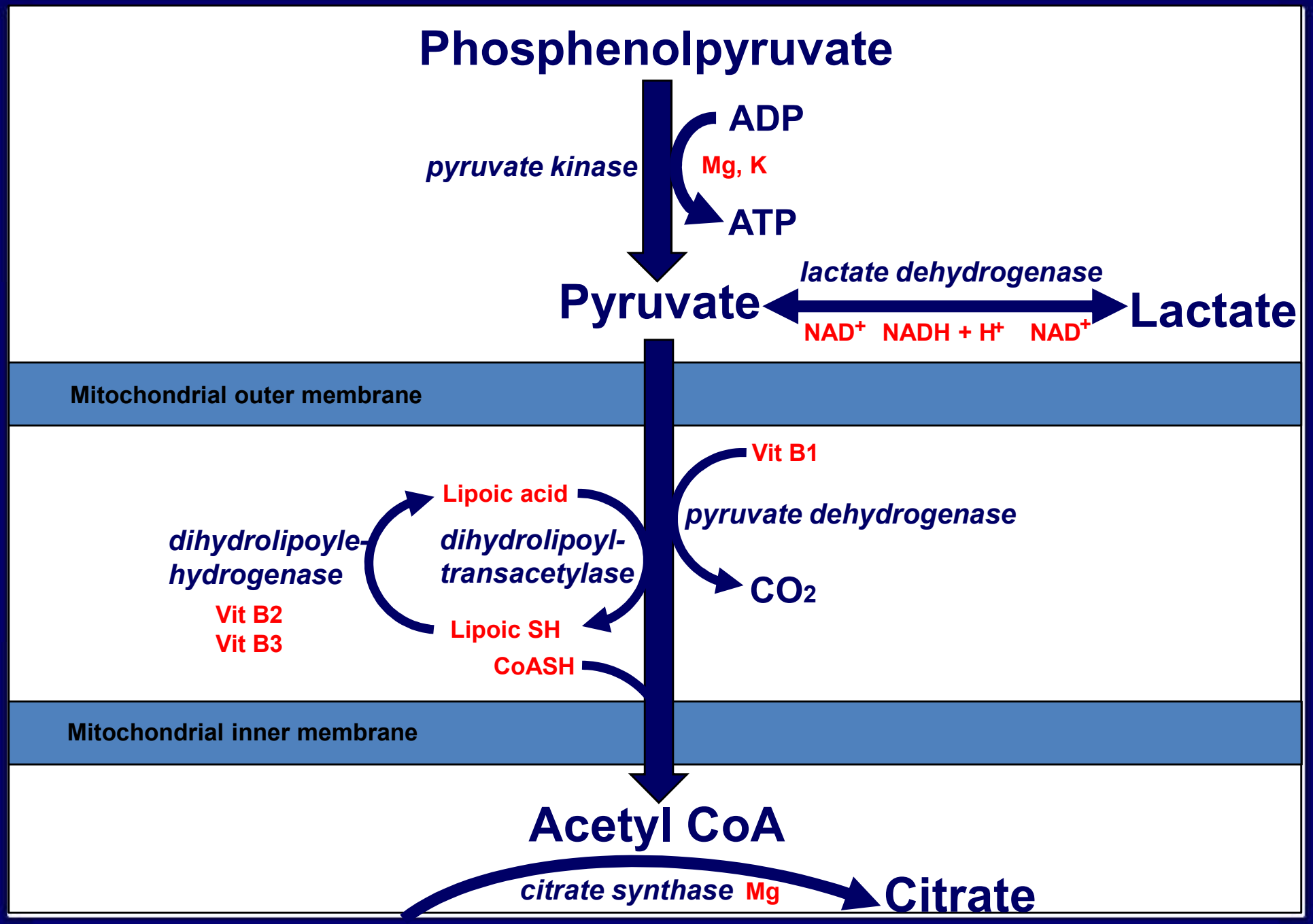
Energy pathway



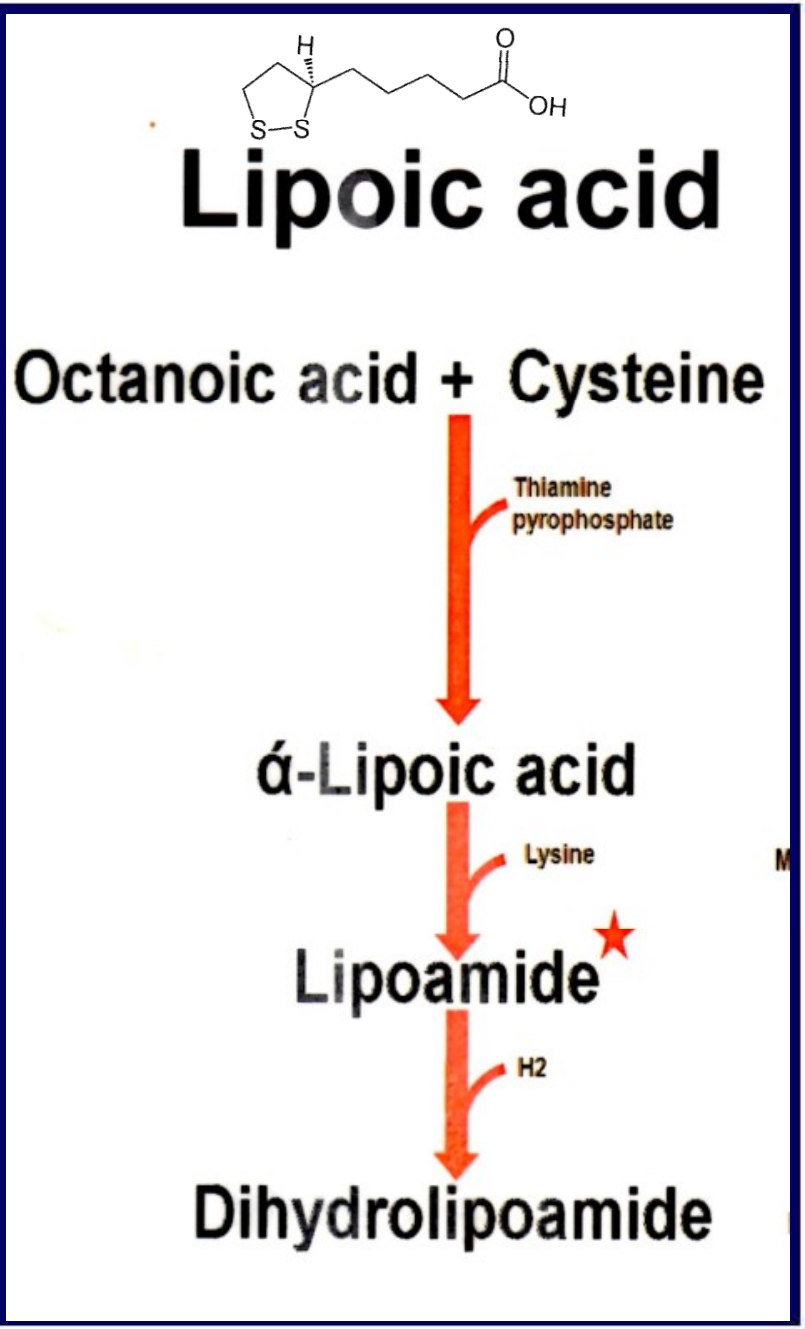
Glycolysis

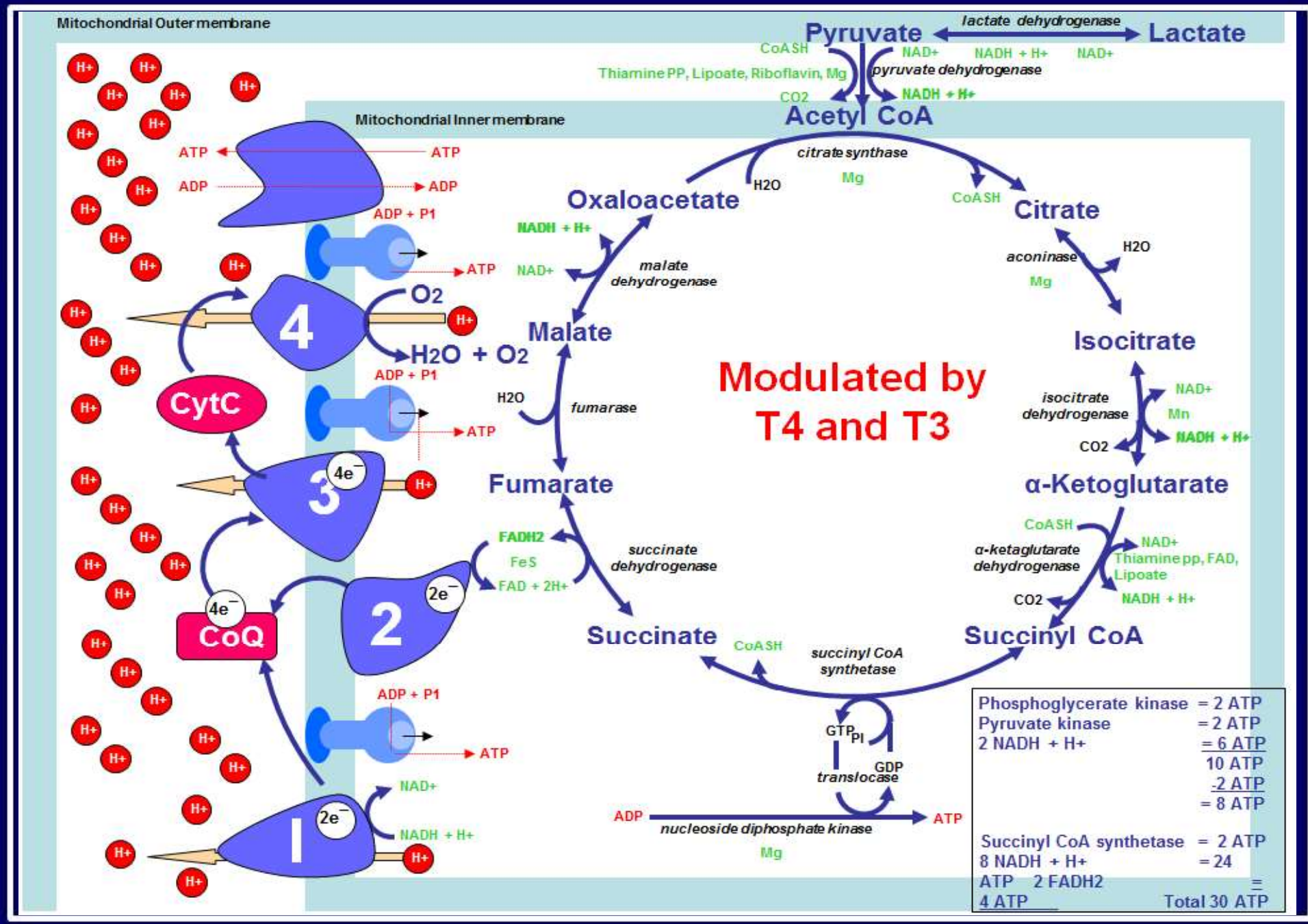
Electron transport or Oxidative phos-phorylation pathway

