CARBOHYDRATE METABOLISM

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INTRODUCTION

NUTRITION

- Nutrition is defined as " the science of how the body utilizes food to meet requirements for development growth, repair and maintenance."

CARBOHYDRATES

FATS

PROTEINS

VITAMINS

MINERALS

WATER

Daily Intake

- **Nutrient Quantity Per Day**

✓ Energy = 8,700 kilojoules

✓ Protein = 50 grams

✓ Fat = 70 grams

✓ Carbohydrates = 310 grams

✓ Sugars = 90 grams

✓ Sodium (salt) = 2.3 grams

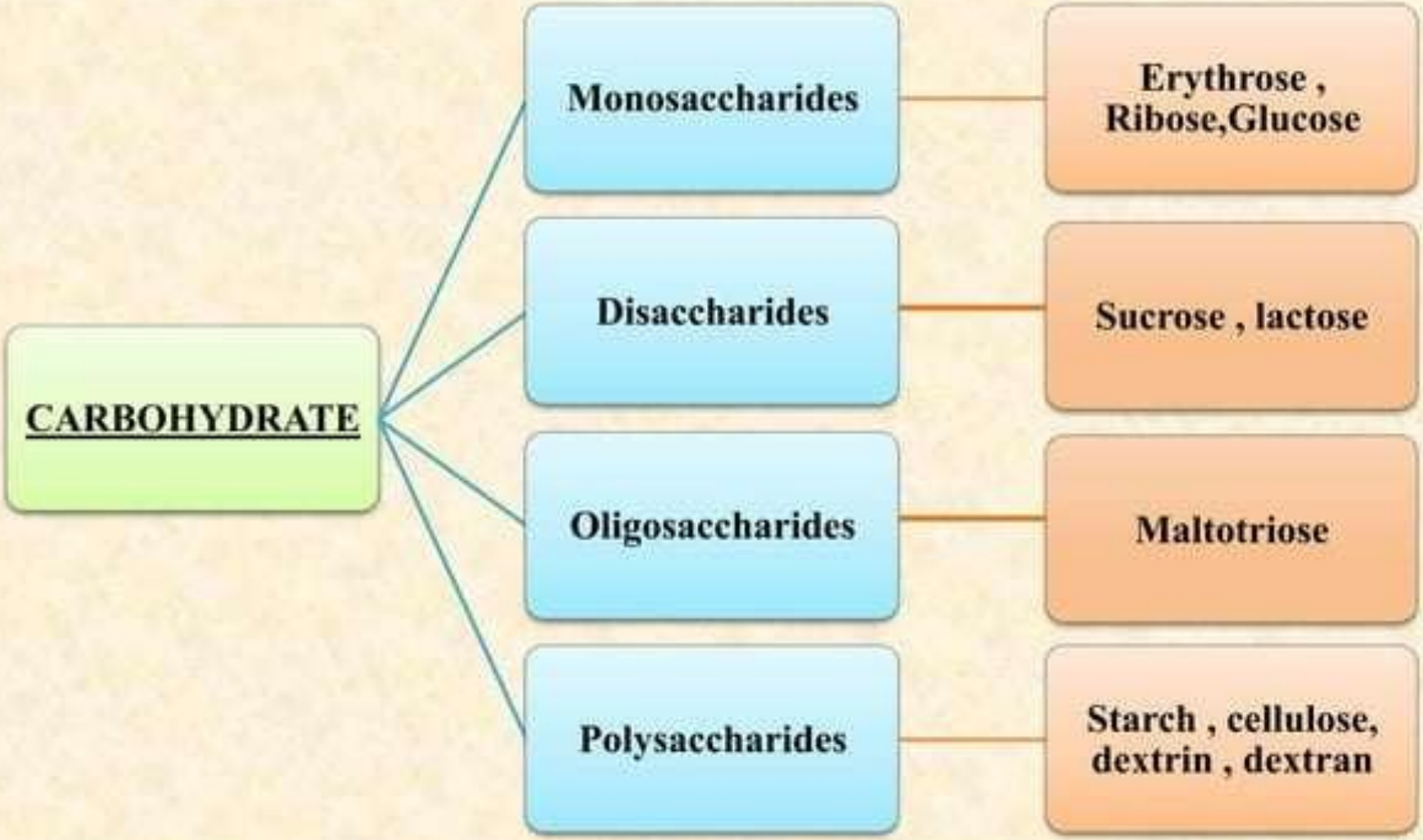
✓ Dietary Fibre = 30 grams

✓ Saturated Fatty Acids = 24 grams

CARBOHYDRATE:

- Most abundant organic molecule on earth.
- *Carbohydrates are defined as aldehyde or keto derivatives of polyhydric alcohols.*
- For example: Glycerol on oxidation is converted to D-glyceraldehyde, which is a carbohydrate derived from the trihydric alcohol (glycerol).
- All carbohydrates have the general formula $C_nH_{2n}O_n$ [or it can be re-written as $C_n(H_2O)_n$] .

CLASSIFICATION OF CARBOHYDRATE



FUNCTIONS OF CARBOHYDRATES

- Main source of energy in the body. Energy production from carbohydrates will be 4 k calories/g (16 k Joules/g).
- Storage form of energy (starch and glycogen).
- Excess carbohydrate is converted to fat.
- Glycoproteins and glycolipids are components of cell membranes and receptors.
- Structural basis of many organisms. For example, cellulose of plants,exoskeleton of insects etc.

Biomedical Importance Of Glucose

- Glucose is a major carbohydrate
- It is a major fuel of tissues
- It is converted into other carbohydrates

✓ Glycogen for storage.
✓ Ribose in nucleic acids.
✓ Galactose in lactose of milk.
✓ They form glycoproteins & proteoglycans
✓ They are present in some lipoproteins (LDL) .
✓ Present in plasma membrane:glycocalyx.
✓ Glycophorin is a major intergral membrane glycoprotein of human erythrocytes.

