Module 1

Principles of Essential Nutrition

Dr George Goodheart founder of Applied Kinesiology
1918 - 2008

The Major Causes of Disease

TABLE 1–2 The Major Causes of Disease *

1. **Physical agents:** Mechanical trauma, extremes of temperature, sudden changes in atmospheric pressure, radiation, electric shock.
2. **Chemical agents, including drugs:** Certain toxic compounds, therapeutic drugs, etc.
3. **Biologic agents:** Viruses, bacteria, fungi, higher forms of parasites.
4. **Oxygen lack:** Loss of blood supply, depletion of the oxygen-carrying capacity of the blood, poisoning of the oxidative enzymes.
5. **Genetic disorders:** Congenital, molecular.
6. **Immunologic reactions:** Anaphylaxis, autoimmune disease.
7. **Nutritional imbalances:** Deficiencies, excesses.
8. **Endocrine imbalances:** Hormonal deficiencies, excesses.

*Harpers Biochemistry 29th edition p3

LIFE and HEALTH

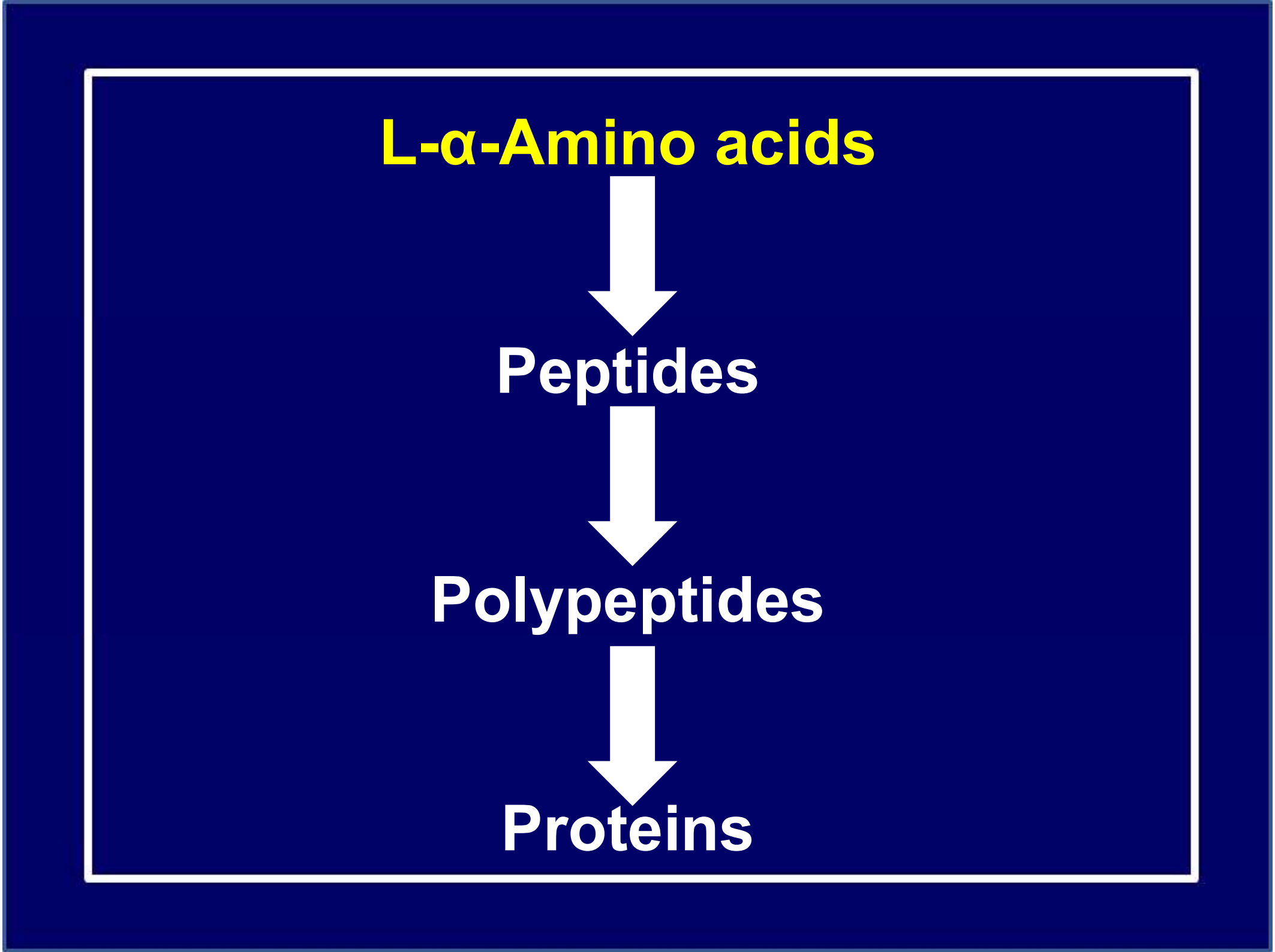
ARE DEPENDANT

UPON

ADEQUATE NUTRITIONAL

INTAKE

Amino acids and Peptides


L-α-Amino acids
Peptides
Polypeptides
Proteins

The L-α-amino acids and their derivatives build proteins, neurotransmitters, biosynthesis of porphyrins, purines, pyrimidines and urea.

Peptides form hormones, hormone releasing factors, neuromodulators and neurotransmitters.*

*Harpers Biochemistry 29th edition p17

Proteins

1. Build tissues
2. Transport molecules
3. Form antibodies
4. Form enzymes

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Proteins are physically and functionally complex macromolecules that perform multiple critically important roles.

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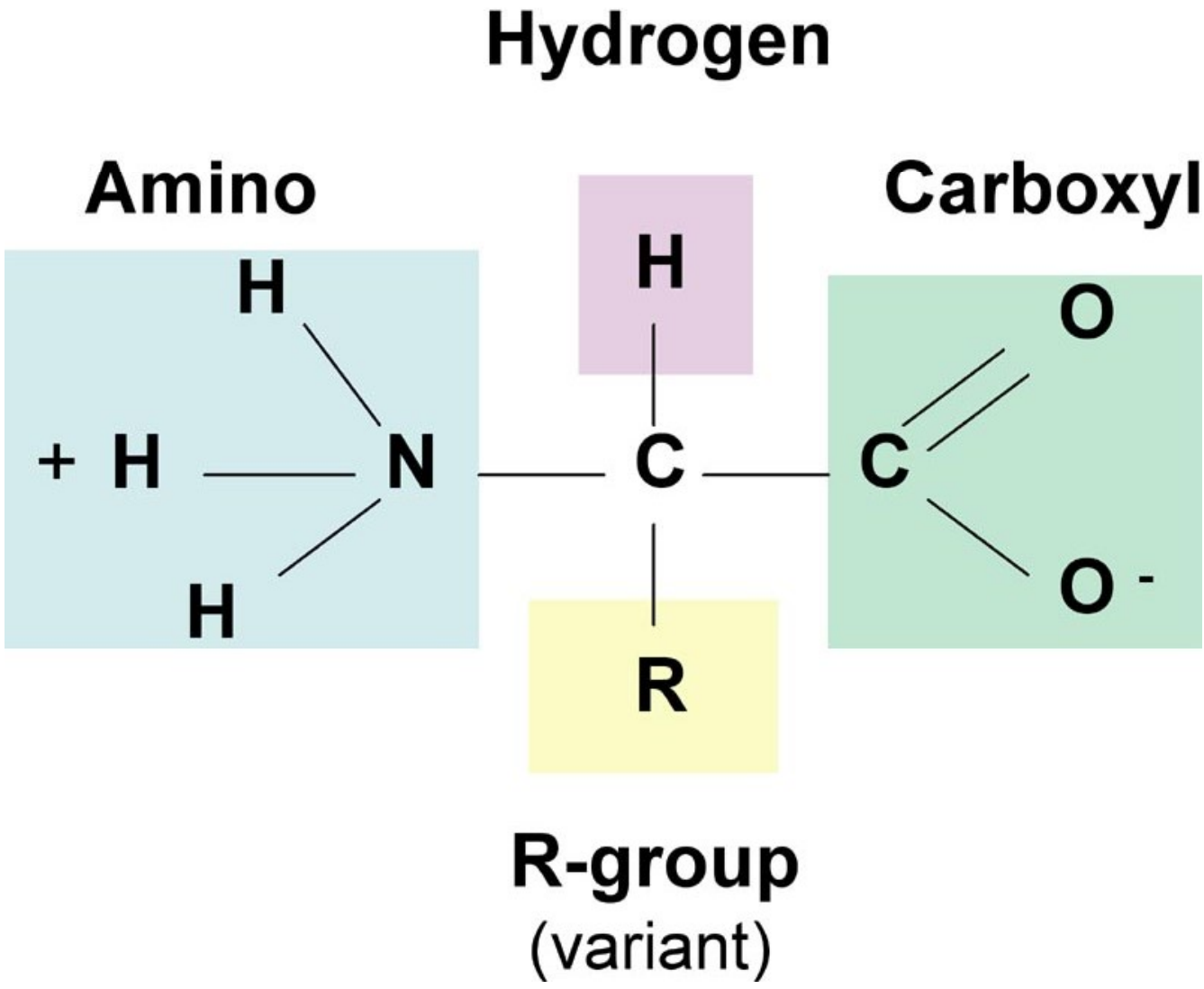
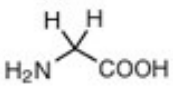
Amino acids are biologically important organic compounds composed of amine (-NH_2) and carboxylic acid (-COOH) functional groups, along with a side-chain specific to each amino acid. The key elements of an amino acid are carbon, hydrogen, oxygen and nitrogen.*