Citric Acid Cycle

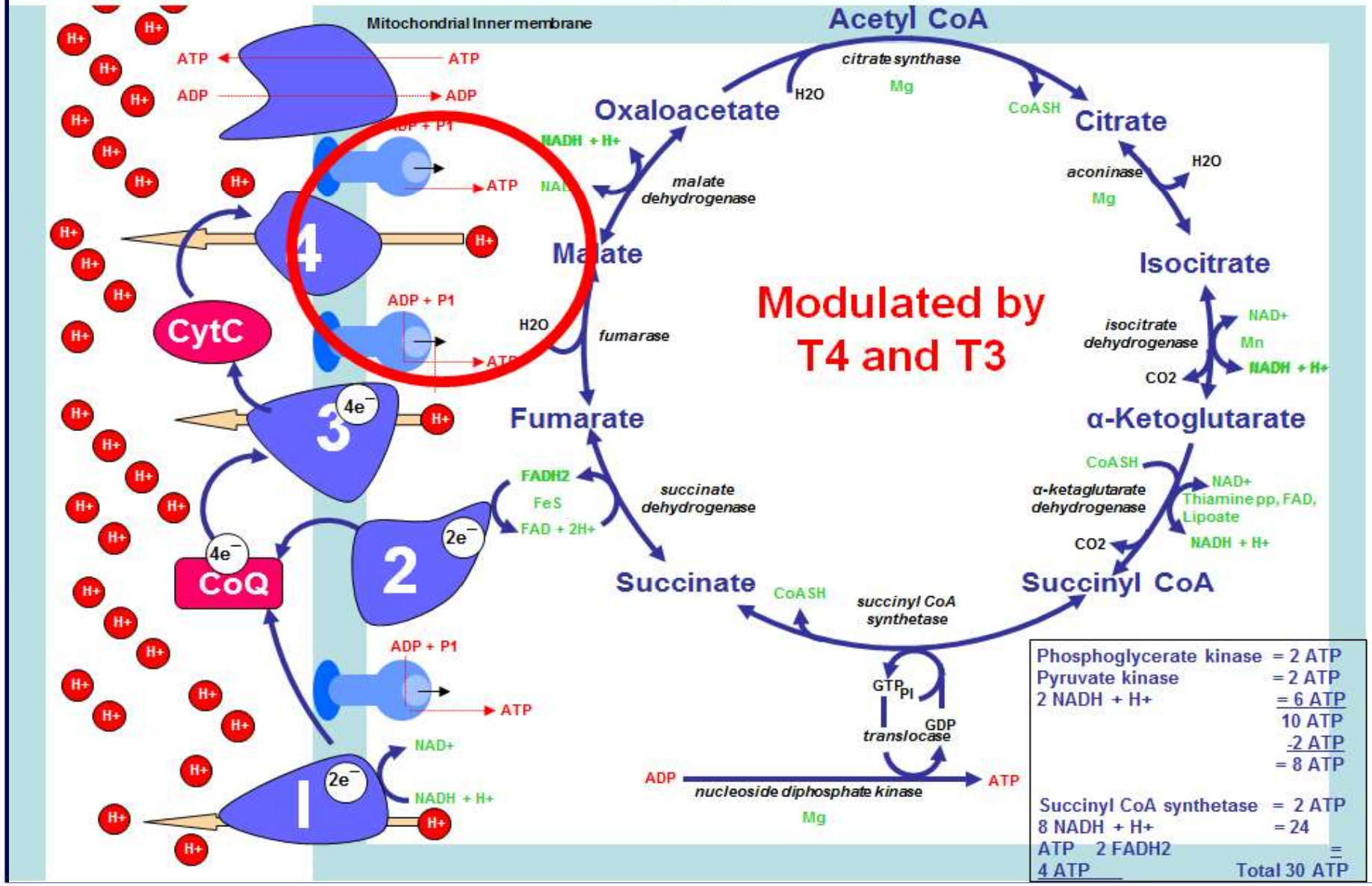

Phosphenolpyruvate
ADP
pyruvate kinase
Mg, K
ATP
lactate dehydrogenase
Pyruvate
Lactate
NAD+ NADH + H+ NAD+
Mitochondrial outer membrane
Vit B1
Lipoic acid
pyruvate dehydrogenase
dihydrolipoyle-hydrogenase
dihydrolipoyl-transacetylase
CO2
Vit B2
Vit B3
Lipoic SH
CoASH
Mitochondrial inner membrane
Acetyl CoA
citrate synthase Mg
Citrate

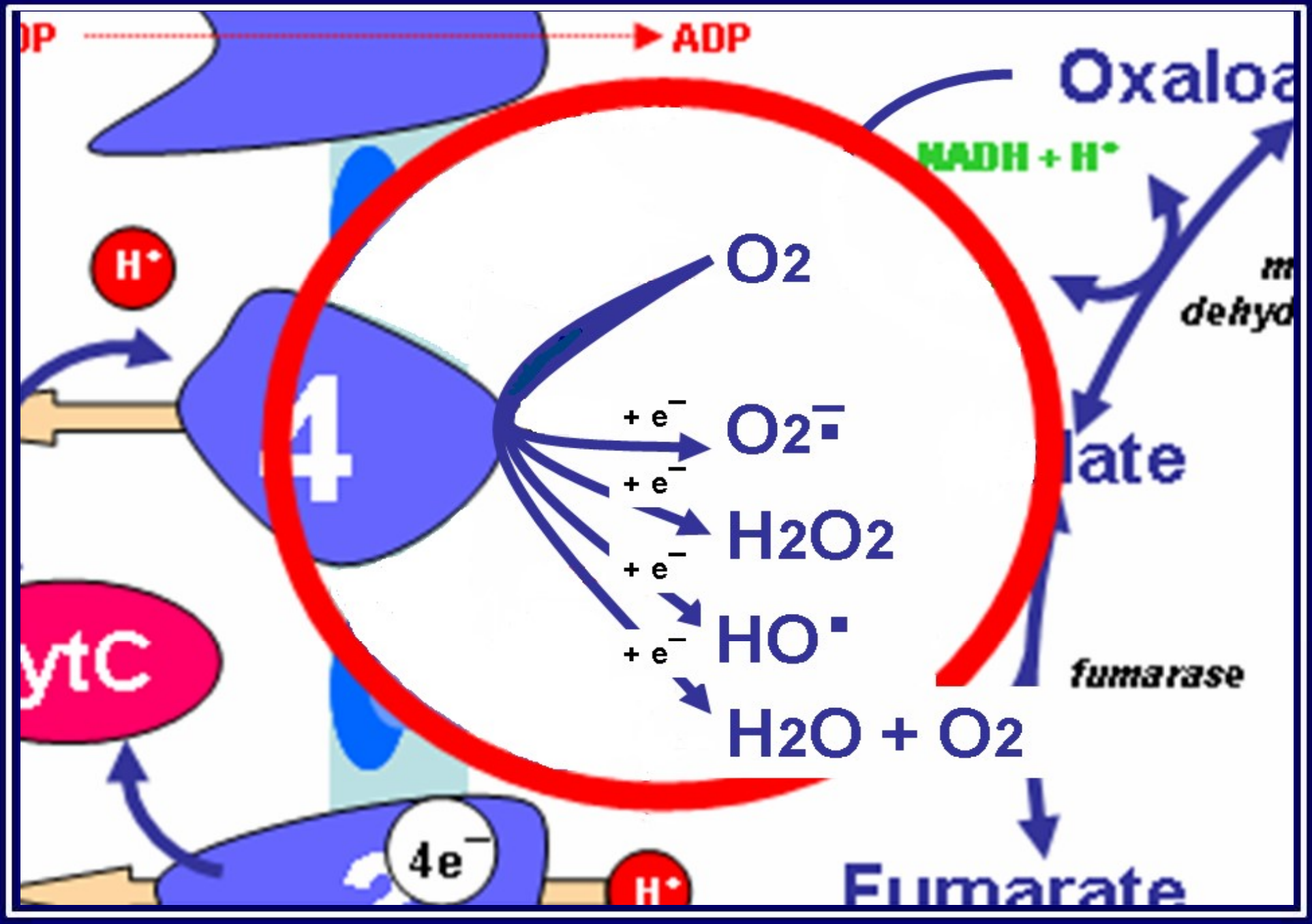
Alpha Lipoic Acid catalyses the conversion of Pyruvate to Acetyl CoA and the conversion of alpha Keto-glutarate to Succinyl CoA