METABOLISM

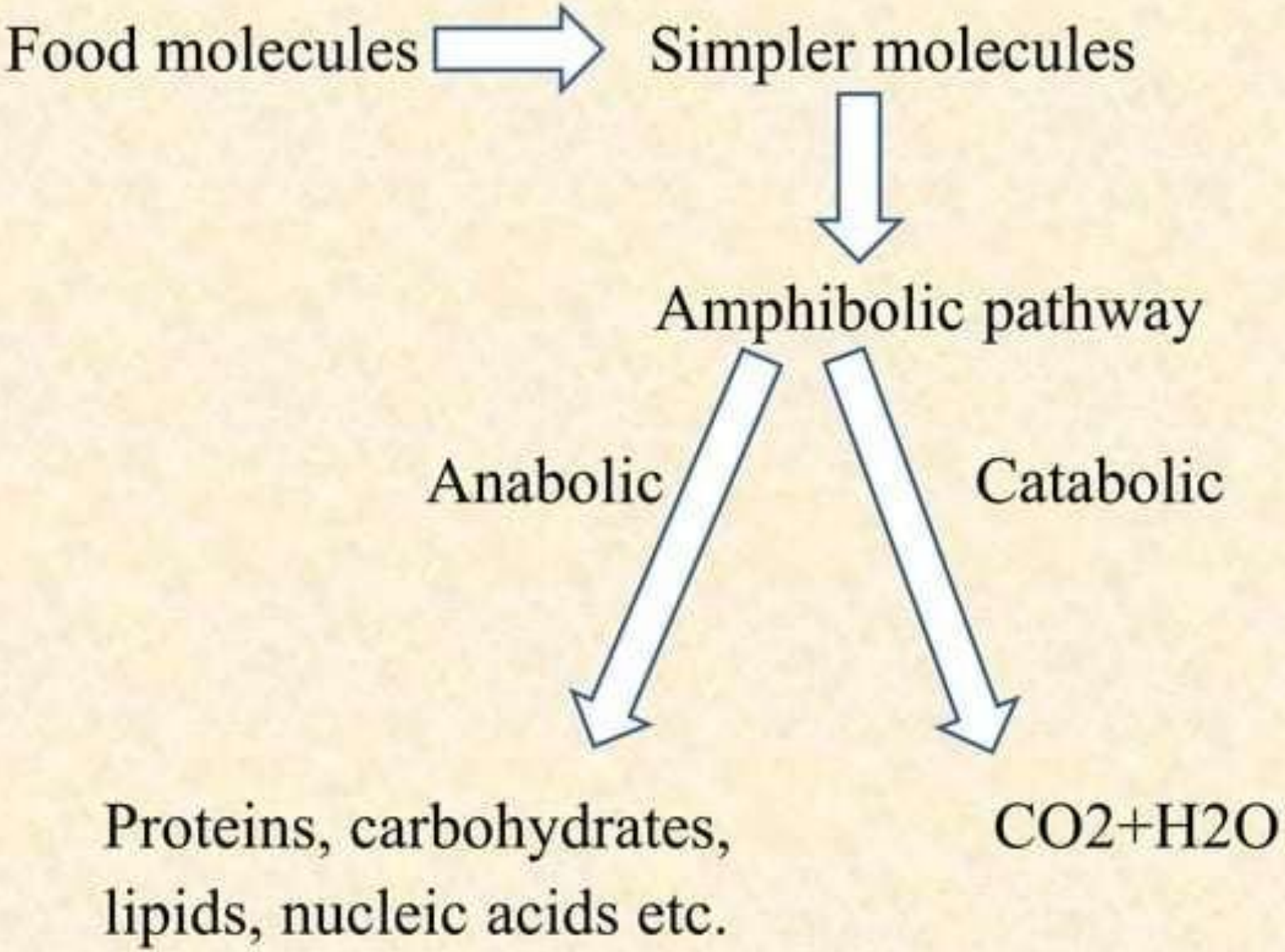
Thousands of chemical reactions are taking place inside a cell in an organized, well co-ordinated and purposeful manner; all these reactions are called as METABOLISM.

TYPES OF METABOLIC PATHWAY:

- ✓Catabolic Pathway
- ✓Anabolic Pathway
- ✓Amphibolic Pathway

STAGES AND PHASES OF METABOLISM:

- ✓Primary
- ✓Secondary
- ✓Tertiary


Food molecules
Simpler molecules
Amphibolic pathway
Anabolic
Catabolic
Proteins, carbohydrates, lipids, nucleic acids etc.
CO2+H2O