*Wagner I, Musso H (November 1983). "New Naturally Occurring Amino Acids".22 (11): 816–28

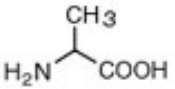
Amino Acid Structure



Small

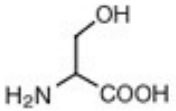


Glycine (Gly, G)
MW: 57.05

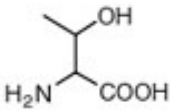


Alanine (Ala, A)
MW: 71.09

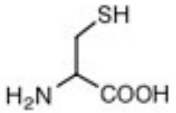
Nucleophilic



Serine (Ser, S)
MW: 87.08, pK_a ~ 16

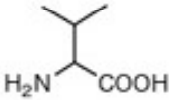


Threonine (Thr, T)
MW: 101.11, pK_a ~ 16

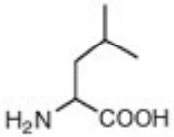


Cysteine (Cys, C)
MW: 103.15, pK_a = 8.35

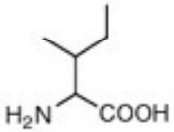
Hydrophobic



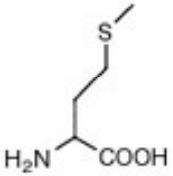
Valine (Val, V)
MW: 99.14



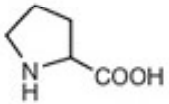
Leucine (Leu, L)
MW: 113.16



Isoleucine (Ile, I)
MW: 113.16

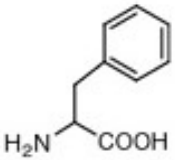


Methionine (Met, M)
MW: 131.19

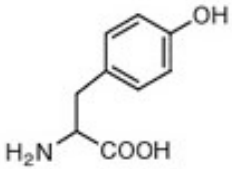


Proline (Pro, P)
MW: 97.12

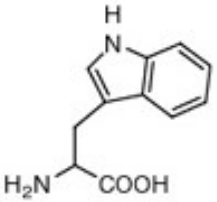
Aromatic



Phenylalanine (Phe, F)
MW: 147.18

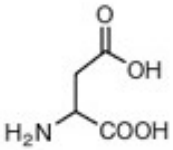


Tyrosine (Tyr, Y)
MW: 163.18

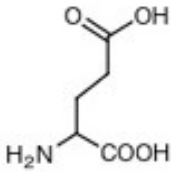


Tryptophan (Trp, W)
MW: 186.21

Acidic

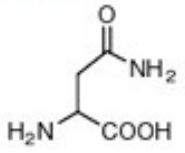


Aspartic Acid (Asp, D)
MW: 115.09, pK_a = 3.9

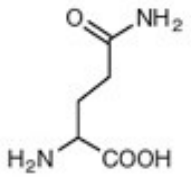


Glutamic Acid (Glu, E)
MW: 129.12, pK_a = 4.07

Amide

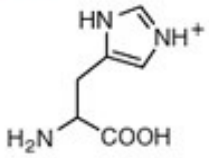


Asparagine (Asn, N)
MW: 114.11

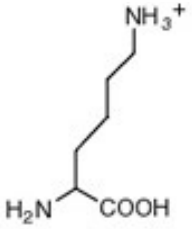


Glutamine (Gln, Q)
MW: 128.14

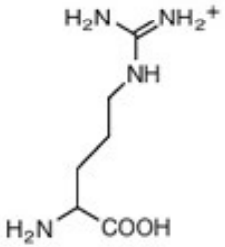
Basic



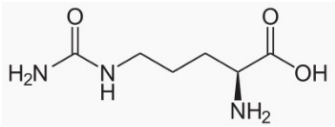
Histidine (His, H)
MW: 137.14, pK_a = 6.04



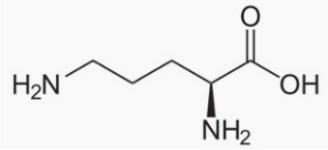
Lysine (Lys, K)
MW: 128.17, pK_a = 10.79



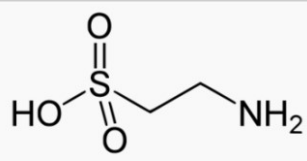
Arginine (Arg, R)
MW: 156.19, pK_a = 12.48



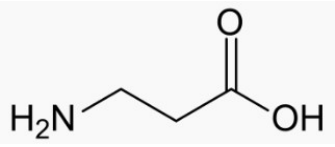
Citrulline



Ornithine



Taurine



B-Alanine

***Harpers Biochemistry 29th edition p18**

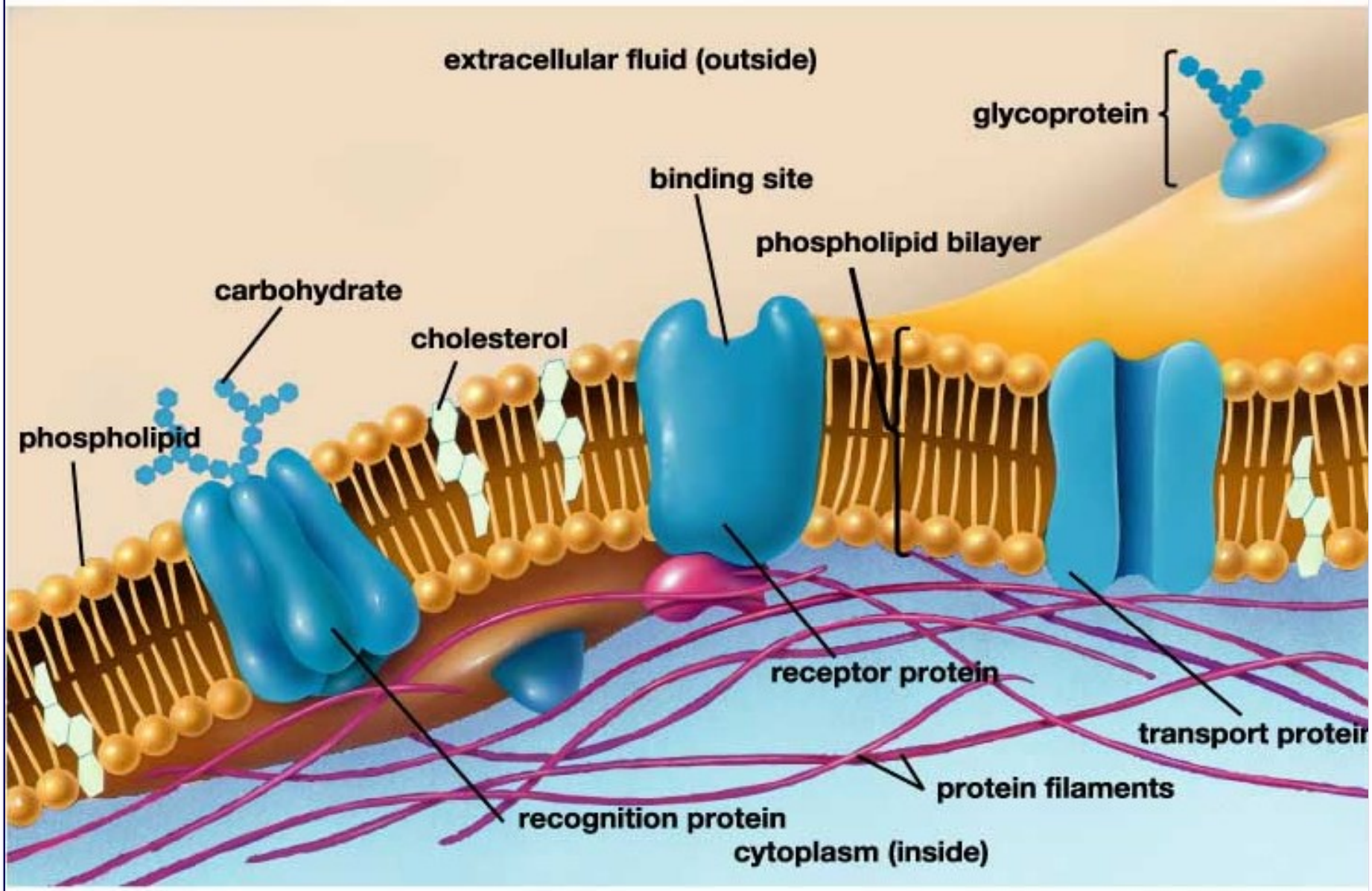
Essential			Nonessential		
Histidine	N 90%,	Hydrophobic	Alanine	N	Hydrophobic
Isoleucine	N	Hydrophobic	Arginine	+ve	Hydrophilic
Leucine	N	Hydrophobic	Asparagine	N	Hydrophilic
Lysine	+ve	Hydrophilic	Aspartic acid	–ve	Hydrophilic
Methionine	N	Hydrophobic	Cysteine	N	Hydrophobic
Phenylalanine	N	Hydrophobic	Glutamic acid	–ve	Hydrophilic
Threonine	N	Hydrophilic	Glutamine	N	Hydrophilic
Tryptophan	N	Hydrophobic	Glycine	N	Hydrophobic
Valine	N	Hydrophobic	Ornithine		
			Proline	N	Hydrophobic
			Selenocysteine		
			Serine	N	Hydrophilic
			Tyrosine	N	Hydrophilic

