Module 6
Fatty Acids

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

The most common toxins are naturally occurring chemicals within the foods we eat or drink or natural endogenously produced reactive intermediates (metabolic toxins) which fail to be metabolised completely.

Jeff Bland Dec 2010 Functional Medicine Update

Common naturally endogenously occurring reactive intermediates

Acetaldehyde
Acetic acid
Acetone
Ammonia*
Butyric acid
Cyanide
Ethane
Formaldehyde
Formic acid*
Glutamate
Hydrogen sulfite
4-Hydroxynonenol*
Indole
Lactic acid D
Lactic acid D/L
Lactic acid L
Malondialdehyde*
Methane
Methanol
Oxalate
Phenol*
Propionic acid*
Toluene
Tyramine*
Uric acid

* Most common

Common naturally occurring food and drink chemicals

Alpha Solanene
Betaine
Caffeine
Cysteine
Glutamate
Histamine
Isothiocyanate
Malondialdehyde
Oxalates
Salicylates
Sulfites
Thiobromine
Tomato toxin
Tyramine
Uric acid

Vitamin C stimulates HDL and decreases LDL and triglycerides. Binds cholesterol to make bile salts.

"Linus Pauling"

Regular omega-3 intake during pregnancy could boost baby brain and vision: Study

By Nathan Gray

02-Oct-2018 - Last updated on 02-Oct-2018 at 13:28 GMT

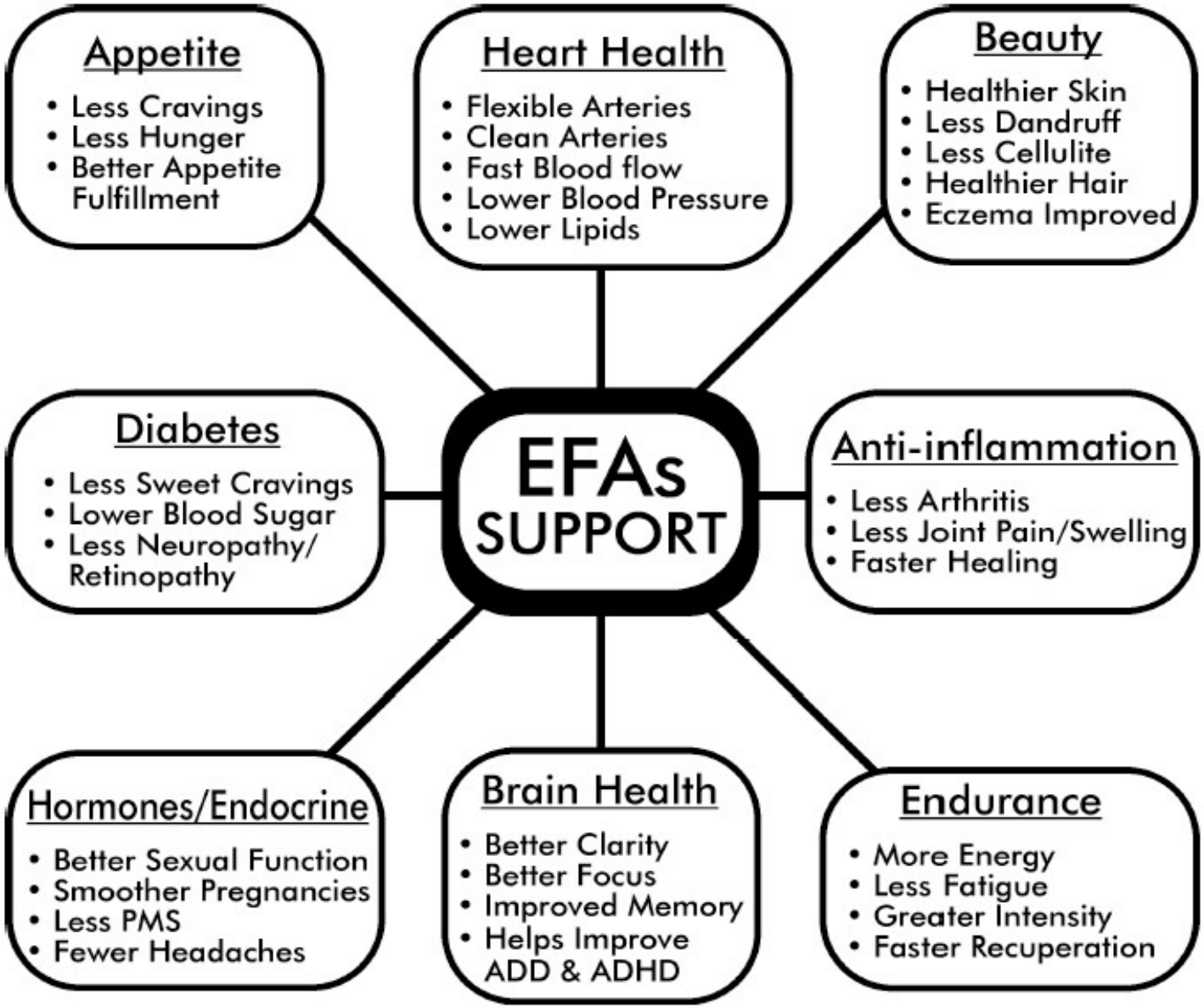
iStock / Serquan

Women could enhance the development of their unborn child's eyesight and brain function by regularly eating omega-3 rich fatty fish during pregnancy, say researchers from Finland.

The small-scale study, published in *Pediatric Research,* supports previous research that demonstrates the importance of diet and lifestyle during pregnancy on the development of a baby – finding that adjusting the diet of healthy pregnant women to include higher levels of omega-3 could be beneficial to their babies.

The Missing Link: *EFA "Oxygen Magnets"*

Prof. Brian Peskin



Cell Membrane

- Transport of substances across the cell membrane
- Neurotransmitters – glutamate, dopamine, acetylcholine
- Sodium and calcium have to be pumped out of the cell
- Oxygen
- Require a flexible, permeable, fluid cell membrane

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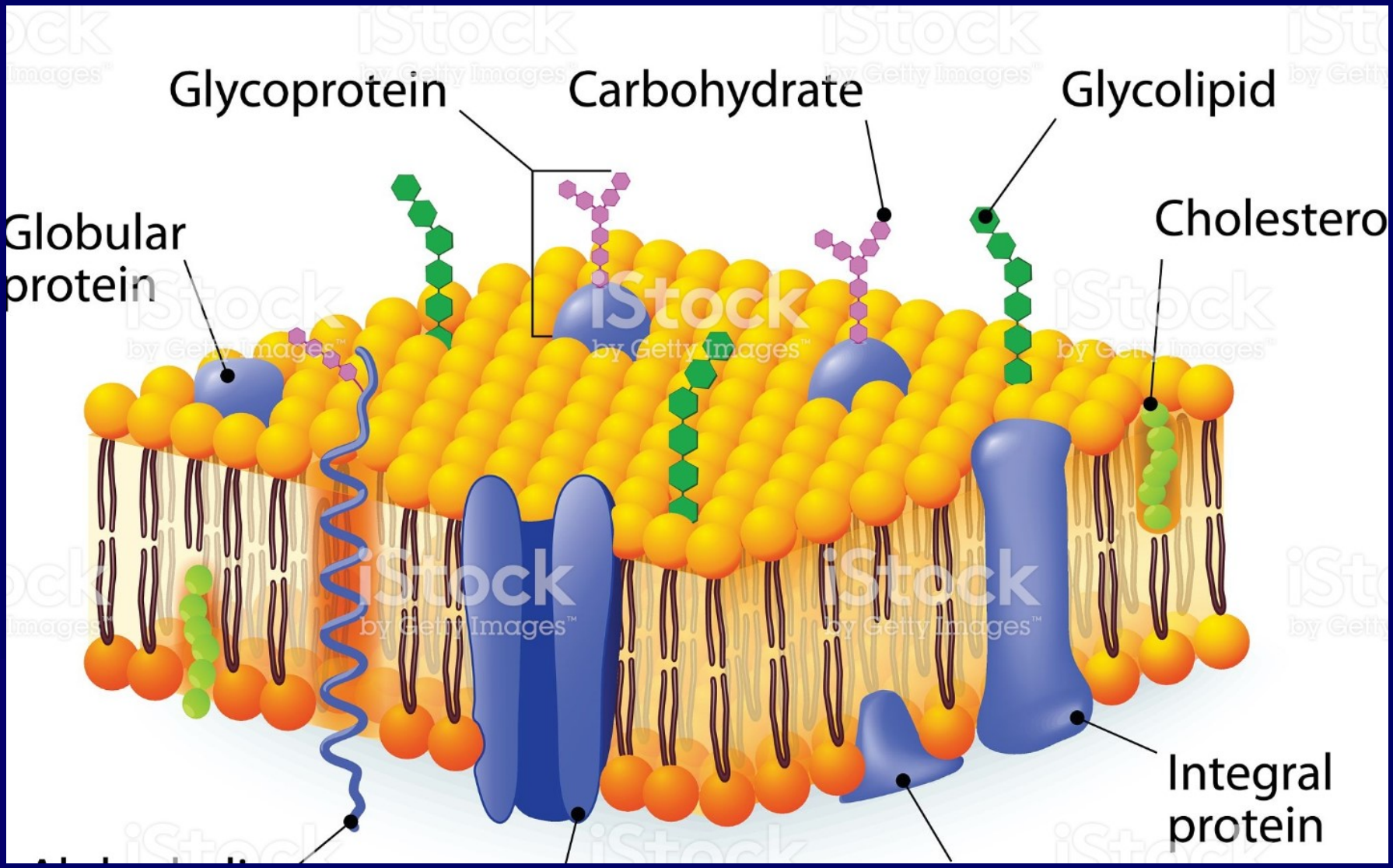
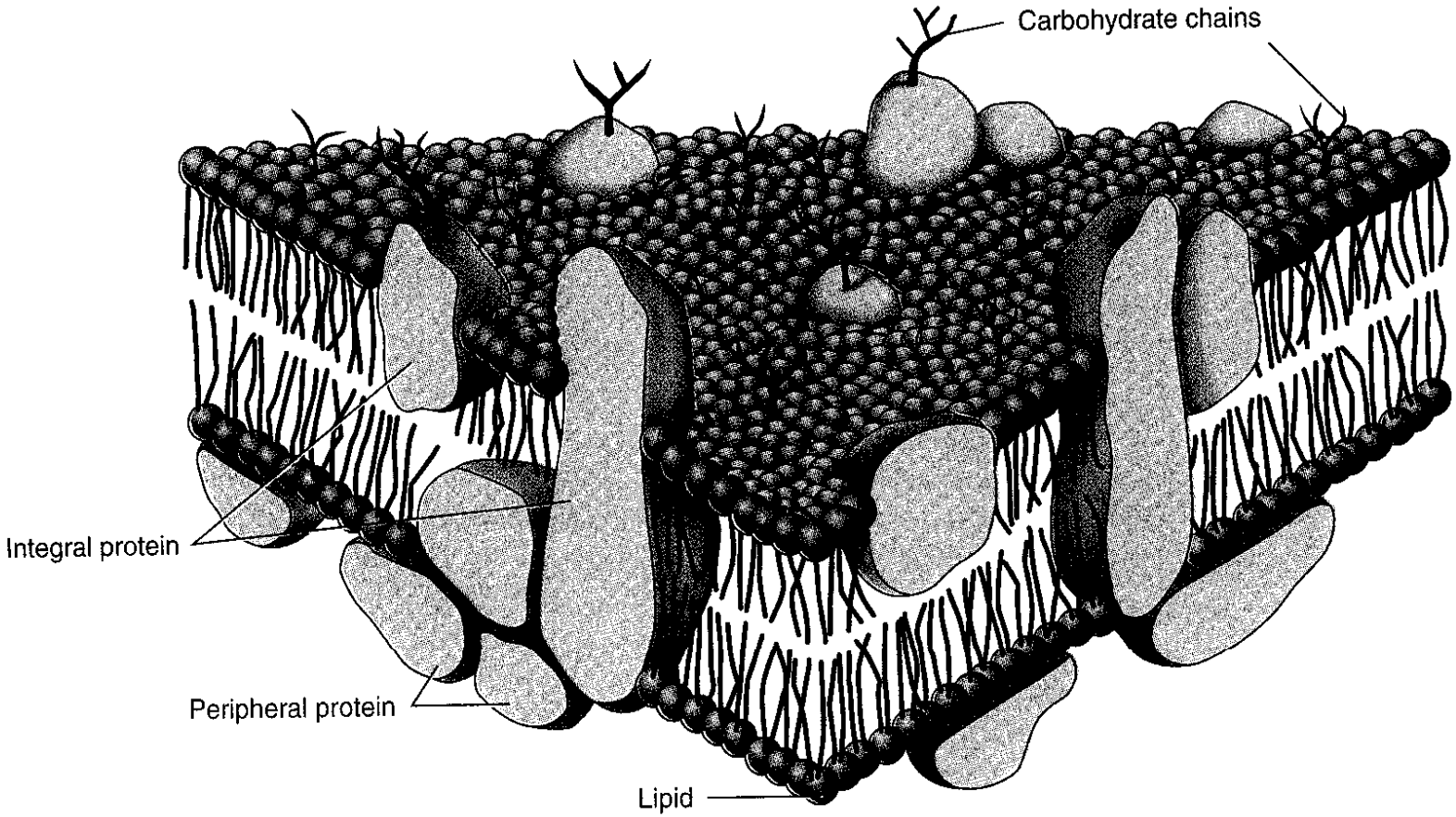

Glycoprotein
Carbohydrate
Glycolipid
Globular protein
Cholestero
Integral protein

Cell Membranes



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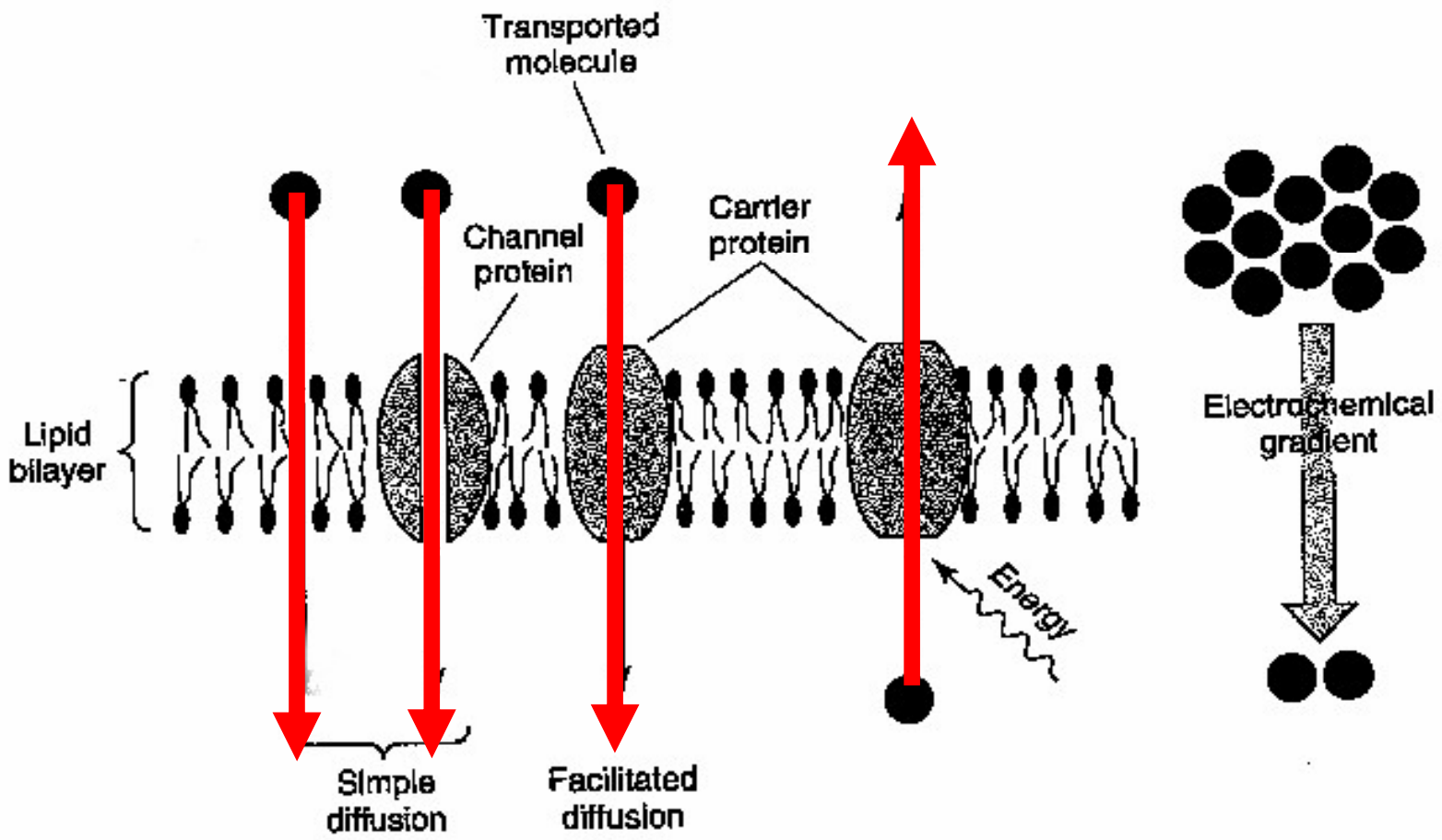
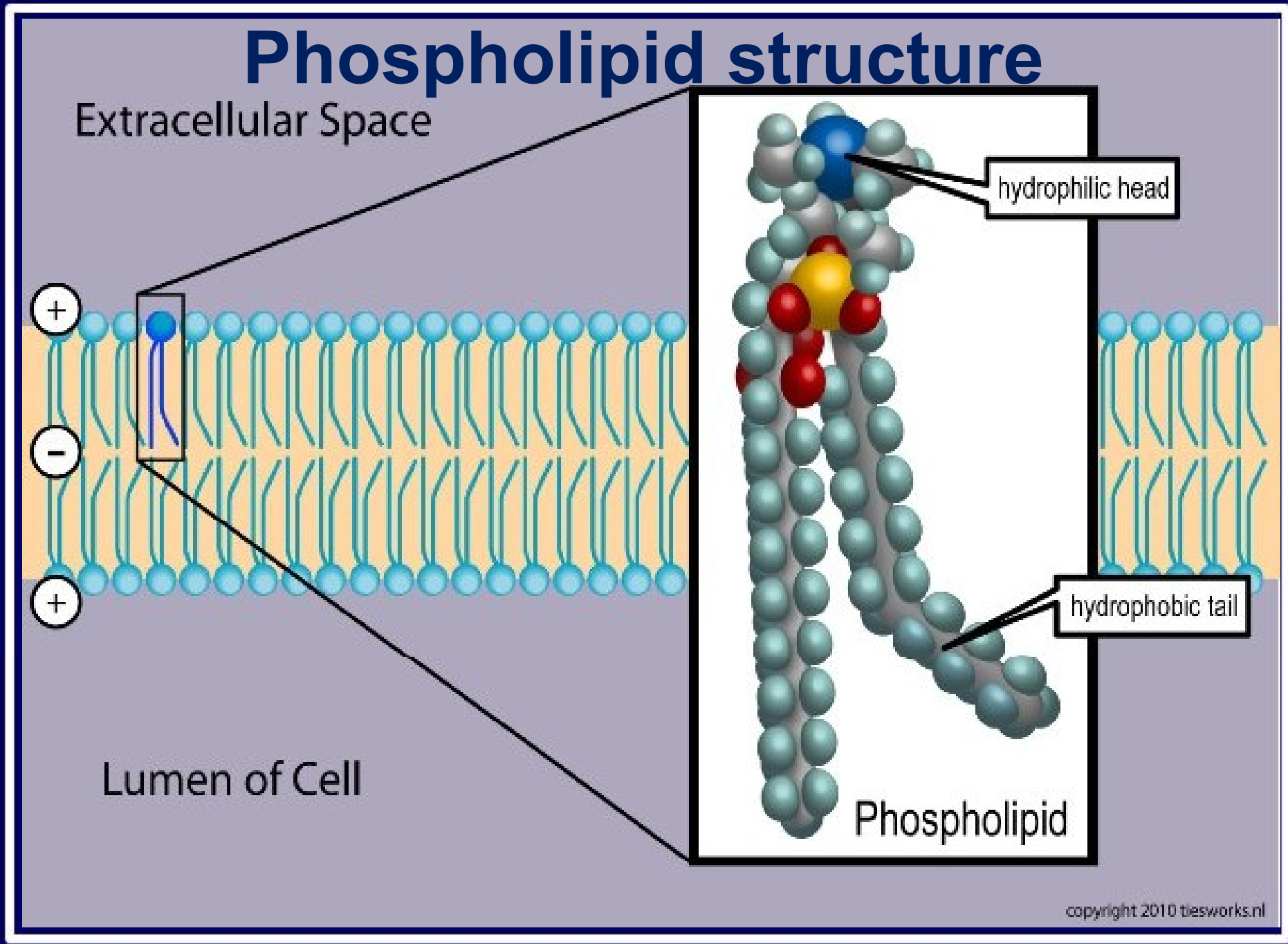
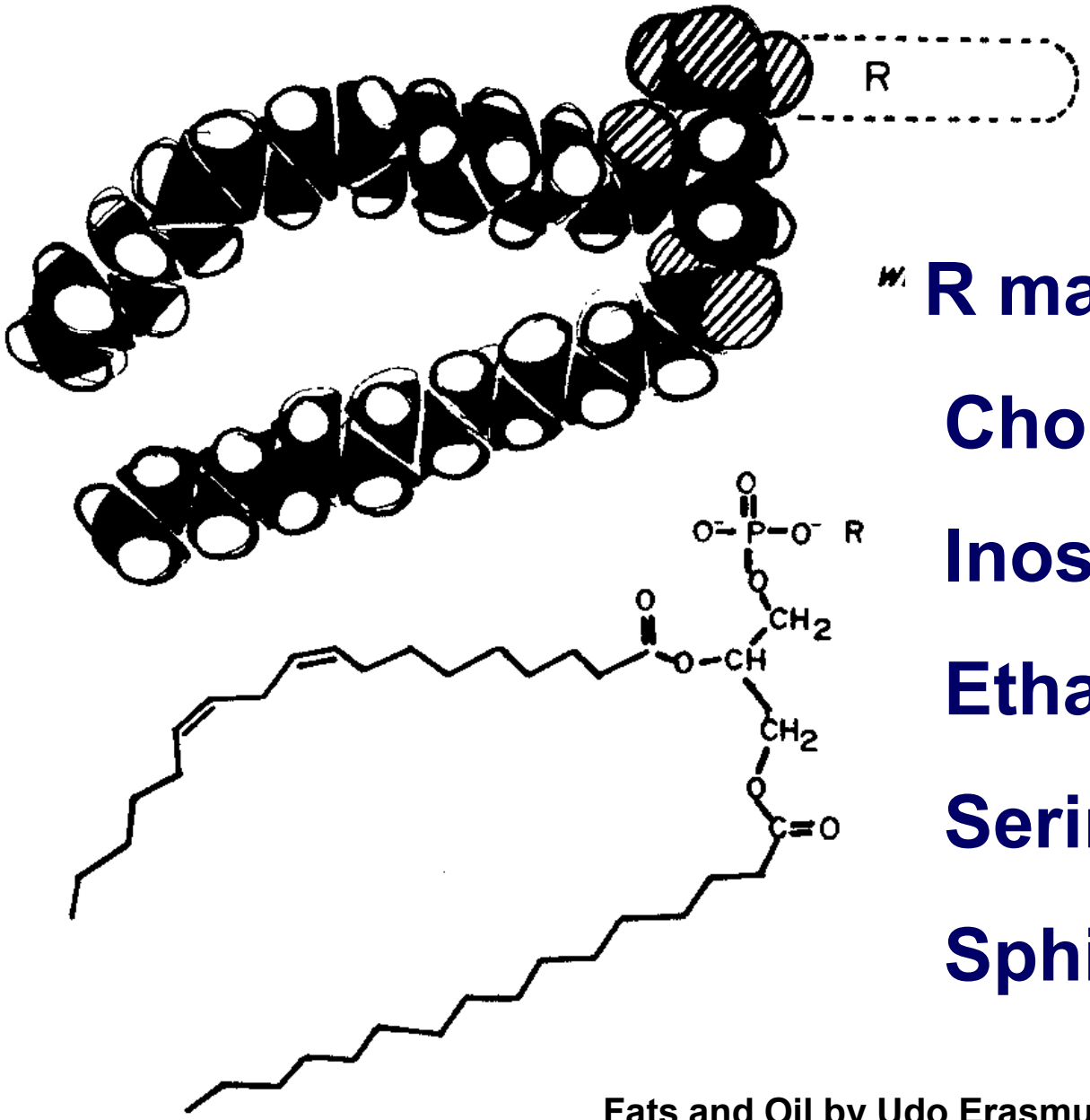


Passive transport **Active transport**

Phospholipids

Phospholipid structure





- R maybe
  - Choline
  - Inositol
  - Ethanolamine
  - Serine
  - Sphingomyelin

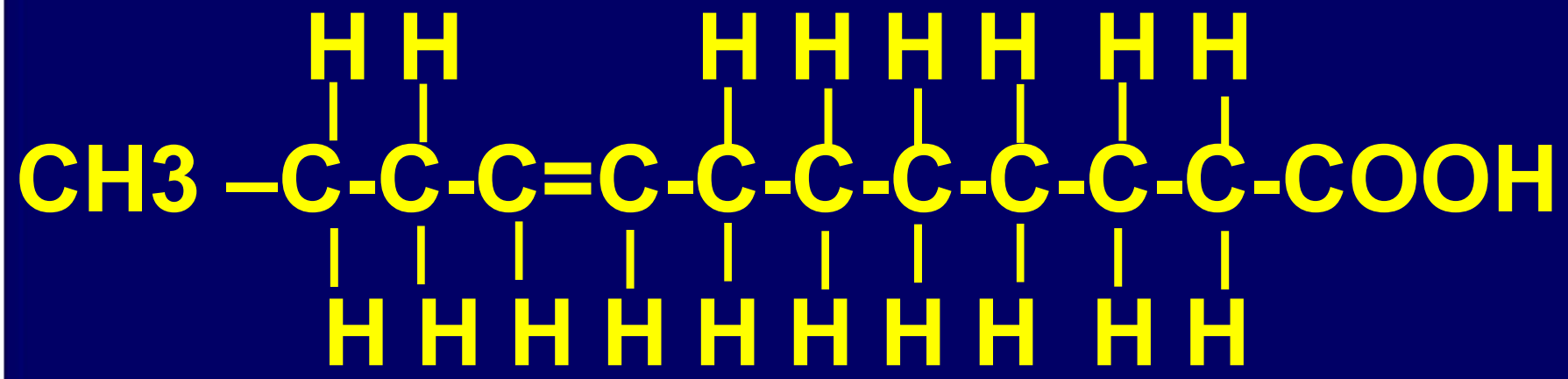
Fats and Oil by Udo Erasmus page 51

Simple lipids are

a) Saturated (no double bonds)
CH3 ----------COOH

b) Unsaturated (mono or poly double bonds)
CH3 ----=---------- COOH
(Methyl (w) end Carboxyl end)

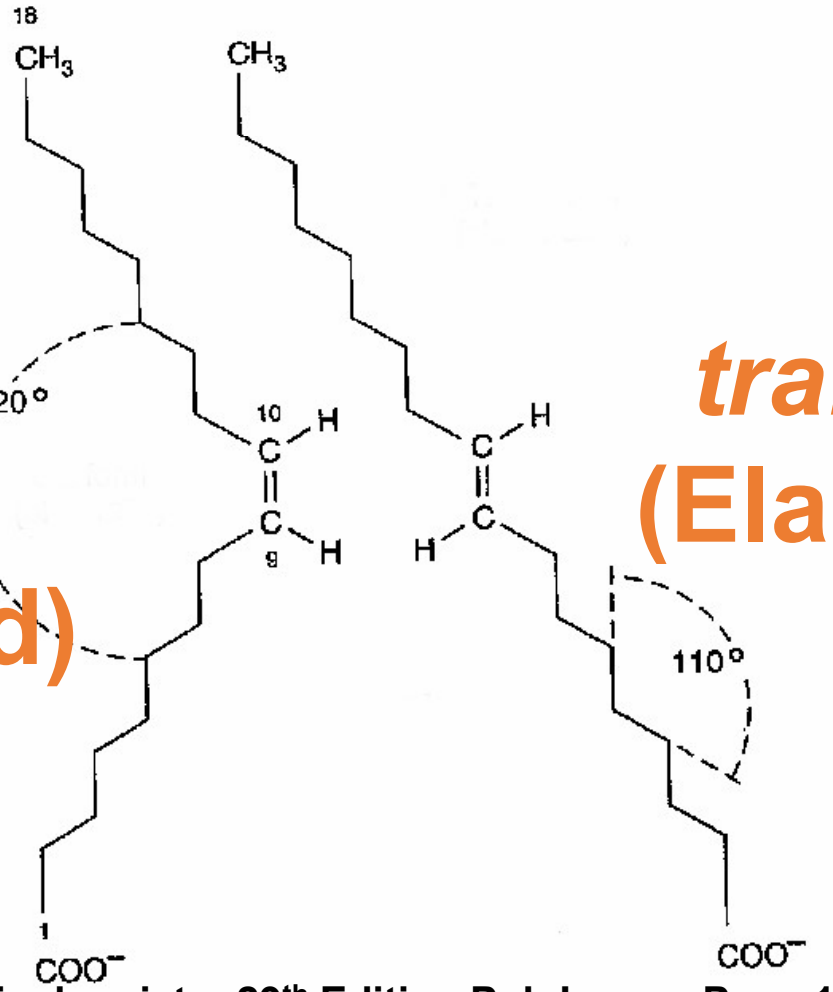
An unsaturated fatty acid (Omega 3)



(Methyl (w) end Carboxyl end)

Fats and Oil by Udo Erasmus page 5

Unsaturated fatty acids can be in either *cis* or *trans* forms



cis form (Oleic acid)

trans form (Elaidic acid)

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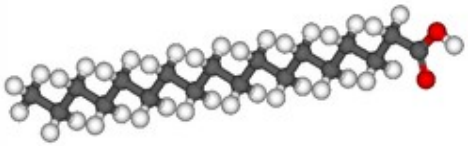
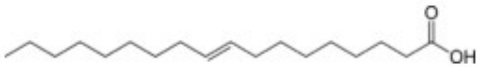
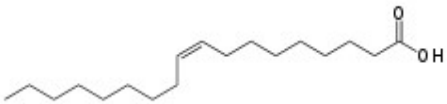
Trans (Elaidic acid)	Cis (Oleic acid)	Saturated (Stearic acid)
Elaidic acid is the principal *trans* unsaturated fatty acid often found in partially hydrogenated vegetable oils.[33]	Oleic acid is a *cis* unsaturated fatty acid that comprises 55–80% of olive oil.[34]	Stearic acid is a saturated fatty acid found in animal fats and is the intended product in full hydrogenation. Stearic acid is neither *cis* nor *trans* because it has no carbon-carbon double bonds.
		
		

Neuronal membranes

The lipid portion of cell membranes is just 3 nanometres thick.

If you were to stack sheets of them one upon the other – it would take 10,000 membranes to make up the thickness of a piece of paper.