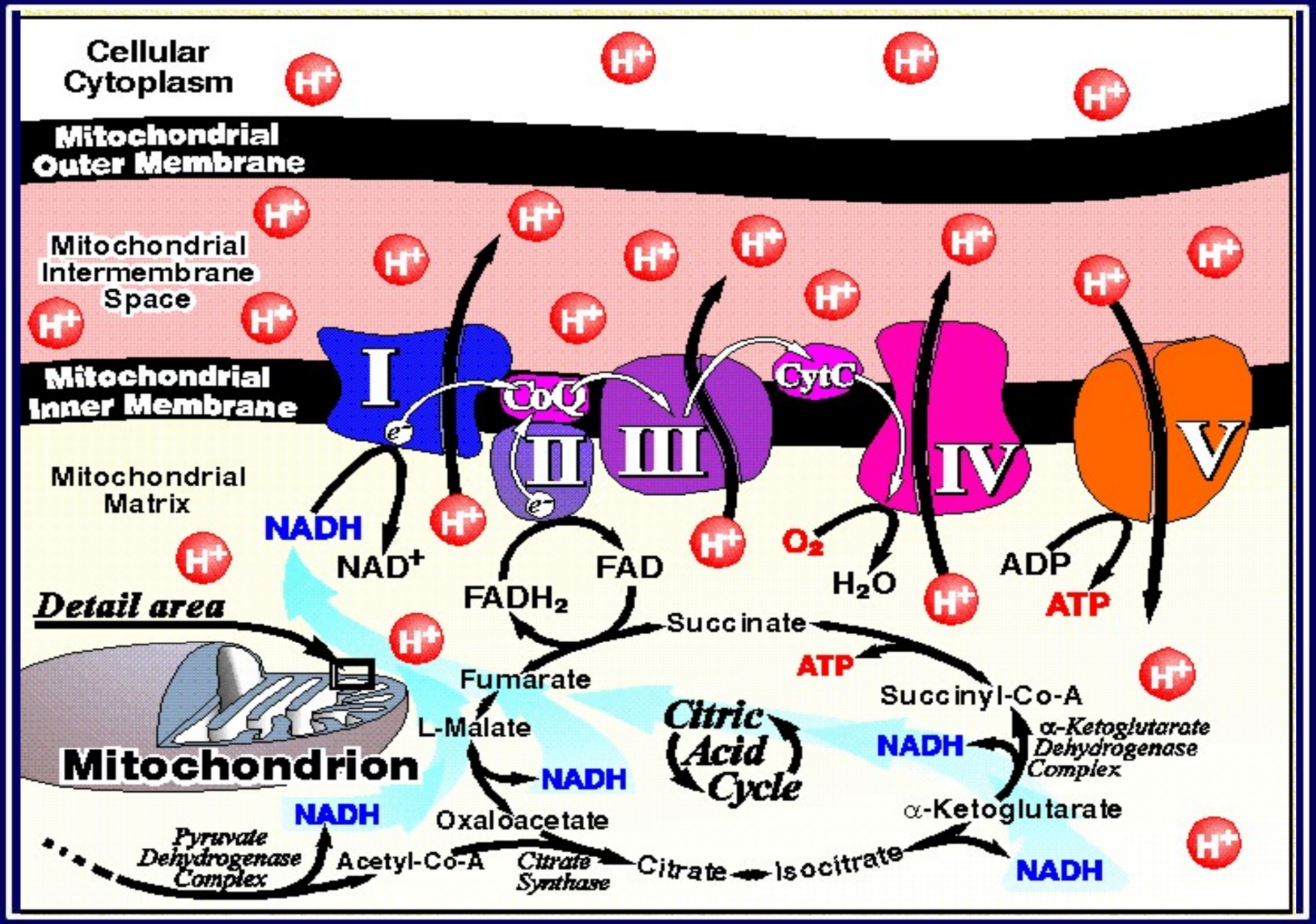



Mitochondrial Outer membrane
Mitochondrial Inner membrane
Pyruvate
lactate dehydrogenase
Lactate
CoASH
NAD+
NADH + H+
NAD+
Thiamine PP, Lipoate, Riboflavin, Mg
pyruvate dehydrogenase
CO2
NADH + H+
Acetyl CoA
citrate synthase
Mg
H2O
CoASH
Oxaloacetate
Citrate
NADH + H+
NAD+
malate dehydrogenase
aconinase
H2O
Mg
Malate
Isocitrate
Modulated by T4 and T3
H2O
fumarase
isocitrate dehydrogenase
NAD+
Mn
NADH + H+
CO2
Fumarate
α-Ketoglutarate
FADH2
FeS
FAD + 2H+
succinate dehydrogenase
CoASH
α-ketaglutarate dehydrogenase
NAD+
Thiamine pp, FAD, Lipoate
NADH + H+
CO2
Succinate
CoASH
succinyl CoA synthetase
Succinyl CoA
GTP
PI
GDP
translocase
ADP
ATP
nucleoside diphosphate kinase
Mg
ATP
ADP
ADP + P1
ATP
O2
H2O + O2
CytC
4e−
2e−
CoQ
NAD+
NADH + H+
H+
Phosphoglycerate kinase = 2 ATP
Pyruvate kinase = 2 ATP
2 NADH + H+ = 6 ATP
10 ATP
-2 ATP
= 8 ATP
Succinyl CoA synthetase = 2 ATP
8 NADH + H+ = 24
ATP 2 FADH2 =
4 ATP
Total 30 ATP

Reactive Oxygen Species

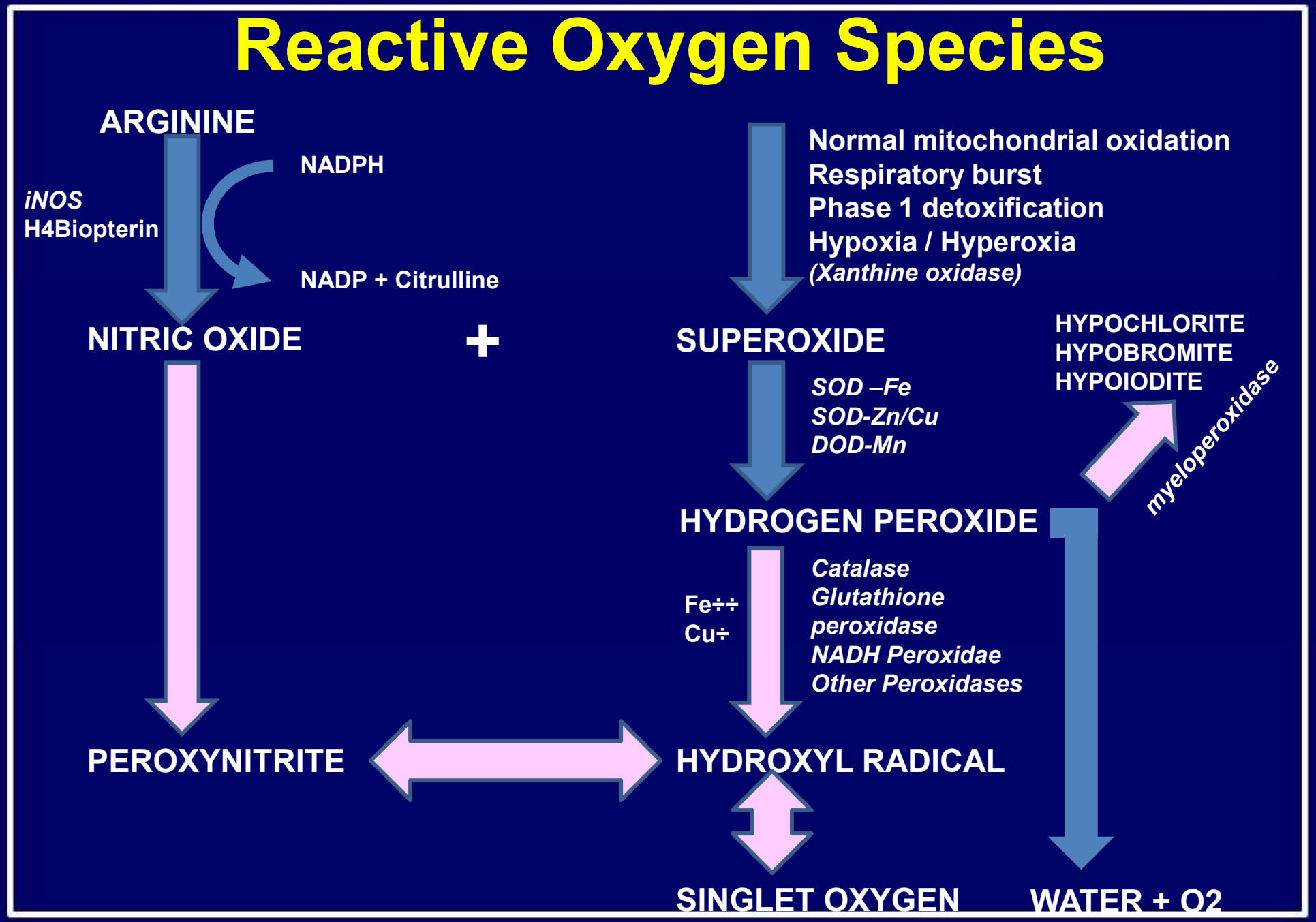



ADP
Oxaloa
NADH + H•
H•
O2
+ e−
O2−•
+ e−
H2O2
+ e−
HO•
+ e−
H2O + O2
4
ytC
4e−
H•
fumarase
Fumarate

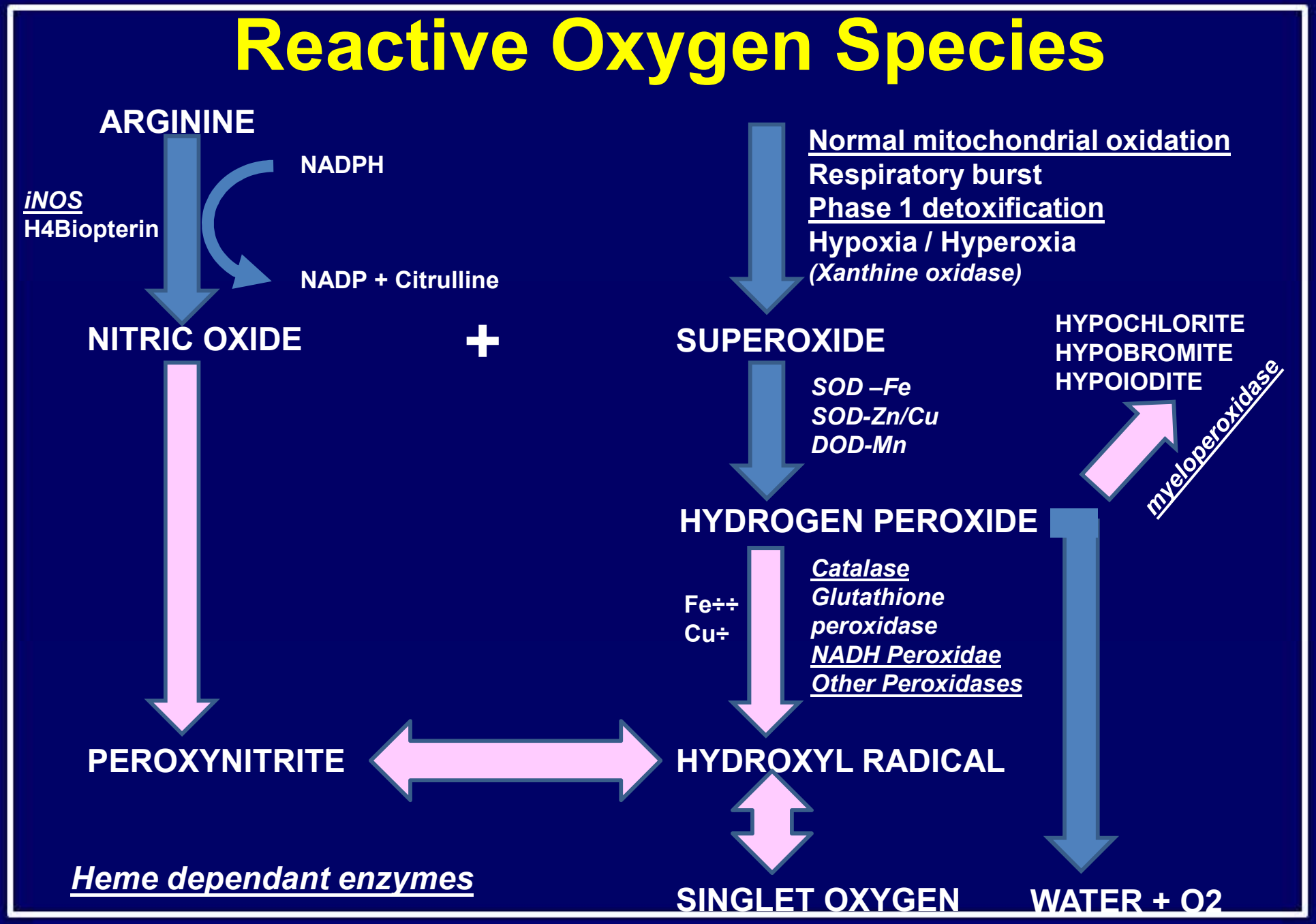

Cellular Cytoplasm
Mitochondrial Outer Membrane
Mitochondrial Intermembrane Space
Mitochondrial Inner Membrane
Mitochondrial Matrix
I
II
III
IV
V
CoQ
CytC
NADH
NAD+
FADH2
FAD
O2
H2O
ADP
ATP
Detail area
Mitochondrion
Succinate
Fumarate
L-Malate
Oxaloacetate
Citric Acid Cycle
Succinyl-Co-A
α-Ketoglutarate Dehydrogenase Complex
α-Ketoglutarate
Pyruvate Dehydrogenase Complex
Acetyl-Co-A
Citrate Synthase
Citrate
Isocitrate
H+