Left brain weakness give hydrophilic
Right brain weakness give hydrophobic*

*Collected Published Articles and Reprints by Dr G. Goodheart

Both D-Amino acids and non α-amino acids occur in nature but only L-α-amino acids are present in proteins.*

*Harpers Biochemistry 29th edition p24


extracellular fluid (outside)
glycoprotein
binding site
phospholipid bilayer
carbohydrate
cholesterol
phospholipid
receptor protein
transport protein
protein filaments
recognition protein
cytoplasm (inside)

Protein conformation

When a protein changes shape it performs movement i.e. as it changes from **Conformation A** to **Conformation B**.* This movement is harnessed by the cell to carry out functions such as digestion, respiration, muscle contraction etc.

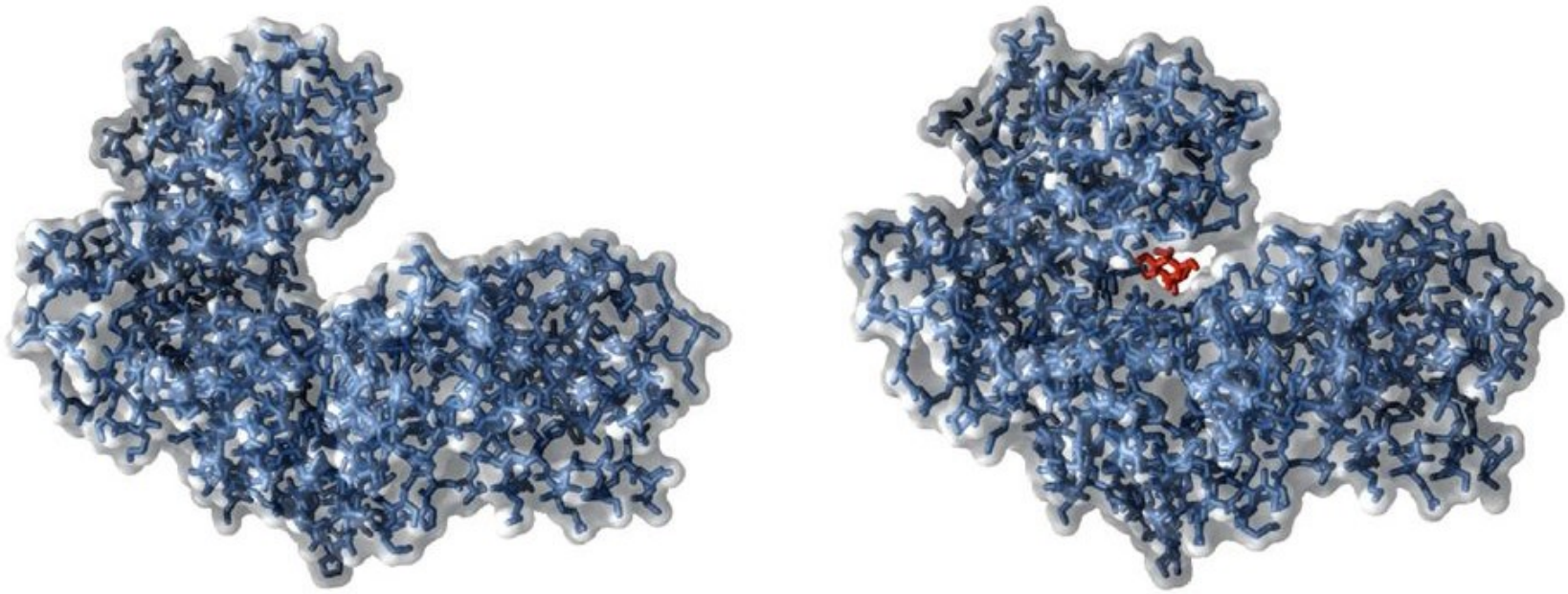
Compare a human cadaver to a living human. <u>Life is movement.**</u>

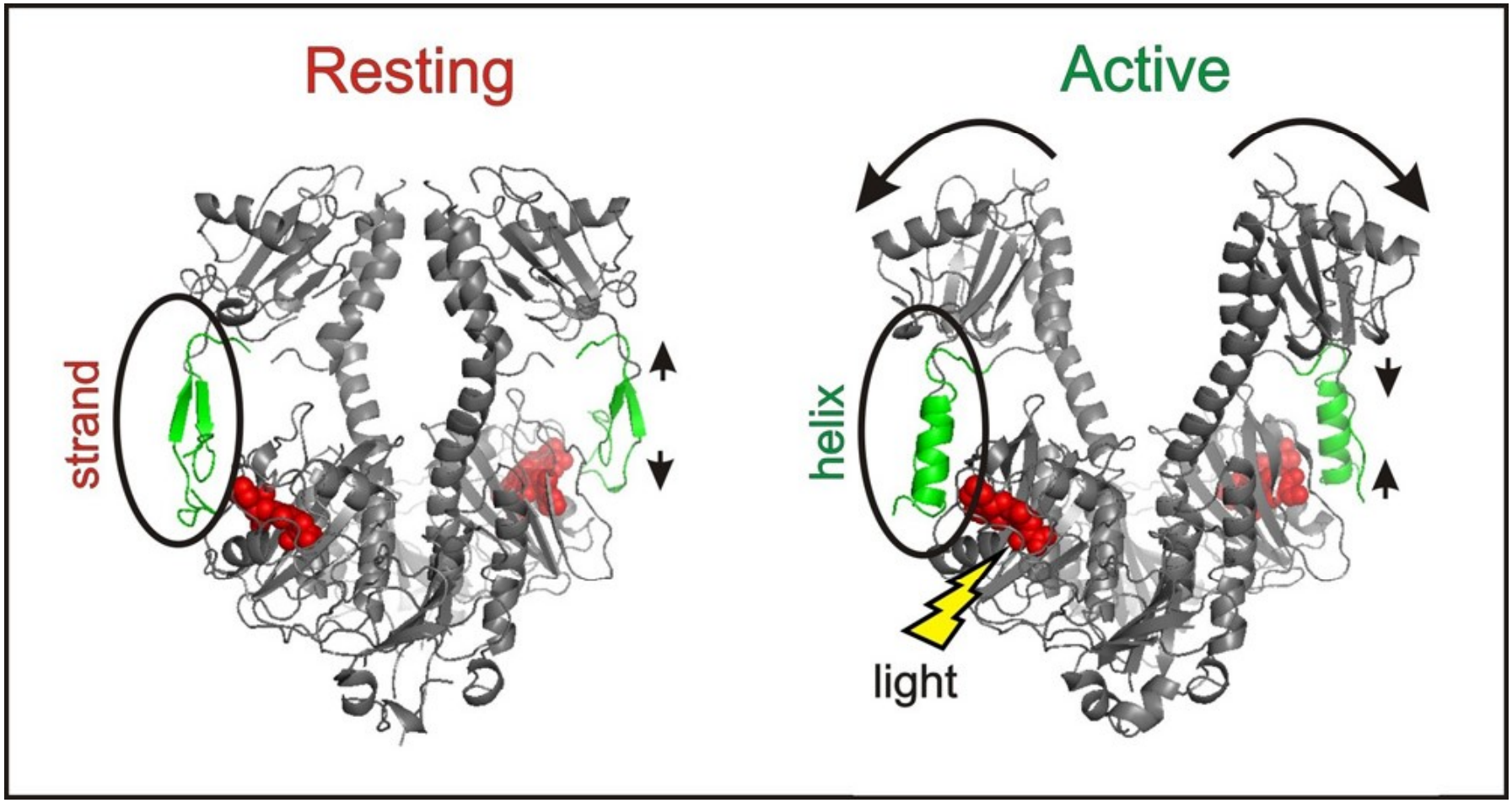
*Harpers Biochemistry 29th edition Pg35 **The Biology of Belief13 Oct 2015 by Bruce H. Lipton

Conformation A Conformation B

Example

Hexokinase undergoes a conformational change on binding glucose (induced fit)




Resting
Active
strand
helix
light

What makes a protein change shape is called a signal. A signal may be a particle (drugs, hormones, growth factors etc) or an energy (such as light (e.g. acetates), sound, EM waves (e.g biomarker vials), thoughts). Normal shape of a protein is when all the positive and negative charges are balanced.