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Lipids are classified as

1. Simple lipids – oils and fats
2. Complex lipids
   a) Phospholipids
   b) Glycosphingolipids containing a fatty acid, sphingosine and a CHO
   c) Lipoproteins

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Simple lipids are

a) Saturated (no double bonds)
CH3 ----------COOH

b) Unsaturated (mono or poly double bonds)
CH3 ----=---------- COOH
(Methyl (w) end Carboxyl end)

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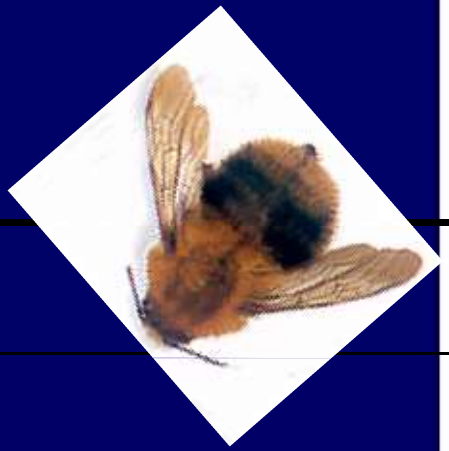
Saturated fatty acids



Name	Number	
Formic	1	Bee stings
Acetic	2	Rumen fermentation
Propionic	3	Rumen fermentation
Butyric	4 (-8°C)	Rumen fermentation
Valeric	5	Rumen fermentation
Caproic	6 (-3°C)	Coconut

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Caprylic	8 (17°)	Coconut
Nonanoic	9	Licorice root. Clove
Capric	10 (32 °)	Coconut
Undecanoic	11	Castor bean oil
Lauric	12 (44 °)	Breast milk, Coconut
Myristic	14 (54 °)	Nutmegs, Coconut
Palmitic	16 (63 °)	Animal and plant fats
Stearic	18 (70 °)	Animal and plant fats
Arachidic	20 (75 °)	Peanuts
Behenic	22 (80 °)	Seeds
Lignoceric	24 (84 °)	Cerebrosides, Peanuts

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Unsaturated Fatty Acids

Monoenoic acid (one double bond)

Number	Series	Common Name	Systematic Name	Source
16:1:9	w7	Palmitoleic	Cis-9-hexadecenoic	Sea Buckthorn
18:1:9	w9	Oleic	Cis-9-Octadecenoic	Olive
18:1:9	w9	Elaidic	Trans-9-Octadecenoic	Hydrogenated fats
22:1:13	w9	Erucic	Cis-13-Docosenoic	Rapeseed
24:1:15	w9	Nervoic	Cis-15-Tetracosenoic	Cerebrosides Honesty seed

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Dienoic acids (two double bonds)

18:2:9,12	w6	Linoleic	all-cis-9,12-Octadenenoic	Corn, peanut, soybean

Trienoic acids (three double bonds)

18:3:6,9,12	w6	γ-Linolenic	all-cis-6,9.12-Octadecatrienoic	EPO, BSO, Borage
18:3:9,12,15	w3	α-Linolenic	all-cis-8,12,15-Octadecatrienoic	Flax, walnut, pumpkin

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Tetraenoic acids (four double bonds)

20:4:5,8,11,14	w6	Arachidonic	all-cis-5,8,11,14-Eicotetraenoic	Peanut

Pentaenoic acids (five double bonds)

20:5:5,8,11,14,17	w3	Timnodonic (EPA)	all--cis-5,8,11,14,17-Eicosapentaenoic	Fish oil, Canola, Eggs
22:5:7,10,13,16,19	w3	Clupanodonic (DPA)	all-cis-7,10,13,16,19, Docosapenaenoic	Fish oil

Hexaenoic acids (six double bonds)

22:6:4,7,10,13,16,19	**w3**	**Cervonic (DHA)**	**all-cis-4,7,10,13,16,19-Docosahexaenoic**	**Fish oil Algae, Eggs**

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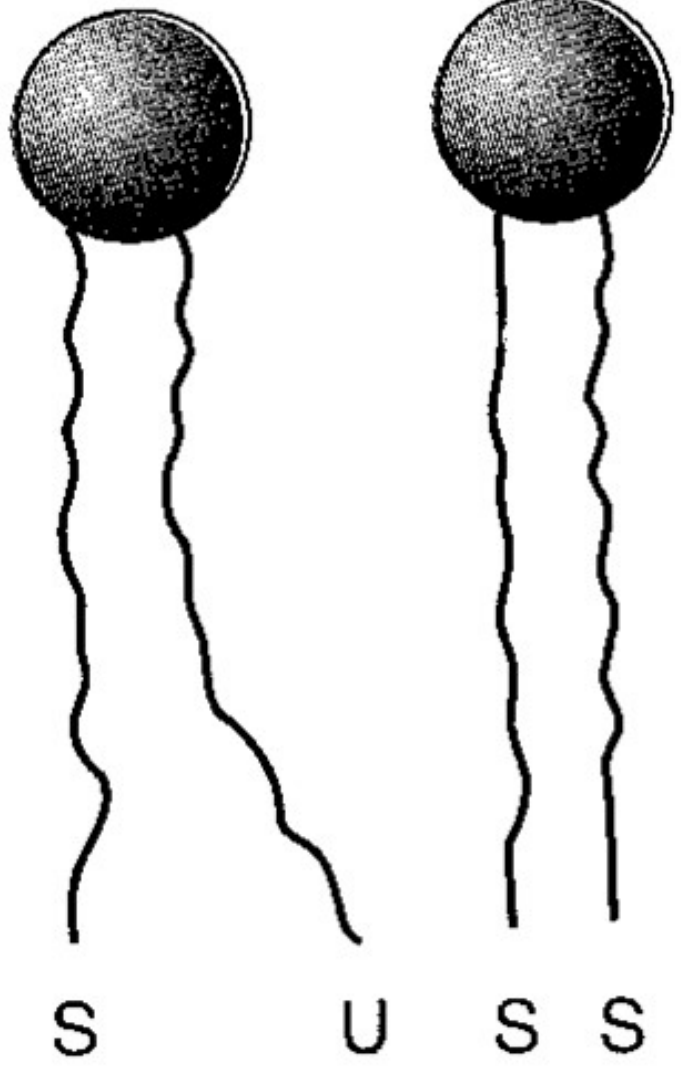
Fatty Acid Properties

	18:0	18:1 w9 c	18:1 w9 t	18:2 w6c,c	18:3 w3c,c,c
Stacking (acid end / fatty end)					
Saturation	Saturated	Unsaturated 1 double bond cis-configuration	Unsaturated 1 double bond trans-	Unsaturated 2 double bonds cis,cis-	Unsaturated 3 double bonds cis,cis,cis-
Melting Point	70°c sfa sticky	13°c cis-single slightly anti-sticky	44°c trans-single slightly sticky	-5°c cis,cis-double anti-sticky	-12°c cis,cis,cis-triple very anti-sticky
Repelling Charges	no charge	1 neg. charge	1 neg. charge	2 neg. charges	3 neg. charges

Fats that Heal, Fats that Kill by Udo Erasmus page 41

A Phospholipid



Polar head group

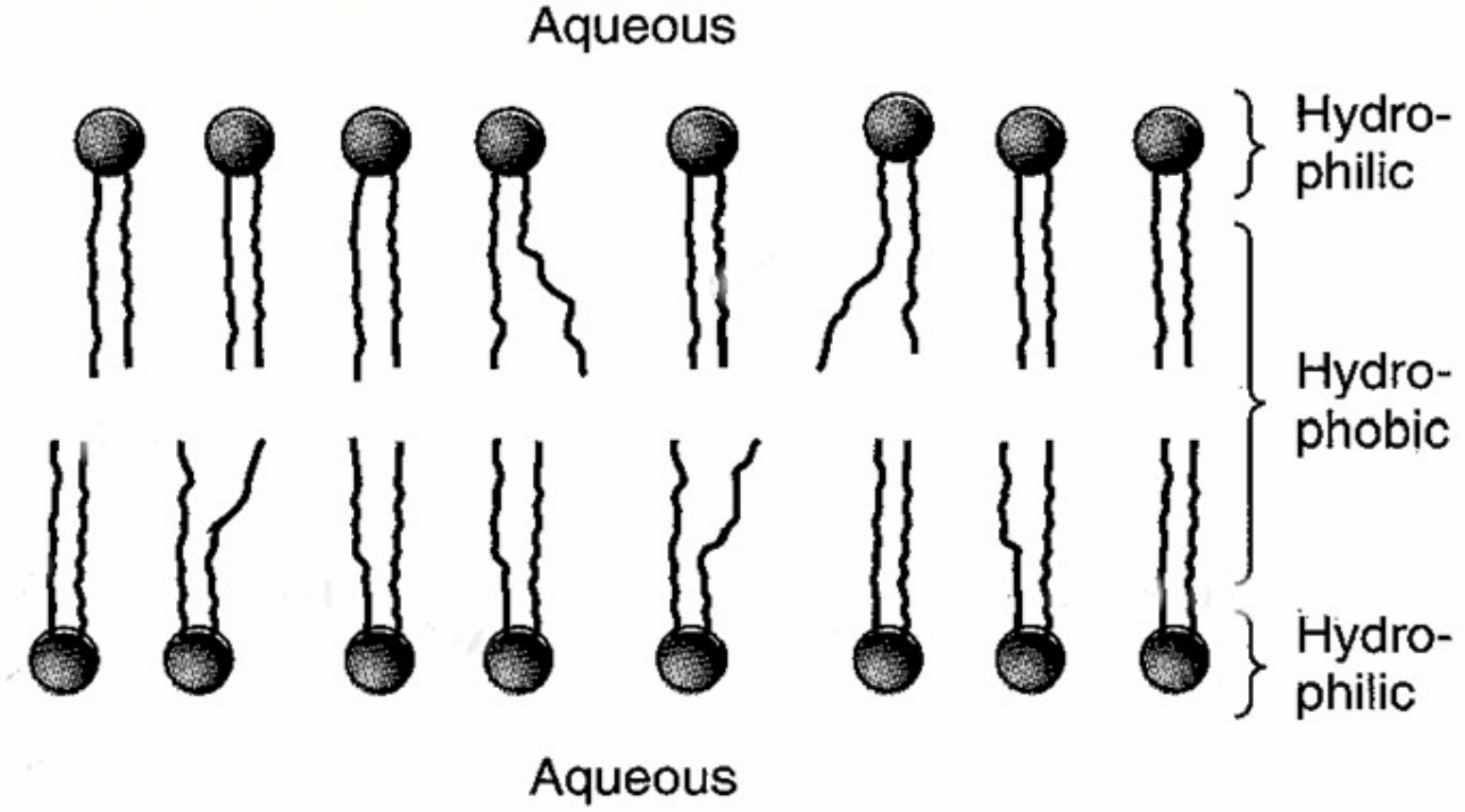
(hydrophilic)

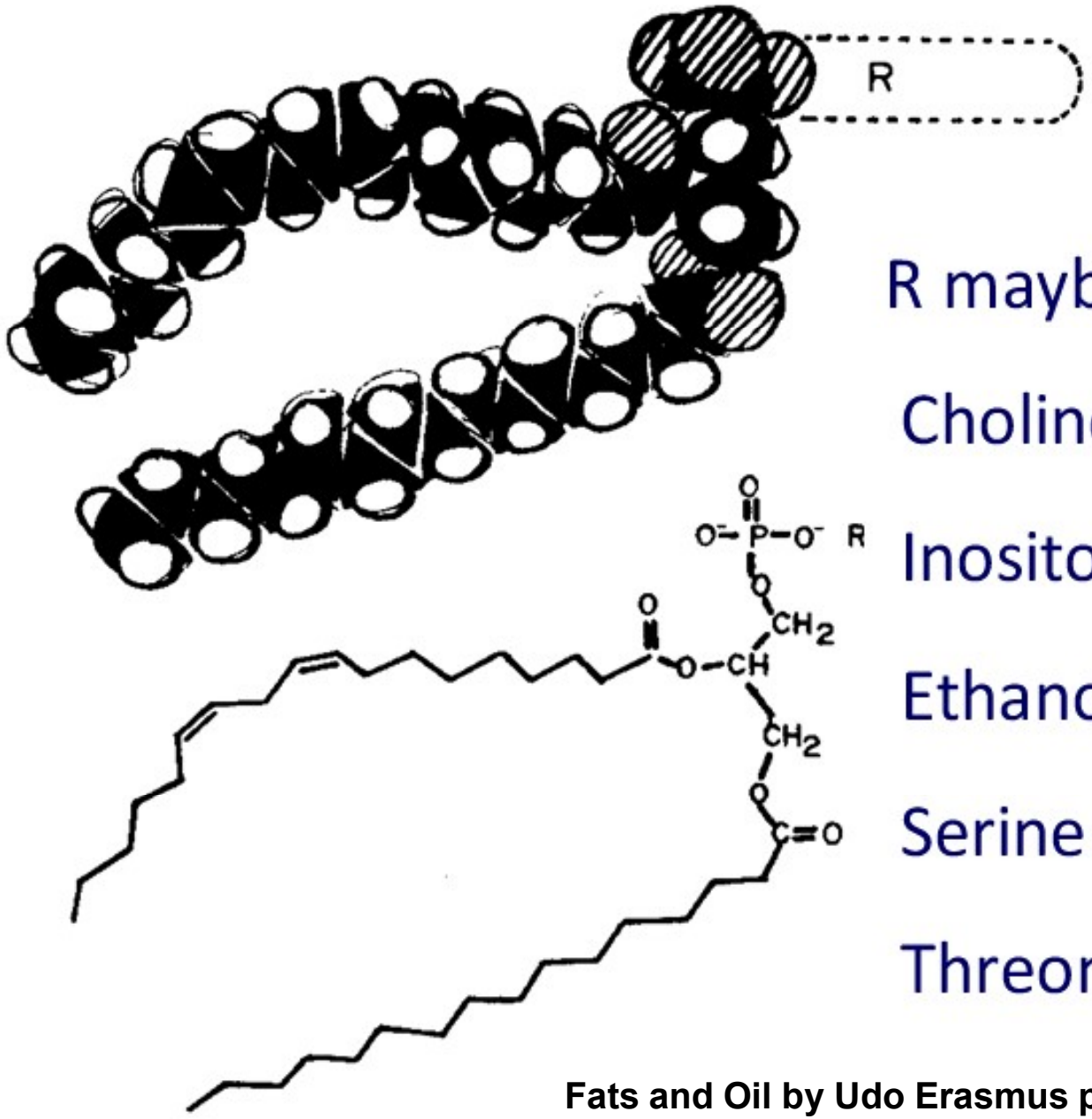
Apolar, hydrocarbon tails

(hydrophobic)

S U S S

The unsaturated fatty acid tails are kinked and lead to more spacing between the polar heads and hence more movement.





R maybe

Choline

Inositol

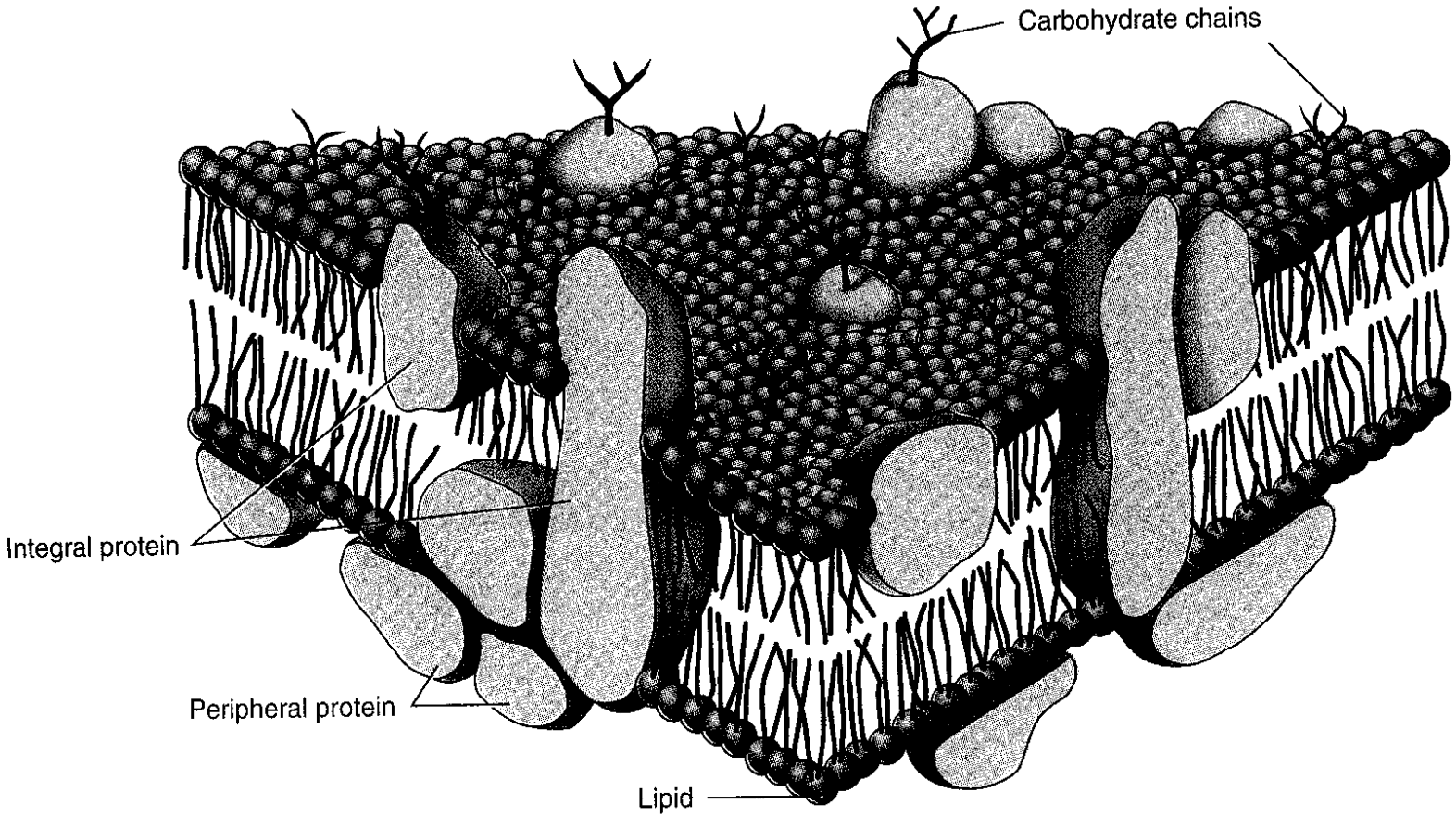
Ethanolamine

Serine

Threonine

Fats and Oil by Udo Erasmus page 51

Cell Membranes



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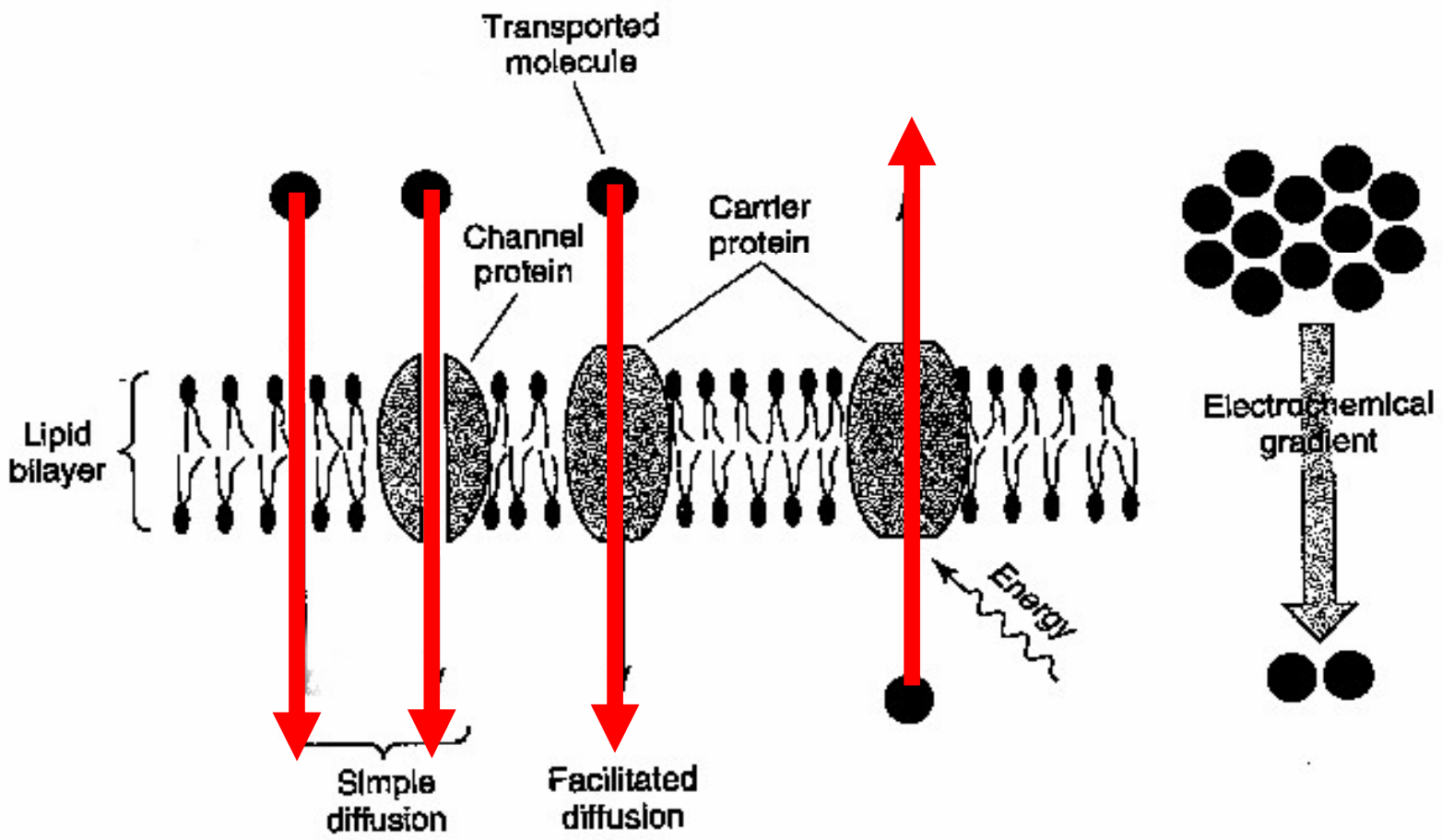


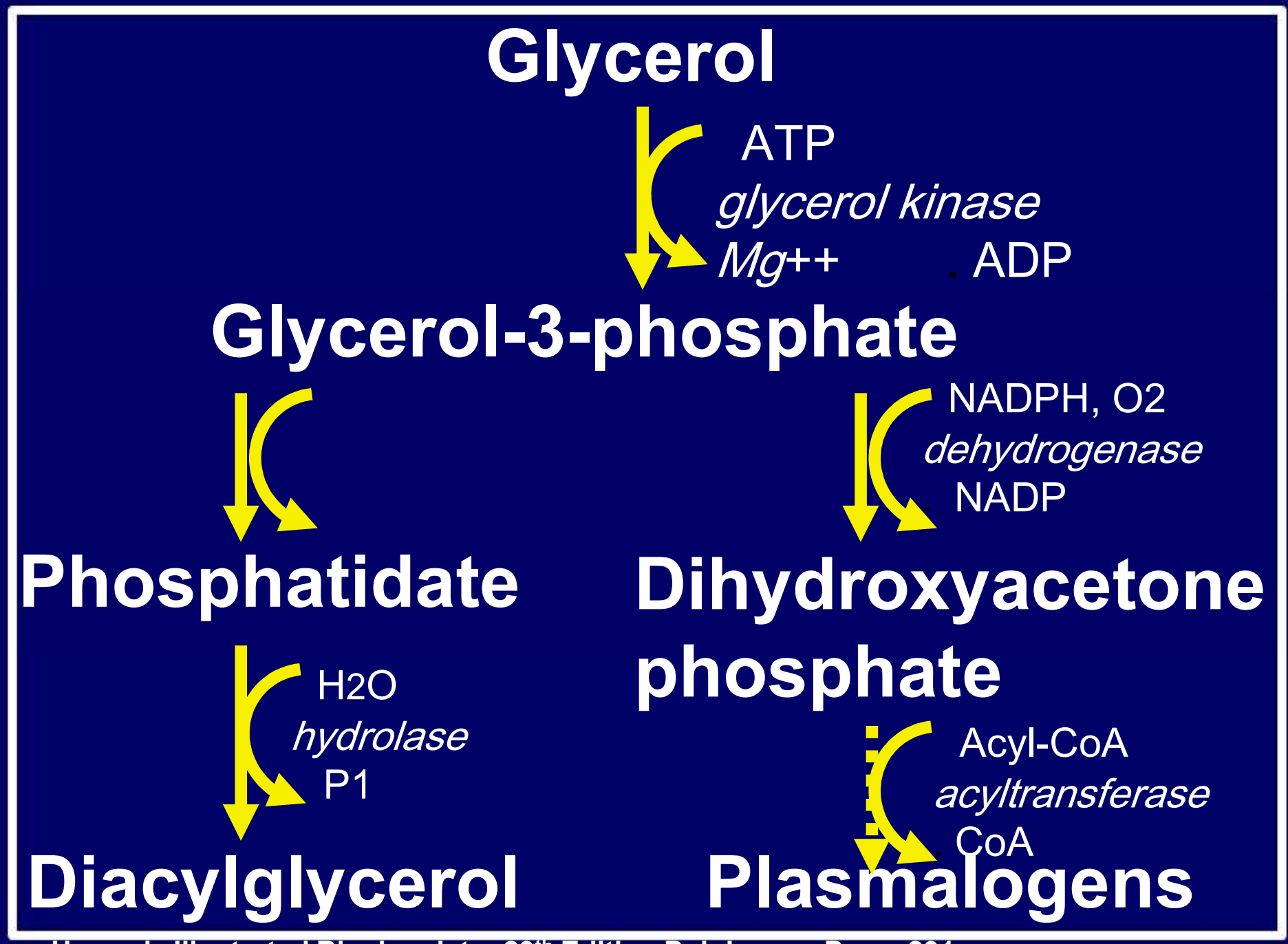
Passive transport **Active transport**

Neuronal cell membranes

Glial cells – the C1 position is taken by a saturated fatty acid and C2 by an unsaturated fatty acid

Neurones – in many neurones the C1 position is taken by Arachidonic acid and C2 by DHA.

Retina – both C1 and C2 positions are taken by DHA.



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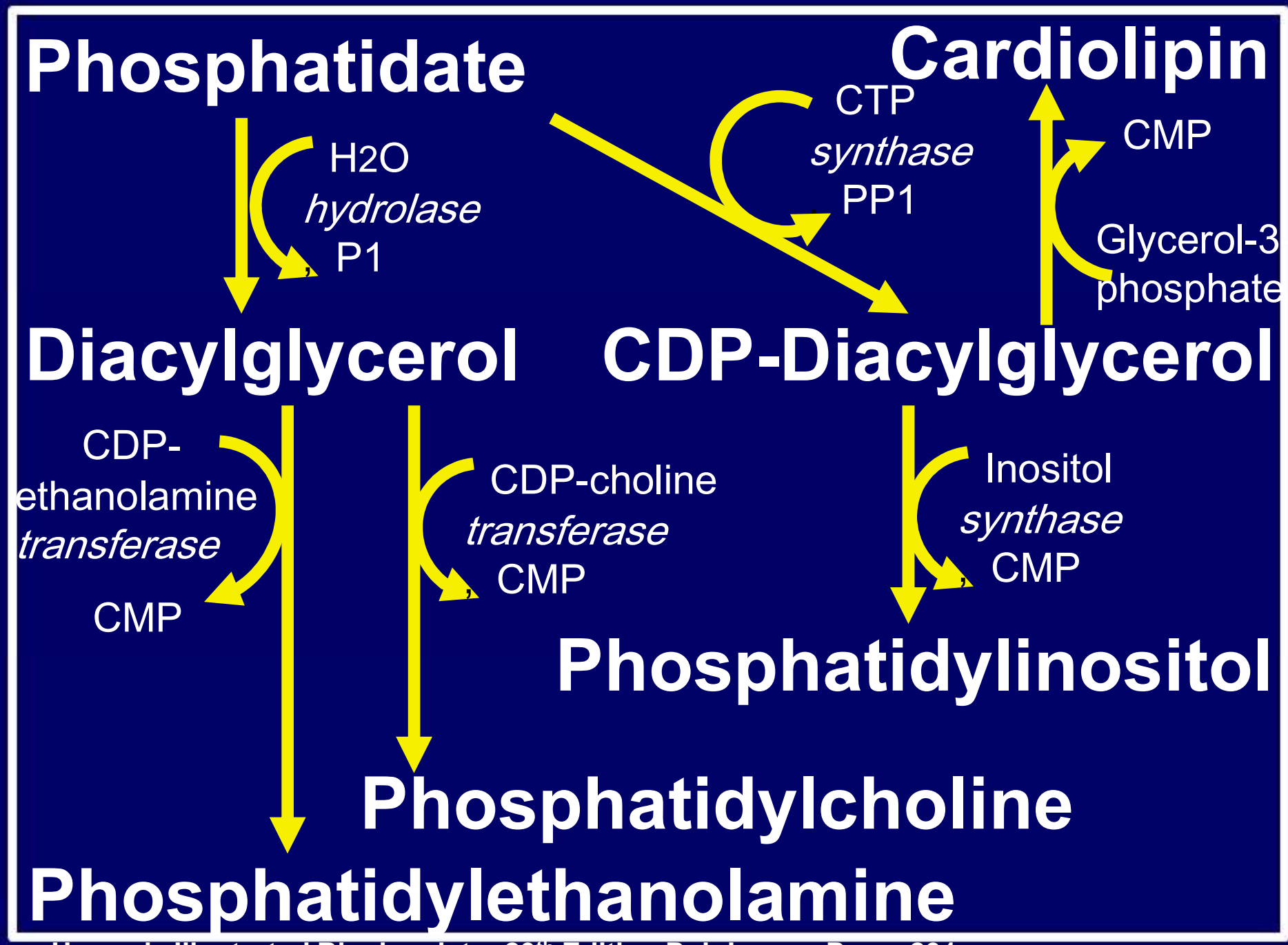

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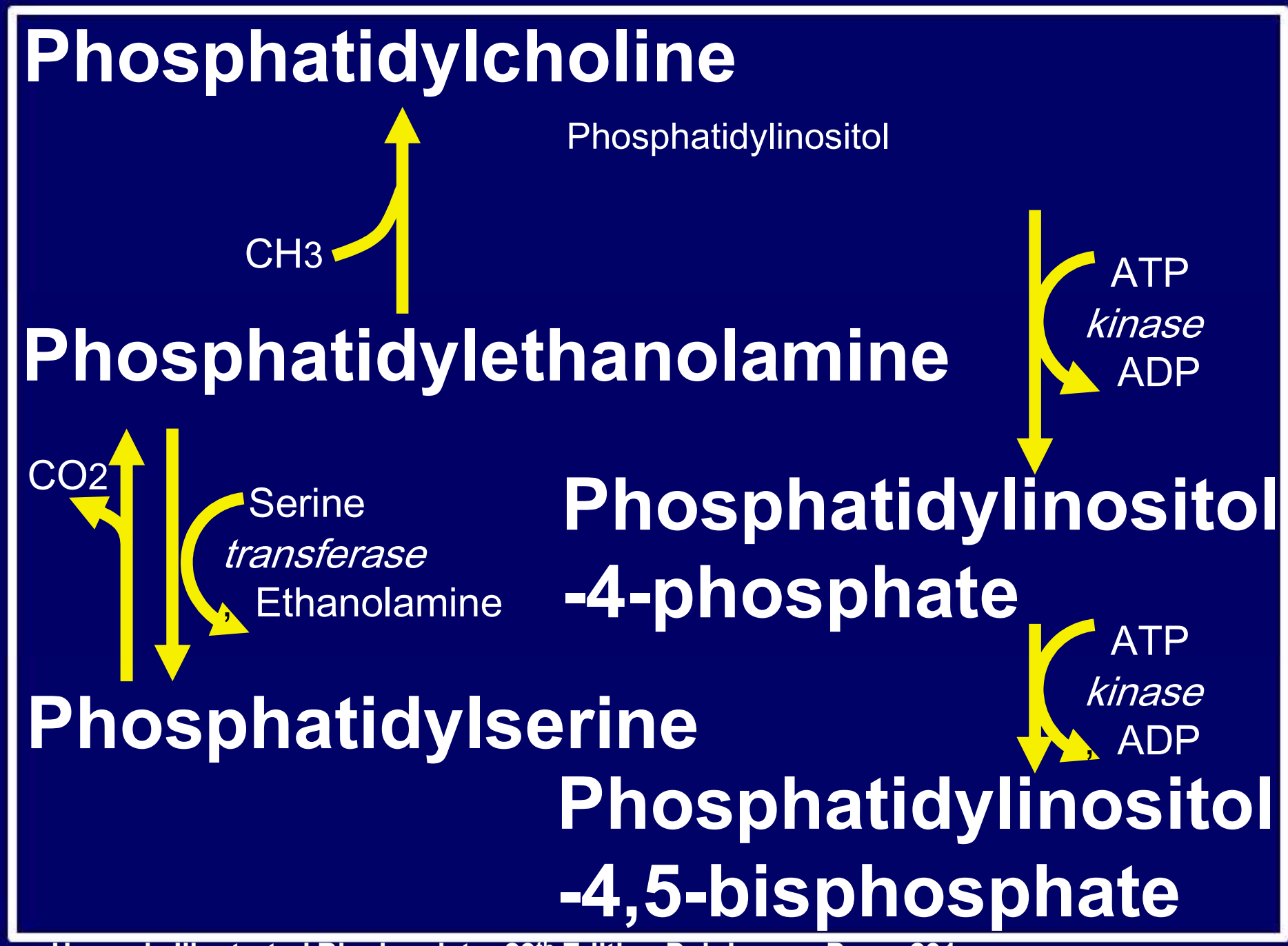