Reactive Oxygen Species

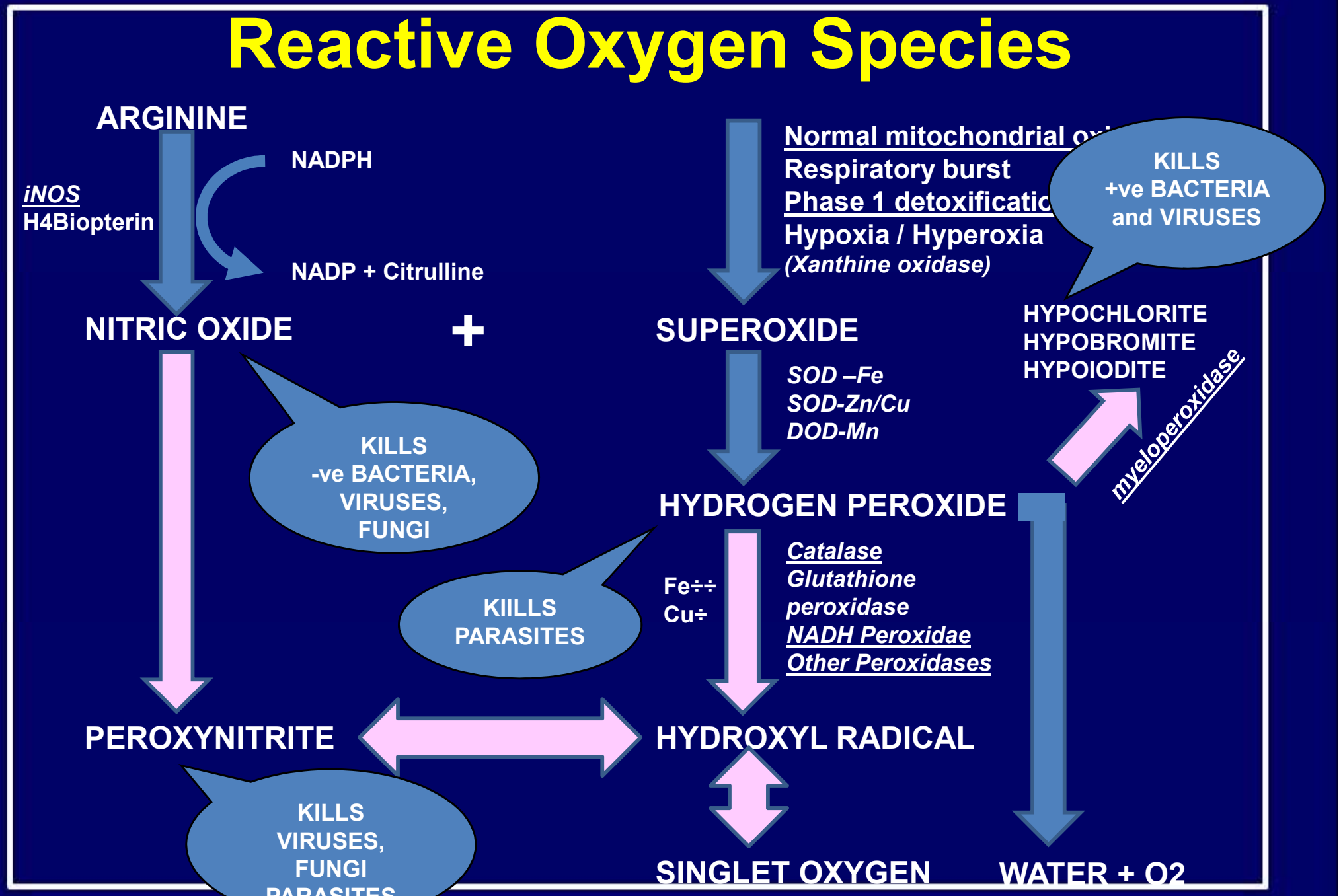
Reactive Oxygen Species



Reactive Oxygen Species



Reactive Oxygen Species



Challenging for Energy

1. Weak muscle strengthens to ATP
2. Strong muscle weakens to ADP

For Glycolysis challenge for strengthening against Pyruvate

For Krebs cycle challenge

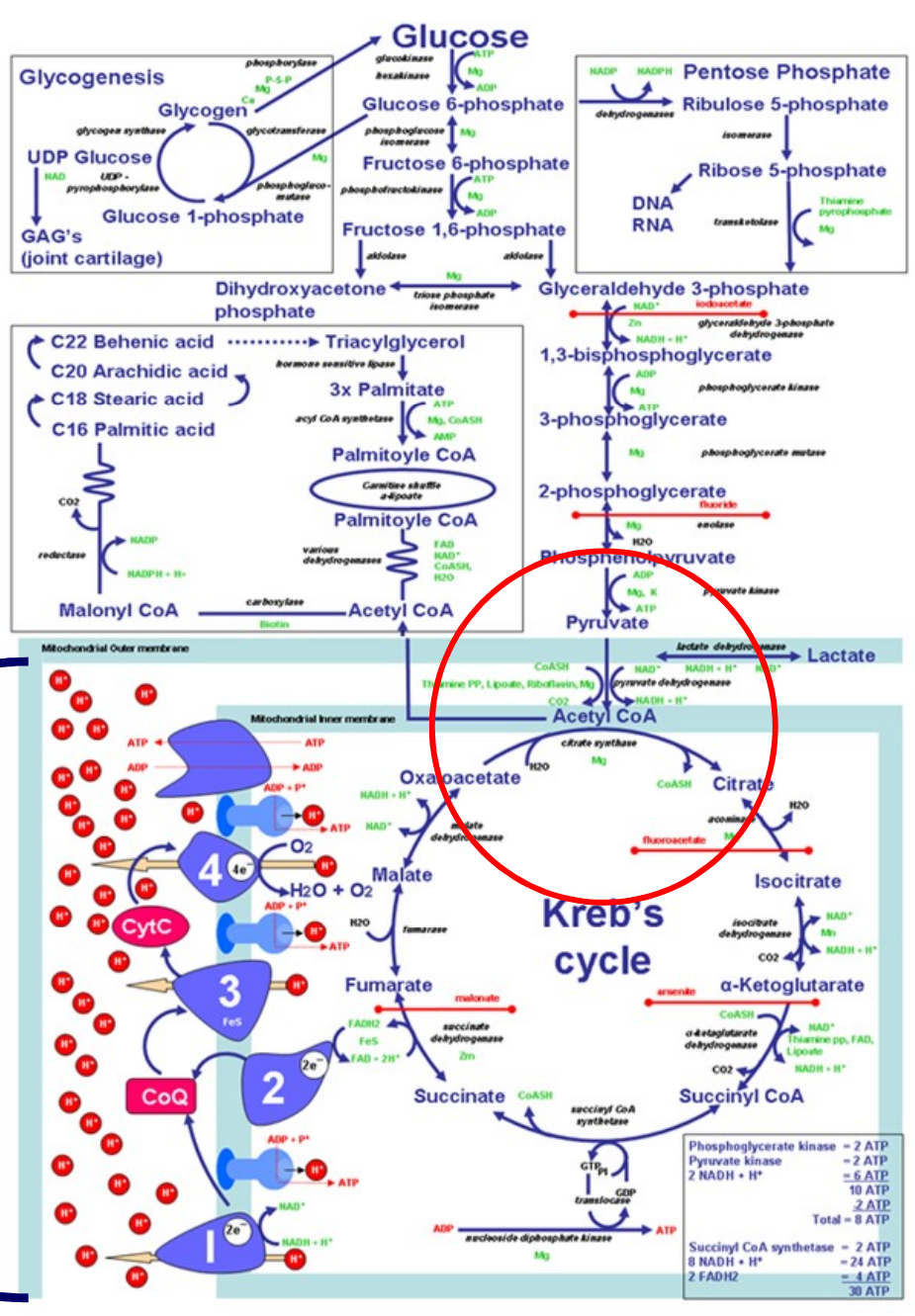
for strengthening against NADH or FADH2

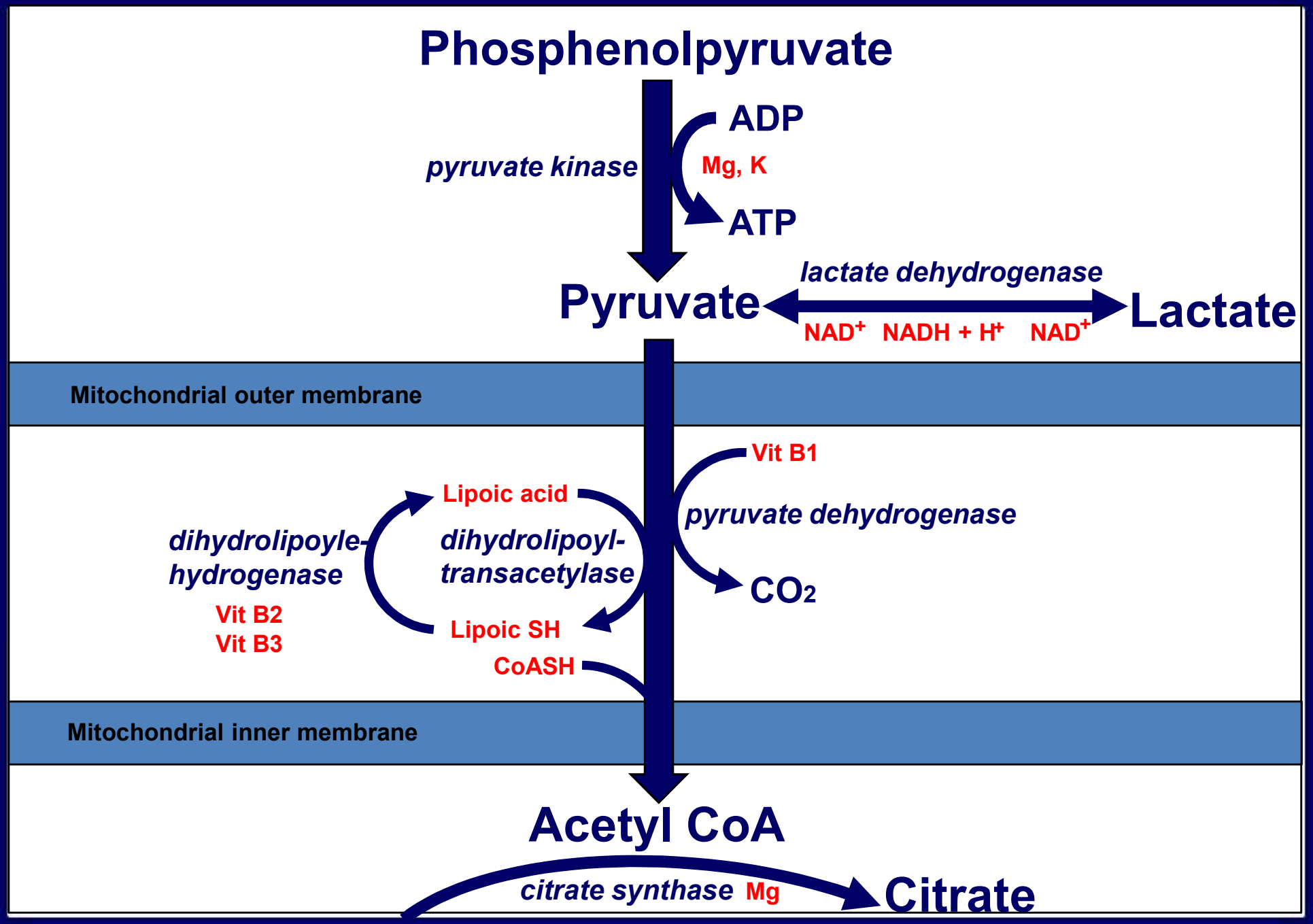
Challenge against individual Krebs cycle intermediates