The Biology of Belief13 Oct 2015 by Bruce H. Lipton

These proteins have little clefts on their surfaces that can plug into signals.

So the protein can be like a baseball glove and the signal is like the baseball.

So the signal has to have the precise shape to fit into the glove and the right charge so it can attach.

The Biology of Belief13 Oct 2015 by Bruce H. Lipton

This changes the charge and thus the protein conformation. So the movement can be used to drive work which creates functions e.g. a digestive enzyme and a food particle. The movement crushes the food particle. If a number of fragments are inserted into the glove then synthesise will occur.

The Biology of Belief13 Oct 2015 by Bruce H. Lipton

Genes provide the blueprint of the protein but the life of the protein is determined by the signal.

Thus two parts to life – the physical part (protein) and the signal part that controls the movement.

Behaviour is thus dependant on the proteins and the signals.

The Biology of Belief13 Oct 2015 by Bruce H. Lipton

Disease is a defect in the behaviour of the proteins and the signals.

< 5% is due to defects in the protein from birth defects (but more as we age)

95+% is due to defects in the signal.

FATTYACIDS

1. Form cell membranes
2. Are sources of energy
3. Are stores of energy
4. Protect organs
5. Act as electrical and thermal insulators
6. Build steroid hormones

Carbohydrates

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

Vitamins

1. Act as co-enzymes

in specific enzyme pathways

2. Act as antioxidants

3. Involved with blood clotting

4. Part of cell membranes

CO-ENZYMES

Thiamine pyrophosphate	Decarboxylation
Thiamine triphosphate	Acetylation
Flavin coenzymes	Oxidation - reduction
Nicotinamide coenzymes	Oxidation – reduction
Co-enzyme A	Acyl transfer
Pyridoxal-5-phosphate	Amine group transfer
Biotin	Carboxylation
Folates	One carbon transfer

Adenosylcobalamin	Alkylation
Methylcobalamin	Methylation
Tetrahydro Biopterin	Hydroxylation
Vitamin C	Oxidation – reduction
SAM	Methylation
Alpha Lipoic acid	Acyl transfer
Co-enzyme Q10	Oxidation - reduction


ENZYMES

Enzymes are efficient catalyst whose stringent specificity extends to the kind of reaction catalysed and typically to a single substrate.

*Harpers Biochemistry 29th edition Pg68

Organic and inorganic prosthetic groups, co-factors (minerals) and co-enzymes play import roles in catalysis.

Co-enzymes are largely derived from Vitamin Bs and serve as shuttles for commonly used groups such as amines, electrons and acetyl groups.

*Harpers Biochemistry 29th edition Pg68

During catalysis enzymes frequently redirect the conformational changes induced by the substrate binding to effect complementary changes in the substrate that facilitate its transformation into product.

*Harpers Biochemistry 29th edition Pg68

Enzymes are protein catalysts that regulate the rates at which physiological processes take place. They are encoded by specific genes which in turn are stimulated by hormones.

There are 7341enzymes catalogued in the **BRENDA ENZYME DATABASE.***

*http://www.brenda-enzymes.org/all_enzymes.php

There are two types

1) those that require a coenzyme such as the oxido-reductases. 22% of known enzymes require coenzymes to function.

2) those that do not require a coenzyme such as the digestive enzymes.

Four parts

1.The **apoenzyme** is the protein part of an enzyme.

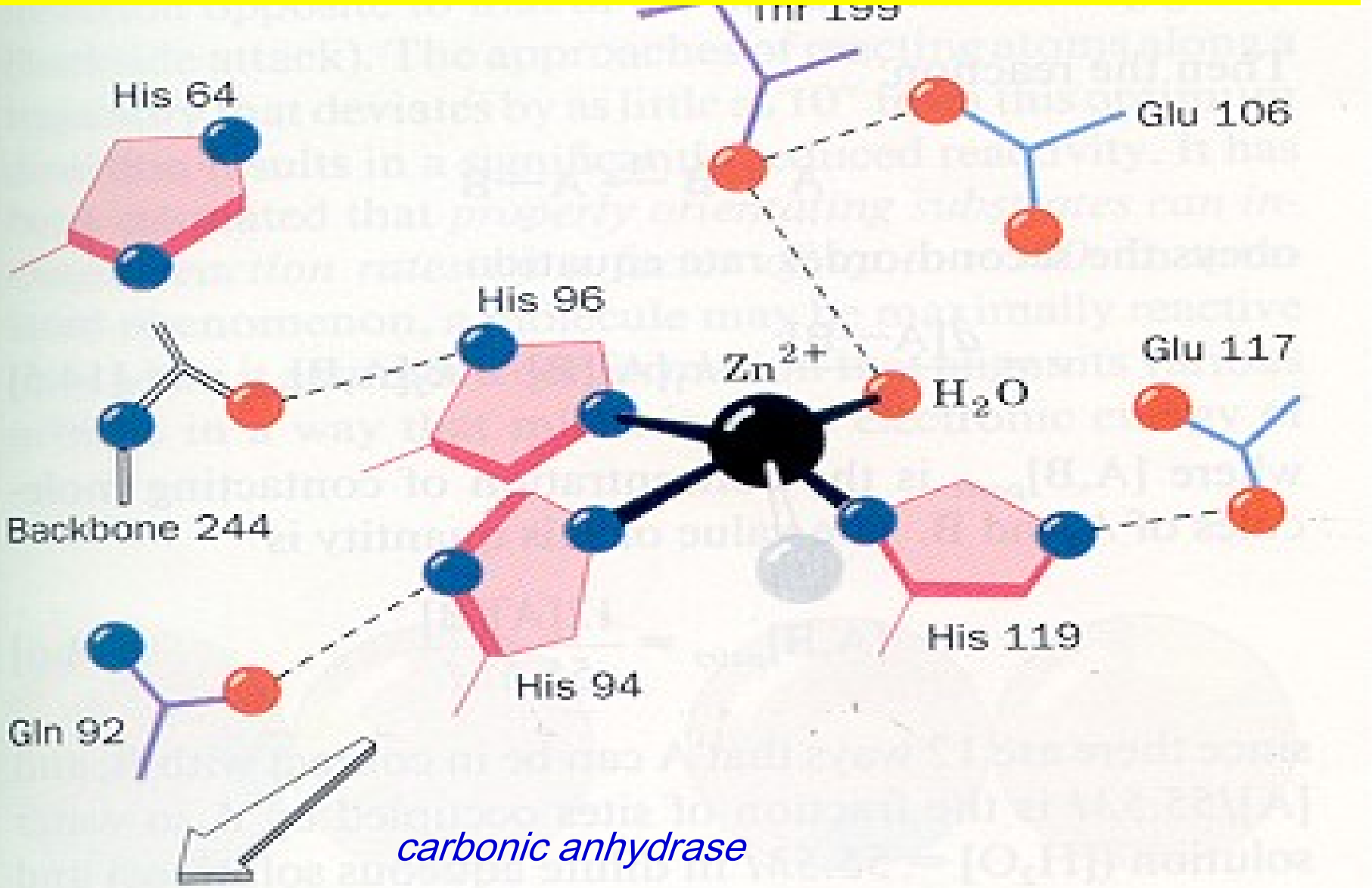
2.The **coenzyme** is required for the activation of an enzyme.

3.Metal ion catalysts

a) Metalloenzymes contain tightly bound metal ions most commonly transition metal ions such as Fe^{2+}, Fe^{3+}, Cu^{2+}, Zn^{2+},Mn^{2+} or Co^{3+}.