MAJOR PATHWAYS OF CARBOHYDRATE METABOLISM

1) Glycolysis

2) Citric Acid Cycle

3) Gluconeogenesis

4) Glycogenesis

5) Glycogenolysis

6) Hexose monophosphate shunt

7) Uronic Acid Pathway

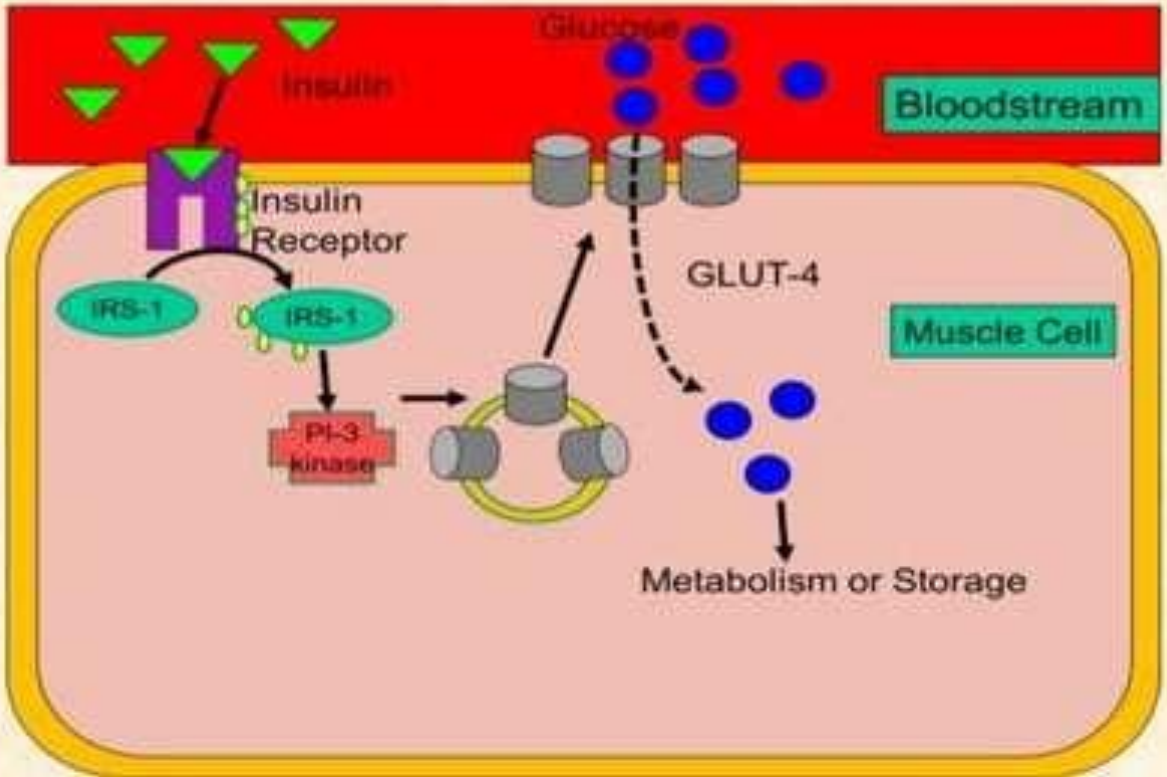
8) Galactose Metabolism

9) Fructose Metabolism

10) Amino sugar metabolism

Entry of Glucose into cells

1) Insulin-independent transport system of glucose: Not dependent on hormone insulin. This is operative in – hepatocytes, erythrocytes (GLUT-1) and brain.
2) Insulin-dependent transport system: Muscles and adipose tissue (GLUT-4).



Type 2 diabetes melitus:
-Due to reduction in the quantity of GLUT-4 in insulin deficiency.
-Insuin resistance is observed in tissues.

GLYCOLYSIS

EMBDEN-MEYERHOF PATHWAY

(OR)

E.M.PATHWAY

Definition:

Glycolysis is defined as the sequence of reactions converting glucose (or glycogen) to pyruvate or lactate, with the production of ATP

Salient features:

1) Takes place in all cells of the body.
2) Enzymes present in "cytosomal fraction" of the cell.
3) Lactate – end product – anaerobic condition.
4) Pyruvate(finally oxidized to CO2 & H2O) – end product of aerobic condition.
5) Tissues lacking mitochondria – major pathway – ATP synthesis.
6) Very essential for brain – dependent on glucose for energy.
7) Central metabolic pathway
8) Reversal of glycolysis – results in gluconeogenesis.