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Key nutrients for synthesising the phospholipids

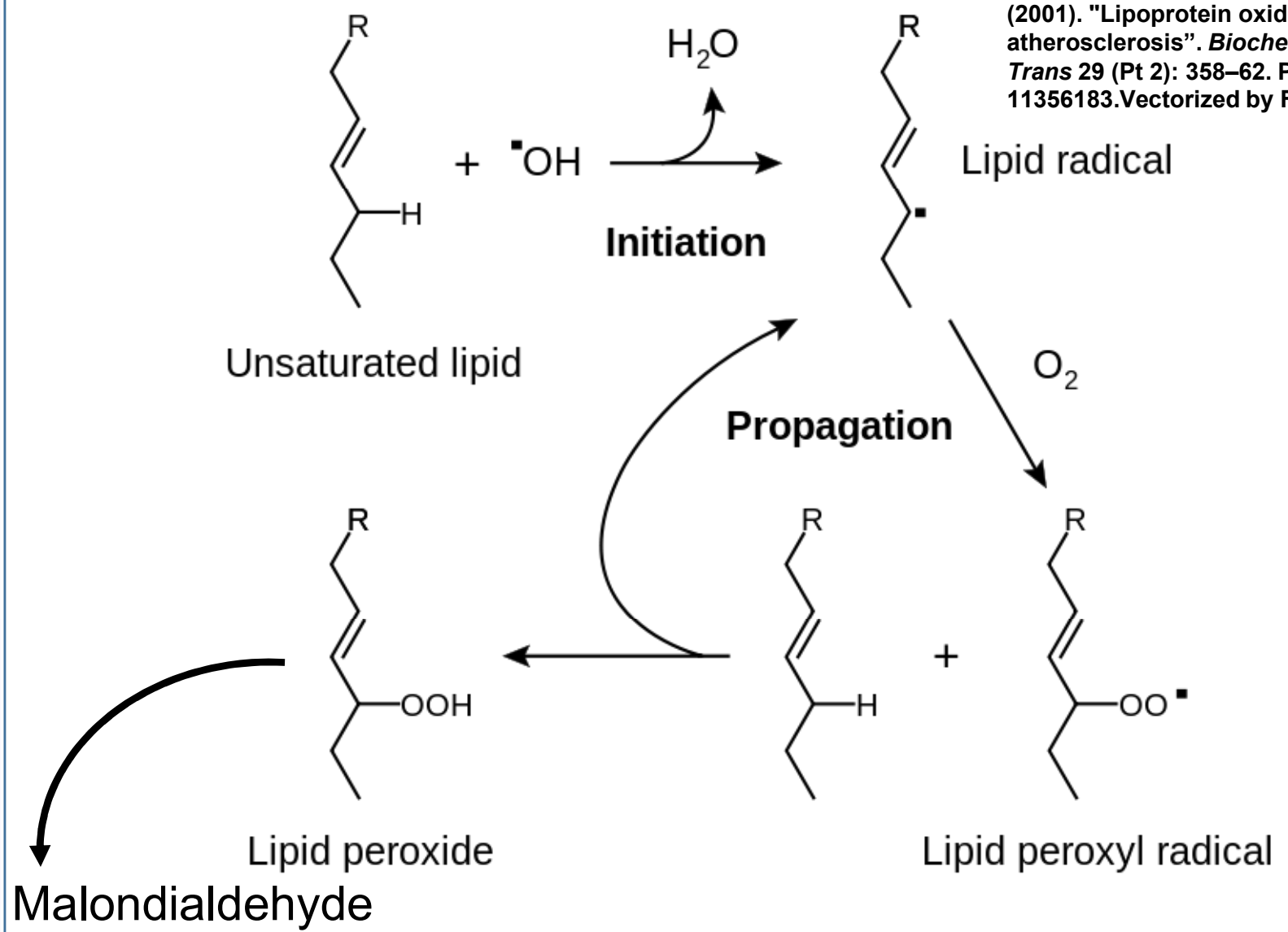
Acetyl CoA (Vit B5)
NAD, NADPH (Vit B3)
Mg, Zn, SAM (Mg, B6, Folates, B12)
Choline
Ethanolamine
Serine
Inositol
Saturated fatty acids C16-18 (palmitic – stearic)
Unsaturated fatty acids C18-24

Rancid Fats

- Primarily occurs with unsaturated fats
- More susceptible to rancidity because of structure with many double bonds
- Fats turn rancid in the presence of free radicals or reactive oxygen species

Erich Lück and Gert-Wolfhard von Rymon Lipinski "Foods, 3. Food Additives" in *Ullmann's Encyclopedia of Industrial Chemistry*, 2002, Wiley-VCH, Weinheim.

RANCID FATS



Tim Vickers, after Young IS, McEneny J (2001). "Lipoprotein oxidation and atherosclerosis". *Biochem Soc Trans* 29 (Pt 2): 358–62. PMID 11356183.Vectorized by Fvasconcellos

Rancid Fats

- Reactive oxygen species degrade polyunsaturated lipids forming malondialdehyde
- Reactive aldehyde causes toxic stress in cells and forms advanced lipoxidation end products
- Lead to loss of membrane integrity

Sergey,, Bylikin,. *Chemistry : course companion*. Horner, Gary,, Murphy, Brian,, Tarcy, David, (2014 ed.). Oxford.

Rancid Fats

- **Malondialdehyde** is used as a biomarker to assess the oxidative stress of a person
- It reacts with deoxyadenosine and deoxyguanosine in DNA to form DNA combinations which can be mutagenic.

•V. Nair, C. L. O'Neil, P. G. Wang "Malondialdehyde", *Encyclopedia of Reagents for Organic Synthesis*, 2008, John Wiley & Sons, New York. doi:10.1002/047084289X.rm013.pub2 Article Online Posting Date: March 14, 2008

•** Moore K, Roberts LJ (1998). "Measurement of lipid peroxidation". *Free Radic. Res.* 28 (6): 659–71.

Malondialdehyde, a colourless liquid, is a highly reactive compound that occurs as the enol*. It occurs naturally and is a marker for oxidative stress.**

•V. Nair, C. L. O'Neil, P. G. Wang "Malondialdehyde", *Encyclopedia of Reagents for Organic Synthesis*, 2008, John Wiley & Sons, New York. doi:10.1002/047084289X.rm013.pub2 Article Online Posting Date: March 14, 2008

•** Moore K, Roberts LJ (1998). "Measurement of lipid peroxidation". *Free Radic. Res.* 28 (6): 659–71.

Malondialdehyde is reactive and potentially mutagenic. It has been found in heated edible oils such as sunflower and palm oils. MDA also can be found in tissue sections of joints from patients with osteoarthritis.*

*Tiku ML, Narla H, Jain M, Yalamanchili P (2007). "Glucosamine prevents in vitro collagen degradation in chondrocytes by inhibiting advanced lipoxidation reactions and protein oxidation". *Arthritis Research & Therapy*. 9 (4): R76.

<u>Oils should be</u>
Organic.
Cold pressed.
In small dark glass bottles.
Kept away from heat.
Tops kept on.

Rancid Fats

- Measure the oxidative stability of an oil
- Rancimat method measures the progress of the oxidation reaction
- Measures the volatile oxidation products, largely formic acid
- Biomarker Formic acid to test rancid oils

Allen, J.C. & Hamilton, R.J. (1994). *Rancidity in Foods*. Springer-Verlag GmbH. p. 47.

4-Hydroxynonenal (4-HNE) is an α,β-unsaturated hydroxyalkenal that is produced by lipid peroxidation in cells.

4-HNE has 3 reactive groups: an aldehyde, a double-bond at carbon 2, and a hydroxy group at carbon 4.

*Awasthi, Y. C.; Yang, Y.; Tiwari, N. K.; Patrick, B.; Sharma, A.; Li, J.; Awasthi, S. (2004). "Regulation of 4-hydroxynonenal-mediated signaling by glutathione S-transferases". *Free Radical Biology and Medicine*. 37 (5): 607–619.

It is found throughout all tissues, and in higher quantities during oxidative stress due to the increase in the lipid peroxidation chain reaction.*

*Awasthi, Y. C.; Yang, Y.; Tiwari, N. K.; Patrick, B.; Sharma, A.; Li, J.; Awasthi, S. (2004). "Regulation of 4-hydroxynonenal-mediated signaling by glutathione S-transferases". *Free Radical Biology and Medicine*. 37 (5): 607–619.

4-HNE has been hypothesized to play a key role in cell signal transduction, in a variety of pathways from cell cycle events to cellular adhesion.*

*Awasthi, Y. C.; Yang, Y.; Tiwari, N. K.; Patrick, B.; Sharma, A.; Li, J.; Awasthi, S. (2004). "Regulation of 4-hydroxynonenal-mediated signaling by glutathione S-transferases". *Free Radical Biology and Medicine*. 37 (5): 607–619.

4-Hydroxynonenal is generated in the oxidation of lipids containing polyunsaturated omega-6 acyl groups, such as arachidonic or linoleic groups respectively. *

*Riahi, Y.; Cohen, G.; Shamni, O.; Sasson, S. (2010). "Signaling and cytotoxic functions of 4-hydroxyalkenals". *AJP: Endocrinology and Metabolism*. 299 (6):

These compounds can be produced in cells and tissues of living organisms or in foods during processing or storage, and from these latter can be absorbed through the diet.*

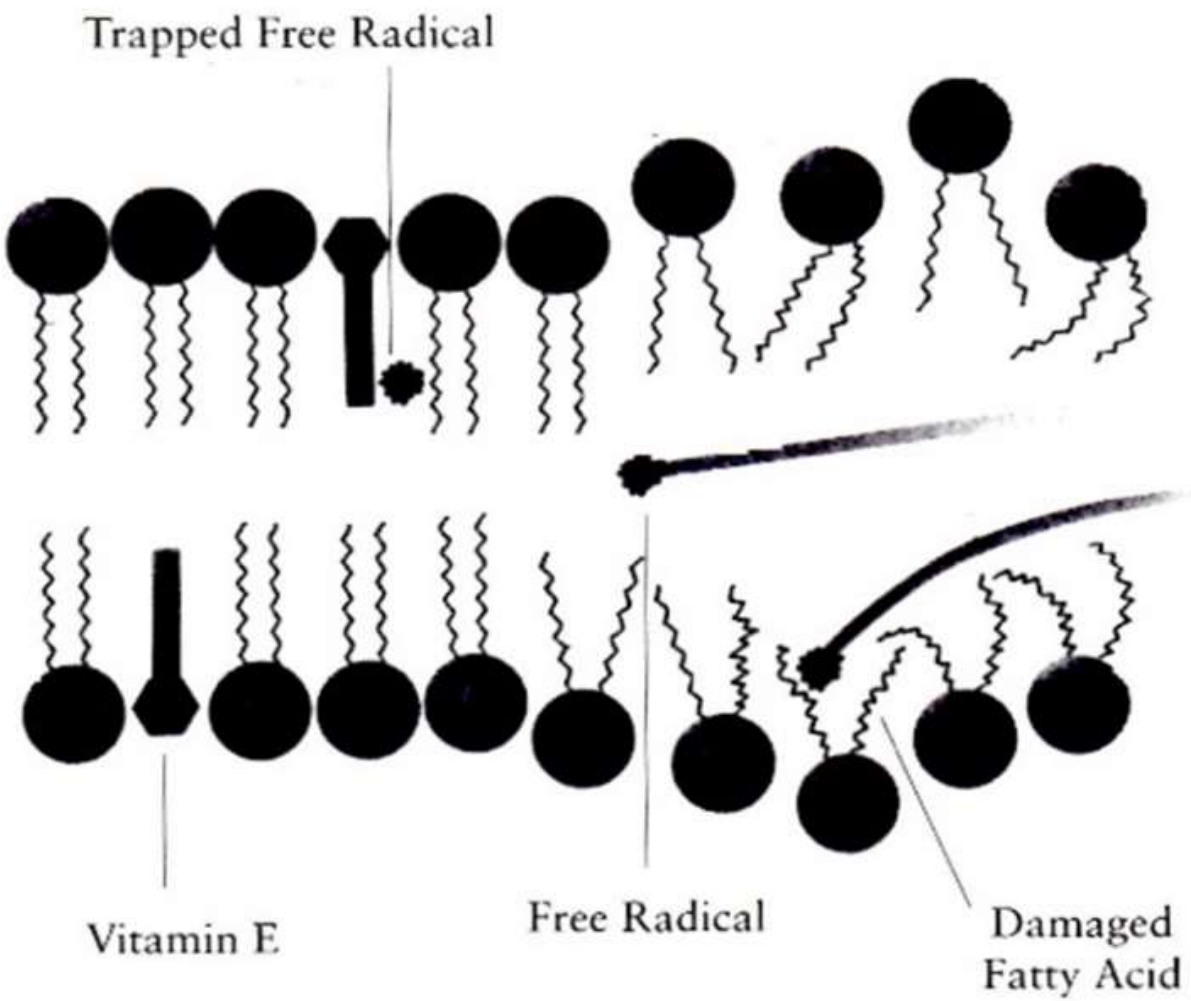
*Riahi, Y.; Cohen, G.; Shamni, O.; Sasson, S. (2010). "Signaling and cytotoxic functions of 4-hydroxyalkenals". *AJP: Endocrinology and Metabolism*. 299 (6)

Since 1991, they are receiving a great deal of attention because they are being considered as possible causal agents of numerous diseases, such as chronic inflammation, neurodegene rative diseases, adult respiratory distresssyndrome, atherogenesis, diabetes and different types of cancer.*

*Zarkovic, N. (2003). "4-Hydroxynonenal as a bioactive marker of pathophysiological processes". *Molecular Aspects of Medicine*. 24(4–5): 281–291.

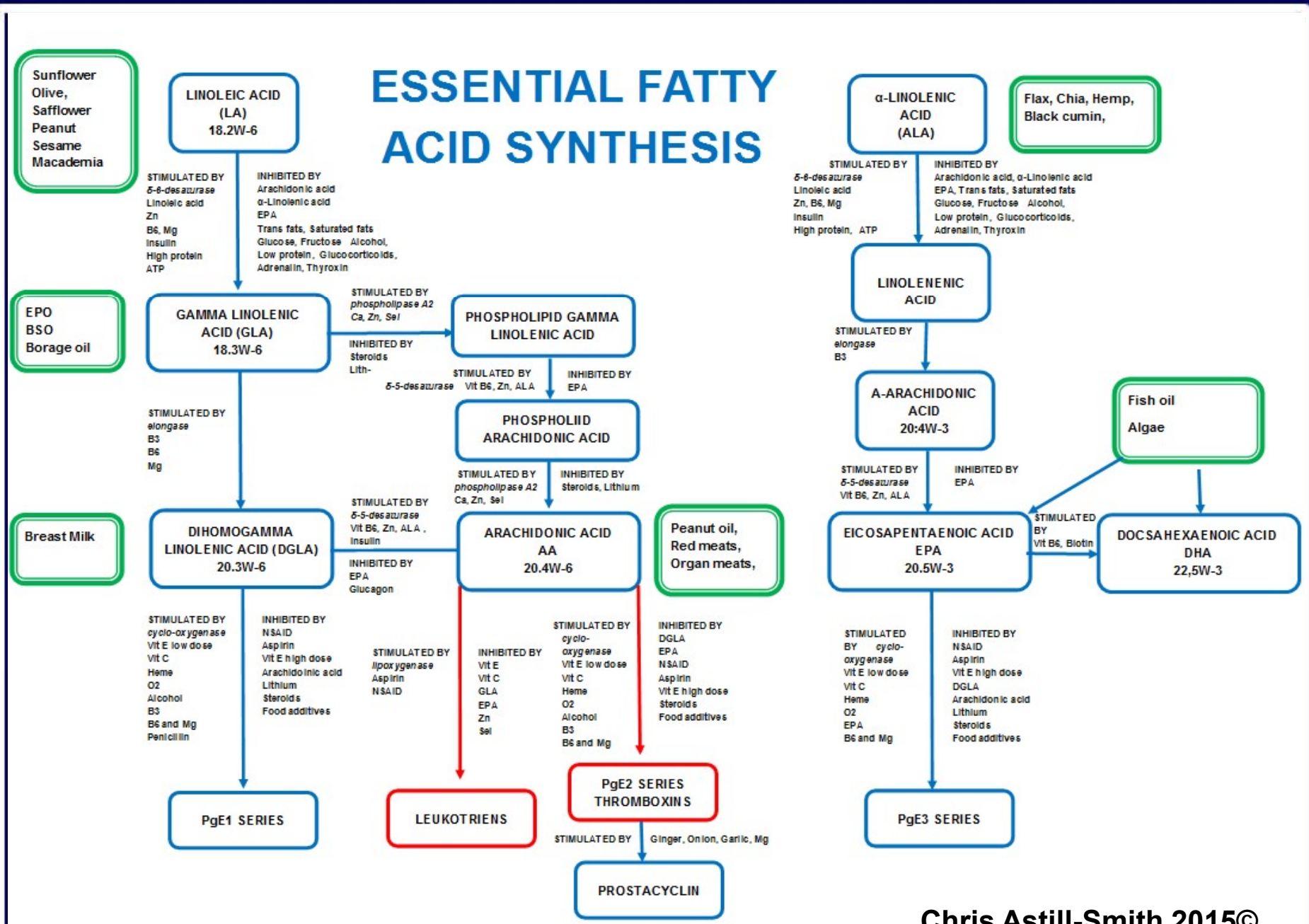
A small group of enzymes are specifically suited to the detoxification and removal of 4-HNE from cells.
Within this group are
Glutathione S-transferases (GSTs),
Aldose reductase,
Aldehyde dehydrogenase.

Vitamin E Activity in Cell membrane



Organic Wheatgerm Oil

- One of highest sources of Vitamin E - 150mg per 100g
- Tocopherols and tocotrienols
- Combination of enzymes, catalysts, plant compounds, minerals
- Synergy of natural components
- Optimal Vitamin E complex


ESSENTIAL FATTY ACID SYNTHESIS
Sunflower
Olive,
Safflower
Peanut
Sesame
Macademia
LINOLEIC ACID (LA) 18.2W-6
STIMULATED BY δ-6-desaturase Linoleic acid Zn B6, Mg Insulin High protein ATP
INHIBITED BY Arachidonic acid α-Linolenic acid EPA Trans fats, Saturated fats Glucose, Fructose Alcohol, Low protein, Glucocorticoids, Adrenalin, Thyroxin
EPO
BSO
Borage oil
GAMMA LINOLENIC ACID (GLA) 18.3W-6
STIMULATED BY phospholipase A2 Ca, Zn, Sel
INHIBITED BY Steroids Lith-
PHOSPHOLIPID GAMMA LINOLENIC ACID
STIMULATED BY Vit B6, Zn, ALA
INHIBITED BY EPA
δ-5-desaturase
PHOSPHOLIID ARACHIDONIC ACID
STIMULATED BY elongase B3 B6 Mg
STIMULATED BY phospholipase A2 Ca, Zn, Sel
INHIBITED BY Steroids, Lithium
Breast Milk
DIHOMOGAMMA LINOLENIC ACID (DGLA) 20.3W-6
STIMULATED BY δ-5-desaturase Vit B6, Zn, ALA , Insulin
INHIBITED BY EPA Glucagon
ARACHIDONIC ACID AA 20.4W-6
Peanut oil, Red meats, Organ meats,
STIMULATED BY cyclo-oxygenase Vit E low dose Vit C Heme O2 Alcohol B3 B6 and Mg Penicillin
INHIBITED BY NSAID Aspirin Vit E high dose Arachidonic acid Lithium Steroids Food additives
PgE1 SERIES
STIMULATED BY lipoxygenase Aspirin NSAID
INHIBITED BY Vit E Vit C GLA EPA Zn Sel
LEUKOTRIENS
STIMULATED BY cyclo-oxygenase Vit E low dose Vit C Heme O2 Alcohol B3 B6 and Mg
INHIBITED BY DGLA EPA NSAID Aspirin Vit E high dose Steroids Food additives
PgE2 SERIES THROMBOXINS
STIMULATED BY Ginger, Onion, Garlic, Mg
PROSTACYCLIN
α-LINOLENIC ACID (ALA)
Flax, Chia, Hemp, Black cumin,
STIMULATED BY δ-6-desaturase Linoleic acid Zn, B6, Mg Insulin High protein, ATP
INHIBITED BY Arachidonic acid, α-Linolenic acid EPA, Trans fats, Saturated fats Glucose, Fructose Alcohol, Low protein, Glucocorticoids, Adrenalin, Thyroxin
LINOLENENIC ACID
STIMULATED BY elongase B3
A-ARACHIDONIC ACID 20:4W-3
Fish oil
Algae
STIMULATED BY δ-5-desaturase Vit B6, Zn, ALA
INHIBITED BY EPA
EICOSAPENTAENOIC ACID EPA 20.5W-3
STIMULATED BY Vit B6, Biotin
DOCSAHEXAENOIC ACID DHA 22,5W-3
STIMULATED BY cyclo-oxygenase Vit E low dose Vit C Heme O2 EPA B6 and Mg
INHIBITED BY NSAID Aspirin Vit E high dose DGLA Arachidonic acid Lithium Steroids Food additives
PgE3 SERIES
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High GLA content oils

- Blackcurrant seed oil
- Evening Primrose oil
- Borage oil

Flider, Frank J (May 2005). "GLA: Uses and New Sources"

Fats and Oils in Dementia

- PUFAs used in brain DHA & AA
- DHA is primarily in cerebral cortex used for working memory and short term memory*
- AA is primarily in hippocampus used for consolidation of short term to long term memory and spatial navigation

*Cederholm T, Salem N Jr, Palmblad J (2013). "ω-3 fatty acids in the prevention of cognitive decline in humans". *Adv Nutr*. 4 (6): 672–6.

DHA Docosahexaenoic acid

- Most abundant omega 3 fatty acid
- 40% of PUFAs in brain
- 50% weight of neuron's membrane
- Synthesized from ALA
- Diet: Fish oil, egg yolks, breast milk

*Cederholm T, Salem N Jr, Palmblad J (2013). "ω-3 fatty acids in the prevention of cognitive decline in humans". *Adv Nutr*. 4 (6): 672–6.

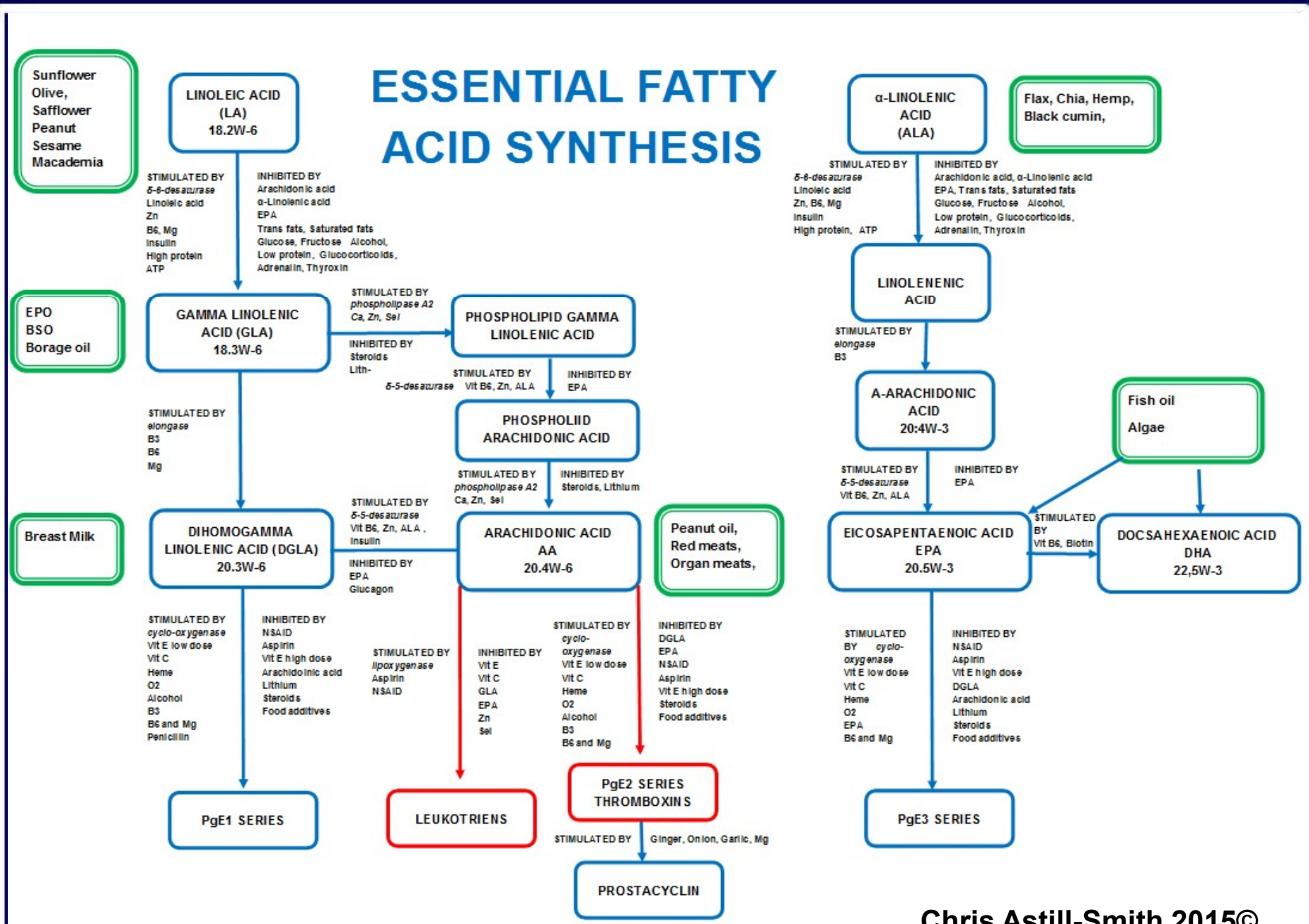
DHA

- Most unsaturated fatty acid in brain
- Structurally comprises 22 carbons and 6 cis double bonds
- Greater tendency to oxidation
- Increases the fluidity of cell membranes

Burdge, G. C.; Wootton, S. A. (2002). "Conversion of alpha-linolenic acid to eicosapentaenoic, docosapentaenoic and docosahexaenoic acids in young women". British Journal of Nutrition.

Synthesis of DHA

"Biosynthesis of docosahexaenoic acid (DHA, 22:6-4, 7,10,13,16,19): two distinct pathways". Prostaglandins, Leukotrienes and Essential Fatty Acids. 68 (2): 181–186. 2003-02-01.


ESSENTIAL FATTY ACID SYNTHESIS
Sunflower
Olive,
Safflower
Peanut
Sesame
Macademia
LINOLEIC ACID (LA) 18.2W-6
STIMULATED BY δ-6-desaturase Linoleic acid Zn B6, Mg Insulin High protein ATP
INHIBITED BY Arachidonic acid α-Linolenic acid EPA Trans fats, Saturated fats Glucose, Fructose Alcohol, Low protein, Glucocorticoids, Adrenalin, Thyroxin
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BSO
Borage oil
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INHIBITED BY Steroids, Lithium
Breast Milk
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Peanut oil, Red meats, Organ meats,
STIMULATED BY cyclo-oxygenase Vit E low dose Vit C Heme O2 Alcohol B3 B6 and Mg Penicillin
INHIBITED BY NSAID Aspirin Vit E high dose Arachidonic acid Lithium Steroids Food additives
PgE1 SERIES
STIMULATED BY lipoxygenase Aspirin NSAID
INHIBITED BY Vit E Vit C GLA EPA Zn Sel
LEUKOTRIENS
STIMULATED BY cyclo-oxygenase Vit E low dose Vit C Heme O2 Alcohol B3 B6 and Mg
INHIBITED BY DGLA EPA NSAID Aspirin Vit E high dose Steroids Food additives
PgE2 SERIES THROMBOXINS
STIMULATED BY Ginger, Onion, Garlic, Mg
PROSTACYCLIN
α-LINOLENIC ACID (ALA)
Flax, Chia, Hemp, Black cumin,
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INHIBITED BY Arachidonic acid, α-Linolenic acid EPA, Trans fats, Saturated fats Glucose, Fructose Alcohol, Low protein, Glucocorticoids, Adrenalin, Thyroxin
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INHIBITED BY EPA
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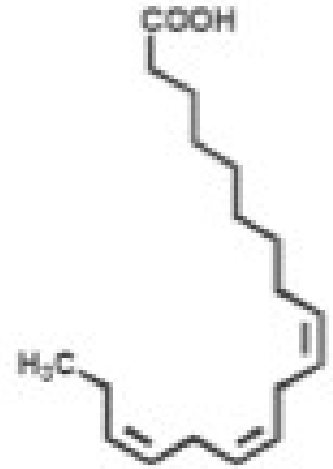
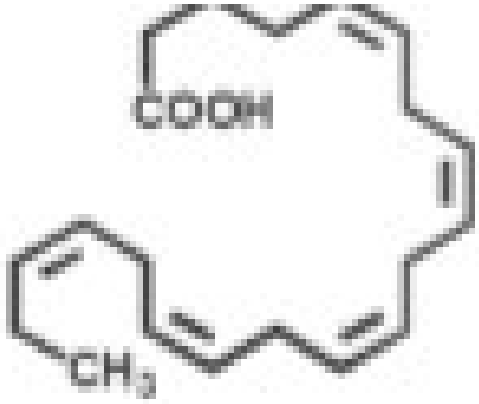
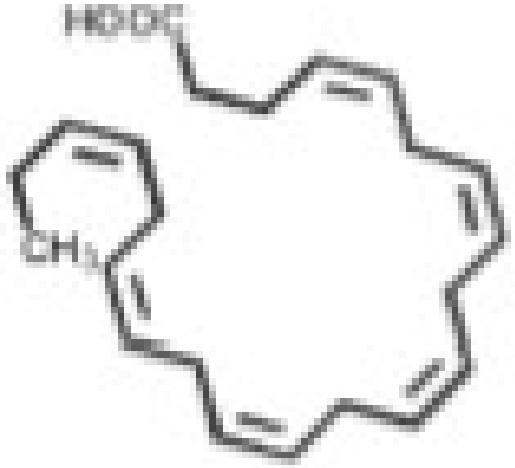
INHIBITED BY NSAID Aspirin Vit E high dose DGLA Arachidonic acid Lithium Steroids Food additives
PgE3 SERIES
Chris Astill-Smith 2015©



α-Linolenic acid
18:3.ω3 (ALA)



Eicosapentaenoic acid
20:5.ω3 (EPA)



Docosahexaenoic acid
22:6.ω3 (DHA)

Enzymes in EFA conversion

- Desaturation – addition of a double bond
- Delta-6-desaturase
- Delta-5-desaturase
- Elongation – addition of 2 carbon atoms
- Elongase enzyme

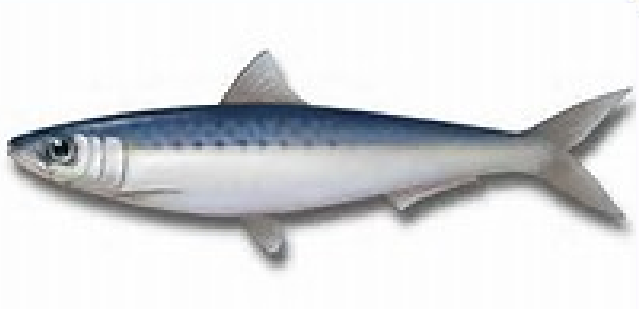
"Biosynthesis of docosahexaenoic acid (DHA, 22:6-4, 7,10,13,16,19): two distinct pathways". Prostaglandins, Leukotrienes and Essential Fatty Acids. 68 (2): 181–186. 2003-02-01.