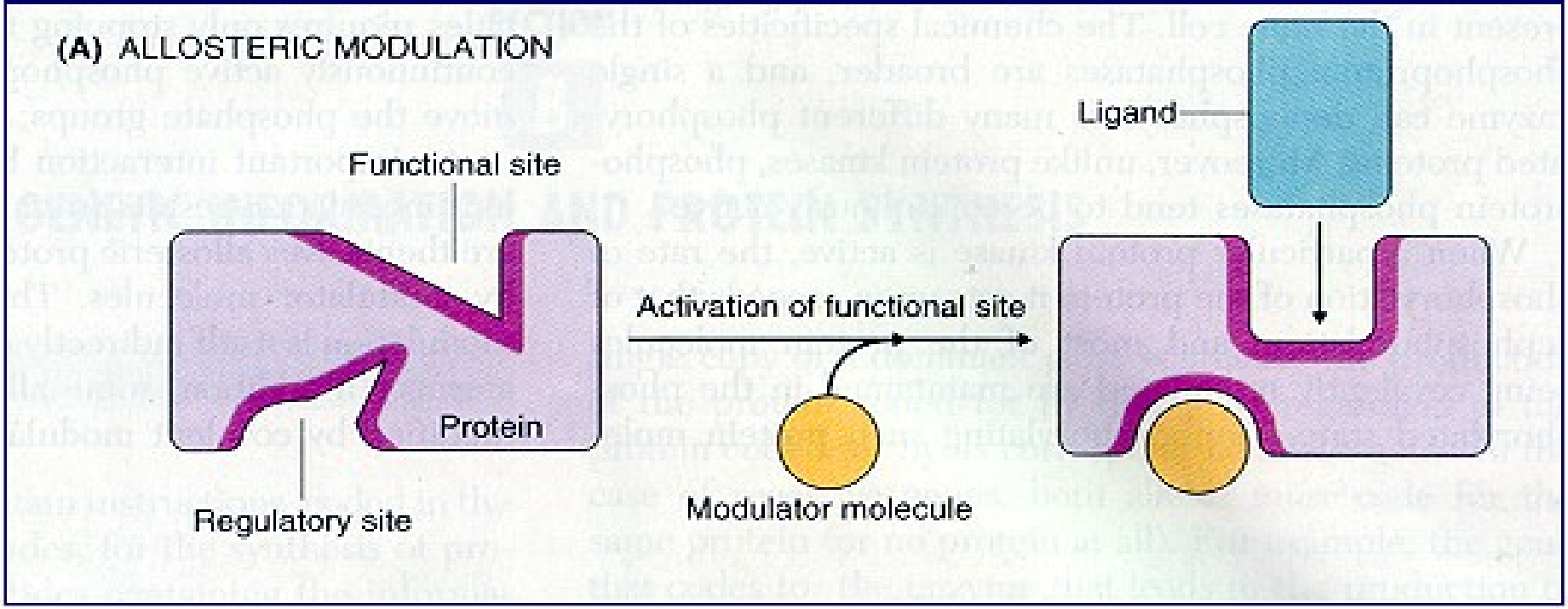
b) Metal activated enzymes loosely bind metal ions from solution, usually alkaline earth metal ions Na^{+}, K^{+}, Mg^{2+} or Ca^{2+}

$CO_2 + H_2O = H_2CO_3 = H^+ + HCO_3^-$



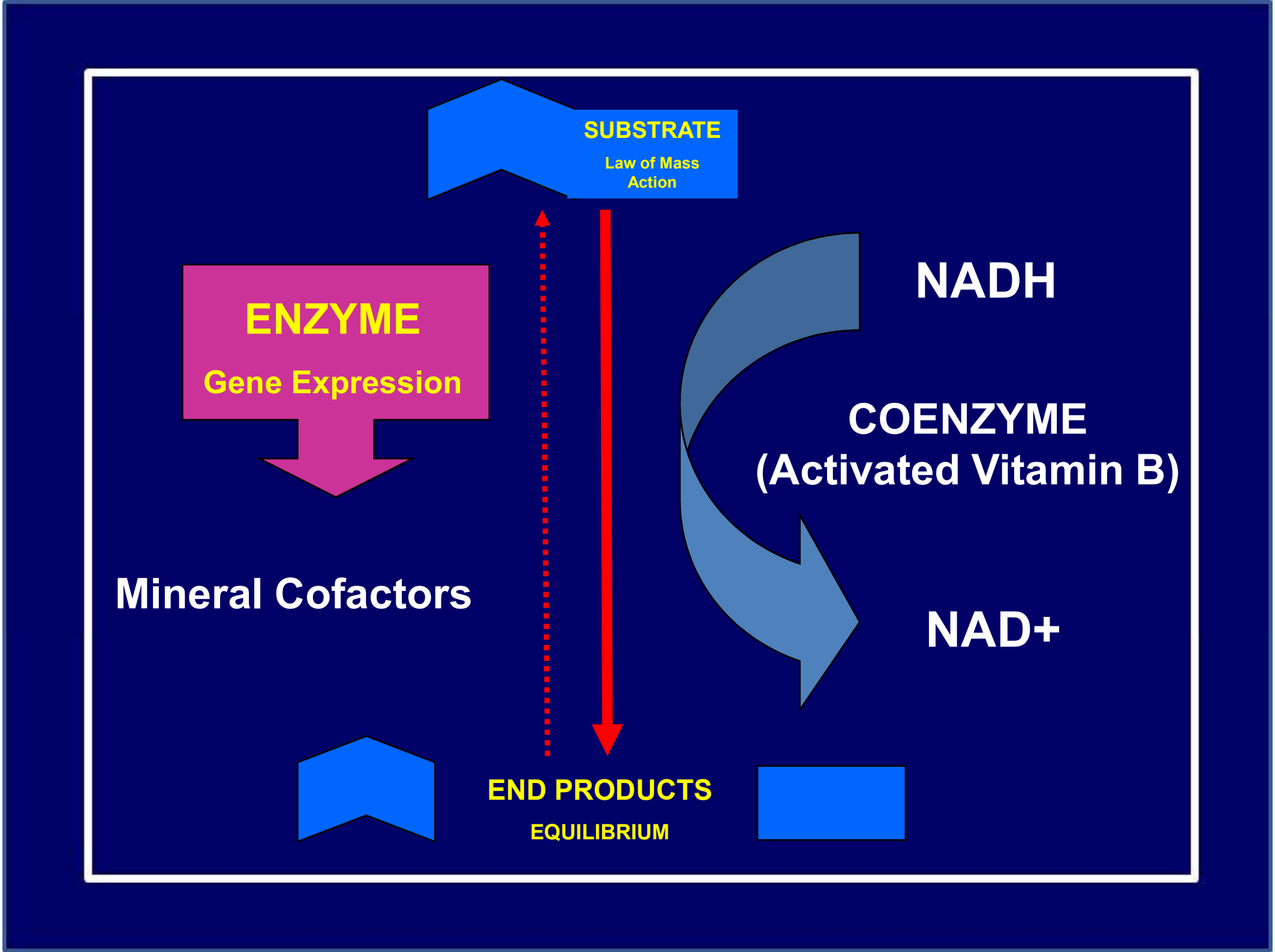
carbonic anhydrase

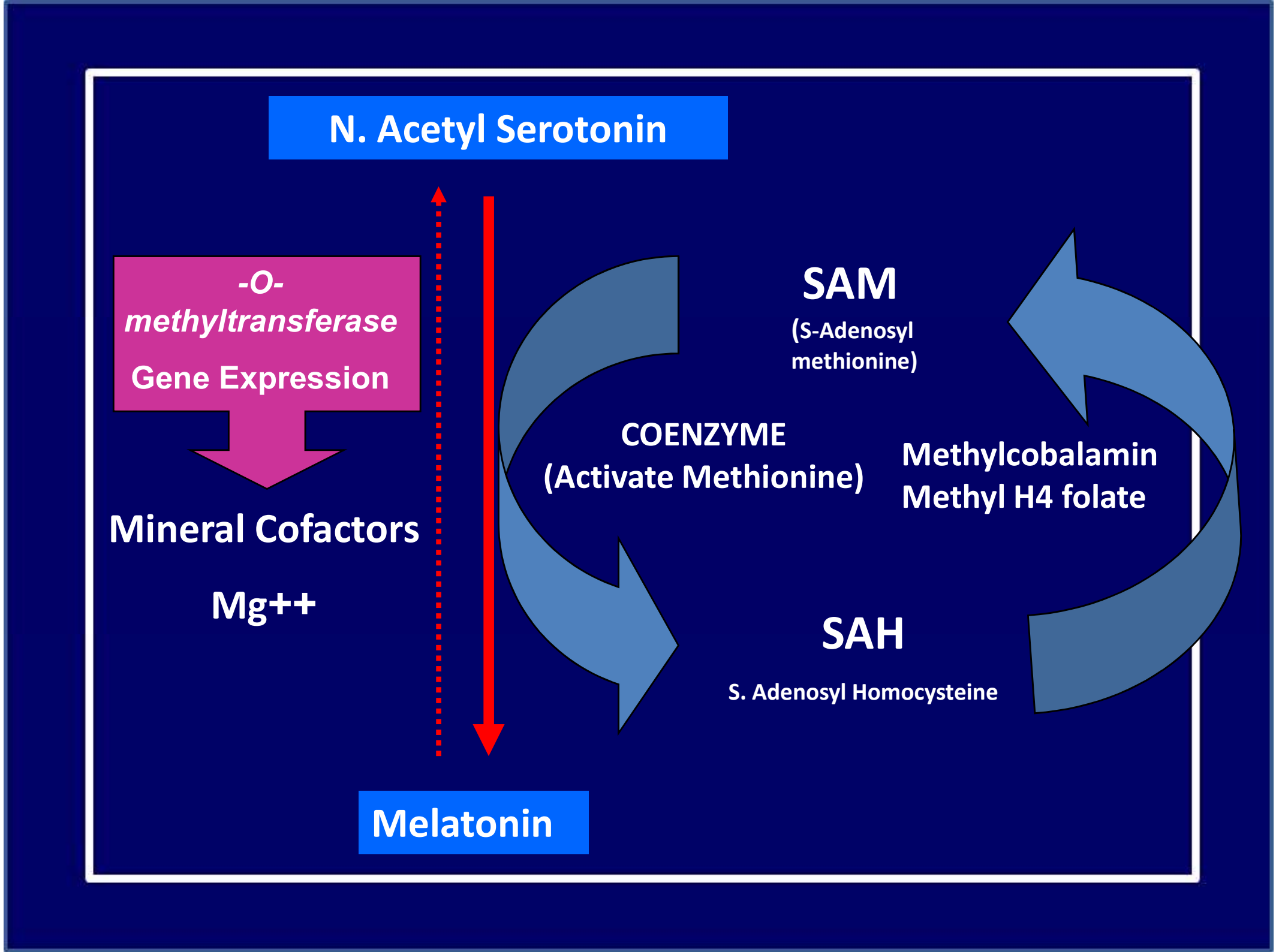
4. Low molecular weight allosteric effectors modulate the catalytic activity of certain regulatory enzymes.