Reactions of Glycolysis

1) Energy Investment phase (or) priming phase

2) Splitting phase

3) Energy generation phase

Energy Investment Phase

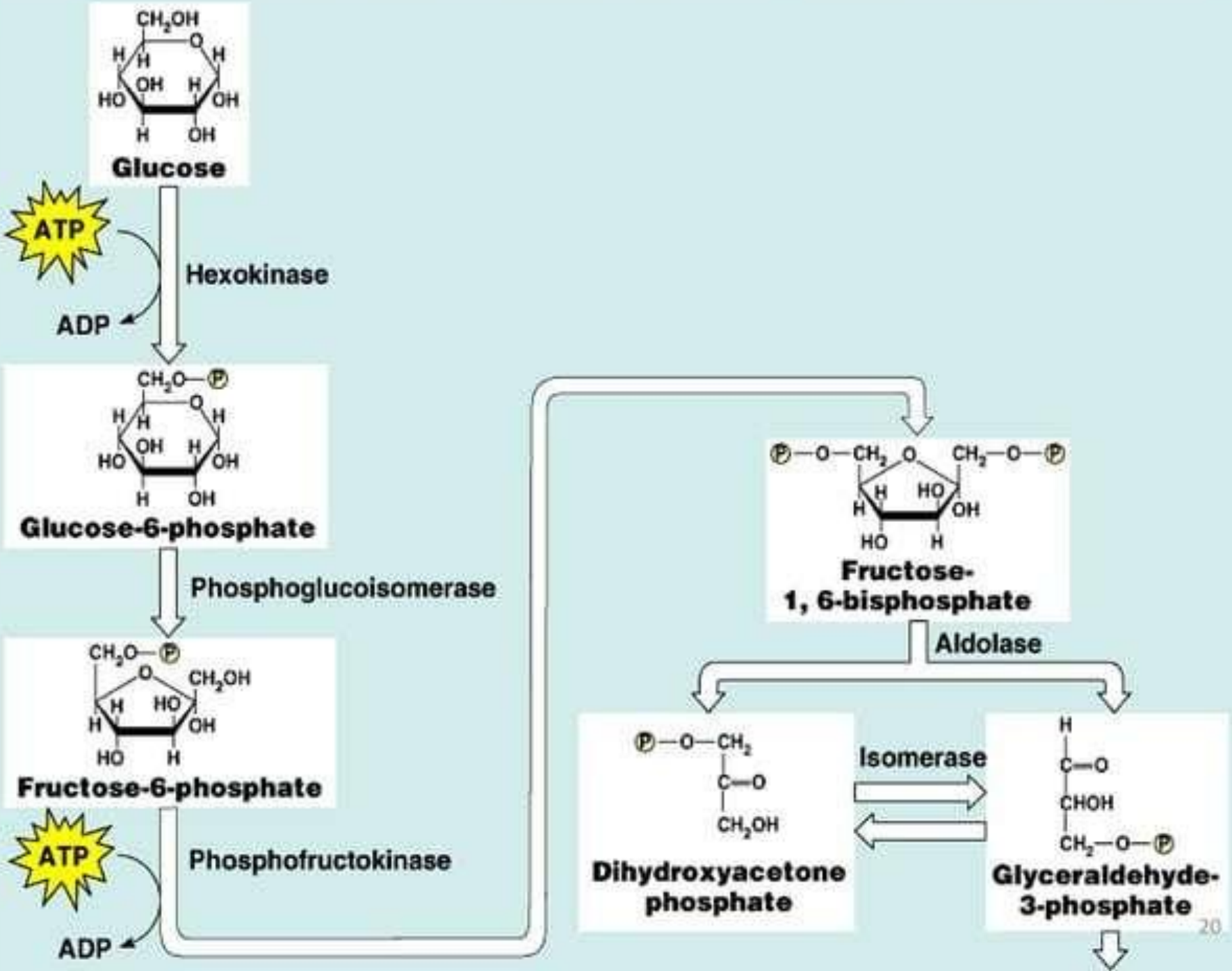
- **Glucose** is phosphorylated to **glucose-6-phosphate** by *hexokinase (or) glucokinase.*
- Glucose-6-phosphate undergoes isomerization to give **fructose -6- phosphate** in the presense of *phospho-hexose isomerase* and Mg^{2+}
- Fructose-6-phosphate is phoshorylated to **fructose 1,6-bisphosphate** by *phosphofructokinase.*