For Electron Transport challenge

for strengthening against
Complex 1 (NADH)
Complex 11 (FADH2)
Complex 111 (CoQ10)
Complex IV (cytochrome c oxidase)

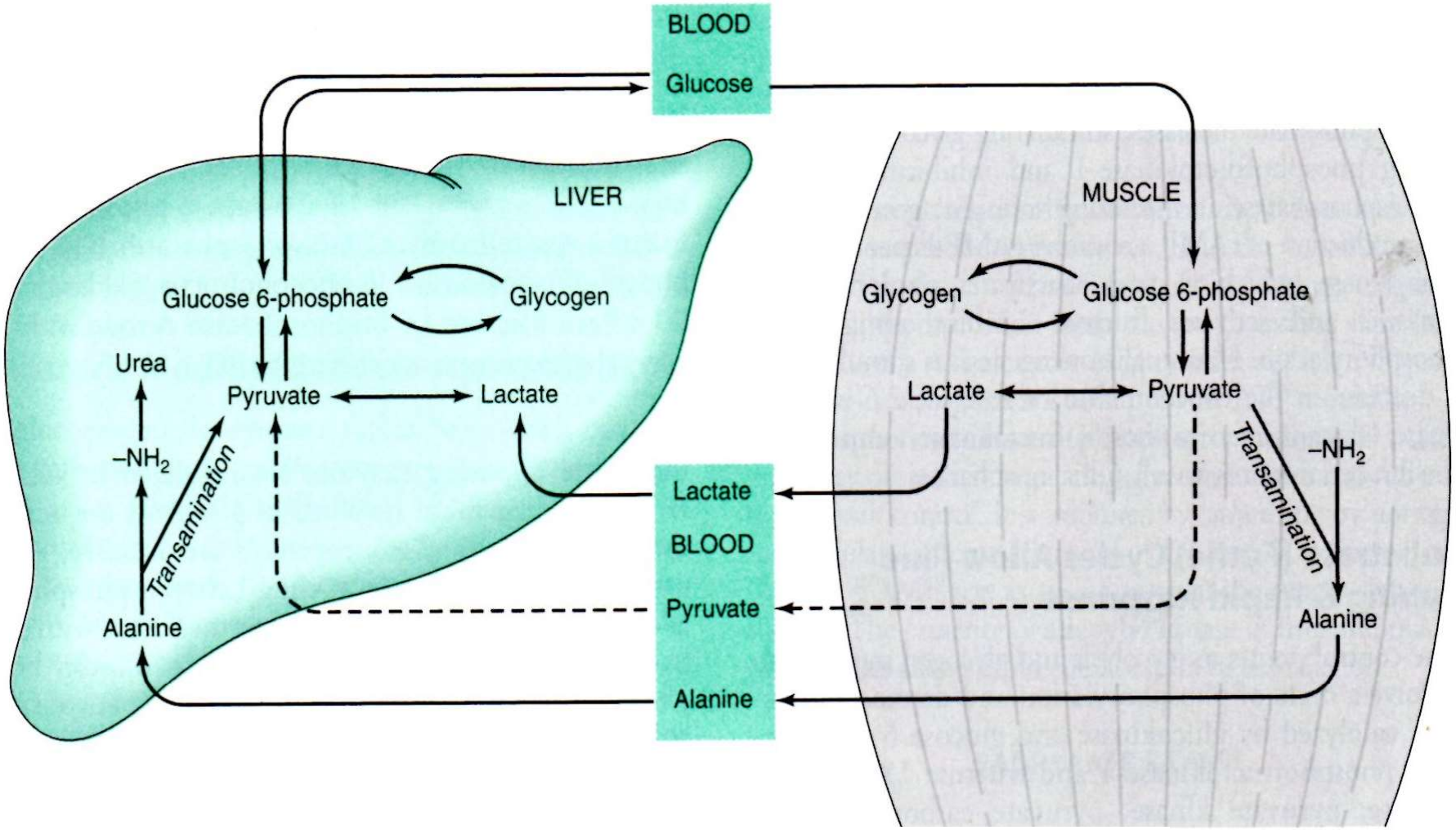
Energy pathway




Phosphenolpyruvate
ADP
pyruvate kinase
Mg, K
ATP
lactate dehydrogenase
Pyruvate
Lactate
NAD+ NADH + H+ NAD+
Mitochondrial outer membrane
Vit B1
Lipoic acid
pyruvate dehydrogenase
dihydrolipoyle-hydrogenase
dihydrolipoyl-transacetylase
CO2
Vit B2
Vit B3
Lipoic SH
CoASH
Mitochondrial inner membrane
Acetyl CoA
citrate synthase Mg
Citrate

Lactic acid

Tissues that function under hypoxic conditions produce lactic acid.



The Lactic Acid (Cori Cycle)

Under anaerobic conditions NADH cannot be reoxidized through the respiratory chain to oxygen. Pyruvate is reduced by NADH to lactate catalysed by *lactate dehydrogenase*. There are three different specific isoenzymes of *lactate dehydrogenase* that have clinical significance.

The re-oxidation of NADH via lactate formation allows glycolysis to proceed in the absence of oxygen by regenerating sufficient NAD for another cycle of the reaction catalysed by *glyceraldehyde-3-phosphate dehydrogenase*.

Some tissues derive much of their energy from glycolysis and produce lactate –

Erythrocytes	Brain
GI tract	Renal medulla
Retina	Skin

The liver, kidney and heart usually take up lactate and oxidize it but will produce it under hypoxic conditions

SOURCES OF FUEL IN EXERCISE

ANAEROBIC	AEROBIC
Type 11 (glycolytic white) fibres are used predominantly	Type 1 (oxidative red) fibres are used predominantly
1-5 seconds Creatine phosphate is the major energy source	First 4 minutes blood glucose 4-18 minutes liver glycogen
5-10 seconds Glucose derived from muscle glycogen is metabolised by anaerobic glycolysis leading to lactic acid formation	18-70 minutes muscle glycogen 70-4000 minutes Adipose tissue triglycerides
Rapid depletion of muscle glycogen	Gradual depletion of muscle glycogen

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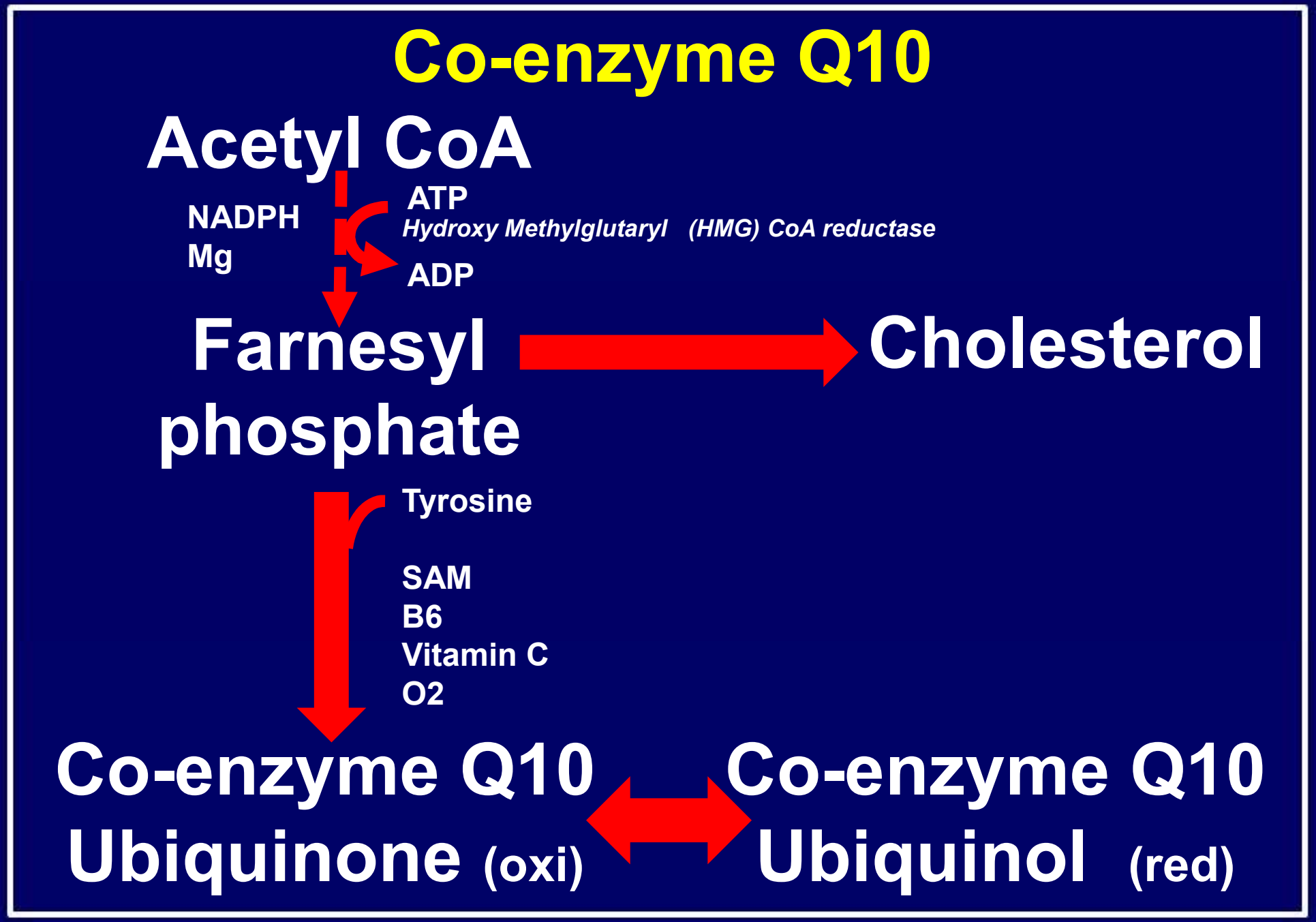
AEROBIC		ANAEROBIC	
GLYCOLYSIS	ATP YIELD	GLYCOLYSIS FROM GLUCOSE	ATP YIELD
Phosphoglycerate kinase	2	Phosphoglycerate kinase	2
Pyruvate kinase	2	Pyruvate kinase	2
Glyceraldehyde 3- phos. Dehydrogenase	6	Minus 2 ATP to activate glycolysis	-2
total	10	Total	2
KREBS CYCLE		GLYCOLYSIS FROM GLYCOGEN	4
Succinyl CoA synthetase	2	Minus 1 ATP to activate glycolysis	-1
total	2	Total	3
OXIDATIVE PHOSPHORYLATION		Grand total	5
8 NADH+H+	24		
2 FADH2	4		
total	28		
Grand total	40		
Minus 2 ATP to activate glycolysis	38		