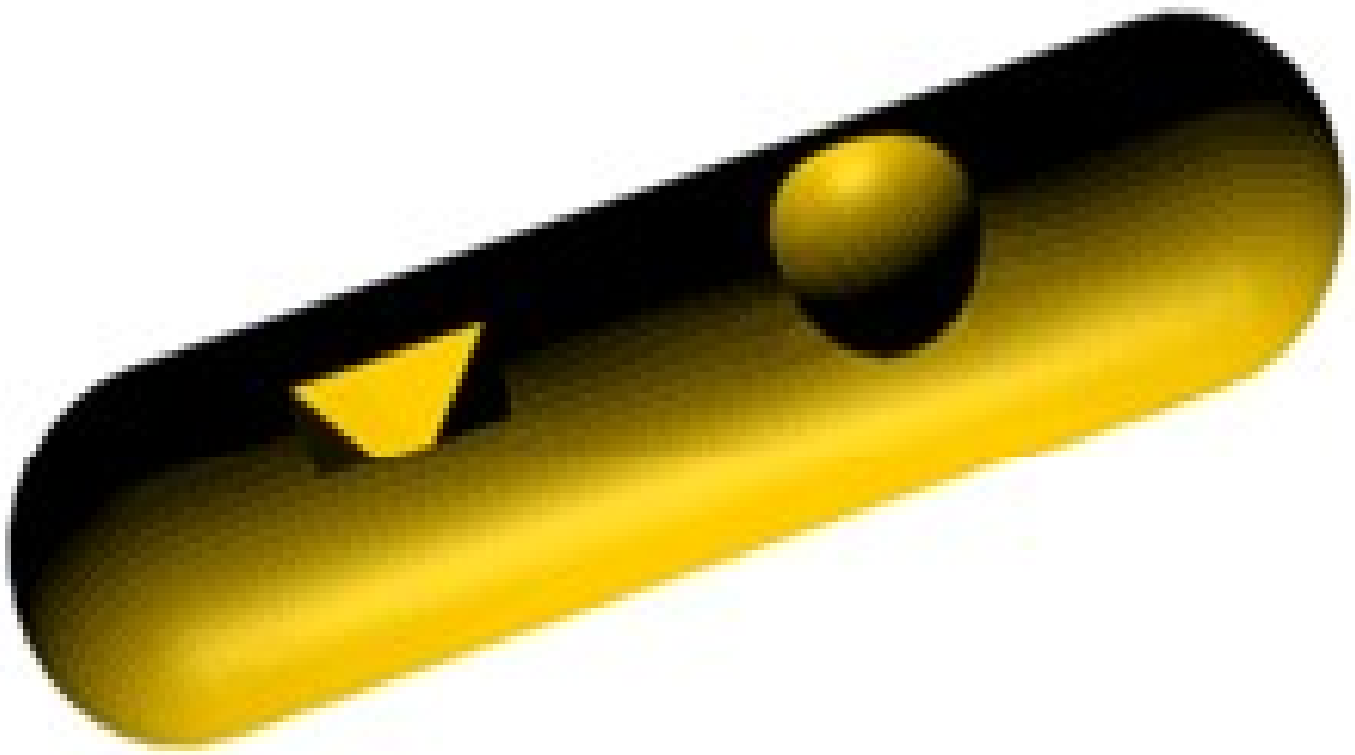
Factors affecting enzyme function

1. Temperature
2. Enzyme concentrations
3. Substrate concentration
4. pH
5. Inhibitors can poison enzymes e.g. certain chemicals e.g. toiletries, cosmetics, toxic metals and mycotoxins.


SUBSTRATE
Law of Mass Action
ENZYME
Gene Expression
NADH
COENZYME
(Activated Vitamin B)
Mineral Cofactors
NAD+
END PRODUCTS
EQUILIBRIUM


N. Acetyl Serotonin
-O-
methyltransferase
Gene Expression
Mineral Cofactors
Mg++
SAM
(S-Adenosyl
methionine)
COENZYME
(Activate Methionine)
Methylcobalamin
Methyl H4 folate
SAH
S. Adenosyl Homocysteine
Melatonin

enzyme molecule
showing the active site

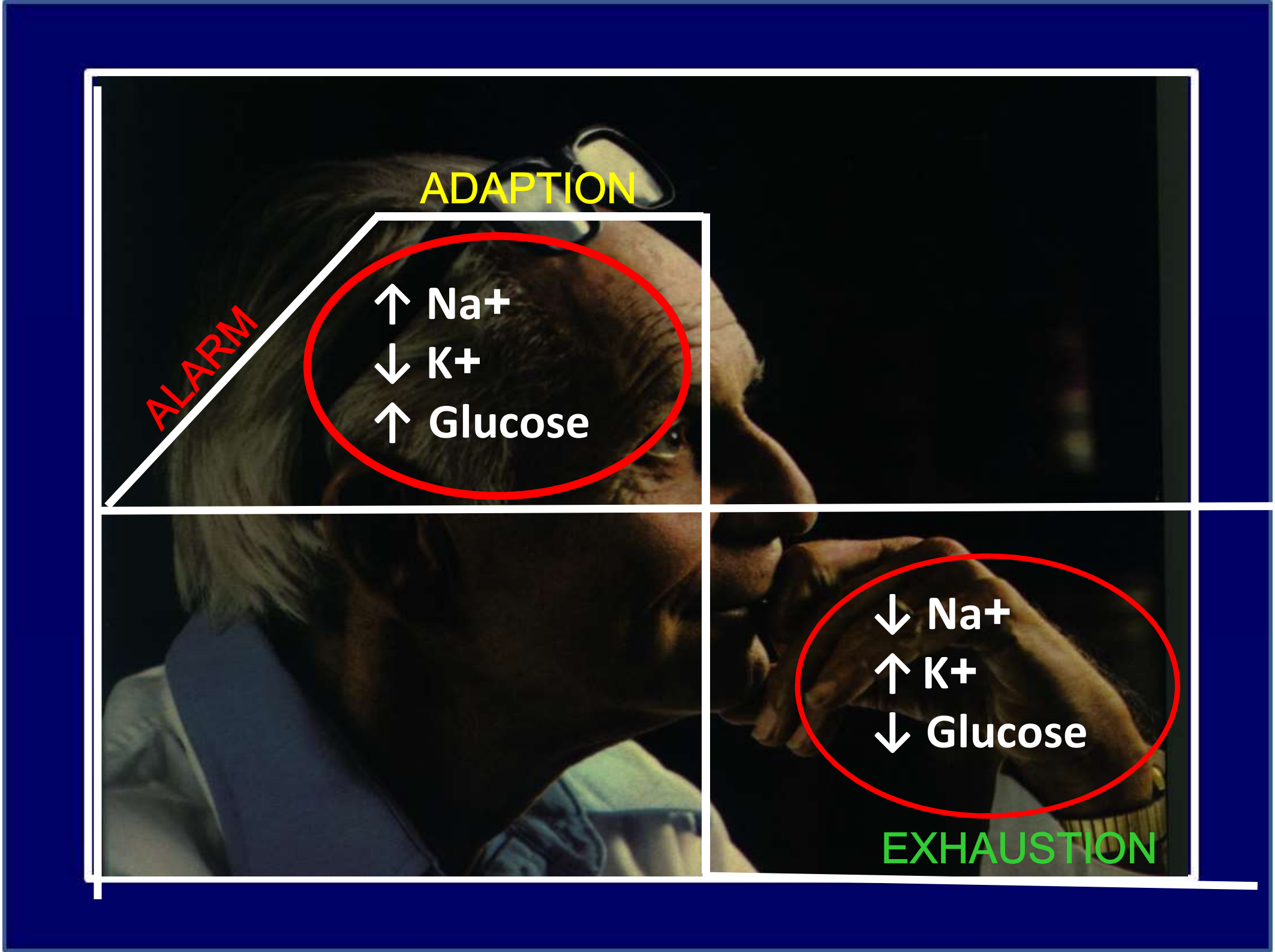


this is a diagrammatic representation

Challenges for enzyme pathway inhibition

1. A weak associated muscle will strengthen to the required end product.
2. A strong indicator muscle will weaken when challenged to the substrate.
3. This weakness will be negated by the mineral cofactors and / or the coenzyme (usually an activated vitamin b)