Enzymes in EFA conversion

- Genetic variability in enzymes involved in fatty acid metabolism influence ability to generate LC PUFAs
- Polymorphisms in genes or acquired defect
- Look for the co-enzyme needed and mineral co-factors

"Biosynthesis of docosahexaenoic acid (DHA, 22:6-4, 7,10,13,16,19): two distinct pathways". Prostaglandins, Leukotrienes and Essential Fatty Acids. 68 (2): 181–186. 2003-02-01.

DHA Dietary Sources



- Top fish sources:
  - Tuna, mackerel, swordfish, salmon, anchovies, herring, sardines, caviar
- Cooked salmon 500 – 1500 mg per 100g
- Recommended daily intake – 650 mg DHA plus EPA
- Algae based oils. Egg yolk

"EPA and DHA Content of Fish Species. Appendix G2". US Department of Agriculture. 2005. Retrieved 15 September 2013.

Research on DHA and Dementia

- Low DHA associated with cognitive decline
- Likely to develop dementia and other cognitive disorders
- Study in Canada – Alzheimer's patients had lower DHA level

Yurko-Mauro, K; McCarthy, D; Rom, D; Nelson, E. B.; Ryan, A. S.; Blackwell, A; Salem Jr, N; Stedman, M; Midas, Investigators (2010). "Beneficial effects of docosahexaenoic acid on cognition in age-related cognitive decline". *Alzheimer's & Dementia*. 6 (6): 456–64.

Research on DHA and Dementia

- DHA accumulates in phosphotidylserine (PS)
- PS controls apoptosis and low DHA levels lower neural cell PS and increases neural cell death
- Particularly in hippocampus associated with memory consolidation

Yurko-Mauro, K; McCarthy, D; Rom, D; Nelson, E. B.; Ryan, A. S.; Blackwell, A; Salem Jr, N; Stedman, M; Midas, Investigators (2010). "Beneficial effects of docosahexaenoic acid on cognition in age-related cognitive decline". *Alzheimer's & Dementia*. 6 (6): 456–64.

DHA and Alzheimer's

- Sufferers have much lower levels in neurons of hippocampus
- Hippocampus is severely affected in Alzheimer's – working memory
- DHA supplementation improves memory in Alzheimer's and age related memory loss

Yurko-Mauro, K; McCarthy, D; Rom, D; Nelson, E. B.; Ryan, A. S.; Blackwell, A; Salem Jr, N; Stedman, M; Midas, Investigators (2010). "Beneficial effects of docosahexaenoic acid on cognition in age-related cognitive decline". *Alzheimer's & Dementia*. 6 (6): 456–64.

DHA and Alzheimer's

- Studies in mice and primates show DHA depleted diets impair learning and memory
- Re-feeding DHA diet reverses these impairments
- Chicago and Rotterdam studies found a 60% reduction in Alzheimer's taking omega 3 oils

Yurko-Mauro, K; McCarthy, D; Rom, D; Nelson, E. B.; Ryan, A. S.; Blackwell, A; Salem Jr, N; Stedman, M; Midas, Investigators (2010). "Beneficial effects of docosahexaenoic acid on cognition in age-related cognitive decline". *Alzheimer's & Dementia*. 6 (6): 456–64.

DHA and Alzheimer's

- Also found that intake of plant derived omega 3 ALA was associated with a reduction in risk of Alzheimer's in subjects with APOE4.
- APOE4 is a powerful indicator of Alzheimer's.

Yurko-Mauro, K; McCarthy, D; Rom, D; Nelson, E. B.; Ryan, A. S.; Blackwell, A; Salem Jr, N; Stedman, M; Midas, Investigators (2010). "Beneficial effects of docosahexaenoic acid on cognition in age-related cognitive decline". *Alzheimer's & Dementia*. 6 (6): 456–64.

Conversion of fatty acids to DHA

- LNA not converted to DHA efficiently in humans
- LNA elongated, desaturated, transported to and from peroxisomes and then shortened from 24:6 to 22:6
- Estimated that 10-40g of flax seed oil to produce 200 mg per day

Clinical Research into DHA

- Marine algae very expensive
- Deficiency in DHA caused by conversion problems

Summary of DHA benefits

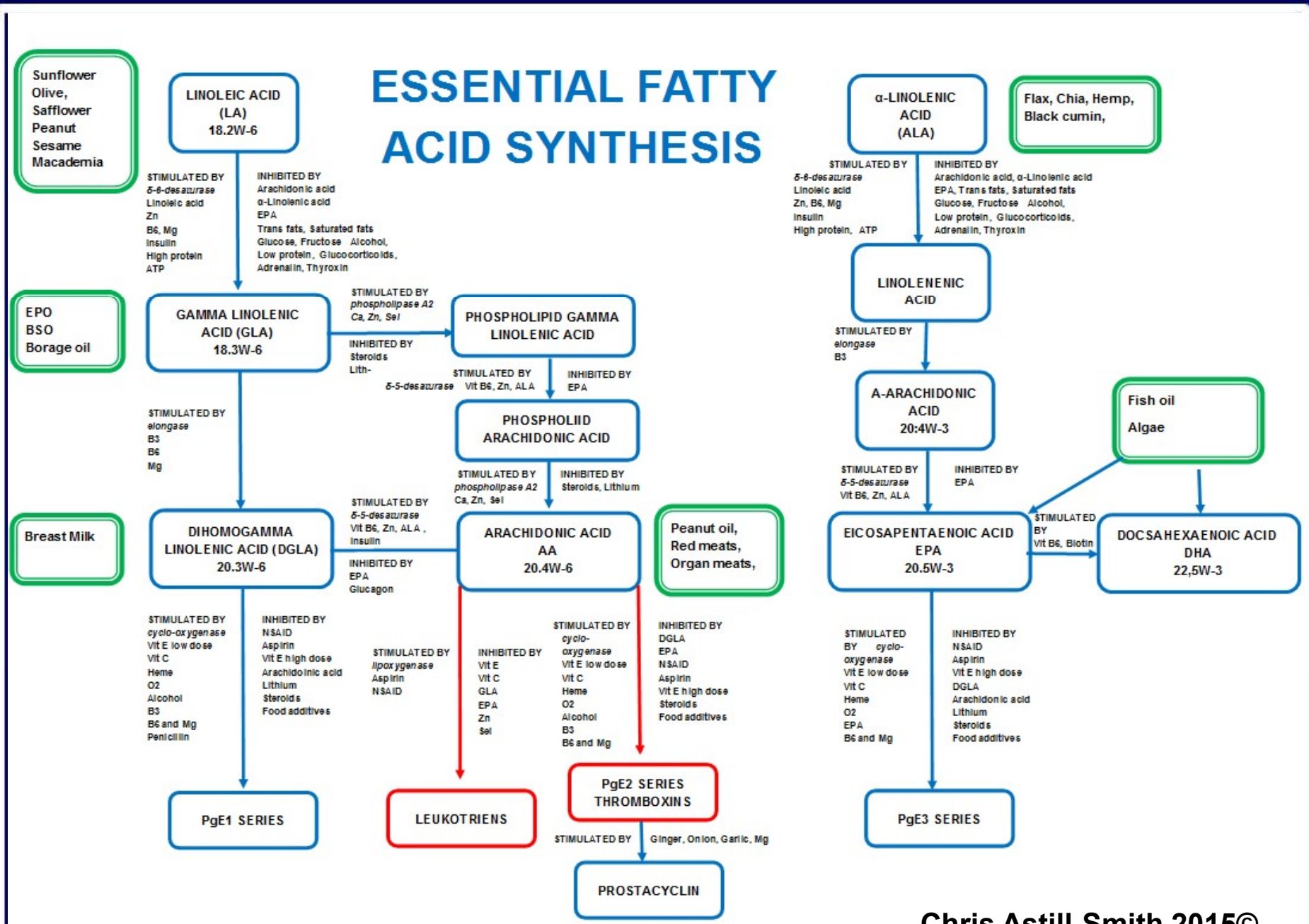
- Crucial for healthy structure and function of brain
- Essential for the adult brain where it impacts brain structure and signalling system
- Promotes nervous system development and optimal memory
- Helps prevent age related memory decline & Alzheimer

Arachidonic Acid (AA)

- Neurological health is dependent on sufficient quantities of AA
- Omega 6 fatty acid, from LA
- Maintains the hippocampal cell membrane fluidity
- Protects brain from oxidative stress
- Dietary source meat, eggs, dairy

Arachidonic Acid (AA)

- Activates syntaxin-3, a protein involved in growth & repair of neurons
- Involved in early neurological development
- In adults the disturbed metabolism contributes to Alzheimer's and dementia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INHIBITED BY EPA
PHOSPHOLIID ARACHIDONIC ACID
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INHIBITED BY NSAID Aspirin Vit E high dose DGLA Arachidonic acid Lithium Steroids Food additives
PgE3 SERIES
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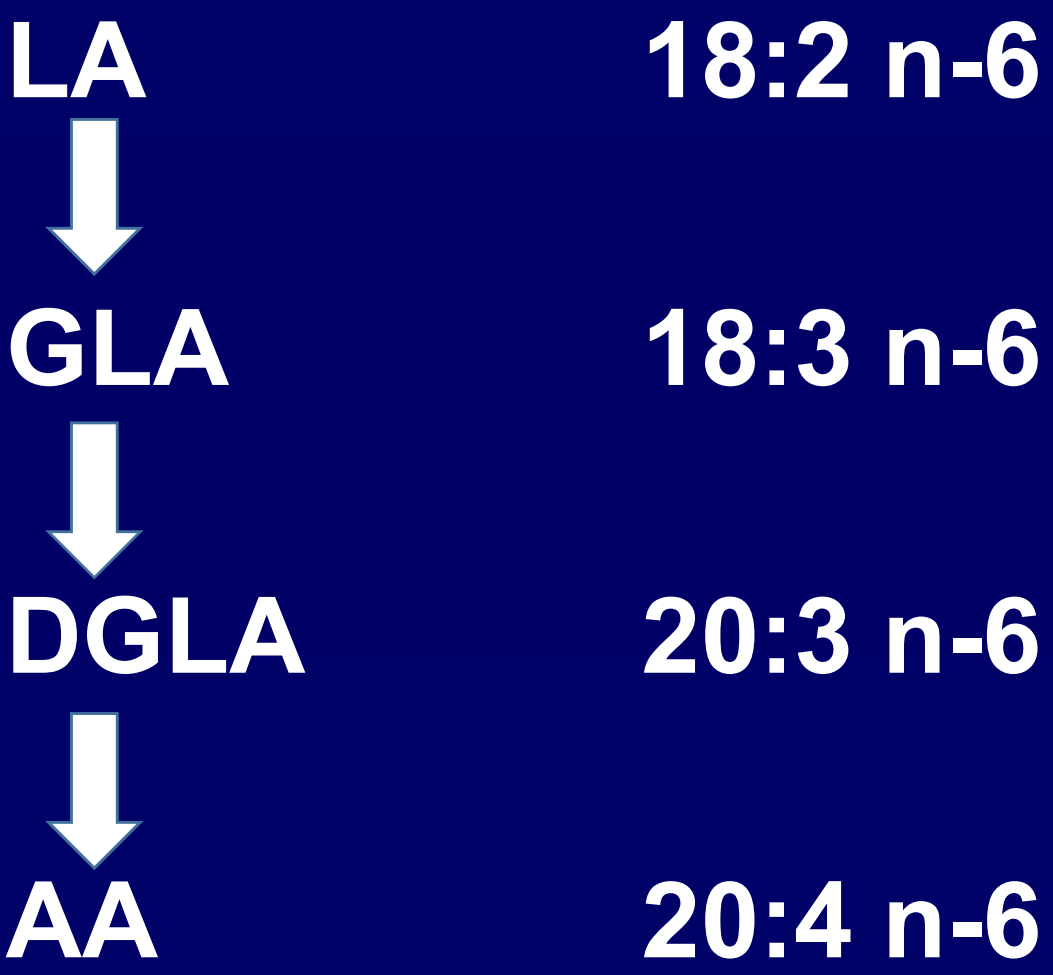
Foods high in AA

- Red meat – beef, lamb, veal, venison, organ meats
- Poultry
- Pork – pork loin in particular
- Oils – peanut, sesame, olive, avocado
- Egg yolks

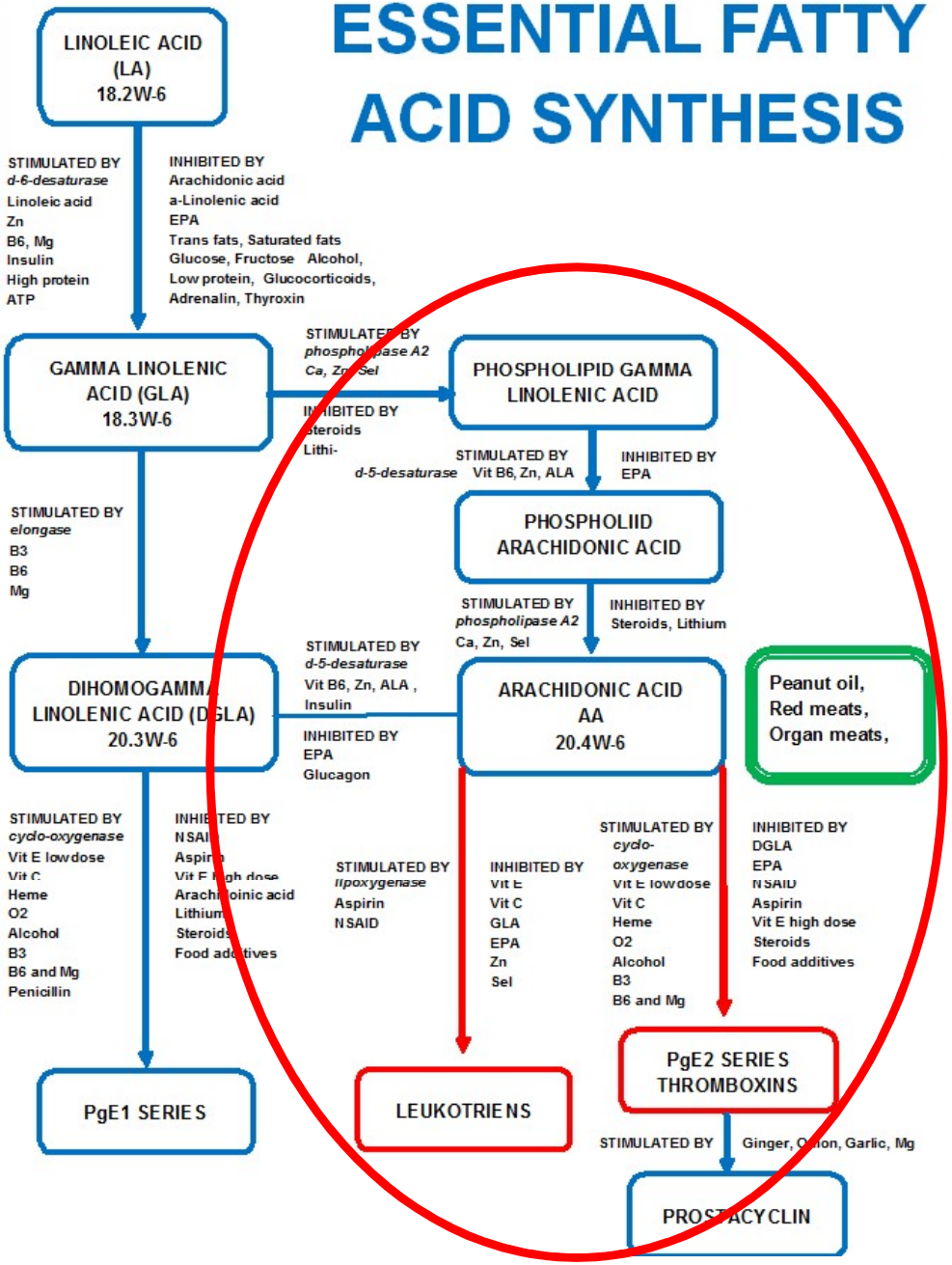
Synthesis of AA



Test for defects in AA synthesis

- Main blockage tends to be between LA and GLA, delta-6-desaturase enzyme, P5P, Zinc, Magnesium
- Pathway affected if have blockages in other parts of EFA synthesis
- Dietary factors – trans fats, saturated fats, glucose, alcohol
- Hormonal imbalances – adrenalin, thyroxin, glucocorticoids

ESSENTIAL FATTY ACID SYNTHESIS



AA as the Bad Guy

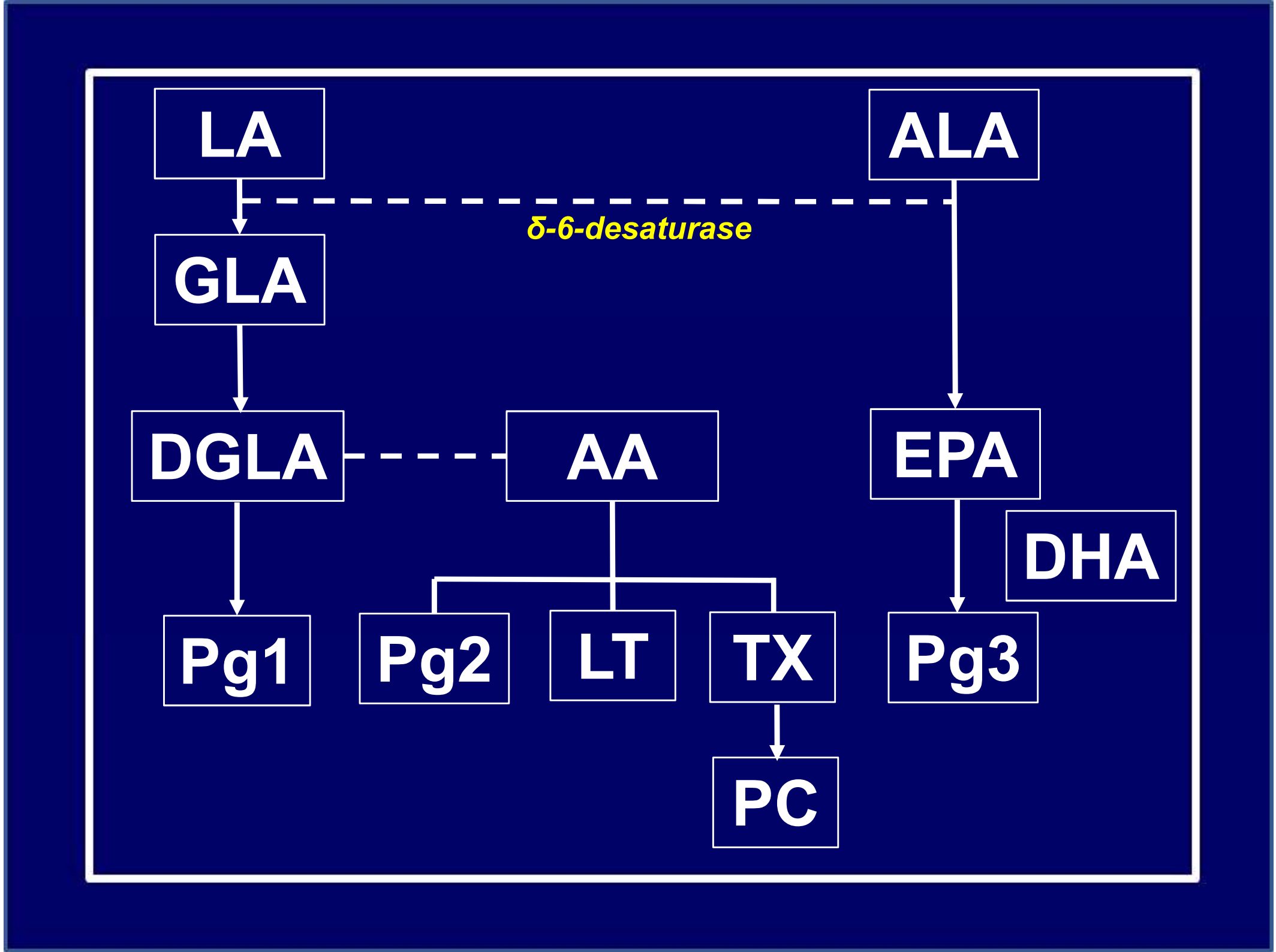
- Key inflammatory intermediate
- AA is metabolised to both pro-inflammatory and anti-inflammatory molecules
- Keep Inflammatory process under control, balance of hormones
- Continual low level inflammation destroys organs including brain

AA as the Bad Guy

- Remove building blocks of excess
- Reduce intake of bad omega 6 fats, trans & hydrogenated fats
- Keep insulin levels low and stable
- Nutrients from chart
- Introduce ginger, onion, garlic (GOG) into diet

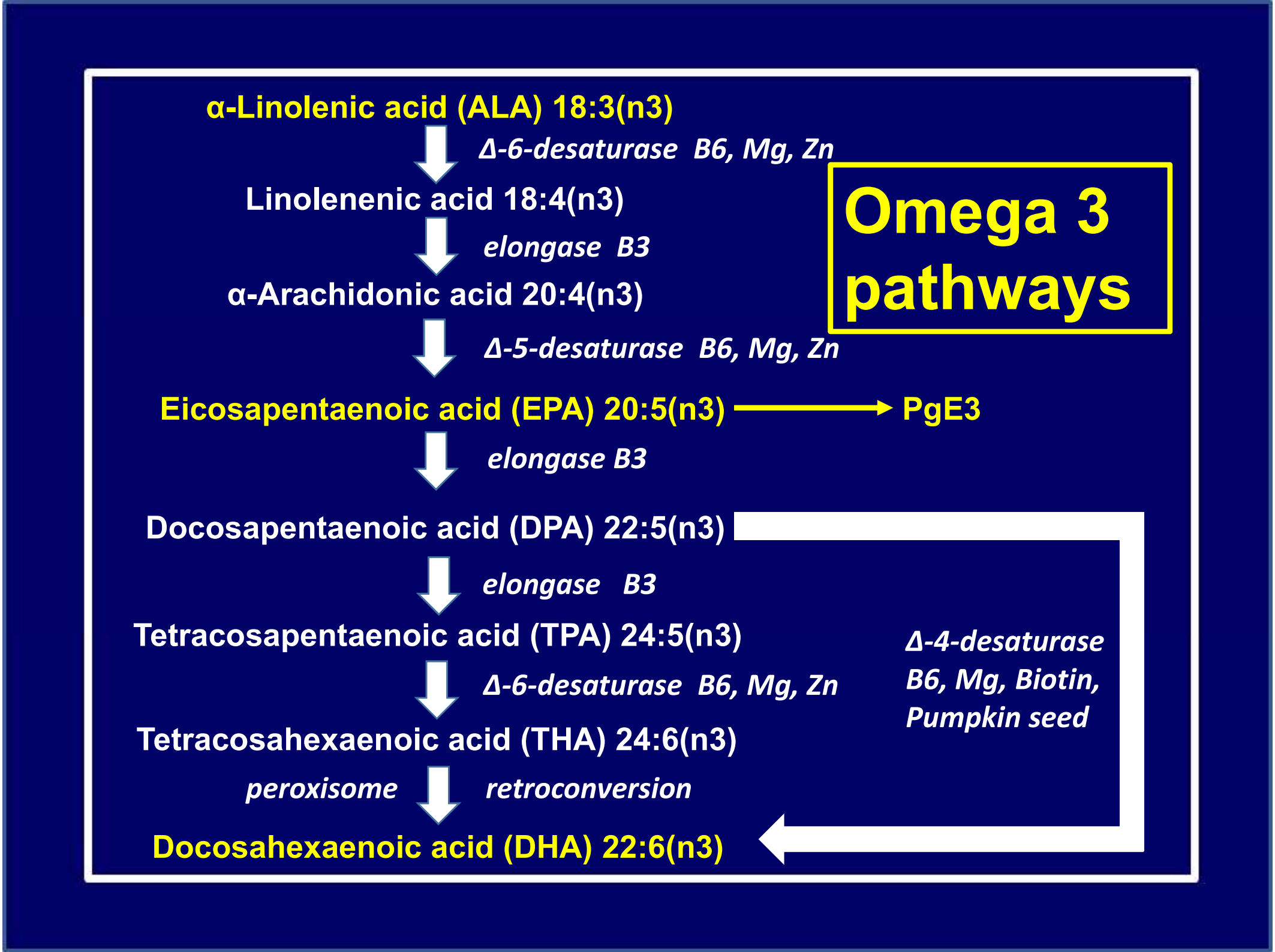
FATS in the Prevention of Memory Loss and Dementia

- Maintain a good supply high quality, cold pressed parent essential oils
- ORGANIC is paramount – toxins cause of neurological decline
- Guard against rancid fats and lipid peroxidation
- Check levels of DHA & AA and fatty acid synthesis


LA
ALA
δ-6-desaturase
GLA
DGLA
AA
EPA
DHA
Pg1
Pg2
LT
TX
Pg3
PC

Key nutrients for synthesising the phospholipids

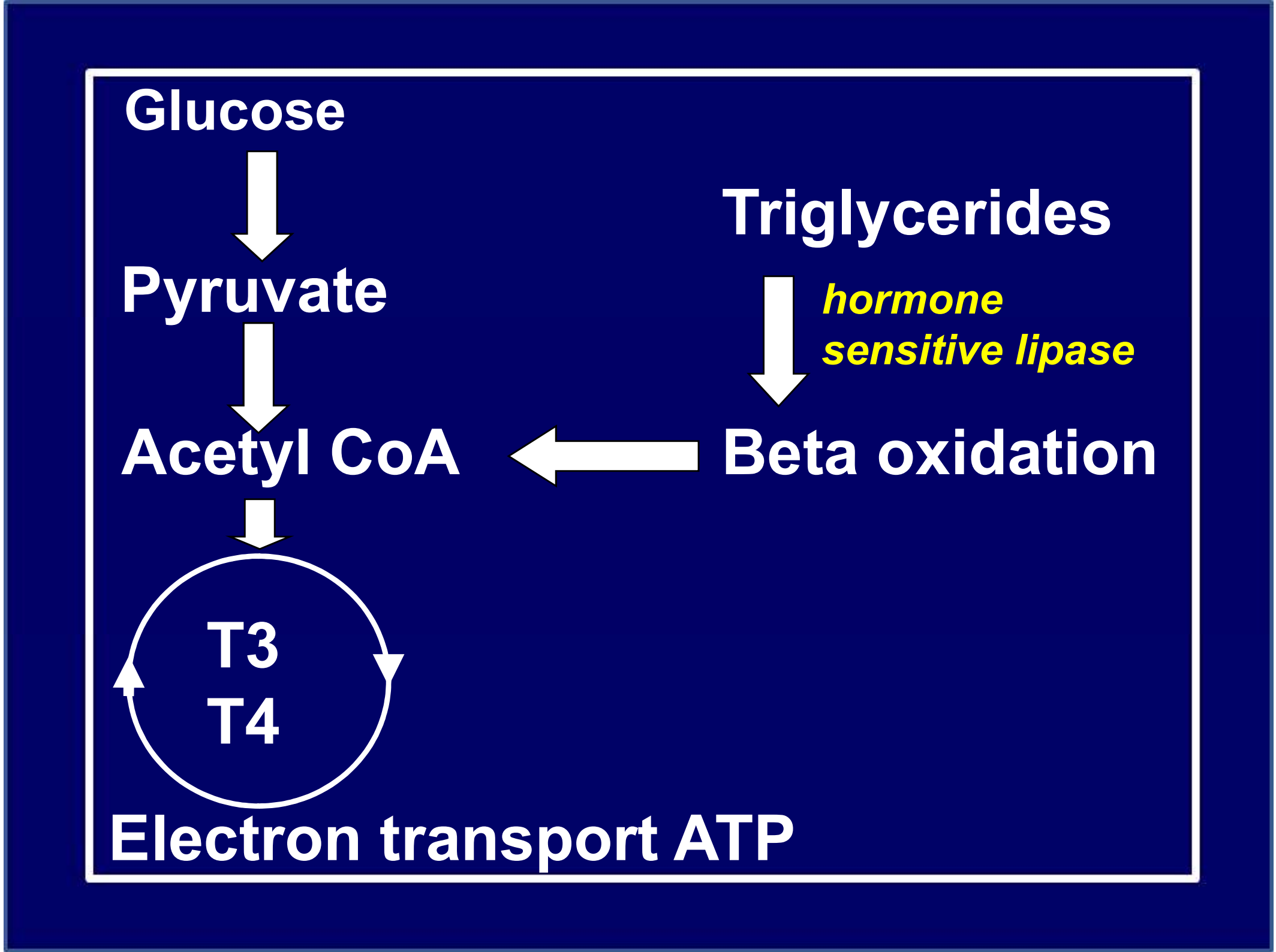
Acetyl CoA (Vit B5, Magnesium, P5P)
NAD, NADPH (Vit B3 complex)
Mg, Zn, SAM (Mg, P-5-P, Folates, B12)
Choline
Serine
Inositol
Saturated fatty acids C16-18
Unsaturated fatty acids C18-24
Lecithin