Challenges

ANAEROBIC	AEROBIC
Contract muscle twice per second 10x and then retest for weakening.	Contract muscle once per second 10x and then retest for weakening.

Co-Enzyme Q10
Ubiquinone - Ubiquinol


Co-enzyme Q10
Acetyl CoA
NADPH
Mg
ATP
Hydroxy Methylglutaryl (HMG) CoA reductase
ADP
Farnesyl phosphate
Cholesterol
Tyrosine
SAM
B6
Vitamin C
O2
Co-enzyme Q10 Ubiquinone (oxi)
Co-enzyme Q10 Ubiquinol (red)

Co-enzyme Q10 is a fat-soluble substance, is present in most eukaryotic cells, primarily in the mitochondria. It is a component of the electron transport chain and participates in aerobic cellular respiration, which generates energy in the form of ATP.*

* Ernster, L.; Dallner, G. (1995). "Biochemical, physiological and medical aspects of ubiquinone function". *Biochimica et Biophysica Acta*. 1271 (1): 195–204

Ninety-five percent of the human body's energy is generated this way.* Therefore, those organs with the highest energy requirements—such as the brain, heart, liver, and kidney—have the highest CoQ_{10} concentrations.

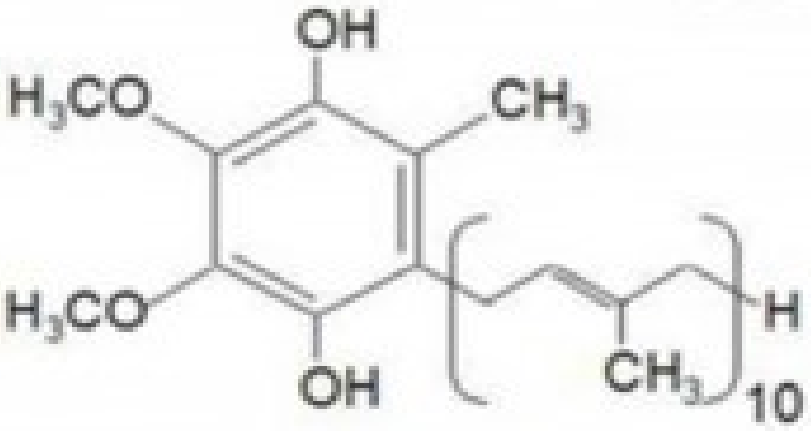
* Okamoto, T.; Matsuya, T.; Fukunaga, Y.; Kishi, T.; Yamagami, T. (1989). "Human serum ubiquinol-10 levels and relationship to serum lipids". *International Journal for Vitamin and Nutrition Research*. 59(3): 288–292.

There are three redox states of CoQ_{10}: fully oxidized (ubiquinone), semiquinone (ubisemiquinone), and fully reduced (ubiquinol).
The capacity of this molecule to act as a two-electron carrier (moving between the quinone and quinol form) and-

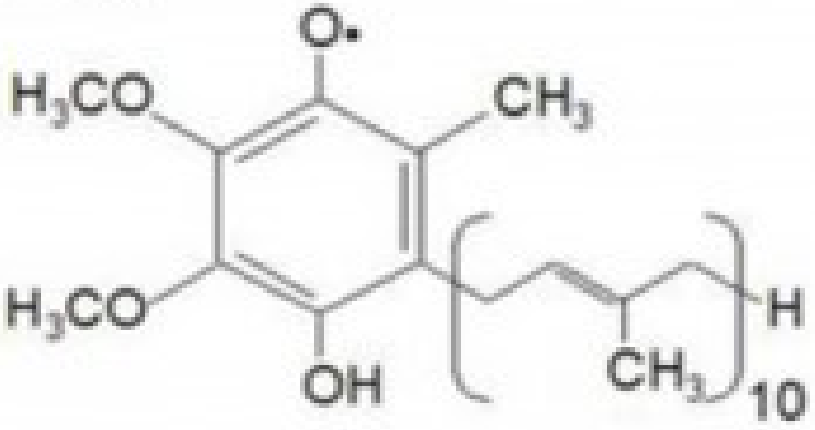
a one-electron carrier (moving between the semiquinone and one of these other forms) is central to its role in the electron transport chain due to the iron–sulfur clusters that can only accept one electron at a time, and as a free radical-scavenging antioxidant.*

** Aberg, F.; Appelkvist, E. L.; Dallner, G.; Ernster, L. (1992). "Distribution and redox state of ubiquinones in rat and human tissues". Archives of Biochemistry and Biophysics. 295 (2): 230–234.*

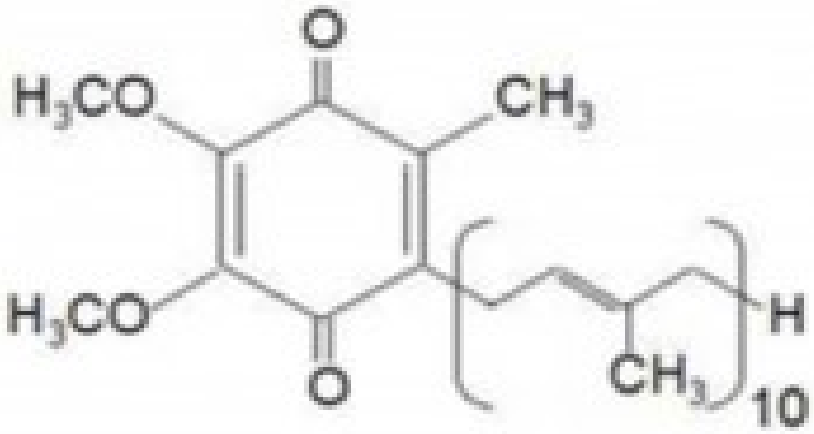
Coenzyme Q_{10}



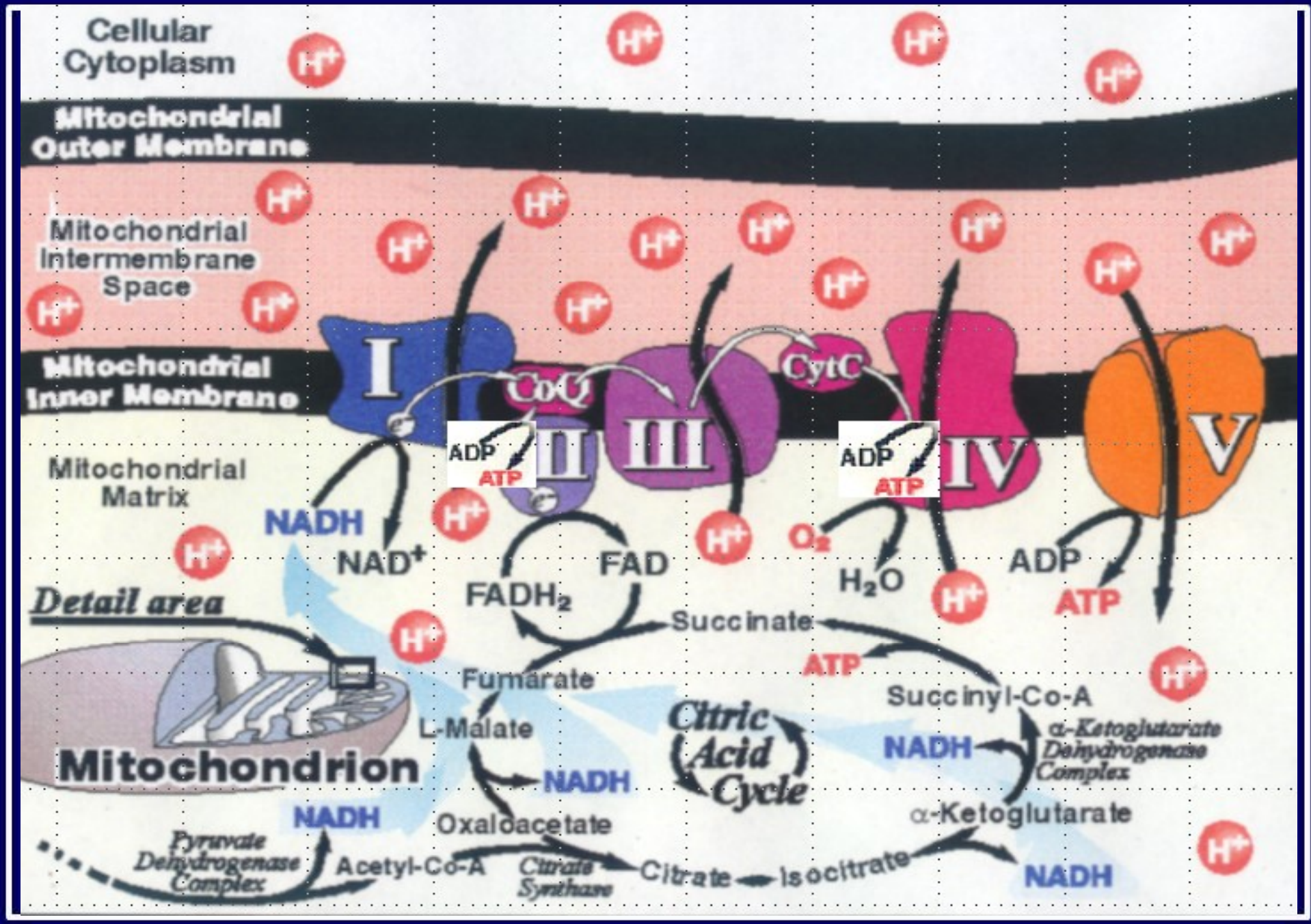
Ubiquinol ($CoQH_2$)