Selye's General Adaption to stress


ADAPTION
ALARM
↑ Na+
↓ K+
↑ Glucose
↓ Na+
↑ K+
↓ Glucose
EXHAUSTION

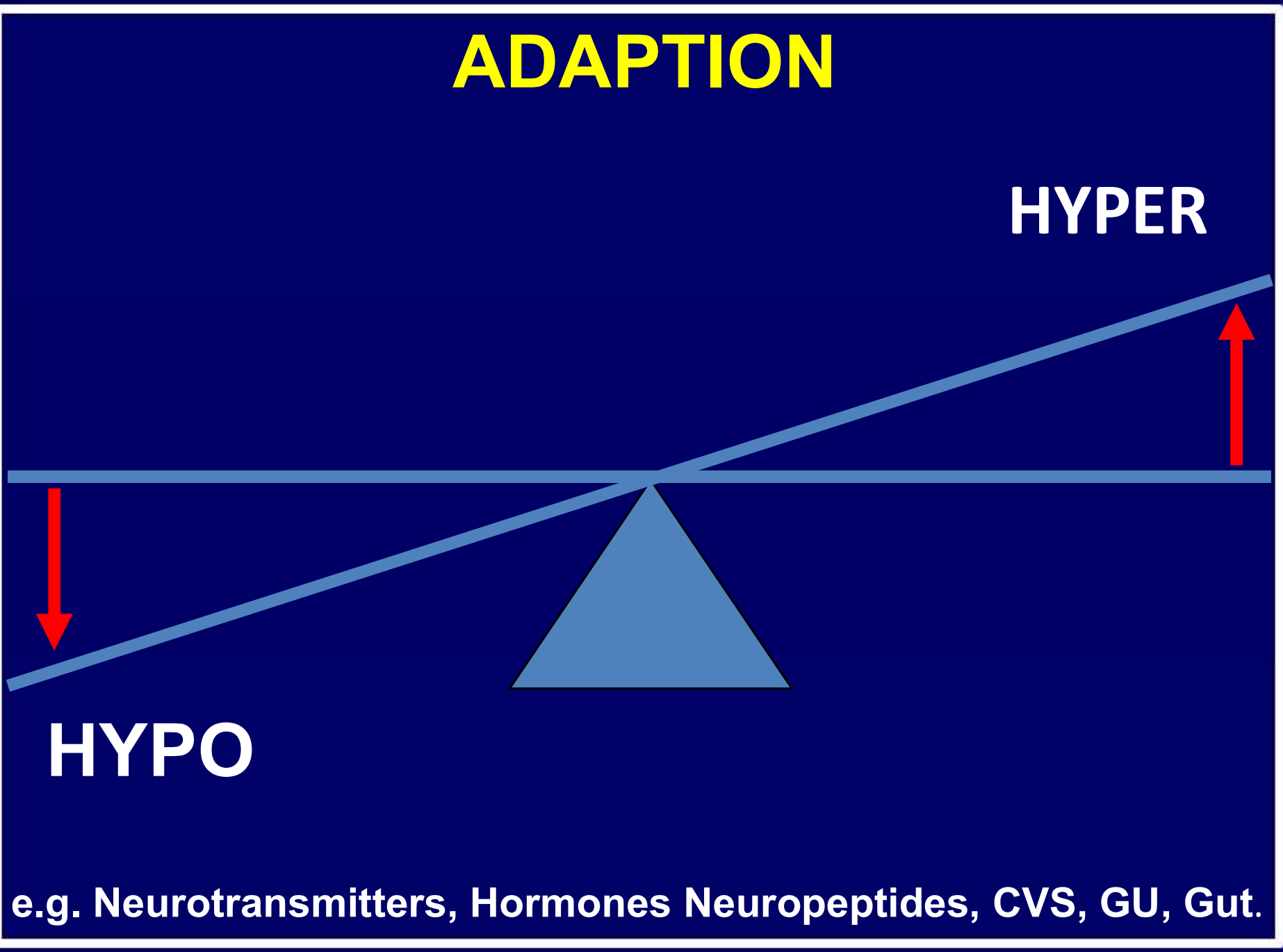
1. Atrophy of the Thymus gland and other Lymphoid Tissues
2. Enlarged Adrenal Glands
3. Gastrointestinal ulcerations

Indicating that the main areas that stress affects are

1. The Immune System
2. The Endocrine System
3. The Gastro-Intestinal System


Normal physiology
Dysfunctioning physiology
Pathology
Death

ADAPTION



e.g. Neurotransmitters, Hormones Neuropeptides, CVS, GU, Gut.

Adaption occurs

Initially in the Nervous System (Neural)

Secondarily in the Endocrine System (Humoral)

Applied Kinesiology was first developed by **Dr George Goodheart Jn,** an American chiropractor from Detroit, some 55 years ago.

Applied Kinesiology
is a diagnostic tool
used to evaluate
the motor response
of the central nervous system
to a sensory challenge.*