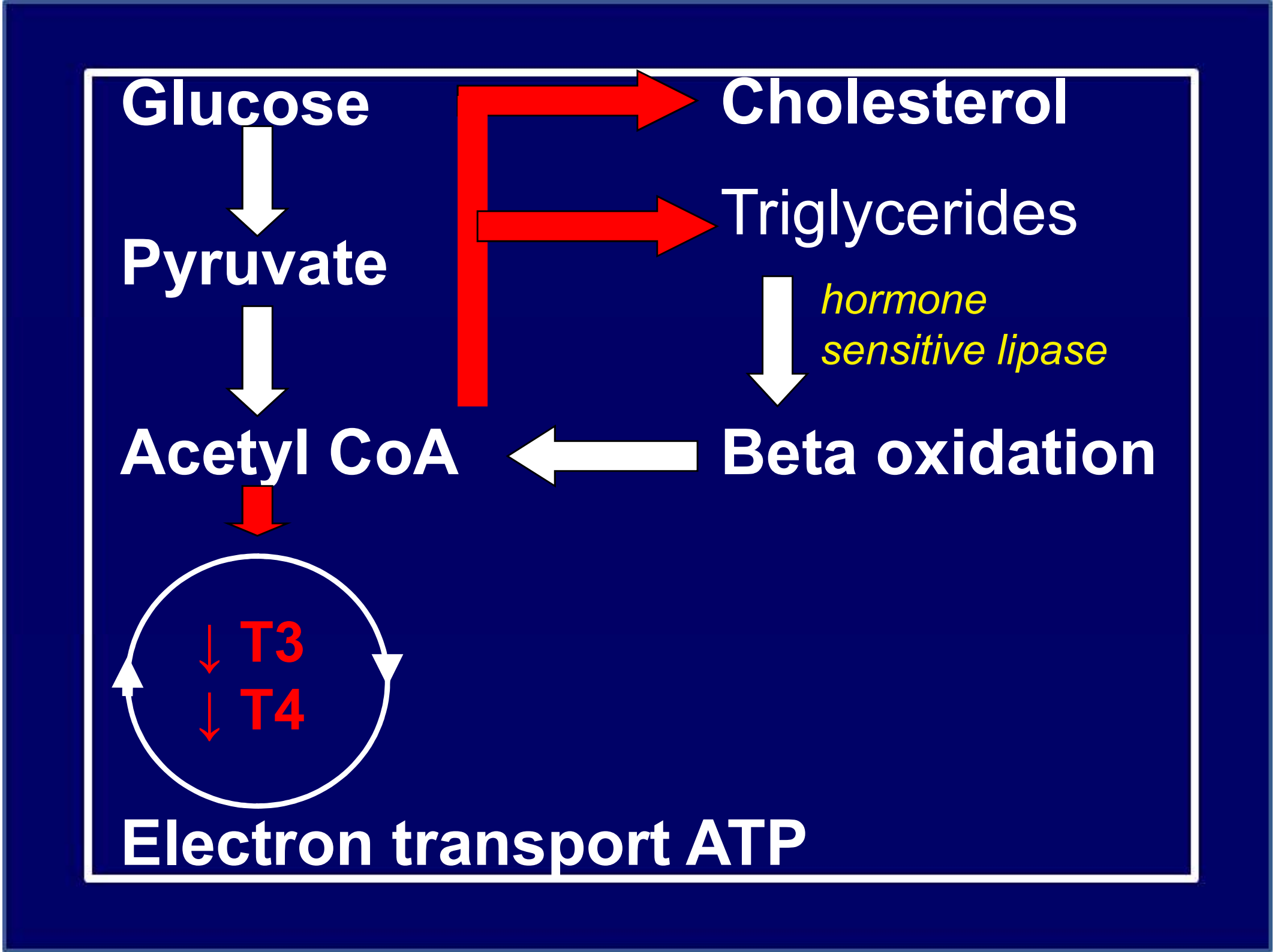

α-Linolenic acid (ALA) 18:3(n3)
Δ-6-desaturase B6, Mg, Zn
Linolenenic acid 18:4(n3)
elongase B3
α-Arachidonic acid 20:4(n3)
Δ-5-desaturase B6, Mg, Zn
Eicosapentaenoic acid (EPA) 20:5(n3)
PgE3
elongase B3
Docosapentaenoic acid (DPA) 22:5(n3)
elongase B3
Tetracosapentaenoic acid (TPA) 24:5(n3)
Δ-6-desaturase B6, Mg, Zn
Tetracosahexaenoic acid (THA) 24:6(n3)
peroxisome
retroconversion
Docosahexaenoic acid (DHA) 22:6(n3)
Δ-4-desaturase
B6, Mg, Biotin,
Pumpkin seed
Omega 3
pathways

CHOLESTEROL

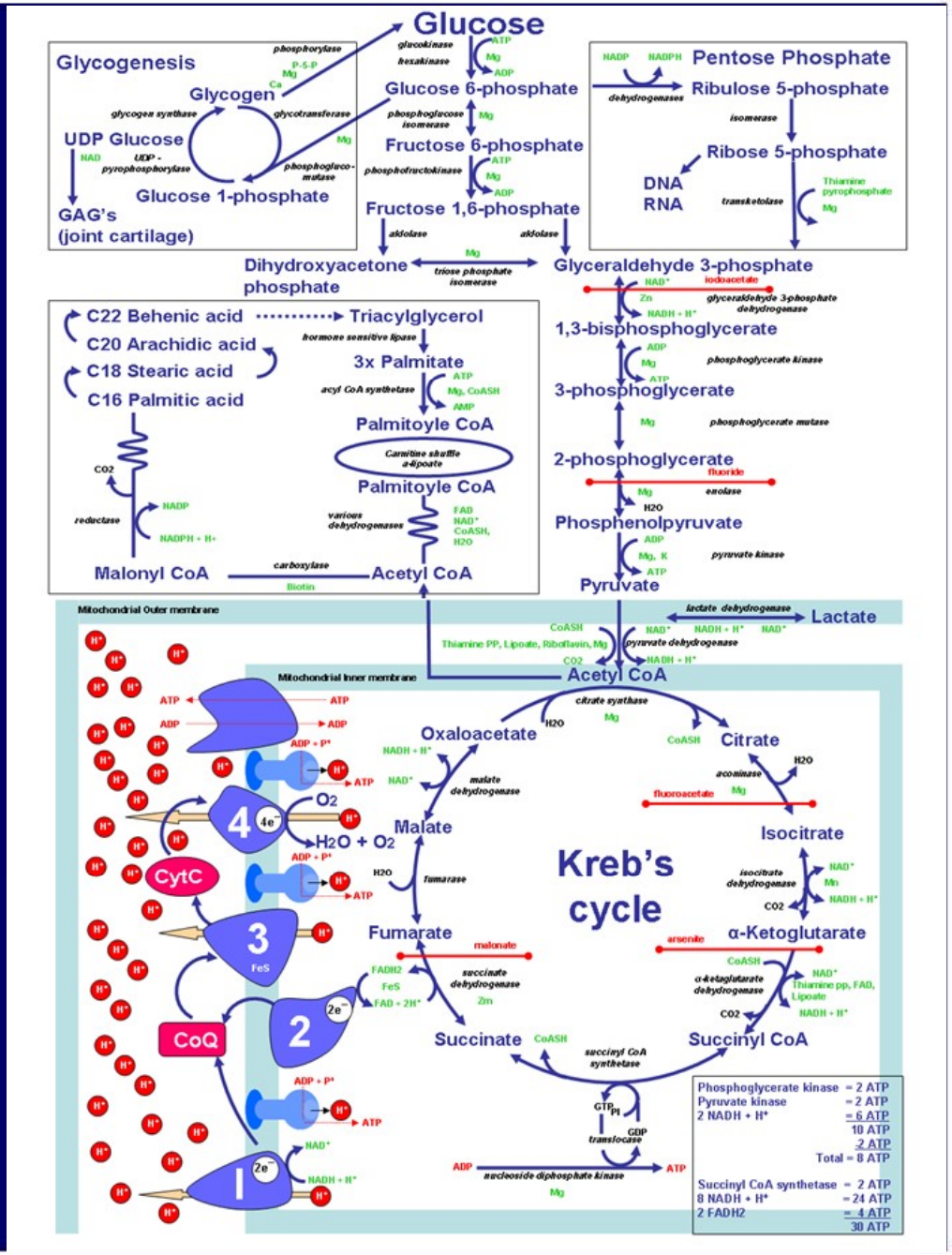
and

TRIGLYCERIDES


Glucose
Pyruvate
Acetyl CoA
T3
T4
Electron transport ATP
Triglycerides
hormone
sensitive lipase
Beta oxidation


Glucose
Pyruvate
Acetyl CoA
Cholesterol
Triglycerides
hormone
sensitive lipase
Beta oxidation
↓ T3
↓ T4
Electron transport ATP

Energy



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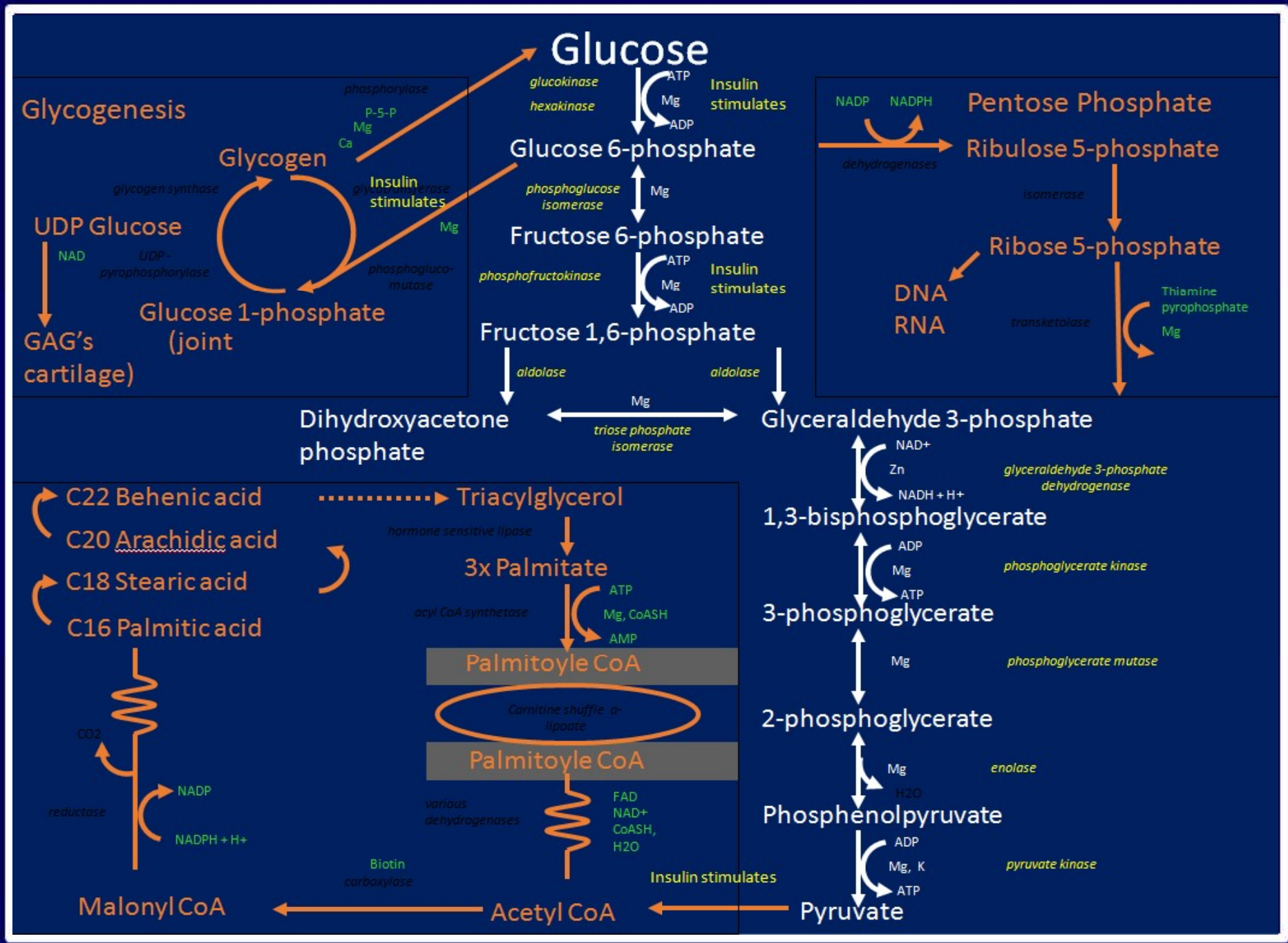
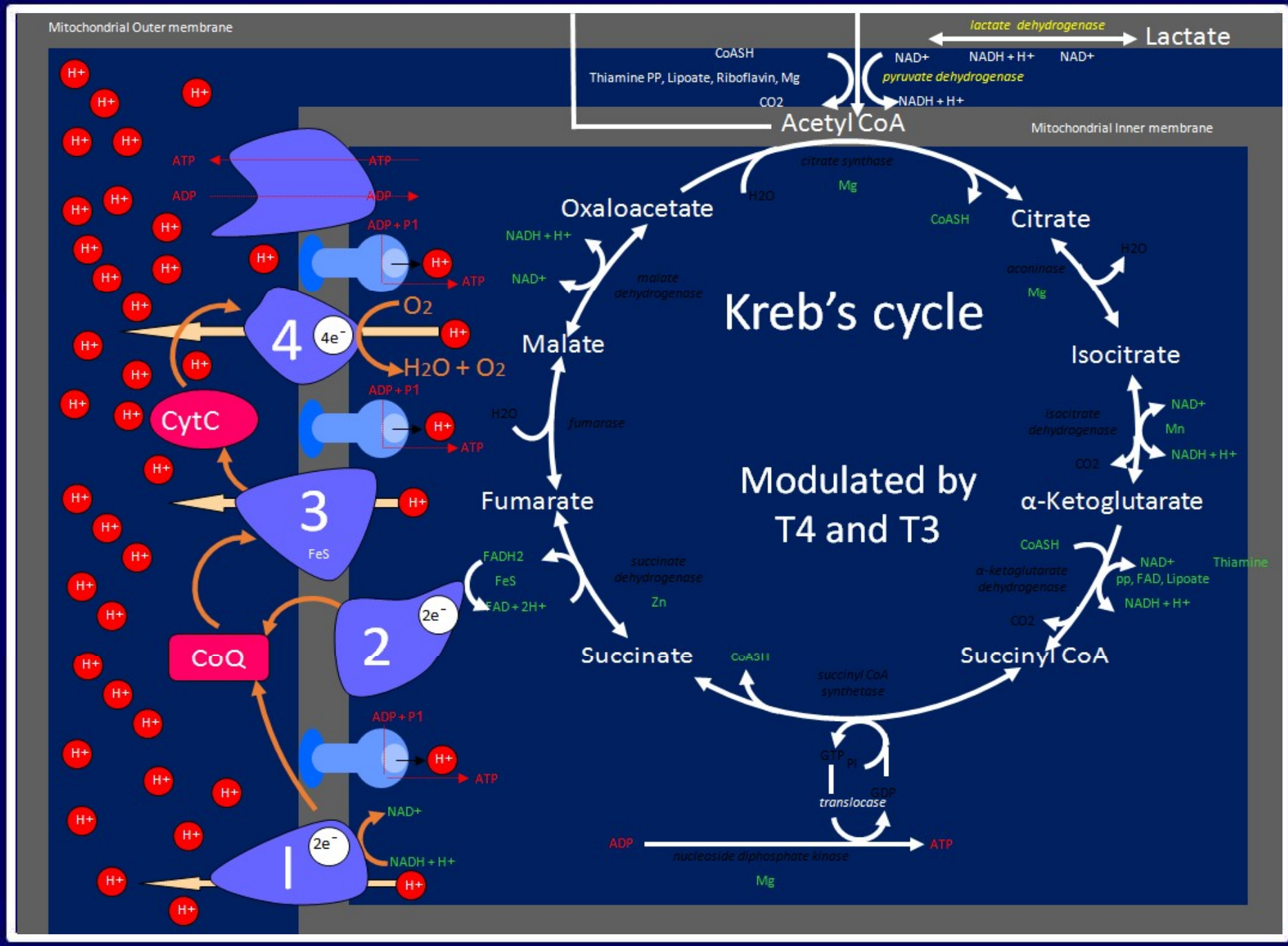
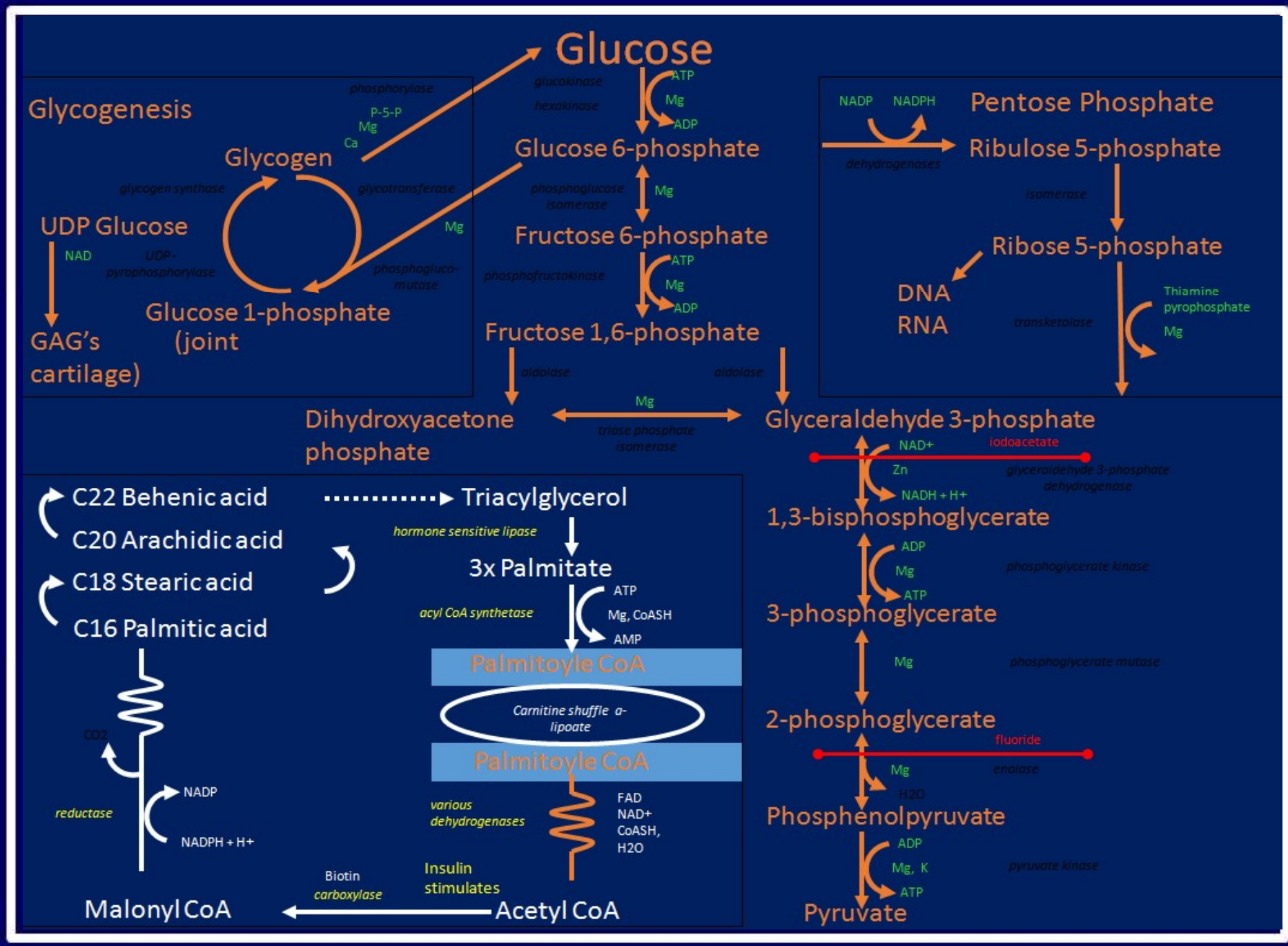

Glucose
glucokinase
hexakinase
ATP
Mg
ADP
Insulin stimulates
Glycogenesis
phosphorylase
P-5-P
Mg
Ca
Glycogen
glycogen synthase
glycogen debranching enzyme
Insulin stimulates
Mg
UDP Glucose
NAD
UDP-pyrophosphorylase
phosphoglucomutase
Glucose 1-phosphate
GAG's cartilage)
(joint
Glucose 6-phosphate
phosphoglucose isomerase
Mg
Fructose 6-phosphate
phosphofructokinase
ATP
Mg
ADP
Insulin stimulates
Fructose 1,6-phosphate
aldolase
aldolase
Mg
triose phosphate isomerase
Dihydroxyacetone phosphate
Glyceraldehyde 3-phosphate
NADP
NADPH
dehydrogenases
Pentose Phosphate
Ribulose 5-phosphate
isomerase
Ribose 5-phosphate
DNA
RNA
transketolase
Thiamine pyrophosphate
Mg
NAD+
Zn
NADH + H+
glyceraldehyde 3-phosphate dehydrogenase
1,3-bisphosphoglycerate
ADP
Mg
ATP
phosphoglycerate kinase
3-phosphoglycerate
Mg
phosphoglycerate mutase
2-phosphoglycerate
Mg
H2O
enolase
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 shuffle a-lipoate
Palmitoyle CoA
CO2
NADP
reductase
NADPH + H+
various dehydrogenases
FAD
NAD+
CoASH,
H2O
Biotin carboxylase
Malonyl CoA
Acetyl CoA
Insulin stimulates


Mitochondrial Outer membrane
Mitochondrial Inner membrane
lactate dehydrogenase
Lactate
NAD+
NADH + H+
NAD+
CoASH
Thiamine PP, Lipoate, Riboflavin, Mg
pyruvate dehydrogenase
CO2
NADH + H+
Acetyl CoA
citrate synthase
Mg
H2O
Oxaloacetate
CoASH
Citrate
NADH + H+
NAD+
malate dehydrogenase
aconinase
Mg
H2O
Kreb's cycle
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
Zn
CoASH
α-ketoglutarate dehydrogenase
NAD+
pp, FAD, Lipoate
NADH + H+
Thiamine
CO2
Succinate
CoASH
Succinyl CoA
succinyl CoA synthetase
GTP
Pi
GDP
translocase
ADP
nucleoside diphosphate kinase
ATP
Mg
ATP
ADP
ADP + P1
ATP
O2
4e-
H2O + O2
CytC
3
FeS
2e-
2
CoQ
2e-
1
NAD+
NADH + H+
H+


Glucose
glucokinase
hexakinase
ATP
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ADP
Glycogenesis
phosphorylase
P-5-P
Mg
Ca
Glycogen
glycogen synthase
glycotransferase
UDP Glucose
NAD
UDP-pyrophosphorylase
Mg
phosphoglucomutase
Glucose 1-phosphate
GAG's cartilage)
(joint
Glucose 6-phosphate
phosphoglucose isomerase
Mg
Fructose 6-phosphate
phosphofructokinase
ATP
Mg
ADP
Fructose 1,6-phosphate
aldolase
aldolase
NADP
NADPH
Pentose Phosphate
dehydrogenases
Ribulose 5-phosphate
isomerase
Ribose 5-phosphate
DNA
RNA
transketolase
Thiamine pyrophosphate
Mg
Dihydroxyacetone phosphate
Mg
triose phosphate isomerase
Glyceraldehyde 3-phosphate
NAD+
iodoacetate
Zn
glyceraldehyde 3-phosphate dehydrogenase
NADH + H+
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 shuffle a-lipoate
Palmitoyle CoA
various dehydrogenases
FAD
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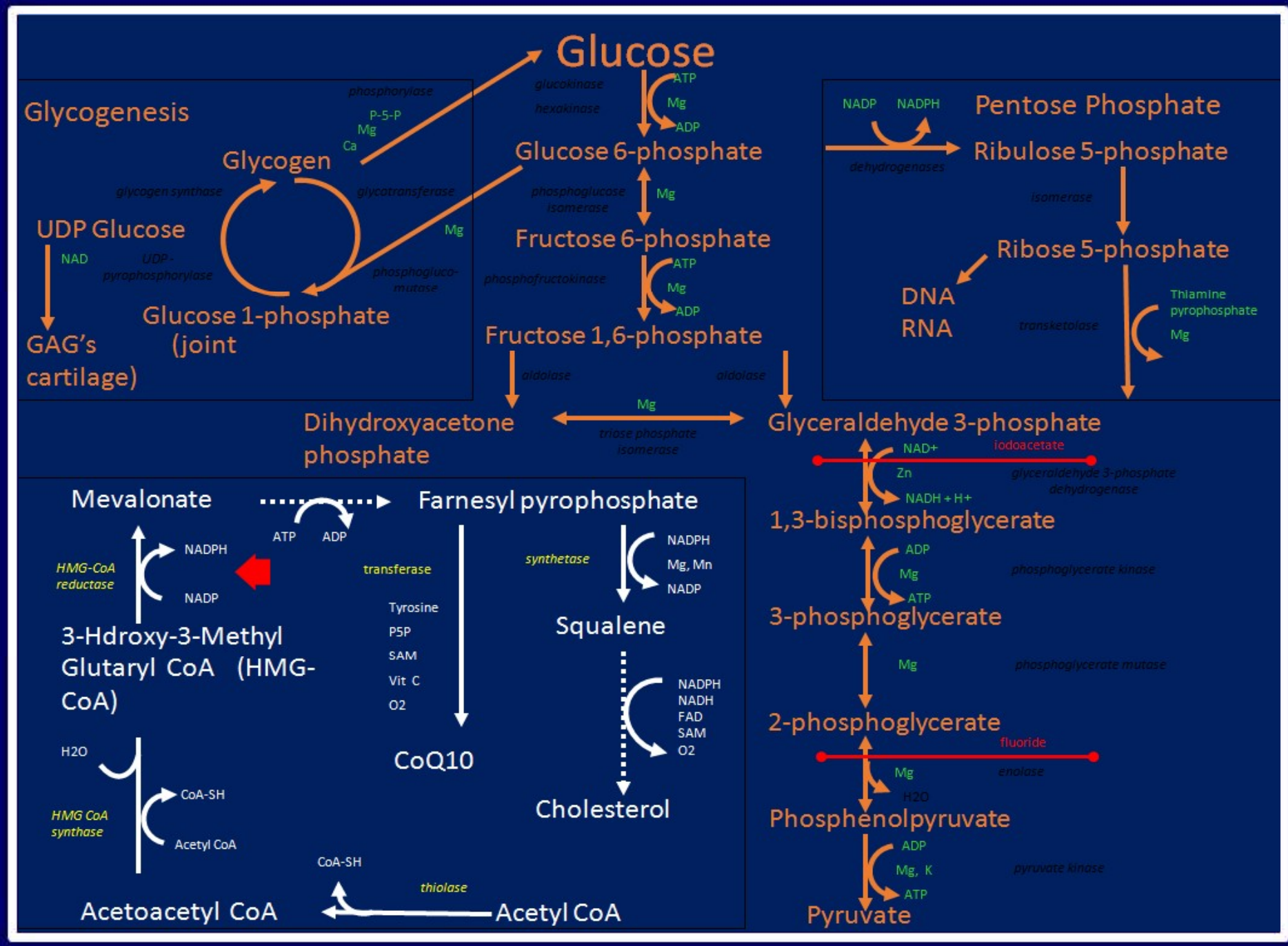
NADPH + H+
Malonyl CoA
Biotin carboxylase
Insulin stimulates
Acetyl CoA