Semiquinone radical ($CoQH^{\cdot}$)



Ubiquinone (CoQ)


Cellular Cytoplasm
Mitochondrial Outer Membrane
Mitochondrial Intermembrane Space
Mitochondrial Inner Membrane
Mitochondrial Matrix
H+
I
II
III
IV
V
CoQ
CytC
ADP
ATP
NADH
NAD+
FAD
FADH2
O2
H2O
Detail area
Mitochondrion
Succinate
Fumarate
L-Malate
Oxaloacetate
Citric Acid Cycle
Succinyl-Co-A
α-Ketoglutarate Dehydrogenase Complex
α-Ketoglutarate
Pyruvate Dehydrogenase Complex
Acetyl-Co-A
Citrate Synthase
Citrate
Isocitrate

CoQ deficiency in humans

Basis	Tissue	% decrease
Age	Myocardium	72
Age	Heart	58
Age	Pancreas	83
Age	Adrenal	50
Age	Kidney	45
Age	Epidermis	75
Age	Liver	17
Statins	Serum	20-30
Diabetes	Serum	65

Food Sources of CoQ10

There are foods with CoQ10 that you can eat if you want to increase your body's levels. It's said that if you consume a balanced diet, you're likely to get enough CoQ10. Notable examples include:[6,7]

- Fish like wild-caught Alaskan salmon and herring
- Grass fed beef and organ meats
- Organic pastured meats
- Sesame seeds
- Broccoli

CoQ10's Potential Capabilities for Your Health

Lately, research has highlighted the impact of eating chlorophyll-rich vegetables and sun exposure in improving the body's conversion of CoQ10 to ubiquinol. Chlorophyll that's consumed is transported to the blood, and once the skin is exposed to significant amounts of sunlight, chlorophyll absorbs solar radiation and promotes CoQ10 conversion into ubiquinol. You can increase your chlorophyll intake by eating these vegetables:[5]

Spinach	Asparagus	Beet greens
Green bell peppers	Bok choy	Brussels sprouts
Green cabbage	Celery	Collard greens
Cucumber	Green beans	Green peas
Kale	Leeks	Mustard greens
Green sea vegetables	Swiss chard	Turnip greens

Fruits like green grapes or kiwis (provided that these are eaten in moderation), as well as parsley and pistachio nuts, are other chlorophyll-rich foods to consider.

Coenzyme Q_{10} has potential to inhibit the effects of warfarin* (Coumadin), a potent anticoagulant, by reducing the INR, a measure of blood clotting.

* *Wyman, M.; Leonard, M.; Morledge, T. (Jul 2010). "Coenzyme Q_{10}: a therapy for hypertension and statin-induced myalgia?". Cleveland Clinic Journal of Medicine. 77 (7): 435–442.*

The structure of coenzyme Q_{10} is very much similar to the structure of Vitamin K, which competes with and counteracts warfarin's anticoagulation effects. Coenzyme Q_{10} should be avoided in patients currently taking warfarin due to the increased risk of clotting.*

** Wyman, M.; Leonard, M.; Morledge, T. (Jul 2010). "Coenzyme Q_{10}: a therapy for hypertension and statin-induced myalgia?". Cleveland Clinic Journal of Medicine. 77 (7): 435–442.*

- it is indispensable for producing energy in the cells in the form of ATP
- it is an essential fat soluble antioxidant
- it helps regenerate other antioxidants especially Vit E
- it stimulates cell growth and inhibits cell death

- It is beneficial for the prevention of cell damage in hypoxia, especially in the cardiac muscle. It has been used for the protection of myocardium in different cardiovascular disorders, such as angina pectoris, hypertension, arrhythmia and congestive heart failure.

- It has been proven to have anti-tumour and immune system enhancing properties when tested in animals.
- Genetic mutations, ageing, cancer and statin-type drugs can cause a decrease in the levels of coenzyme Q10 in tissues and blood.

- Low ratio of coenzyme Q10 to low-density lipoprotein (LDL) cholesterol is a strong indicator of risk of atherosclerosis (clogging of the arteries)

Sources*

Beef heart, liver and muscle, Pork heart liver and muscle, sardines, Red fish, Soy bean, Olive, Grape seed and Rapeseed oils

*Pravst, Igor; Žmitek, Katja; Žmitek, Janko (2010). "Coenzyme Q_{10} Contents in Foods and Fortification Strategies". *Critical Reviews in Food Science and Nutrition*. 50 (4): 269–280. doi:10.1080/10408390902773037. PMID 20301015

FDA Daily Value (RDA)

None known*

Up to 3500mg considered to be safe. **

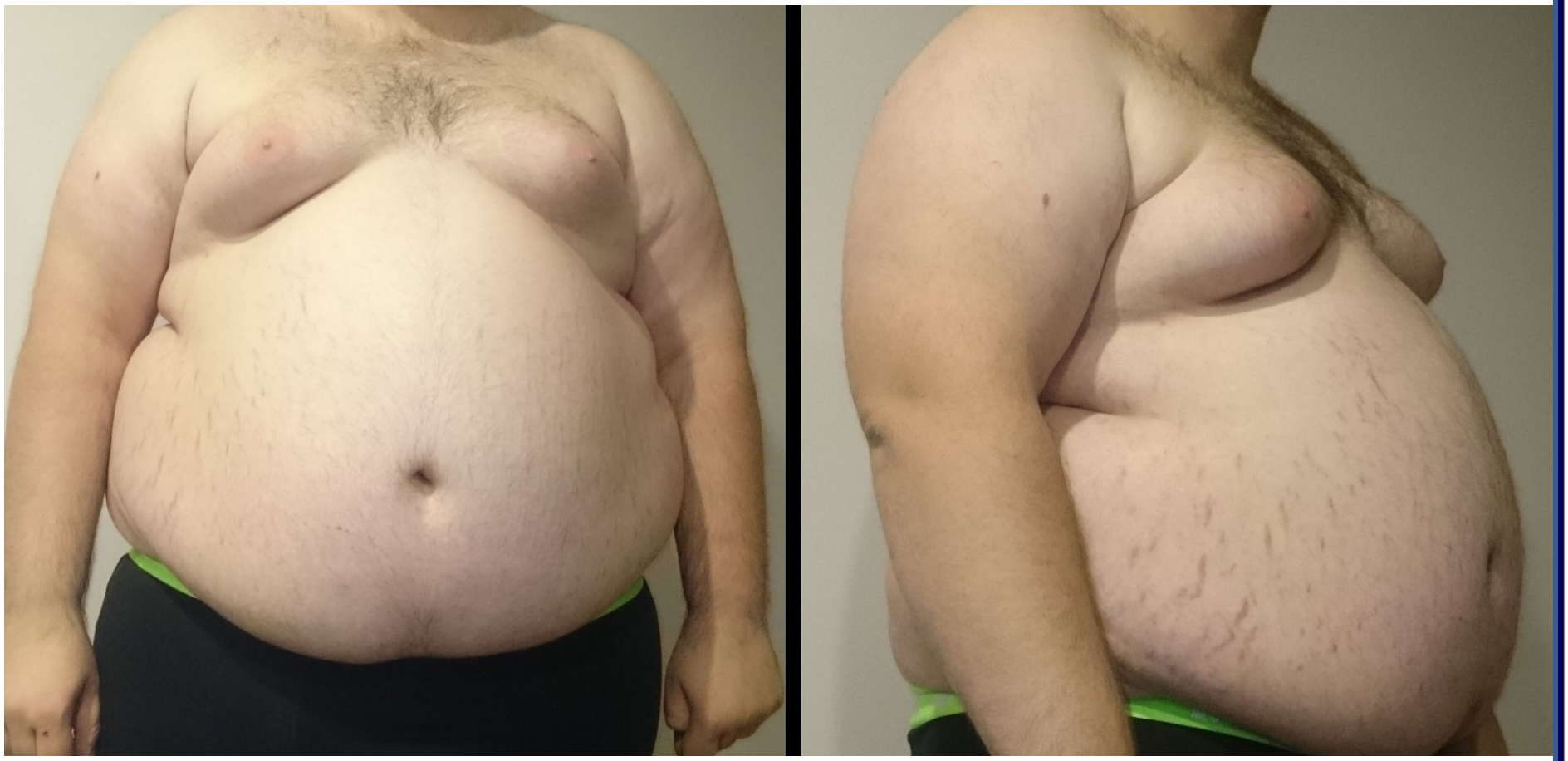
Clinically best taken last thing at night as acid inhibits absorption.

* **Hyson, H. C.; Kieburtz, K.; Shoulson, I.; et al. (Sep 2010). "Safety and tolerability of high-dosage coenzyme Q_{10} in Huntington's disease and healthy subjects". Movement Disorders. 25 (12): 1924–1928.*

*https://www.accessdata.fda.gov/scripts/InteractiveNutritionFactsLabel/factsheets/Vitamin_and_Mineral_Chart.pdf

Metabolic Syndrome

Metabolic syndrome



Central obesity, high blood pressure, high blood sugar, high serum triglycerides, and low serum high-density lipoprotein (HDL).

Metabolic syndrome, is a clustering of at least three of the five following medical conditions: central obesity, high blood pressure, high blood sugar, high serum triglycerides, and low serum high-density lipoprotein (HDL).

Metabolic syndrome is associated with the risk of developing cardiovascular disease and type 2 diabetes.*

Insulin resistance, metabolic syndrome, and pre-diabetes are closely related to one another and have overlapping aspects.

*Kaur J (2014). "A comprehensive review on metabolic syndrome". *Cardiology Research and Practice*. 2014: 1–21.

The most important risk factors are stress, diet (particularly sugar-sweetened beverage consumption), genetics, aging, sedentary behaviour or low physical activity, disrupted chronobiology/sleep, mood disorders/psychotropic medication use, and excessive alcohol use.*

**Vancampfort D, Vansteelandt K, Correll CU, Mitchell AJ, De Herdt A, Sienaert P, Probst M, De Hert M (March 2013). "Metabolic syndrome and metabolic abnormalities in bipolar disorder: a meta-analysis of prevalence rates and moderators". The American Journal of Psychiatry. 170 (3): 265–74.*

A number of markers of systemic inflammation, including C-reactive protein, are often increased, as are fibrinogen, interleukin 6, tumour necrosis factor-alpha (TNF-α), and increased uric acid levels caused by dietary fructose.*

*Reiser S, Powell AS, Scholfield DJ, Panda P, Ellwood KC, Canary JJ (May 1989). "Blood lipids, lipoproteins, apoproteins, and uric acid in men fed diets containing fructose or high-amylose cornstarch". *The American Journal of Clinical Nutrition*. 49 (5): 832–39.