*Chris Astill-Smith 2001

Triad of Health

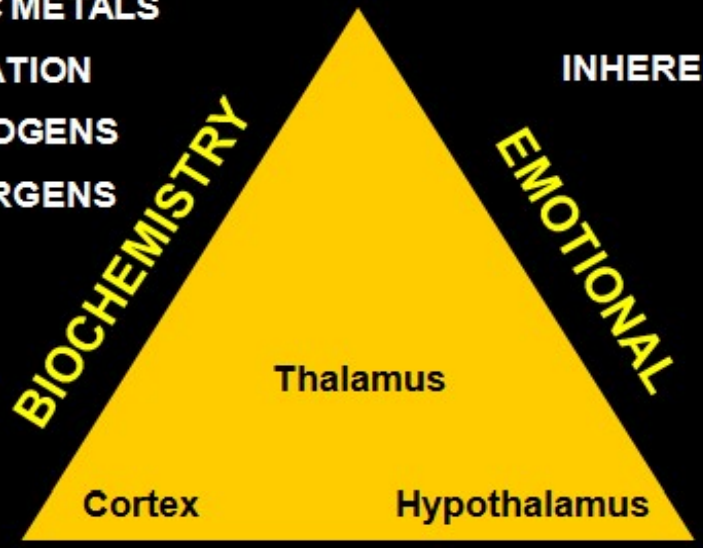
INPUTS

PROTEINS
FATS
CHO
MINERALS
VITAMINS
PROBIOTICS
NUCLEOTIDES
WATER
FIBRE

OUTPUTS

FAECES
URINE
LUNGS
MUCOUS
SKIN

METABOLIC WASTE
+ LIFESTYLE
CHEMICALS
TOXIC METALS
RADIATION
PATHOGENS
ALLERGENS



INHERENT - UNCONSCIOUS

ACQUIRED

STRUCTURE

CRANIO-PELVIS-TMJ
SPINE, EXTREMITIES, SOFT TISSUES,
VISCERA

Introducing two strong indicator muscles

1. Rectus femoris
2. Middle fibres of Deltoid

Principles of Muscle testing

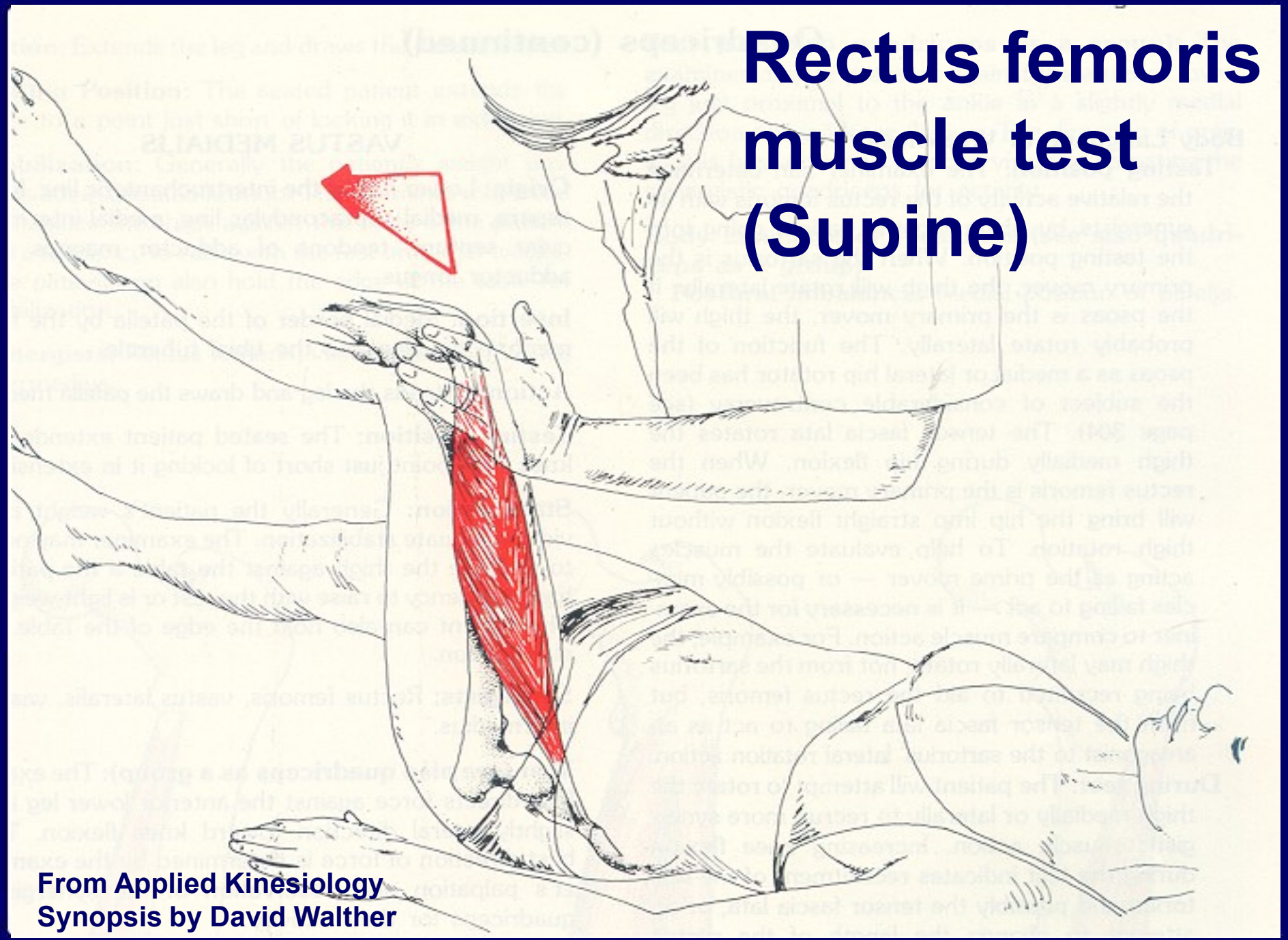
1. Approximate insertion to the origin.
2. Show patient how and where to push.

3. Activation of maximum number of muscle fibres. Important to wait 2/3 seconds for maximum aerobic contraction.

4. Practitioner then applies a little extra pressure.

5. Compare with other side.

Rectus femoris muscle test (Supine)



From Applied Kinesiology
Synopsis by David Walther

Rectus femoris

Origin – Straight head - Anterior inferior iliac spine
Reflected head – groove on upper brim of the acetabulum

Insertion – upper border of the patella with the ligamentum patellae extending to the tibial tubercle

From Applied Kinesiology Synopsis by David Walther

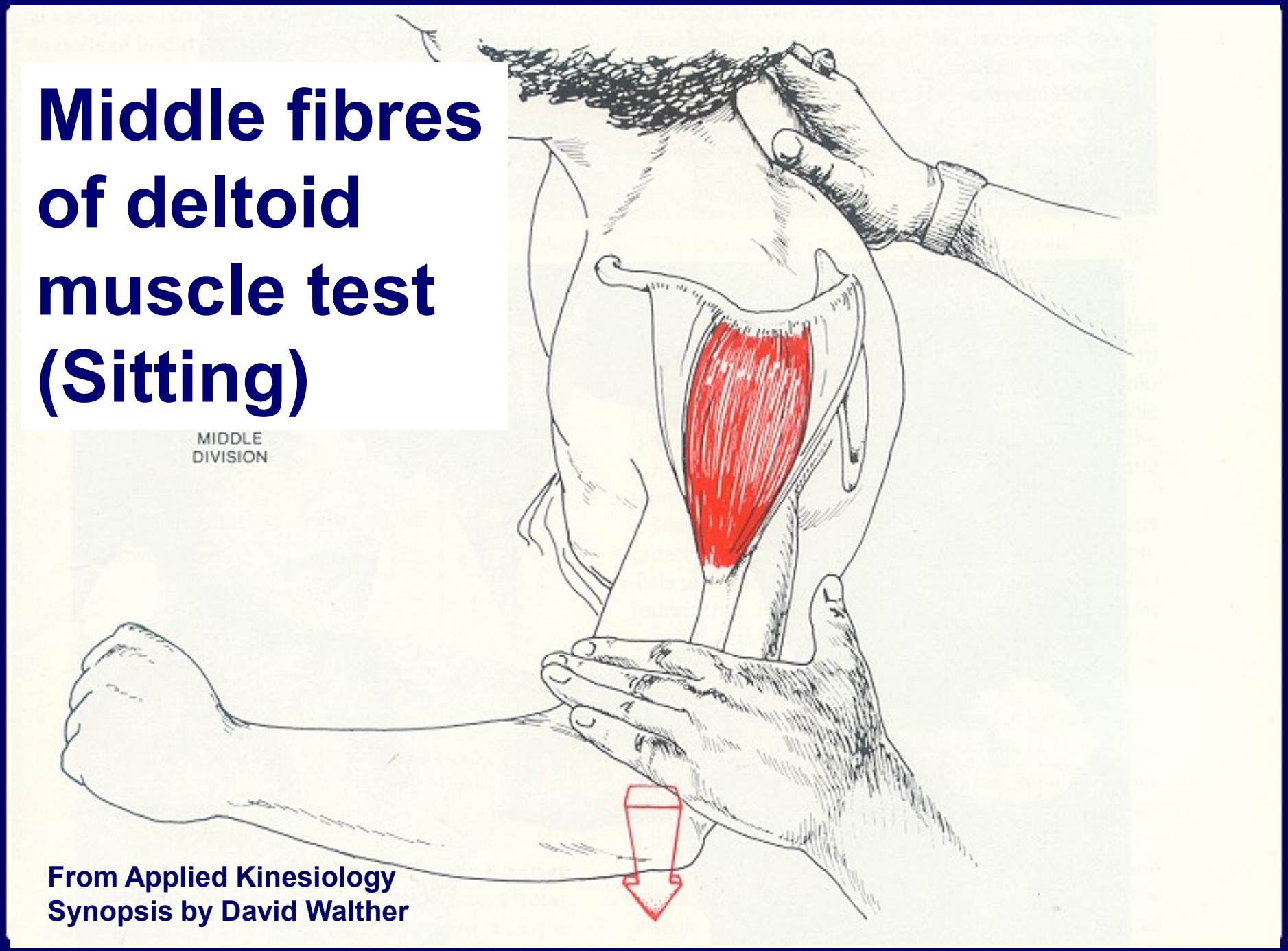
Function – extends the knee and flexes the hip joint

Nerve supply – Femoral L2, 3, 4

Meridian association – Small intestine

From Applied Kinesiology Synopsis by David Walther

Middle fibres of deltoid muscle test (Sitting)



From Applied Kinesiology
Synopsis by David Walther

Deltoid – middle fibres

Origin – upper surface of the acromion process

Insertion – Deltoid tuberosity of the humerus

From Applied Kinesiology Synopsis by David Walther

Function – Abduction of the humerus

Nerve supply – Axillary C5, 6

Meridian association – Lung

From Applied Kinesiology Synopsis by David Walther

Challenges

A challenge maybe

1. Mechanical such as pushing a vertebra in a specific vector.
2. Chemical such as putting wheat on the tongue.
3. Emotional such as thinking of a stressful incident.

From Applied Kinesiology Synopsis by David Walther

- Sensory challenge receptors

- Mechanoreceptors (90%)
- Nociceptors – Therapy localisation (TL)
- Thermal receptors
- Chemoreceptors e.g carotid body with CO_2
- Photoreceptors

Receptors are present in

Muscles, tendons, joints

Mouth (Gustatory)

Nose (Olfactory)

Eyes (Visual)

Ears (Auditory)

Skin (Touch and Electromagnetic)

Next Module

Module 2

Fat soluble Vitamins

Water Soluble Vitamins

Co-Enzymes