Glucose
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ADP
Glycogenesis
phosphorylase
P-5-P
Mg
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Glycogen
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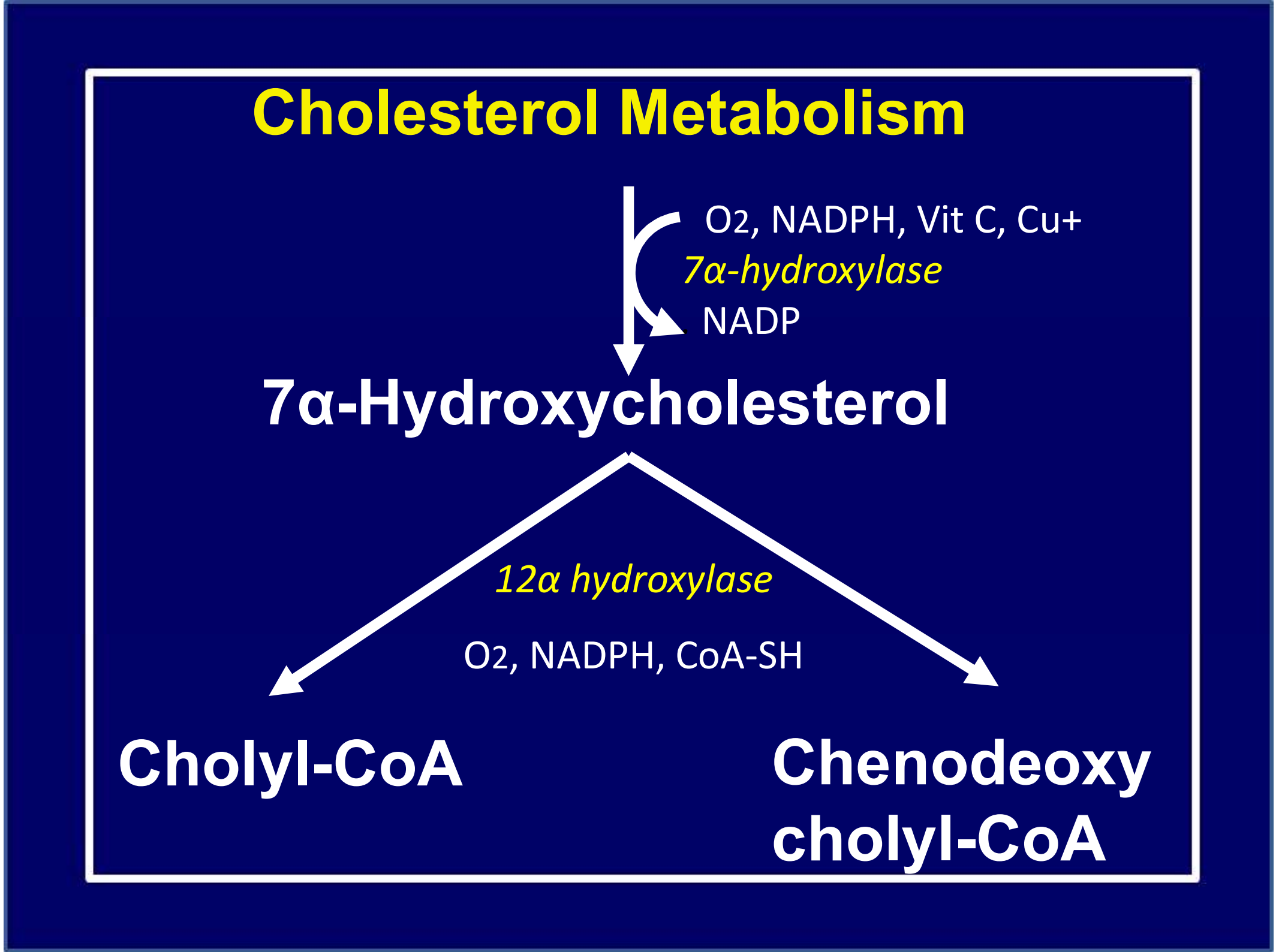
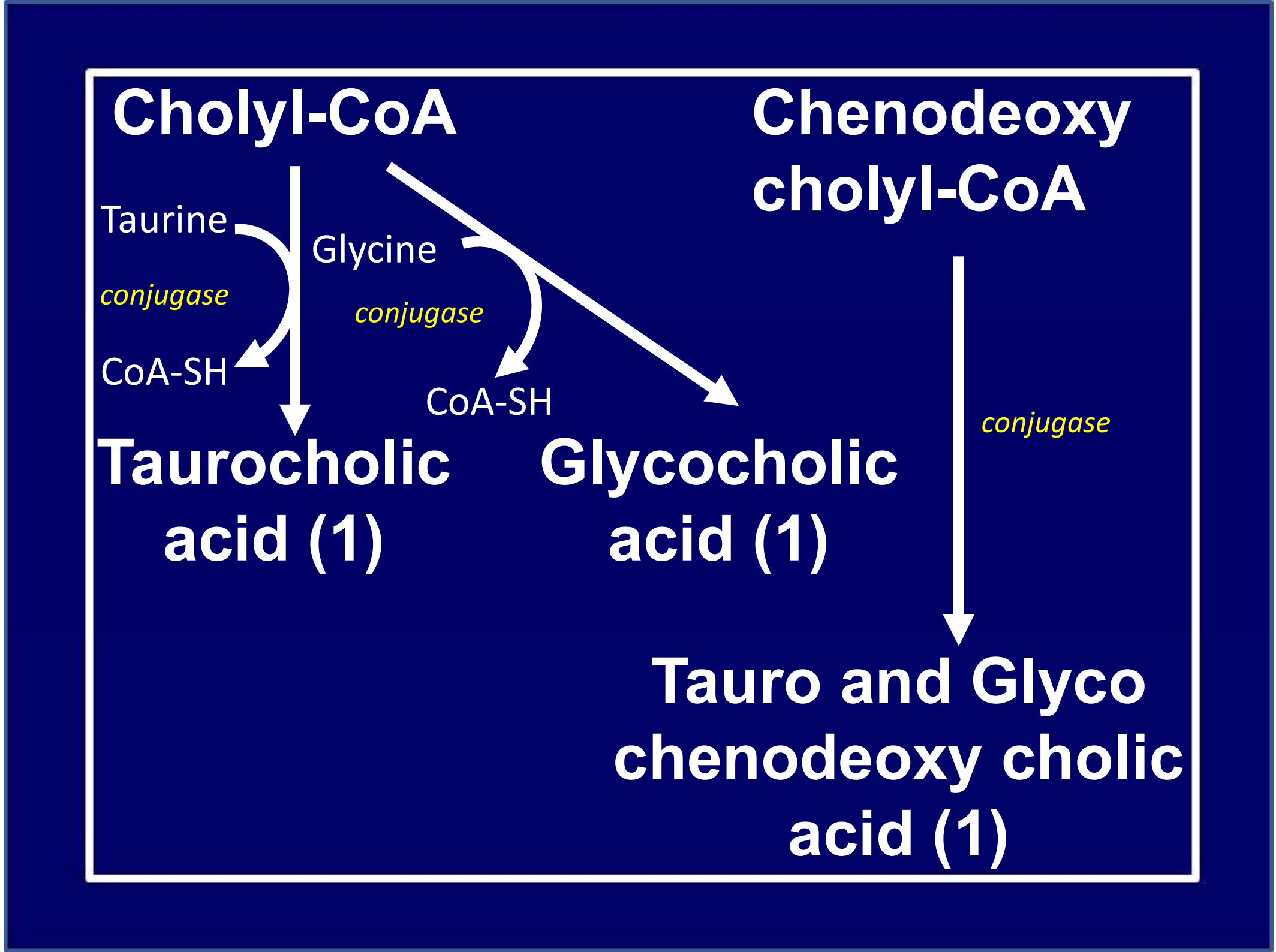
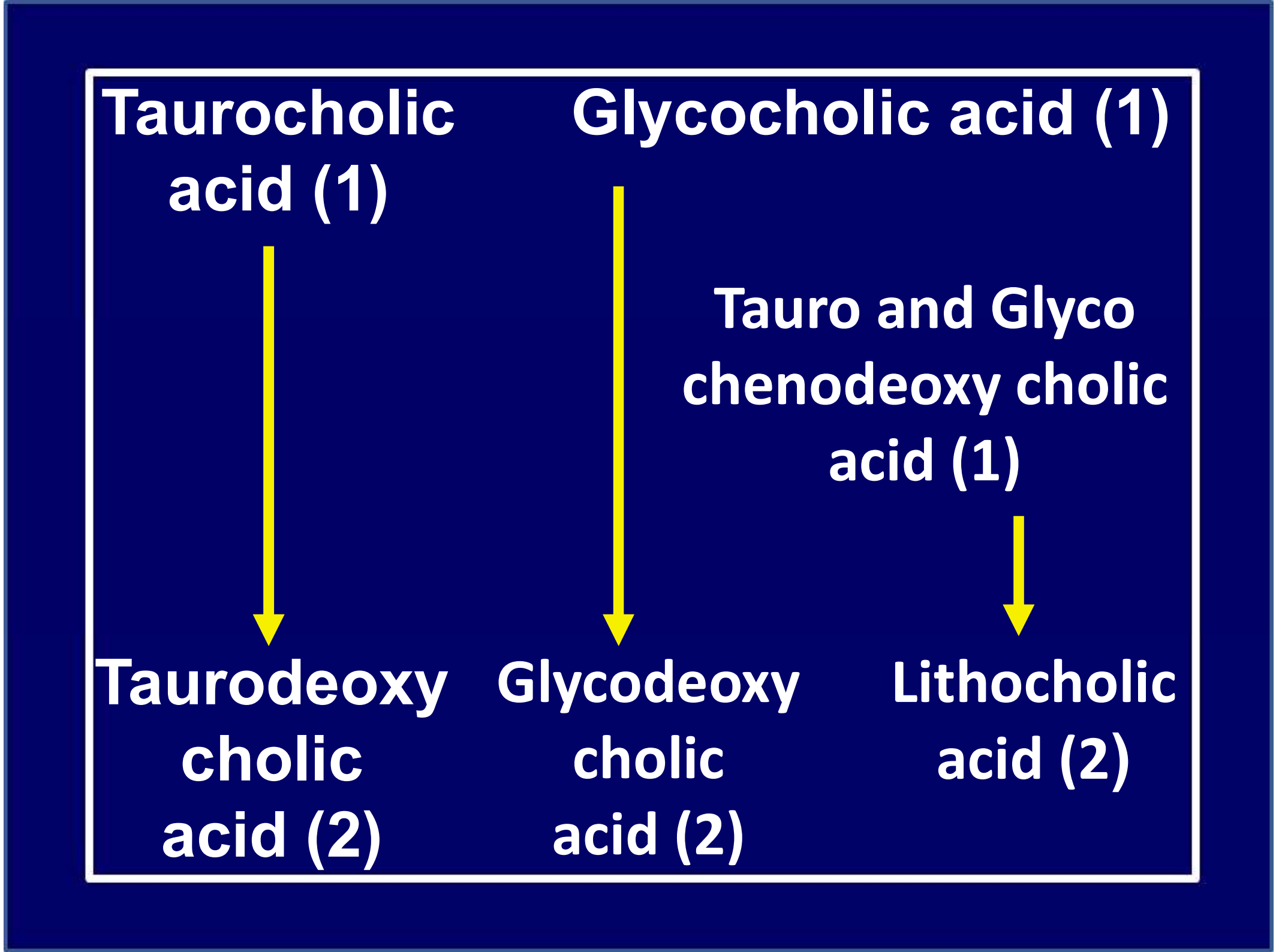
phosphoglucomutase
GAG's cartilage)
Glucose 1-phosphate
(joint
Glucose 6-phosphate
phosphoglucose isomerase
Mg
Fructose 6-phosphate
phosphofructokinase
ATP
Mg
ADP
Fructose 1,6-phosphate
aldolase
aldolase
NADP
NADPH
Pentose Phosphate
dehydrogenases
Ribulose 5-phosphate
isomerase
Ribose 5-phosphate
DNA
RNA
transketolase
Thiamine pyrophosphate
Mg
Dihydroxyacetone phosphate
Mg
triose phosphate isomerase
Glyceraldehyde 3-phosphate
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
Mevalonate
ATP
ADP
HMG-CoA reductase
NADPH
NADP
3-Hdroxy-3-Methyl Glutaryl CoA (HMG-CoA)
Farnesyl pyrophosphate
transferase
Tyrosine
P5P
SAM
Vit C
O2
CoQ10
synthetase
NADPH
Mg, Mn
NADP
Squalene
NADPH
NADH
FAD
SAM
O2
Cholesterol
H2O
HMG CoA synthase
CoA-SH
Acetyl CoA
CoA-SH
thiolase
Acetoacetyl CoA
Acetyl CoA

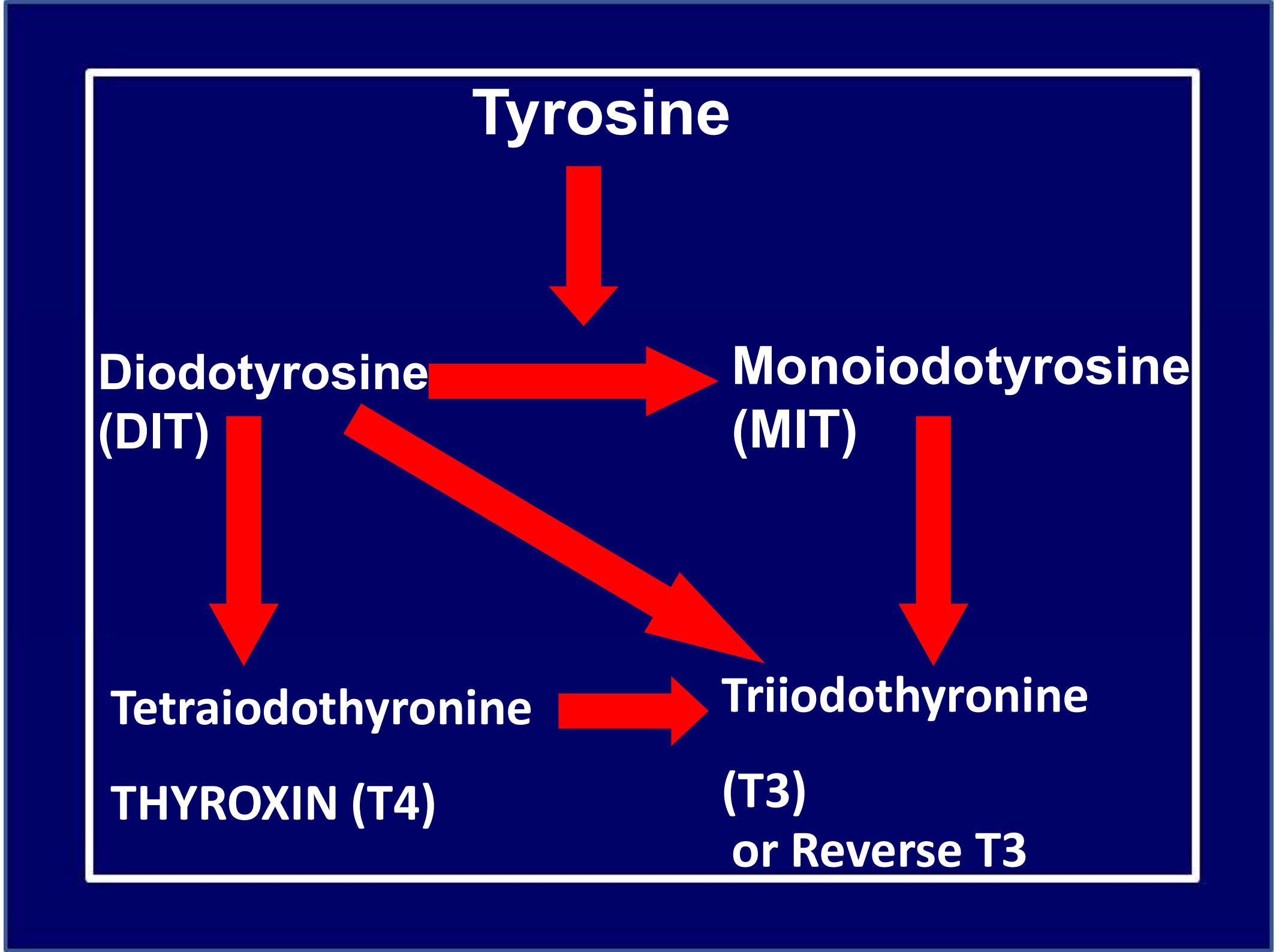

Cholesterol Metabolism
O2, NADPH, Vit C, Cu+
7α-hydroxylase
NADP
7α-Hydroxycholesterol
12α hydroxylase
O2, NADPH, CoA-SH
Cholyl-CoA
Chenodeoxy cholyl-CoA


Cholyl-CoA
Chenodeoxy cholyl-CoA
Taurine
Glycine
conjugase
conjugase
CoA-SH
CoA-SH
conjugase
Taurocholic acid (1)
Glycocholic acid (1)
Tauro and Glyco chenodeoxy cholic acid (1)

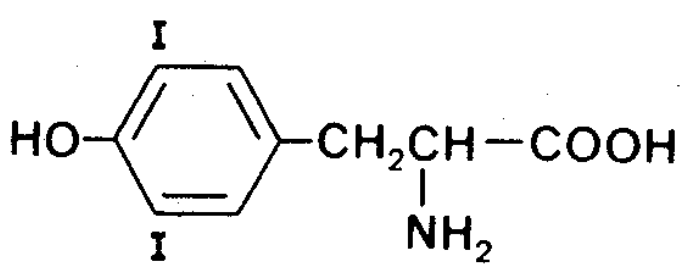

Taurocholic acid (1)
Glycocholic acid (1)
Tauro and Glyco chenodeoxy cholic acid (1)
Taurodeoxy cholic acid (2)
Glycodeoxy cholic acid (2)
Lithocholic acid (2)

Key Nutrients in Cholesterol Metabolism

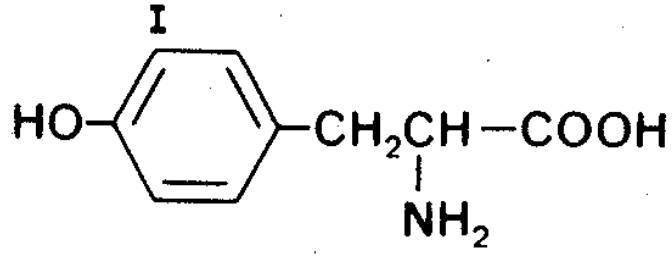
B3 (NADPH or Niasafe)	Vit C
Cu+	O2
B5 (CoA-SH)	Glycine
Taurine	
Phosphatidylcholine	Omega oils
Selenium cysteine	Iodine


Tyrosine
Diodotyrosine
(DIT)
Monoiodotyrosine
(MIT)
Tetraiodothyronine
THYROXIN (T4)
Triiodothyronine
(T3)
or Reverse T3

Tyrosine

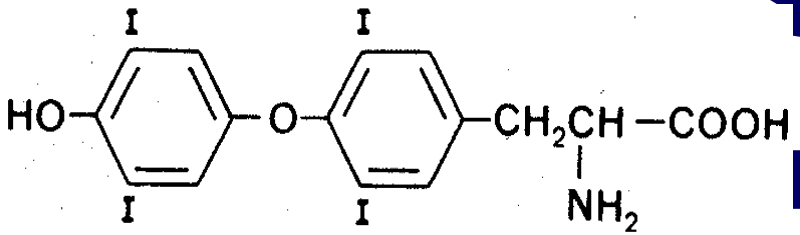


Diodotyrosine (DIT)

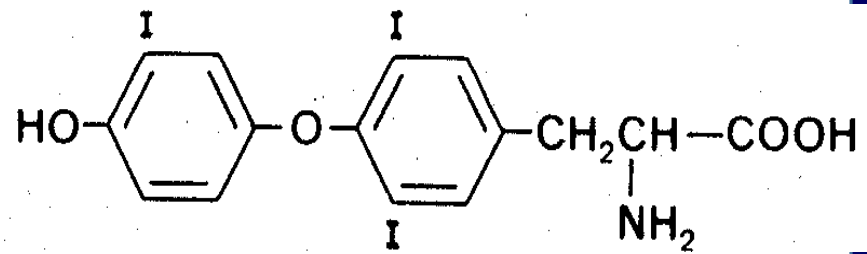


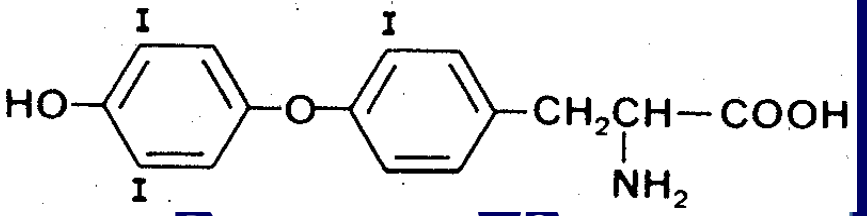
Monoiodotyrosine (MIT)

Triiodothyronine (T3)



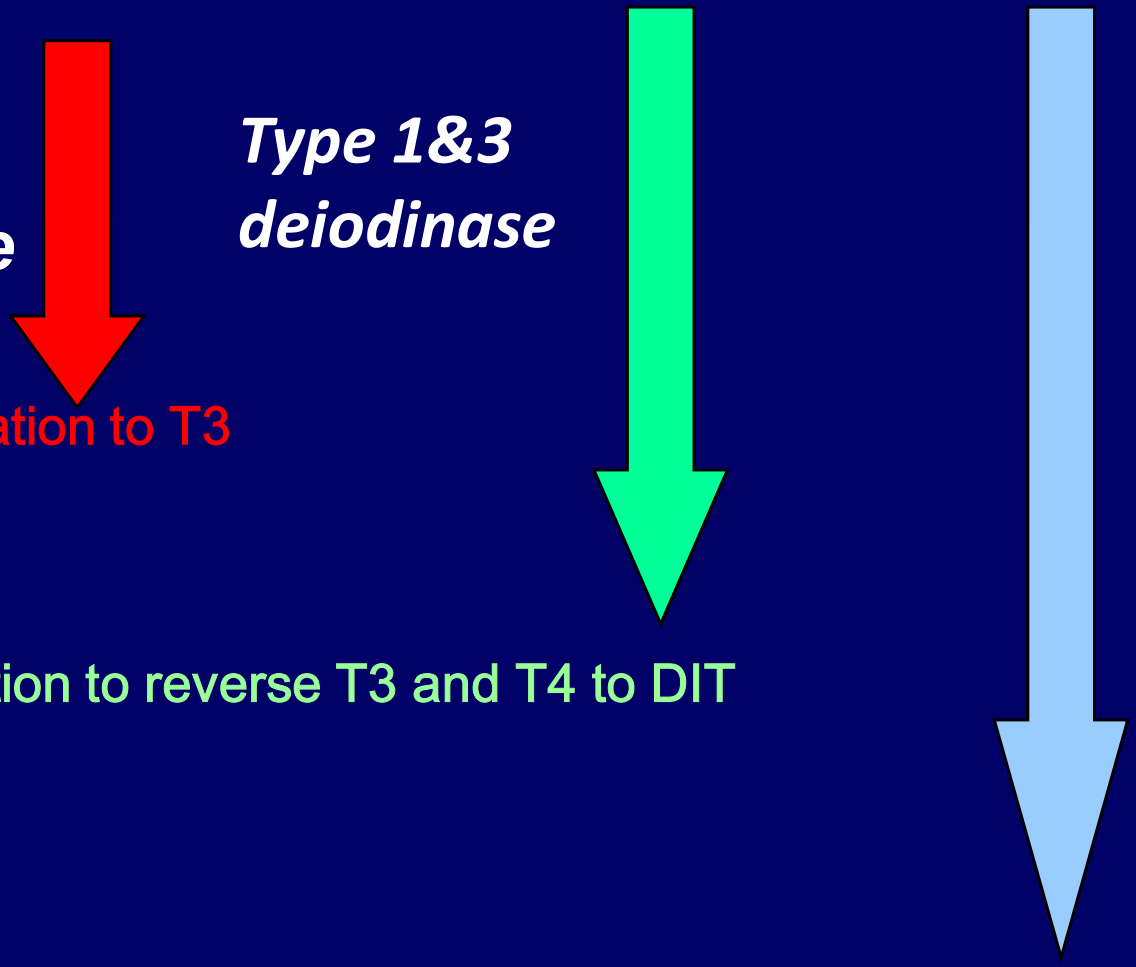
Tetraiodothyronine THYROXIN (T4)





or Reverse T3

Thyroxin (T4)


Type 1&2 deiodinase
Type 1&3 deiodinase
35% deiodination to T3
45% deiodination to reverse T3 and T4 to DIT
20% conjugation mainly with glucuronate in the liver

Metabolic Pathways - Cholesterol

Weakness to Cholesterol

→

Challenge against
B3 (NADPH) Vit C
Cu+
O2 (Adenosylcobalamine, Fe)
B5 (CoA),
Taurine
Glycine
Phosphatidylcholine (Omega oils)
Iodine
Selenium

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

Nutritional and Natural medicines

B3 (NADPH)
Vit C
Cu+
O2 (Adenosylcobalamine, Fe)
B5 (CoA),
Taurine
Glycine
Phosphatidylcholine (Omega oils)
Iodine
Selenium

Guggul

Polycosinol

Beta sitosterol (Plant sterols)

Cayenne pepper

Garlic

Niacin

Niacin is a member of the Vitamin B family, known as vitamin B3. Niacin is a natural cholesterol-lowering agent that alone has been shown to out-perform prescriptive drugs in mild and even moderate cases. It works on the cellular level and increases the health of the digestive system, improves circulation, promotes healthy skin and the sound functioning of the nervous system.

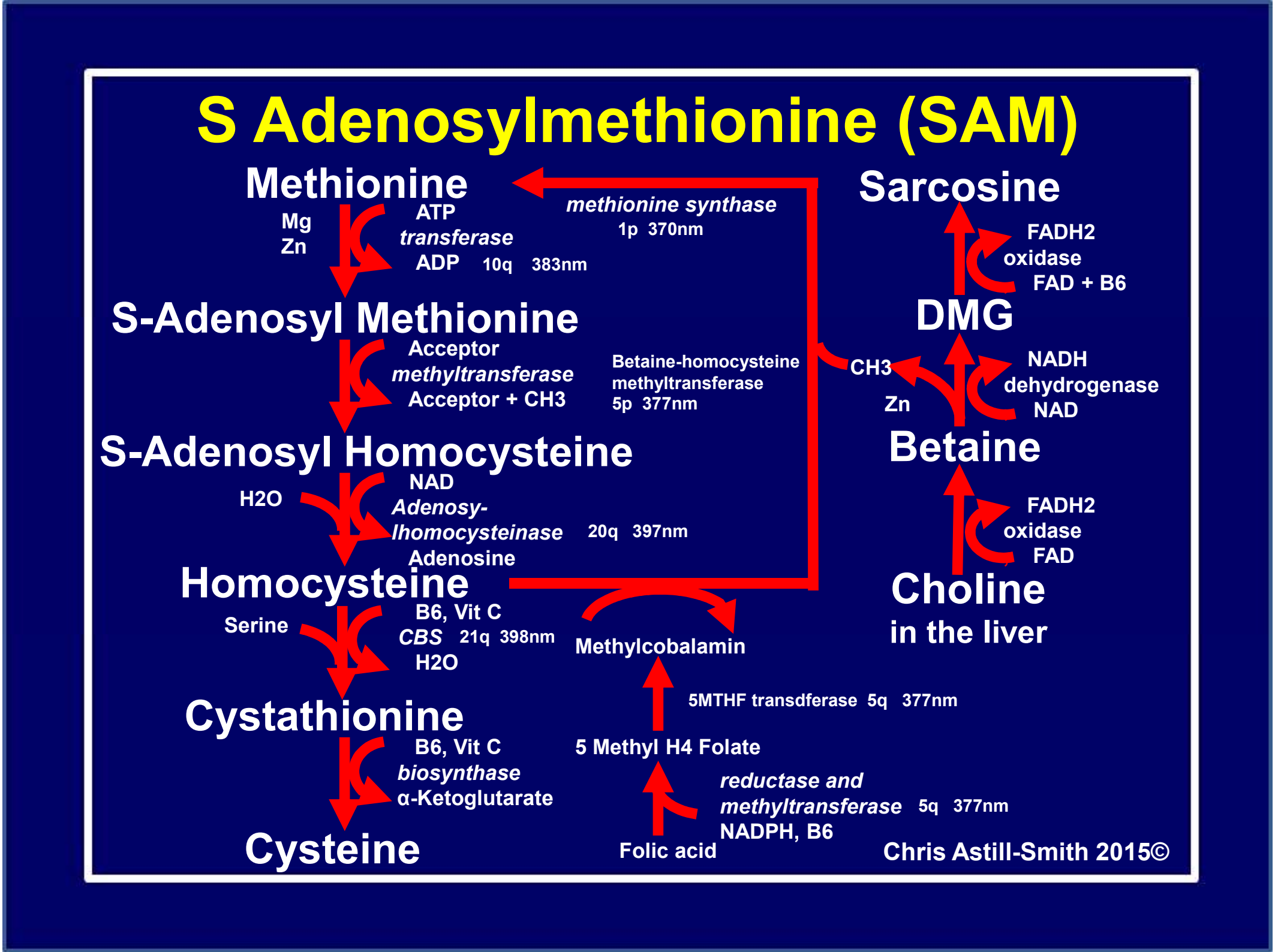
Cayenne

Cayenne has been revered for thousands of years for it's healing powers. Dr. Thao has also recognized cayenne's role as a catalyst, which amplifies the benefits of other herbs when synergistically combined. Cayenne has nutritional attributes as well, being rich in vitamins A and C along with the complete B complex. It is also a source of calcium and potassium, which again benefits the blood and the heart.

Garlic

Garlic is known to reduce cholesterol as well as defend against bacterial and fungal infections. Garlic contains 40 organic compounds and includes over 100 bioavailable chemicals. In 1994 Adesh K. Jain, M.D. of the Clinical Research Center at the Tulane University School of Medicine, New Orleans, reported how garlic can lower blood levels of "total cholesterol" and specifically LDL cholesterol.

Homocysteineamia


S Adenosylmethionine (SAM)
Methionine
Mg
Zn
ATP
transferase
ADP 10q 383nm
methionine synthase
1p 370nm
S-Adenosyl Methionine
Acceptor
methyltransferase
Acceptor + CH3
Betaine-homocysteine
methyltransferase
5p 377nm
S-Adenosyl Homocysteine
H2O
NAD
Adenosy-
lhomocysteinase
20q 397nm
Adenosine
Homocysteine
Serine
B6, Vit C
CBS 21q 398nm
H2O
Cystathionine
B6, Vit C
biosynthase
α-Ketoglutarate
Cysteine
Methylcobalamin
5MTHF transdferase 5q 377nm
5 Methyl H4 Folate
reductase and
methyltransferase 5q 377nm
NADPH, B6
Folic acid
Sarcosine
FADH2
oxidase
FAD + B6
DMG
CH3
Zn
NADH
dehydrogenase
NAD
Betaine
FADH2
oxidase
FAD
Choline
in the liver
Chris Astill-Smith 2015©

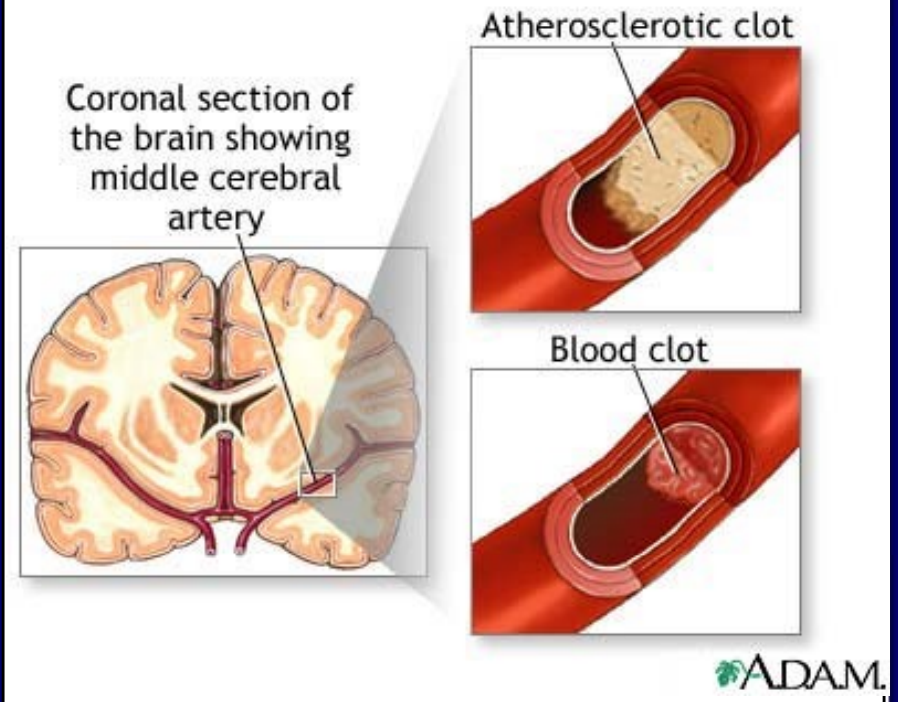
Procollagen is released into the extracellular space (cofactored by Zinc and Vitamin A).

Here it is converted to collagen by peptidase enzymes and strengthened by crosslinking of the microfibrils by lysyl oxidase, a copper dependant enzyme which is inhibited by high levels of homocysteine.

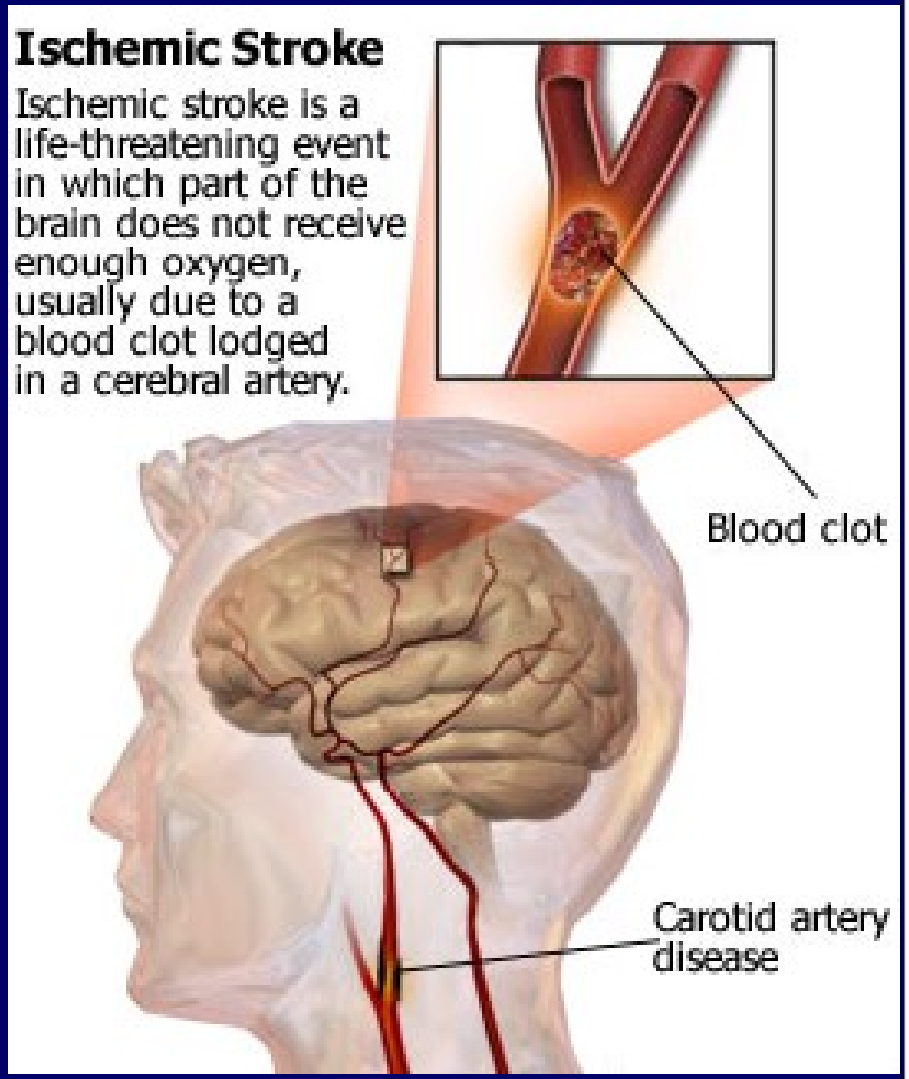
Effect of Hyperhomocysteinemia on Coagulation

Sauls DL, Wolberg AS , Hoffman M: Hyperhomocysteinemia induces alterations in fibrinogen function and fibrin clot structure in a rabbit model. *J of Thromb Haemostas*, In Press for 2003. We are now also studying the mechanism by which elevated plasma homocysteine leads to accelerated atherosclerosis and thrombosis. This is a problem of great importance from a public health point of view. We are using biochemical, biophysical, cellular and mass spectroscopy techniques to examine the effects of homocysteine on key clotting protein in humans and a rabbit model. We believe these studies will lead to a better way of testing which patients have the highest risk of cardiovascular events and might benefit from homocysteine-lowering therapy.