Weight gain is associated with metabolic syndrome. Rather than total adiposity, the core clinical component of the syndrome is visceral and/or ectopic fat (i.e., fat in organs not designed for fat storage) whereas the principal metabolic abnormality is insulin resistance.*

**Ali ES, Hua J, Wilson CH, Tallis GA, Zhou FH, Rychkov GY, Barritt GJ (September 2016). "The glucagon-like peptide-1 analogue exendin-4 reverses impaired intracellular Ca(2+) signalling in steatotic hepatocytes". Biochimica et Biophysica Acta. 1863 (9): 2135–46.*

Since the metabolic syndrome is a disorder of energy distribution and storage, fat accumulation explains for a significant proportion of cardiovascular risk. disease.

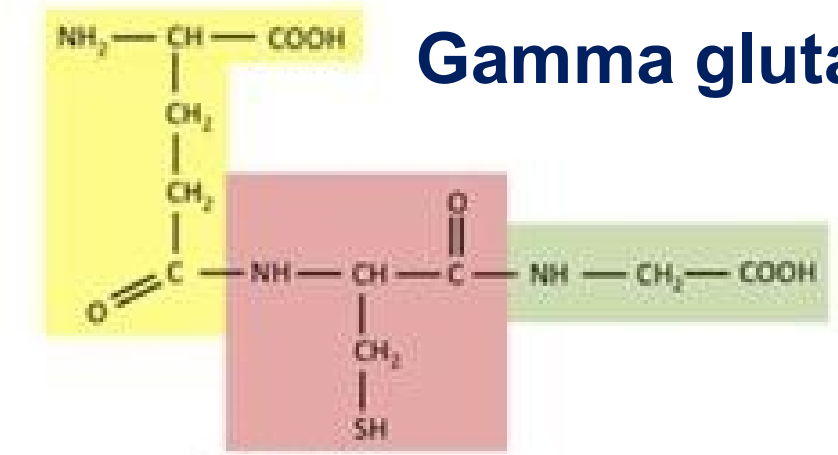
However, obesity without metabolic syndrome does not confer a significant cardiovascular risk, whereas metabolic syndrome without obesity is associated with a significant risk of diabetes and cardiovascular disease.*

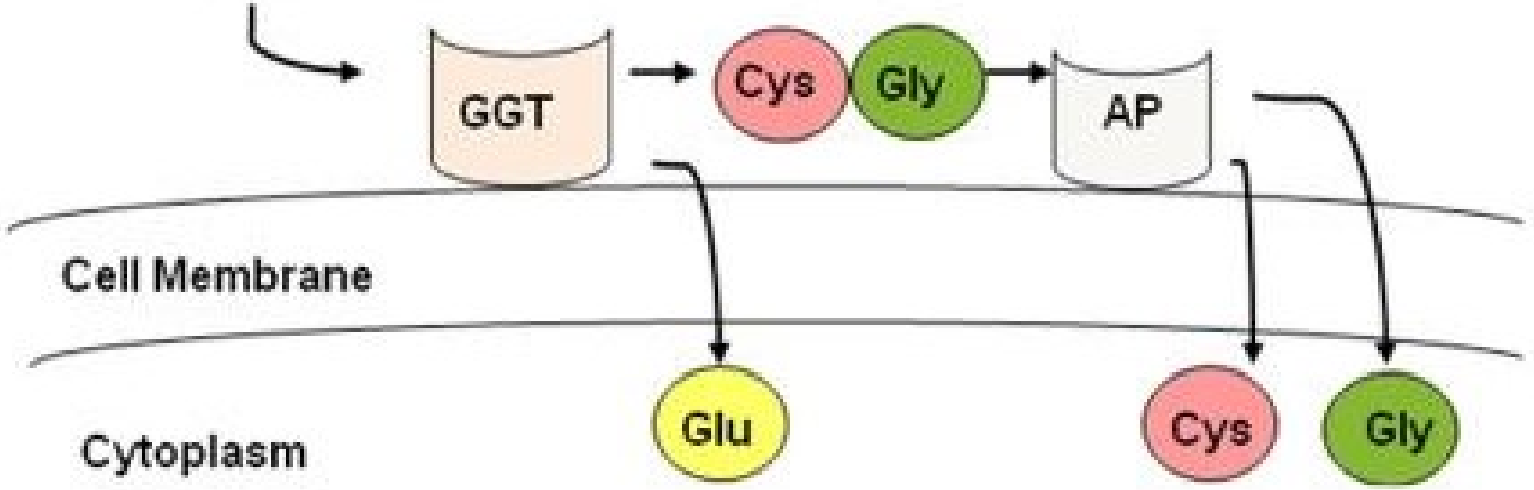
**Kahn R (June 2008). "Metabolic syndrome – what is the clinical usefulness?". Lancet. 371 (9628): 1892–93.*

Role of GGT

Gamma glutamyltransferase (GGT)

22q 399nm





Hydrolysis of extracellular glutathione by GGT. GGT releases glutamate and cysteinyl-glycine. Cysteinyl-glycine hydrolysed by aminopeptidase (AP) releasing cysteine and glycine. All three amino acids can then be taken up into the cell to synthesise glutathione but process not very efficient. Glutathione cannot be taken up intact in most cells.

GGT is present in the cell membranes of many tissues, including the kidneys, bile duct, pancreas, gallbladder, spleen, heart, brain, and seminal vesicles. It is involved in the transfer of amino acids across the cellular membrane and leukotriene metabolism.*

*Raulf M, Stüning M, König W (May 1985). "Metabolism of leukotrienes by L-gamma-glutamyl-transpeptidase and dipeptidase from human polymorphonuclear granulocytes". *Immunology*. 55(1): 135–47.

It is also involved in glutathione metabolism by transferring the glutamyl moiety to a variety of acceptor molecules including water, certain L-amino acids, and peptides, leaving the cysteine product to preserve intracellular homeostasis of oxidative stress. *

*Schulman JD, Goodman SI, Mace JW, Patrick AD, Tietze F, Butler EJ (July 1975). "Glutathionuria: inborn error of metabolism due to tissue deficiency of gamma-glutamyl transpeptidase". *Biochemical and Biophysical Research Communications*. 65 (1): 68–74.

Elevated serum GGT activity can be found in diseases of the liver, biliary system, and pancreas. In this respect, it is similar to alkaline phosphatase (ALP) in detecting disease of the biliary tract.

Slightly elevated serum GGT has also been found to correlate with cardiovascular diseases and is under active investigation as a cardiovascular risk marker. GGT in fact accumulates in atherosclerotic plaques.*

*Emdin M, Pompella A, Paolicchi A (October 2005). "Gamma-glutamyltransferase, atherosclerosis, and cardiovascular disease: triggering oxidative stress within the plaque". *Circulation*. 112 (14): 2078–80.

Elevated GGT may be related to specific pathologies such as metabolic syndrome, alcohol addiction and chronic liver disease. High body mass index is associated with type 2 diabetes only in persons with high serum GGT.

*Lim JS, Lee DH, Park JY, Jin SH, Jacobs DR (June 2007). "A strong interaction between serum gamma-glutamyltransferase and obesity on the risk of prevalent type 2 diabetes: results from the Third National Health and Nutrition Examination Survey".*Clinical Chemistry*. 53 (6): 1092–8.

Gamma-glutamyltransferase (GGT)

High in

Alcohol abuse, Barbituates, NSAIs, Aspirin, St John's Wort.

Biliary, Liver and Pancreas diseases

CVD and Atherosclerosis

Metabolic syndrome. High body mass index is associated with Type 2 diabetes only in persons with high serum GGT.

Lim JS, Lee DH, Park JY, Jin SH, Jacobs DR (June 2007). "A strong interaction between serum gamma-glutamyltransferase and obesity on the risk of prevalent type 2 diabetes: results from the Third National Health and Nutrition Examination Survey". Clinical Chemistry. 53 (6): 1092–8. doi:10.1373/clinchem.2006.079814. PMID 17478563.

Advanced Glycation End Products

Advanced glycation end products (AGEs) are proteins or lipids that become glycated as a result of exposure to sugars.*

**"American Heart Association". Retrieved 5 May 2016.*

They can be a factor in aging and in the development or worsening of many degenerative diseases, such as diabetes, atherosclerosis, chronic kidney disease, and Alzheimer's disease.*

They are also believed to play a causative role in the vascular complications of diabetes mellitus.**

*"American Heart Association". Retrieved 5 May 2016.

**Yan, S. F.; D'Agati, V.; Schmidt, A. M.; Ramasamy, R. (2007). "Receptor for Advanced Glycation Endproducts (RAGE): a formidable force in the pathogenesis of the cardiovascular complications of diabetes & aging". *Current Molecular Medicine*. 7(8): 699–710

In the context of cardiovascular disease, AGEs can induce crosslinking of collagen which can cause vascular stiffening and entrapment of low-density lipoprotein particles (LDL) in the artery walls. AGEs can also cause glycation of LDL which can promote its oxidation.*

*Prasad, Anand; Bekker, Peter; Tsimikas, Sotirios (2012-08-01). "Advanced glycation end products and diabetic cardiovascular disease". *Cardiology in Review*. 20 (4): 177–183.

Oxidized LDL is one of the major factors in the development of atherosclerosis.*
Proteins are usually glycated through their lysine residues**.

*Di Marco, Elyse; Gray, Stephen P.; Jandeleit-Dahm, Karin (2013-01-01). "Diabetes alters activation and repression of pro- and anti-inflammatory signaling pathways in the vasculature". *Frontiers in Endocrinology*. 4: 68.

** Ansari NA, Moinuddin, Ali R (2011). "Glycated lysine residues: a marker for non-enzymatic protein glycation in age-related diseases". *Disease Markers*. 30 (6): 317–324.

When skin is exposed to oxidative stress, the effectiveness of the endogenic antioxidant system can be significantly compromised. Pomegranate, a natural advanced glycation end product (AGEs) inhibitor shows great potential for anti-glycation, a major contributor to slowing the progression of skin damage.

Recent research shows
that the phenolics in pomegranate
play a unique role in the inhibition
of the production of reactive
oxygen species (ROS) and the
formation of AGEs from protein
glycation.

Pomegranate extract offers support against degenerative effects of UV exposure, including glycative stress and collagen cross-linking.

Water

Men are 65% water (15% body fat).

Women are 58% water (25%) body fat.

That is 40-50 litres per person.

Extracellular fluid
has a pH of 7.35 -7.45 maintained by the bicarbonate and ammonia / phosphate buffer systems.

Intracellular fluid pH varies.

1. Water should ideally be in glass bottles.
2. Have a pH of 7.4
3. Not be above 20mg / litre in Sodium
4. Have a 2:1 or less CALCIUM to MAGNESIUM ratio.

FIBRE

1. Soluble fibre is from fruit and vegetables and is fermented by colonic bacteria to fuel the colonocytes.

2. Insoluble fibre is from bran of cereals and helps form faeces along with bacteria, water and mucus. it absorbs bile.