There are two kinds of strokes. An ***ischemic stroke*** occurs when the blood supply to the brain is interrupted, usually by a blood clot.



These clots may be caused by "hardening of the arteries" in the carotid arteries, which feed the head and brain with oxygen-rich blood.



The second kind of stroke is a ***hemorrhagic stroke***, which occurs when there is bleeding into or around the brain.

Ischemic stroke

Probably due to oxidised cholesterol.

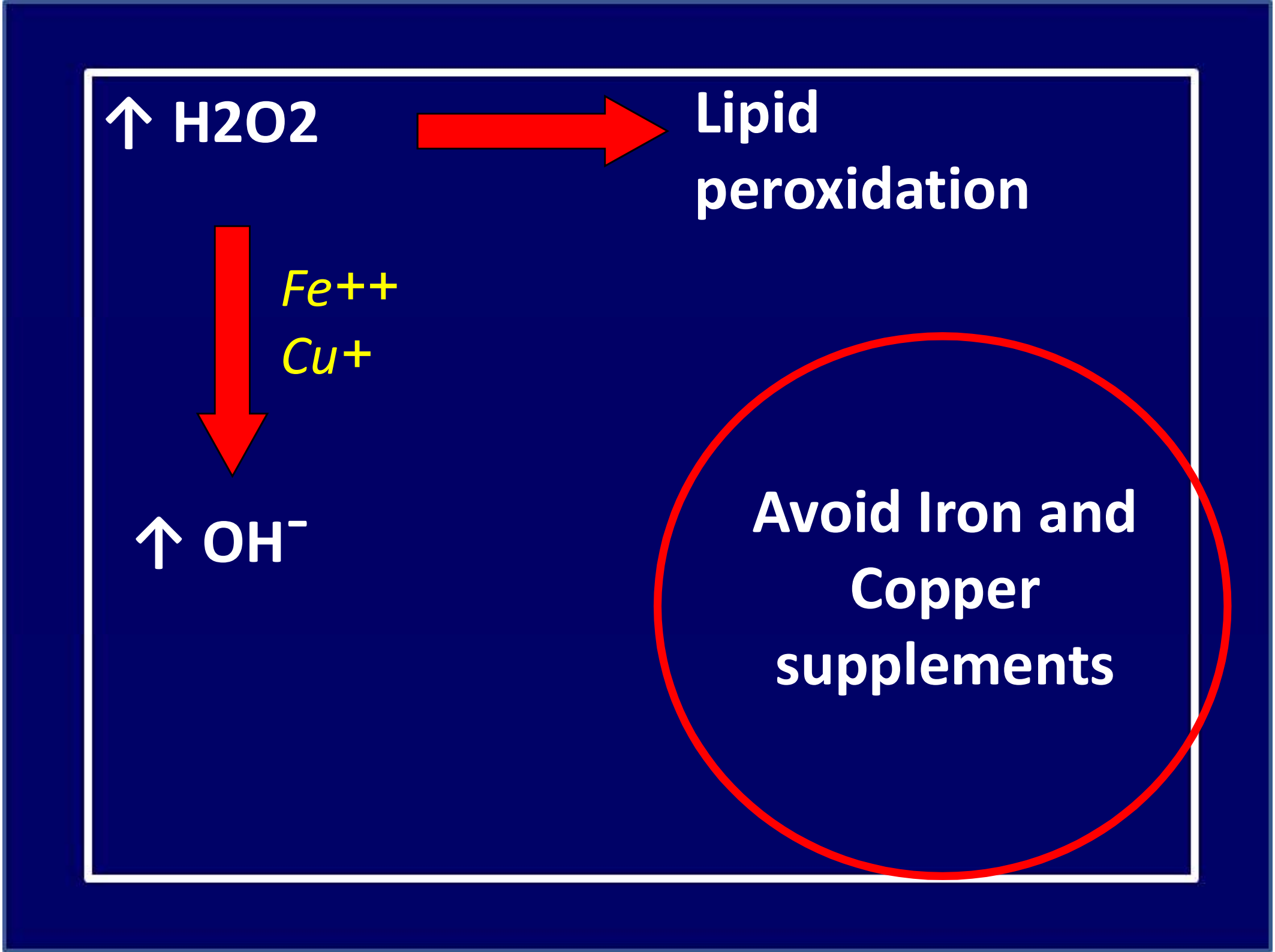
Omega 3 to prevent platelet aggregation.

Magnesium, ginger, garlic to stimulate prostacyclins.

Hemorrhagic stroke

Due to weakened artery endothelial cells due to high homocysteine.

Consider homocysteine factors P5P, Vit C, Methylcobalamin, Methyl H4 folate, Betaine, DMG.


↑ H2O2
Lipid peroxidation
Fe++
Cu+
↑ OH⁻
Avoid Iron and Copper supplements

Nutrients to consider with CVAs

Ischemic CVA

- Omega 3
- Magnesium
- Ginger
- Garlic
- Selenium

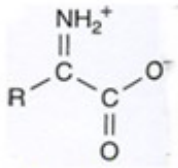
Hemorrhagic CVA

- P5P
- Vitamin C
- Methylcobalamin
- Methyl H4 folate

Post Cerebral Vascular Accident

Patients often weaken to Ammonia.
If positive challenge against
Magnesium citrate
Ornithine
Biotin
Vit C
Zinc
Phosphatidylcholine
P5P
NAD
Manganese

UREA CYCLE



α-imino acid

amino acid oxidase

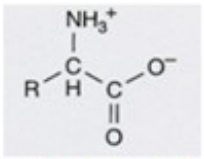
Iron

FADH2 FAD

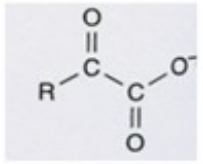
H2O2 O2

catalase

H2O



α-amino acid

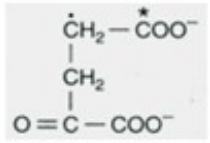


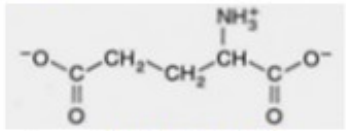
α-keto acid

transaminase

P5P

α-ketoglutarate





glutamate

NADH NAD

deaminase

P5P

glutaminase

H2O

NH4

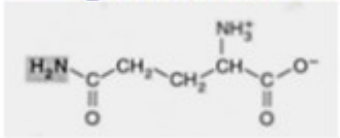
2MgATP

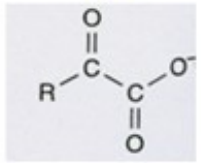
glutamine synthetase

glutamine

H2O

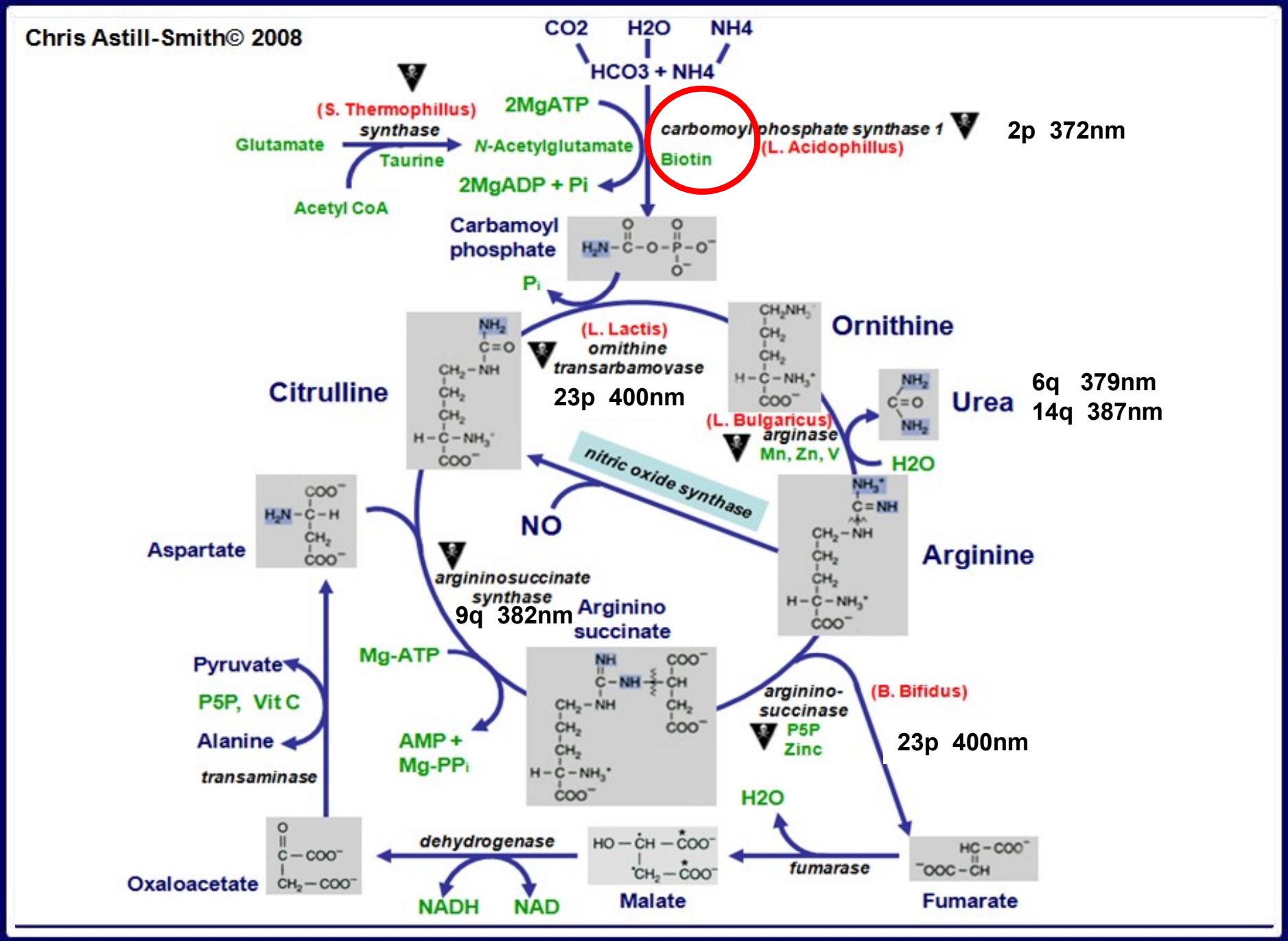
2MgADP + Pi





α-keto acid

AMMONIA NH4 ← Enteric bacteria and parasites


Chris Astill-Smith© 2008
CO2
H2O
NH4
HCO3 + NH4
(S. Thermophillus)
2MgATP
synthase
Glutamate
N-Acetylglutamate
Taurine
2MgADP + Pi
Acetyl CoA
carbomoyl phosphate synthase 1
(L. Acidophillus)
Biotin
2p 372nm
Carbamoyl phosphate
Pi
(L. Lactis)
ornithine transarbamoyase
23p 400nm
Citrulline
Ornithine
(L. Bulgaricus)
arginase
Mn, Zn, V
Urea
H2O
6q 379nm
14q 387nm
nitric oxide synthase
NO
Aspartate
argininosuccinate synthase
9q 382nm
Arginino succinate
Arginine
Mg-ATP
AMP + Mg-PPi
Pyruvate
P5P, Vit C
Alanine
transaminase
arginino-succinase
P5P
Zinc
(B. Bifidus)
23p 400nm
H2O
dehydrogenase
NADH
NAD
Oxaloacetate
Malate
fumarase
Fumarate

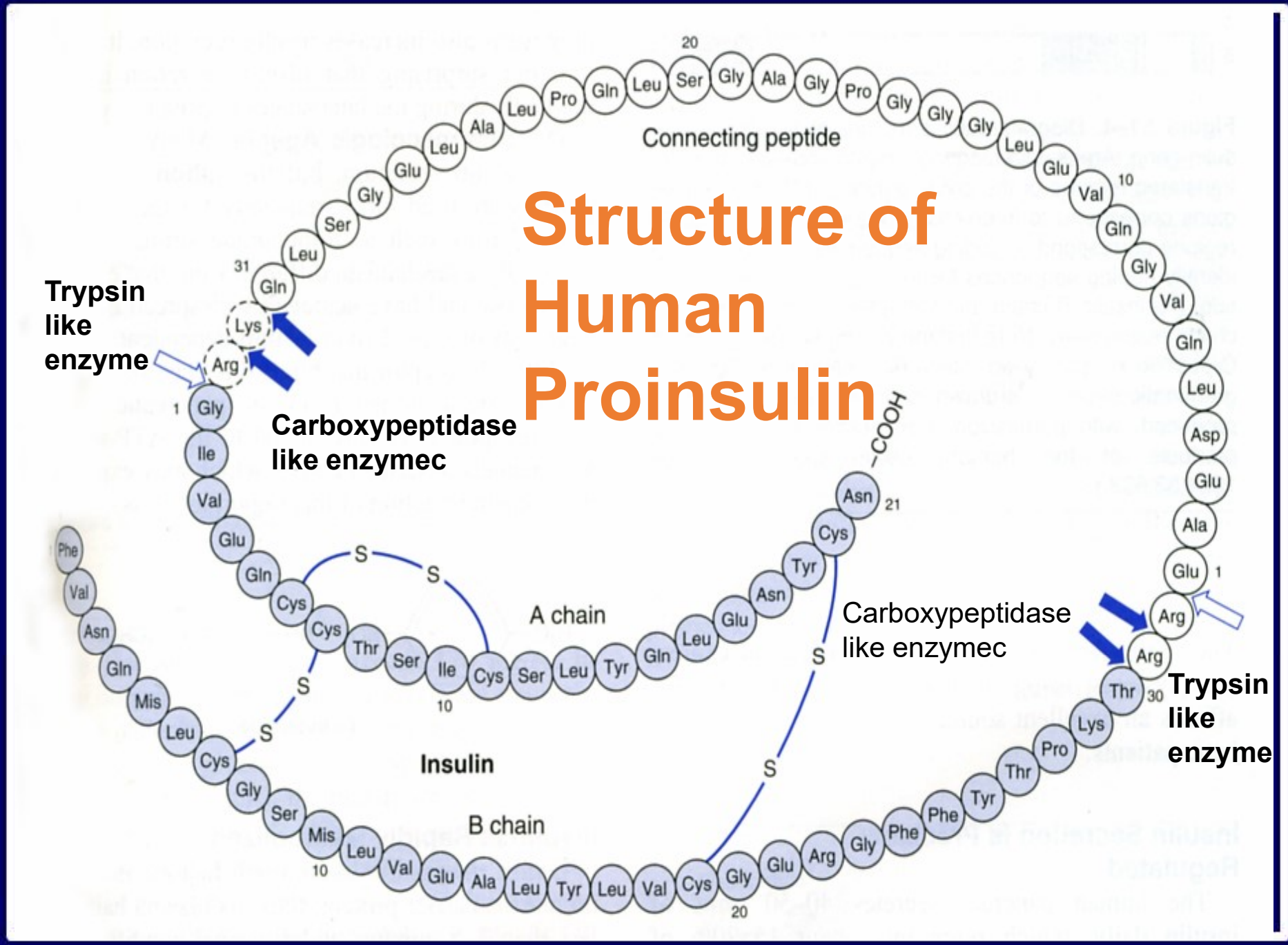
Symptoms of Hyperammonia

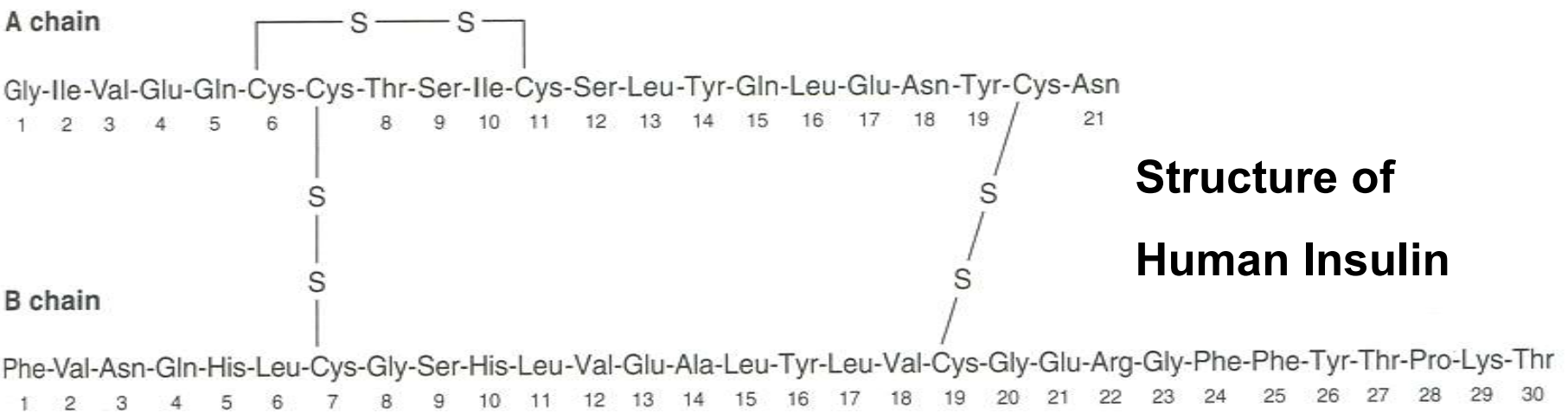
Tremor
Slurred speech
Blurred vision
Joint pains
Muscle pains
Poor sleep
Doom and gloom thoughts
Stinging eyes
Hot flushes
Night sweats
Impaired memory
Cardiac arrhythmia
Skin
Chronic fatigue
pH balance
Muscle fasiculations

DIABETES TYPE 1

10% of diabetics have insulin dependant type 1 diabetes mellitus.

Structure of Human Proinsulin





Structure of Human Insulin

Insulin is a polypeptide consisting of two chains A and B linked by two inter-chain disulfide bridges and a third connecting residues 6 and 11 of A chain.

Zinc is present in high concentration in the B cells of the pancreas and forms complexes with insulin and proinsulin.

B24 (Phe) and B26 (Tyr) each form dimers containing two atoms of zinc respectively.

Insulin regulation

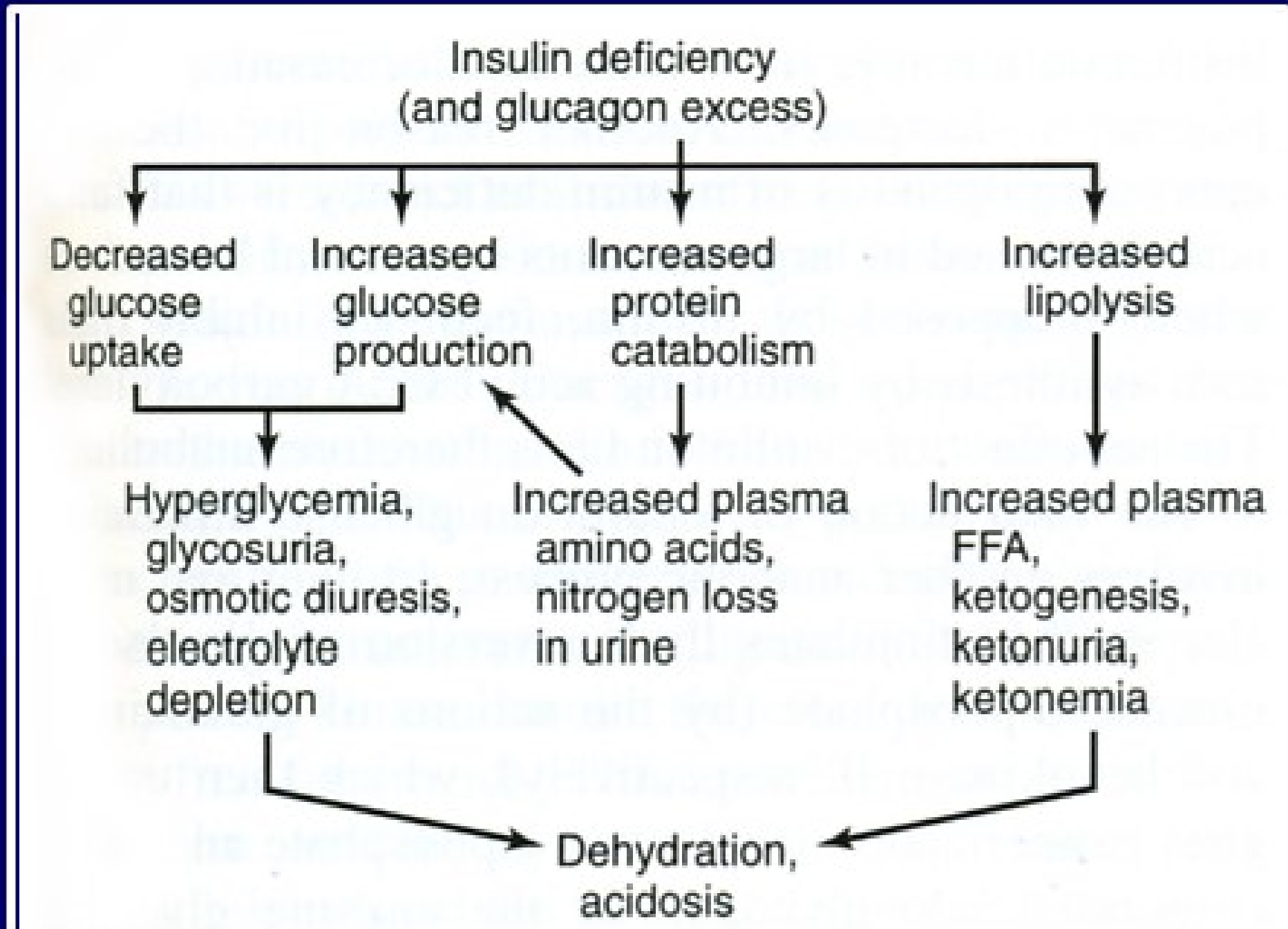
1. High plasma glucose levels indirectly results in an inhibition of ATP-sensitive K+ channels causing depolarisation of the B cell and activation of voltage sensitive Ca++ channels. The Ca++ influx results in insulin secretion.

2. Hormone factors

Adrenalin inhibits insulin release. Cortisol, Estrogens, Progestins, Growth hormone, Placental lactogen all increase insulin secretion. (Insulin secretion is higher in the later stage of pregnancy).

3. Pharmaceutical agents

Sulfonylurea compounds stimulate insulin release via the ATP-sensitive K+ channels.


Insulin deficiency
(and glucagon excess)
Decreased glucose uptake
Increased glucose production
Increased protein catabolism
Increased lipolysis
Hyperglycemia, glycosuria, osmotic diuresis, electrolyte depletion
Increased plasma amino acids, nitrogen loss in urine
Increased plasma FFA, ketogenesis, ketonuria, ketonemia
Dehydration, acidosis

Nutritional and Natural medicines

Carnitine

Zinc
Manganese
Sulfur

Omega 3 / 6

DIABETES TYPE 11

90% of diabetics have non insulin dependant type 2 diabetes mellitus.

Such patients are often obese, have elevated plasma insulin levels and have down regulated insulin receptors.

Characteristics of Type 2 Diabetes

Most common in adults, although more younger people are developing this type.

Usually slow onset with thirst, frequent urination, weight loss developing over weeks to months

Usually runs in families.
Most people who get this type are overweight or obese.

Treatment usually begins with diet and exercise, progressing to use of oral medications and later to insulin as the disease advances

Blood glucose levels may improve with weight loss, change in diet and increased exercise.

May be prevented or delayed in high-risk individuals by moderate weight loss and exercise.

People at high risk, who already had early signs of impaired glucose tolerance, significantly reduced their risk by losing only **5-7 percent of their body weight** and performing moderate physical activity for 30 minutes/day.

Type 2 diabetes is a progressive disease that can cause significant, severe complications such as heart disease, kidney disease, blindness and loss of limbs through amputation.

Treatment differs at various stages of the condition. In its early stages, many people with type 2 diabetes can control their blood glucose levels by losing weight, eating properly and exercising.

Many may subsequently need oral medication, and some people with type 2 diabetes may eventually need insulin shots to control their diabetes and avoid the disease's serious complications.

Patients with diabetes have an approximately threefold risk for all cardiovascular diseases and their relative risk of death from all causes is increased by 75%.

As yet there is no conclusive evidence that improved glucose control with oral agents leads to a decrease in the complications of type 2 diabetes. There is some evidence that improved glucose control delays the onset of complications in type 2 diabetes.

In contrast, there is strong evidence that near-normalisation of blood glucose levels with insulin can delay the development and progression of retinopathy, nephropathy, and neuropathy of patients with type 1 diabetes mellitus (IDDM).

New criteria for the diagnosis of diabetes mellitus

Fasting glucose 7.0 mmol / L.

and/or

2 hour post 75 g glucose load 11.1 mmol / L.

and/or

Symptoms of diabetes plus a single random glucose 11.1 mmol / L.

The risk factors for diabetes are age (≥45 years), family history (first degree relative with diabetes), high-risk ethnic group (aboriginal, Asian, Pacific Islander, Hispanic, African), obesity (BMI ≥ 27 kg/m2), history of gestational diabetes or macrosomic infant (≥4.5 kg), hypertension, coronary artery disease.

Sulfonylureas increase insulin secretion and potentiate insulin action on the liver and peripheral tissues.

Metformin decreases hepatic glucose production, increases glucose uptake and possibly decreases appetite.

White kidney bean extract (Phasiolamine)

Alpha glucosidase inhibitors slow the absorption of carbohydrates.

Troglitazone decreases insulin resistance.

Nutritional and Natural medicines

α-lipoic acid
Biotin

Chromium
Manganese
Zinc
Vanadium
Glucosamine

Banaba
Bitter Melon
Cinnamon
Gymnema sylvestre
Bilberry

Cloves

Banaba

Documented for it's ability to lower blood sugar and act as a glucose transport in the blood stream. Known by some as "botanical insulin".

Bitter Melon

Noted for it's regulative abilities and to improve glucose tolerance. Recommended by Dept. of Health in the Philipines as one of the best herbal medicines for diabetes management.

Cinnamon

USDA research indicates that Cinnamon reduces the amount of insulin necessary for glucose metabolism. Furthermore, Cinamon has been shown to stimulate glucose uptake and glycogen synthesis to similar level as insulin.

Gymnema Sylvestre

A Harvard study indicates the Gymnema can lower blood sugar levels in Type 1 and Type 2 diabetics. A King's College, London, study states that Gymnema acts by increasing cell permatibility, therefore reducing insulin resistance.

Bilberry

Helps to improve circulation in the little capillaries in the hands and feet. Also valuable in balancing the digestion and strengthening the immune system.

Chromium

Starting in the 1960's reports have shown that Chromium helps cells respond properly to naturally produced insulin. Current estimates show that 90% of Americans are lacking in this essential mineral nutrient.

Chromium helps to normalize blood sugar, potentiating the action of insulin (Glucose Tolerance Factor) and plays an important role in the metabolism of fats and carbohydrates.

Zinc

Zinc contributes many factors in correct insulin function. Zinc is necessary for the pancreas to produce insulin and allows insulin to work effectively, it also helps protect the insulin receptor cells. When zinc levels are low, the pancreas may not secrete enough insulin, so glucose levels remain high, and the insulin that is released cannot work as efficiently as it could. When this happens, glucose cannot enter the cells properly causing high levels in the blood.

Biotin

Biotin is a B vitamin needed to process glucose. One study showed that Type 1 diabetics given 16mgs of Biotin for one week cut their fasting glucose by 50%. A type 2 study showed similar results. There is also some indication that Biotin helps relieve pain from diabetic nerve damage.

Arteriosclerosis

Atherosclerosis is a type of arteriosclerosis. It comes from the Greek words athero (meaning gruel or paste) and sclerosis (hardness). It involves deposits of **fatty substances, cholesterol, cellular waste products, calcium and fibrin** (a clotting material in the blood) in the inner lining of an artery.

Atherosclerosis can affect the arteries of the brain, heart, kidneys, other vital organs, and the arms and legs. When atherosclerosis develops in the arteries that supply the brain (carotid arteries), a stroke may occur; when it develops in the arteries that supply the heart (coronary arteries), a heart attack may occur.

In the United States and most other Western countries including the UK, atherosclerosis is the leading cause of illness and death. In the United States alone, it caused almost 1 million deaths in 1992--twice as many as from cancer and 10 times as many as from accidents.

Despite significant medical advances, coronary artery disease (which results from **atherosclerosis** and causes myocardial infarction) and atherosclerotic stroke are responsible for more deaths than all other causes combined.

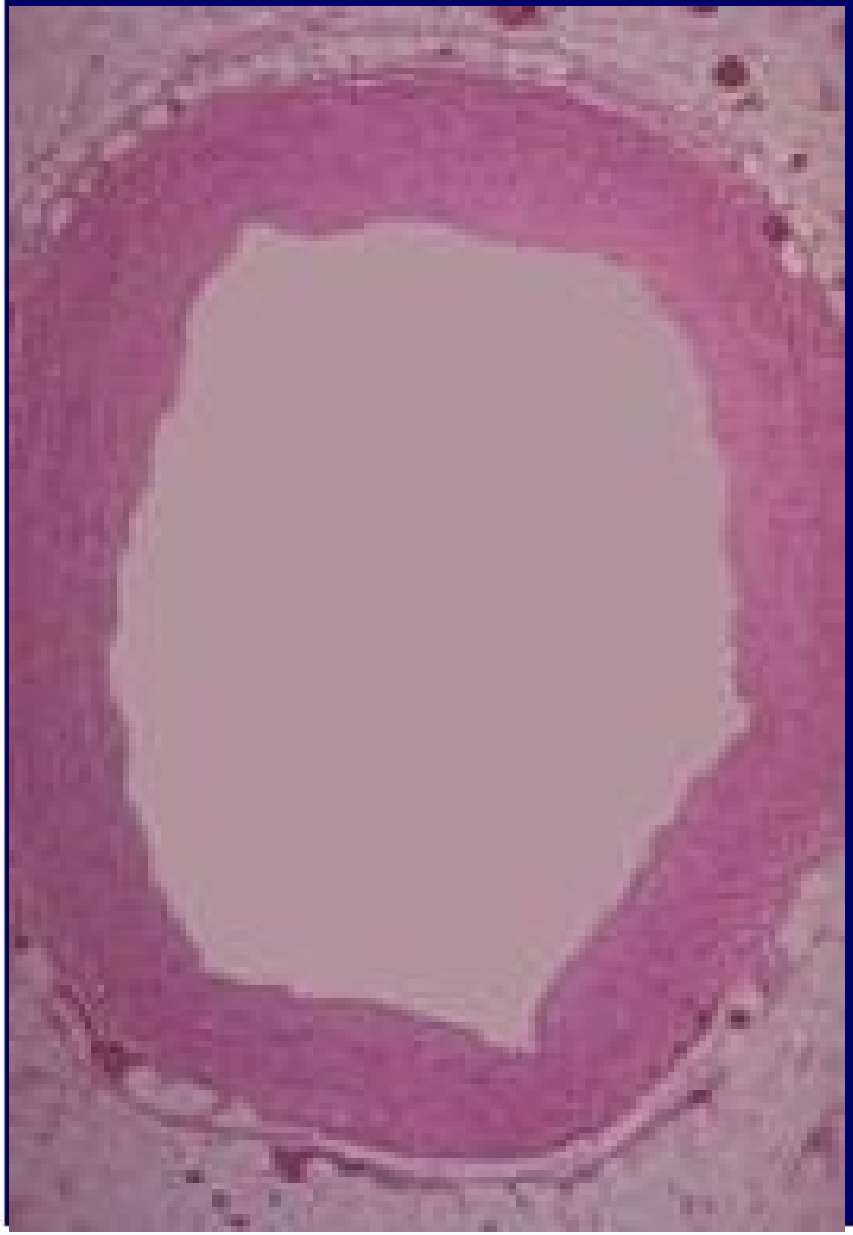
It is a disease of the arterial intima leading to the formation of fibrous (atheromatous) plaques and to stenosis/ occlusion of the lumen. It involves the proliferation of smooth muscle cells and the accumulation of lipids.

Atherosclerosis affects large and medium-sized arteries.

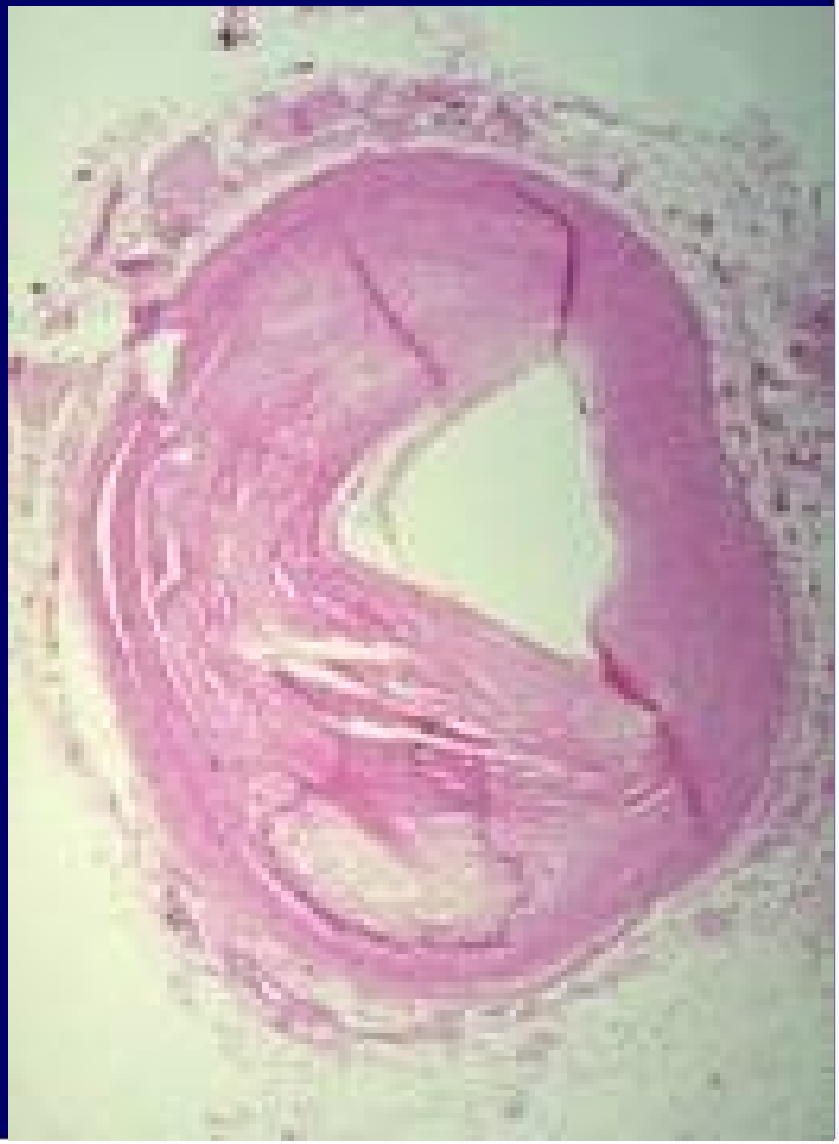
Atherosclerosis is a slow, progressive disease that may start in childhood. In some people this disease progresses rapidly in their third decade.

In others it doesn't become threatening until they're in their fifties or sixties.

Normal coronary artery



Severe calcific coronary atherosclerosis



What causes Atherosclerosis

- Blood vessels lose a certain amount of elasticity with aging.
- A build up of fatty deposits (plaque) occurs in the blood vessel lining.
- Loss of vessel elasticity is termed **arteriosclerosis,** while fatty deposit build-up is termed **atherosclerosis.**

- The process is thought to begin early in life.
- Cigarette smoking is thought to be a causative factor.
- High blood pressure is thought to be a causative factor.
- Diabetes is thought to be a causative factor.
- Obesity is thought to be a causative factor.

Atherosclerosis begins when white blood cells called **monocytes** migrate from the bloodstream into the wall of the artery and are transformed into cells that accumulate fatty materials. In time, these fat-laden monocytes accumulate, leading to a patchy thickening in the inner lining of the artery.

Each area of thickening (called an **atherosclerotic plaque** or atheroma) is filled with a soft cheese-like substance consisting of various fatty materials, principally cholesterol, smooth muscle cells, and connective tissue cells.

Atheromas may be scattered throughout the medium and large arteries, but usually they form where the arteries branch off—presumably because the constant turbulence at these areas injures the arterial wall, making it more susceptible to atheroma formation.

Arteries affected with atherosclerosis lose their elasticity, and as the atheromas grow, the arteries narrow. With time, the atheromas collect **calcium deposits**, may become brittle, and may rupture. Blood may then enter a ruptured atheroma, making it larger, so that it narrows the artery even more.

A ruptured atheroma also may spill its fatty contents and trigger the formation of a blood clot (thrombus). The clot may further narrow or even occlude the artery, or it may detach and float downstream where it causes an occlusion (embolism).

Atherosclerosis causes harm by:

- Occluding the arteries slowly over time.
- Occluding the arteries suddenly by rupture of plaques.
- Weakening of walls of the arteries.
- Arterial changes occurring with age:
- Intimal thickening.
- Reduction of elasticity of elastic tissue.
- Changes in lipoprotein composition.

Many researchers think atherosclerosis begins because the innermost layer of the artery, the endothelium, becomes damaged. Over time, fats, cholesterol, fibrin, platelets, cellular debris and calcium are deposited in the artery wall.

These substances may stimulate the cells of the artery wall to produce still other substances that result in further accumulation of cells in the innermost layer of the artery wall where the atherosclerotic lesions form. These cells accumulate and many of them divide.

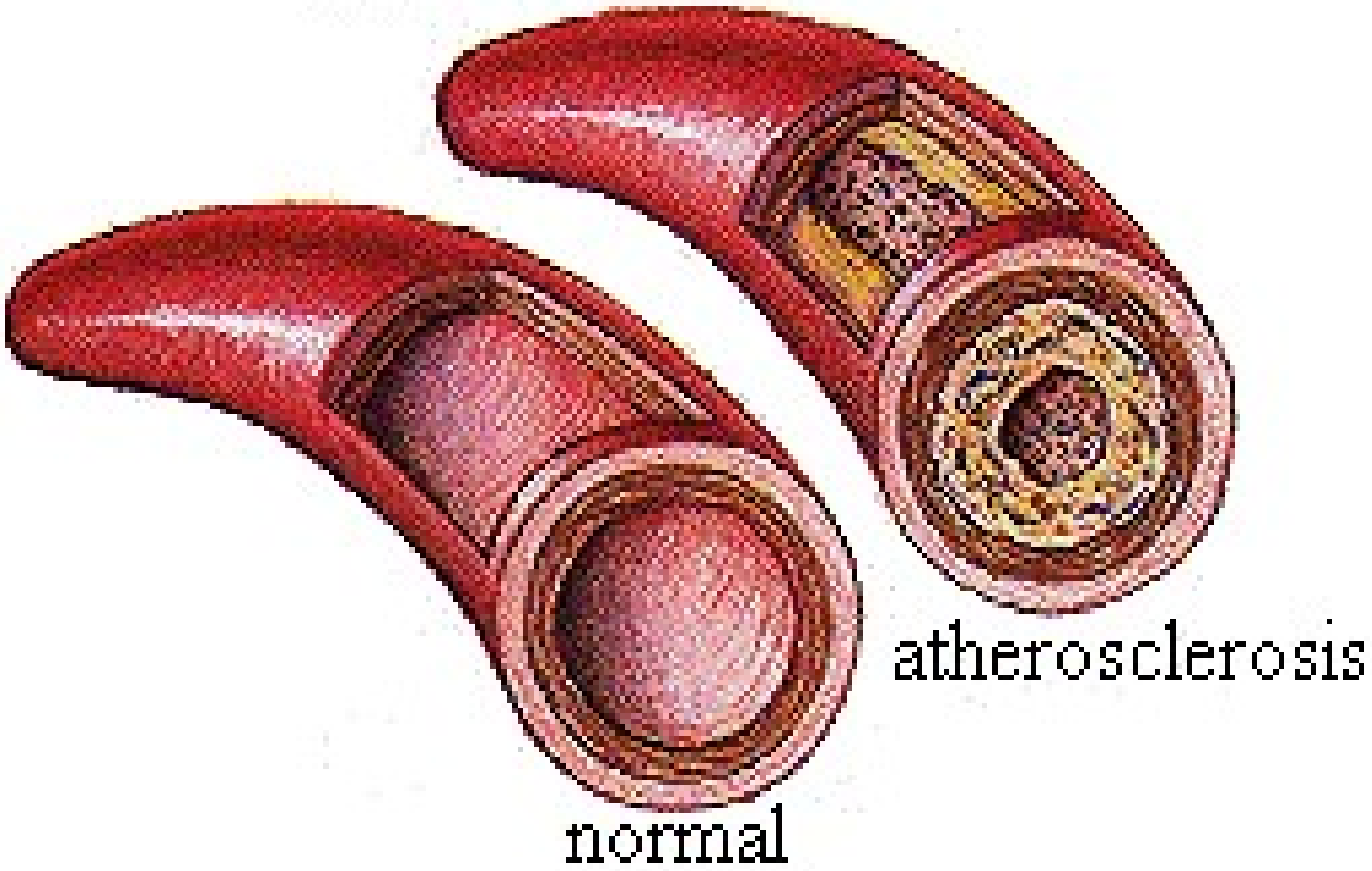
At the same time, fat builds up within these cells and around them. They also form connective tissue. The innermost layer of the artery becomes markedly thickened by these accumulating cells and surrounding material.

If the wall is thickened sufficiently, the diameter of the artery will be reduced and the amount of blood decreased, thus decreasing the oxygen supply. If the oxygen supply to the heart muscle is reduced, a heart attack can occur. If the oxygen supply to the brain is cut off, a stroke can occur.

Researchers are studying other ways in which platelets may play a role in atherosclerosis. For example, they're involved in forming a group of substances called prostaglandins, one of which may damage arteries. They also contain a substance called "platelet growth factor," which can stimulate the growth of smooth muscle cells.

These cells are normally present in the artery wall. But their abnormal growth and increase is believed to be one of the earliest events in the atherosclerosis process.

Blood Vessels



Homocysteine is an amino acid in the blood. Epidemiological studies have shown that too much homocysteine is a risk factor for atherosclerosis. People in the highest 25 percent of homocysteine blood level may be at increased risk.

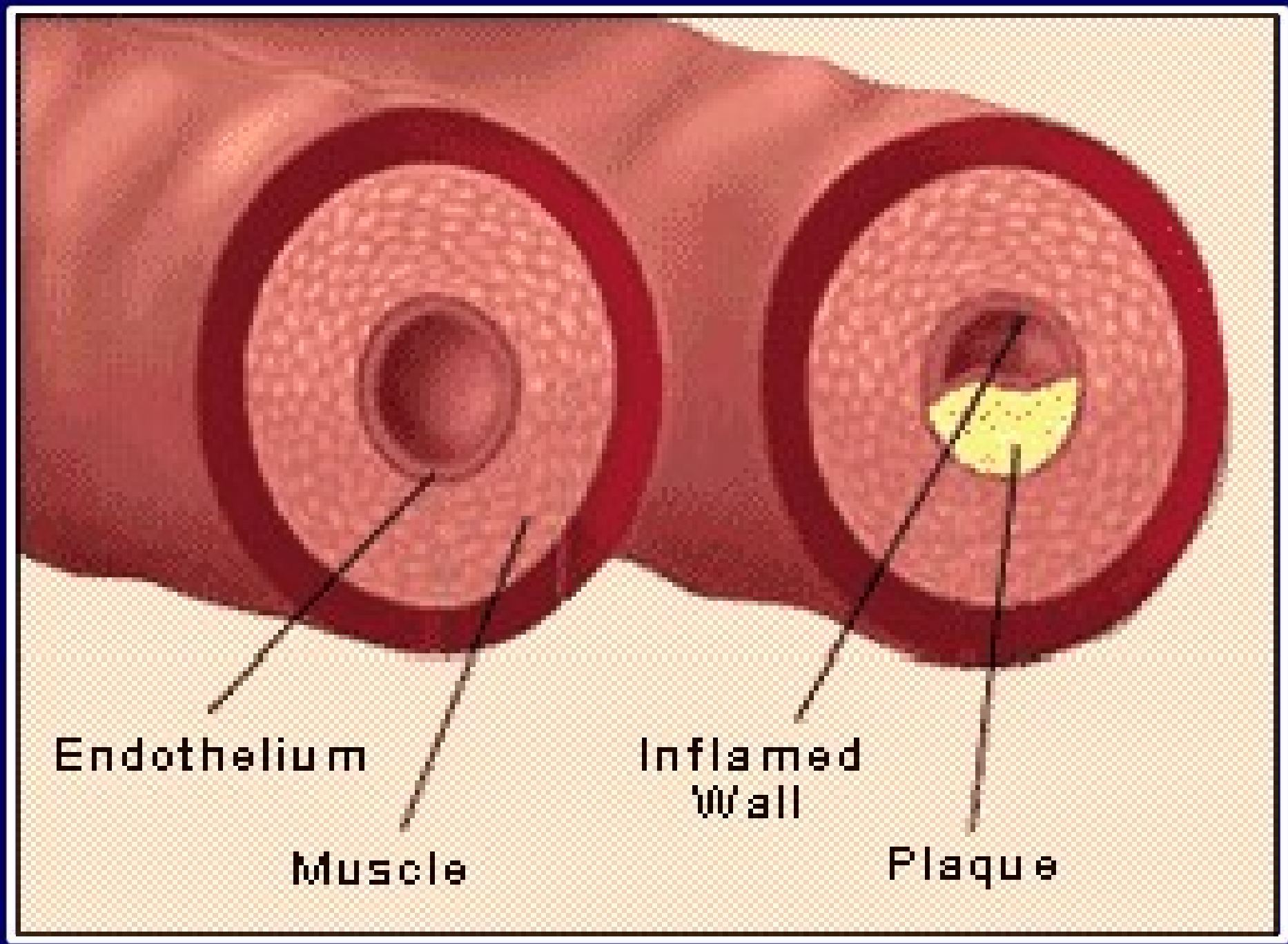
One of the more recent theories suggests that excess lipoproteins, LDL, in the blood are trapped within the artery wall. When this happens and they accumulate, they become oxidized.

That leads to "modified" lipoproteins which are rapidly taken up by smooth muscle cells. This, in turn, leads to the formation of foam cells that lead to the deposition of connective tissue cells and elements.

LDL contains specific functional groups which allow it to be recognised by most cells in the body and remain soluble in blood plasma. Therefore LDL readily passes through the endothelium, contributing to the development of plaques, atheromas.

Over time the fat deposits accumulate and grow, narrowing the lumen of the artery. Subsequent damage to the endothelial wall causes blood platelets to adhere and contributes to blood clot formation. Surrounding smooth muscle tissue also proliferates to form larger plaques.

Hardening of the arterial wall is due to various depositions within the plaque including lipids, cholesterol crystals, and calcium salts. These depositions make the arteries bone-like rigid tubes. The narrowing and hardening of the arteries has dramatic effects on blood pressure, resistance and blood flow.


Endothelium
Muscle
Inflamed
Wall
Plaque

Resistance increases when radius decreases, as friction of blood flow against vessel wall increases. Therefore the circulation of blood flow is reduced, and cells may be deprived of oxygen or experience toxic accumulation of metabolic wastes.

Development of a plaque also **deforms the endothelial wall**, increasing turbulent flow and increasing resistance. The hardening of the arterial walls, increases resistance to flow, as vessel walls lose their distensibility.

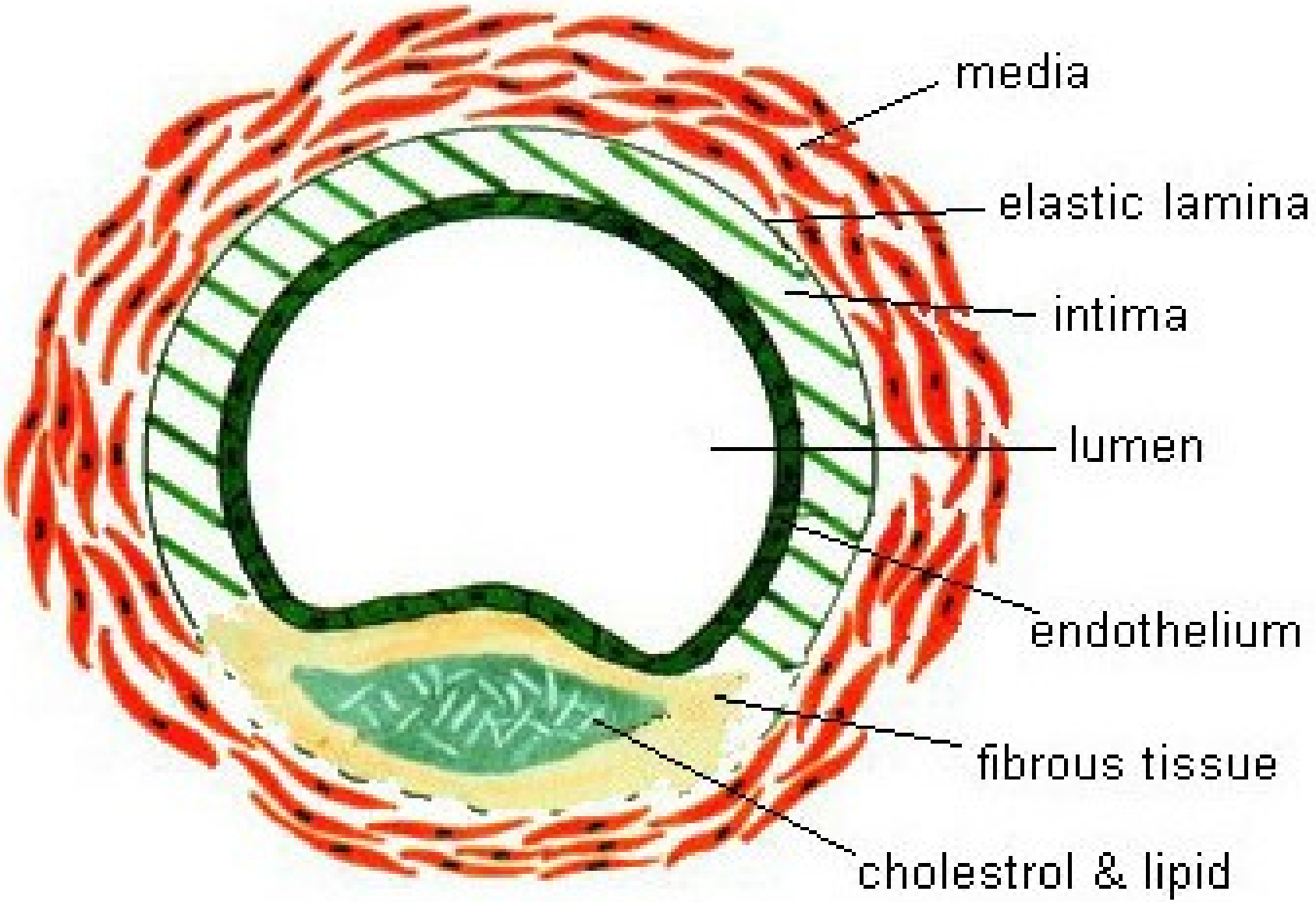
As resistance to flow increases, there is a marked increase in **blood pressure.** Therefore the heart has to work harder to pump blood, causing it to enlarge. This may lead to various heart defects and failures.

Atheromas

Atherosclerosis is a stereo typed response to injury featuring the accumulation of cholesterol-rich fat in the intima of the large and medium sized arteries of the body. Typically these are phagocytes. These masses form plaques, or atheromas.

Lipid streaks are flat or slightly elevated pale yellow areas, of variable size and shape, found throughout the arterial system of patients of all ages. The lipid is deposited in the intima and can be stained with Sudan IV and other fat stains.

Fibrous plaques are raised firm pale areas in the intima of arteries which on cross section reveal central lipid rich debris with surrounding fibrous tissue.


media
elastic lamina
intima
lumen
endothelium
fibrous tissue
cholestrol & lipid

Calcification

Hemorrhage

Ulceration

A local defect or excavation, of the surface of an organ or tissue, which is produced by the sloughing of inflammatory necrotic tissue.

Thrombosis. The formation, development or presence of a thrombus. An aggregation of blood factors, primarily platelets and fibrin with entrapment of cellular elements, frequently causing vascular obstruction at the point of its formation. Some authorities thus differentiate thrombus formation from simple coagulation or clot formation.

Symptoms

Usually, atherosclerosis doesn't produce symptoms until it severely narrows the artery, or until it causes a sudden obstruction. Symptoms depend on where the atherosclerosis develops; thus, they may reflect problems in the heart, the brain, the legs, or almost anywhere in the body.

As atherosclerosis **severely narrows an artery,** the areas of the body it serves may not receive enough blood, which carries oxygen to the tissues. The first symptom of a narrowing artery may be pain or cramps at times when the blood flow can't keep up with the body's demand for oxygen.

For instance, during exercise, a person may feel chest pain (angina) because of a lack of oxygen to the heart, or while walking, a person may feel leg cramps (intermittent claudication) because of a lack of oxygen to the legs.

Typically, these symptoms develop gradually as the atheroma slowly narrows the artery. However, when an obstruction occurs suddenly, for example, when a blood clot lodges in an artery, the symptoms come on suddenly.

Risk Factors

The risk of developing atherosclerosis increases with high blood pressure, high blood cholesterol levels, cigarette smoking, diabetes, obesity, a lack of exercise, and advancing age.

Having a close relative who developed atherosclerosis at an early age also puts a person at risk. Men have a higher risk than women, though after menopause, the risk increases in women and eventually equals that in men.

People with the inherited disease homocystinuria develop extensive atheroma formation, particularly at a young age.

The disease affects many arteries but doesn't primarily affect the coronary arteries, which supply the heart. In contrast, in the inherited disease familial hypercholesterolemia, extremely high levels of blood cholesterol cause atheromas to form in the coronary arteries much more than in other arteries.

Prevention and Treatment

Depending on a particular person's risk factors, prevention may consist of lowering cholesterol levels, lowering blood pressure, quitting smoking, losing weight, and beginning an exercise program.

In people who already have a high risk of heart disease, smoking is particularly dangerous. Cigarette smoking decreases the level of good cholesterol (high-density lipoprotein cholesterol or HDL cholesterol) and increases the level of bad cholesterol (low-density lipoprotein cholesterol or LDL cholesterol).

Smoking also raises the level of carbon monoxide in the blood, which may increase the risk of injury to the lining of the arterial wall, and smoking constricts arteries already narrowed by atherosclerosis, further decreasing the amount of blood reaching the tissues.

Plus, smoking increases the blood's tendency to clot, so it increases the risk of peripheral arterial disease, coronary artery disease, stroke, and obstruction of an arterial graft after surgery.
A smoker's risk of coronary artery disease is directly related to the number of cigarettes smoked daily.

Cholesterol

Cholesterol is an important dietary fat. It is found mainly in meat, egg yolks, dairy products, offal and shellfish.

Cholesterol is a member of the *sterol* group of fats; it is not a triglyceride or a fatty acid.

Cholesterol is made in the body, mostly in the liver, and has many important functions in the body. Levels of cholesterol in the blood are determined by a number of factors including genetics, saturated fat intake, body weight and activity level.

The effect of dietary cholesterol is thought to be minimal in many people. However, if you have raised blood cholesterol levels, reducing the amount of high-cholesterol foods that you eat may help.

Raised blood cholesterol is a significant risk factor for the development of heart disease and circulation problems. In particular, it is the LDL fraction of cholesterol that is harmful. All fats are carried in the blood on *lipoproteins*. These are classified according to how tightly packed their contents are. The proportions of triglyceride vs cholesterol and (other materials) in the lipoprotein particle are important. LDL-cholesterolHDL-cholesterolLow density lipoprotein (LDL) High density lipoprotein (HDL)50 % cholesterol20 % cholesterolLDL takes cholesterol from liver to tissuesHDL mops up spare cholesterol and takes it back to the liverLow LDL reduces health riskHigh HDL reduces health risk

There are several potential mechanisms by which an intake of saturated fat could lead to increased blood cholesterol: unsaturated fatty acids tend to favour the formation of HDLs. Presumably this is linked to the reaction involving the enzyme lecithin cholesterol acyl transferase (LCAT) in which unsaturated fatty acids are transferred from plasma lecithin (a phospholipid) onto the cholesterol molecule to form an ester. HDL then transports this cholesterol to the liver where it is broken down. Hence unsaturated fatty acids would tend to reduce plasma cholesterol. Conversely saturated fatty acid or low levels of unsaturated fatty acid would elevate cholesterol levels in plasma because HDL levels relative to LDL have decreased. low-density lipoprotein (LDL) particles tend to contain cholesterol esters rich in saturated fatty acids, whereas high-density lipoprotein (HDL) contains unsaturated fatty acids.

Thus a high proportion of saturated fatty acids in the diet could (in theory at least) increase LDLs saturated fatty acids and cholesterol tend to be present together in foods such as animal fat. Hence consumption of food rich in saturated fatty acids would raise cholesterol levels concurrently. (However, a recent study by Kromhaut (1999) indicates that this does not apply in the case of stearate which does not raise LDL) saturated fatty acids may have some regulatory effects on cholesterol synthesis which tends to raise plasma cholesterol levels (via LDL), an effect which is not produced by unsaturated fatty acid.

There is clear epidemiological evidence that diets rich in cholesterol and saturated fats lead to an increased risk of coronary heart disease as a consequence of high plasma cholesterol levels promoting the development of atherosclerotic plaques in blood vessel walls.

Polyunsaturated fatty acids are regarded as being protective because they reduce the risk of atheroma development.

Saturated fats taken in with the diet are incorporated into cholesterol esters (and triglycerides) which form part of the LDL complexes in the blood, whereas polyunsaturated fatty acids are taken up mainly into cholesterol esters in HDL.

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The chemical composition of these two particles is quite different: LDL contains 80% fat, of which 50% is made up of cholesterol, and 20% protein. HDL contains 55% fat, of which 25% is cholesterol, and 45% protein. Increased levels of LDL have been linked to an increased risk of coronary heart disease and led to its description as "bad cholesterol". Conversely increased HDL levels reduce the risk of coronary heart disease and HDL is referred to as "good cholesterol". Since cholesterol is an essential component of cells and has numerous other roles throughout the body, for example providing a raw material for the synthesis of steroid hormones, we must be careful in the way we attach such labels and keep in mind the need for an appropriate balance.

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A large proportion of LDL cholesterol is in the form of esters containing saturated fatty acids, whereas HDL contains unsaturated fatty acids. HDL lipoproteins tend to transport excess cholesterol from peripheral tissues back to the liver where it is broken down. HDL is assembled from apolipoprotein-AI, phospholipid, and free cholesterol discs secreted by the liver and intestines. The free cholesterol is esterified in the mature HDL and can then be transferred to LDL or taken up by the liver or steroid-metabolising tissues.

In contrast, LDL particles are involved in the uptake of cholesterol into tissue cells via a receptor-mediated process. A decrease in the number of LDL receptors on tissue cells has been linked to high circulating levels of plasma cholesterol. Decreased receptors means decreased cholesterol uptake and the raised LDL levels that ensue increase the risk of atheroma. About 20 years ago it was discovered that mutations in the gene that encodes the LDL receptor protein causes familial hypercholesterolaemia. In this condition there are high levels of LDL but tissue cells are unable to take up cholesterol from the blood. People affected by this mutation are at a much higher risk of coronary heart disease and stroke than other people (Brown 1984).

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Several factors can influence the levels of circulating HDL (Scott, 1999). For example, there is a difference in levels between women and men: women have higher levels of HDL than men up until the menopause, and this offers them some protection against heart disease. Levels of HDL can also be increased by exercise, weight loss, moderate alcohol intake, and chemicals such as fibric acid derivatives, nicotinic acid, and tamoxifen.

It is currently thought that monounsaturated fatty acids may be beneficial to our health by reducing levels of LDL-cholesterol in the blood. *Myristic acid* is the main saturated fatty acid associated with high blood cholesterol levels.

The n-3 fatty acids can reduce the levels of triglycerides in the blood. The n-6 fatty acids can reduce the amount of LDL-cholesterol circulating in the blood; this is sometimes accompanied by a small rise in HDL-cholesterol, but the overall effect is an increase in the HDL/LDL ratio, which is associated with a reduced risk of heart disease.
However, polyunsaturated fats are vulnerable to chemical alterations within the body, which result in highly damaging ‘lipid peroxides’.

Polyunsaturated fats in the body are vulnerable to attack from ‘free radicals’ and can undergo chemical reactions known as ‘lipid peroxidation’. This results in fats that are more likely to contribute to hardening of the arteries, and also leads to the generation of more ‘free radicals’, which are damaging to health. A healthy supply of antioxidant vitamins can help to reduce the risk of damage from free radicals.

Processing food can result in a subtle alteration of the structure of unsaturated fatty acids, making them *trans* fatty acids. These have similar properties to saturated fatty acids and tend to raise LDL-cholesterol and lower HDL-cholesterol.

Nutritional and Natural medicines

Vitamin E
Phosphatidylcholine

Vitamin K2

B3 (NADPH)
Vit C
Cu+
O2 (Adenosylcobalamin, Fe)
B5 (CoA),
Taurine
Glycine
Omega oils
Iodine
Selenium

Guggul

Polycosinol

Plant sterols

Cayenne pepper
Garlic

Cloves

Challenge with Oxidised Cholesterol

Elevated C-Reactive Protein is associated with an increased 10-year risk of Coronary Heart Disease - C-reactive protein (CRP) is a marker for inflammation that has been reported to be a risk factor for myocardial infarction in many studies. High CRP is associated with increased coronary heart disease. In a study conducted by Mary Cushman M.D., MSc et al from the Departments of Medicine and Pathology at the University of Vermont, baseline CRP and 10-year incidence of first MI or CHD death were compared. This observational cohort study, published in the July 5, 2005 issue of *Circulation,* determined that in older men and women, elevated CRP measurement was associated with an increased 10-year risk of CHD.

Omega 3 Fish Oil
Great For Your Blood And Blood Vessels

Scientists were baffled by the low incidence of heart disease amongst Greenland's Inuit community as they had such a high fat content in their diet. They finally discovered the reason, *Omega 3* fatty acids in the fish the Inuit's were eating! Studies have confirmed that these fatty acids play a vital role in protecting the heart.

Omega 3 oils are vital to a range of bodily functions such as regulating blood pressure and blood clotting, and boosting the immune system.

Omega 3's Help Regulate Blood Pressure And Boost The Immune System!

Fish oils reduce the risk of heart disease in several important ways. By lessening the 'stickiness' of the blood, Omega3 oils reduce the risk of blood clots that lead to heart attacks. They can reduce the blood fats called triglycerides that are carried with cholesterol and lower blood pressure. Recent research also shows that Omega3 oils strengthen the heart's electrical system preventing heart rhythm defects.

Omega3 oils help prevent inflammation within artery walls which helps prevent a build-up of plaque. This makes therapeutic doses of fish oil supplements an important way to help prevent re-blockage of small arteries after angioplasty and also makes fish oil helpful for Reynaud's disease.


Next module 7
Digestive Enzymes, Probiotics,
Common GI disorders