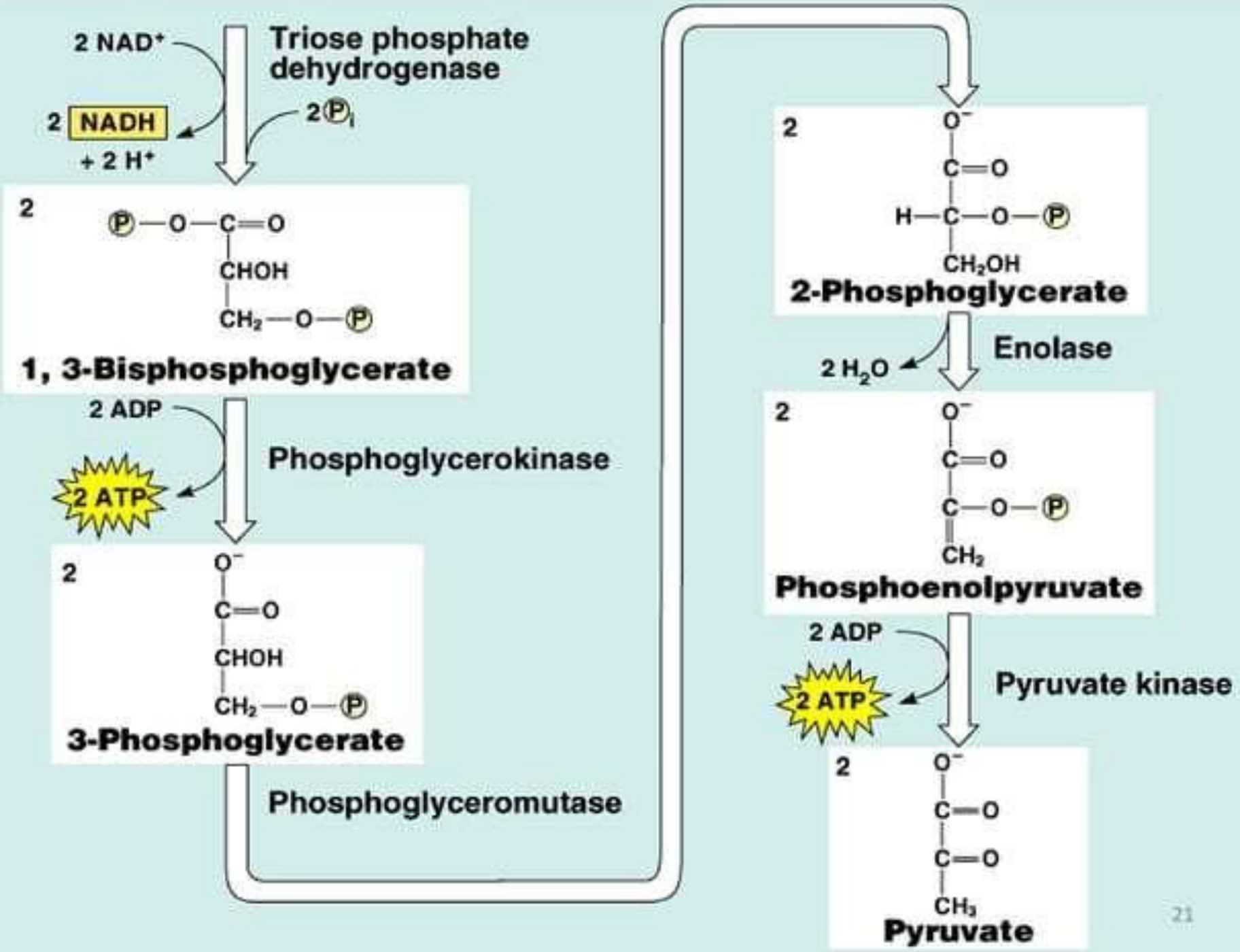
Splitting Phase

- Fructose 1,6-bisphosphate → **glyceraldehyde 3-phosphate** + **dihydroxyacetone phosphate.**(*aldolase enzyme*)
- **2 molecules** of glyceraldehyde 3-phosphate are obtained from 1 molecule of glucose

Energy Generation Phase

- Glyceraldehyde 3-phosphate → **1,3-bisphosphoglycerate**(*glyceraldehyde 3-phosphate hydrogenase)*
- 1,3-bisphosphoglycerate → **3-phosphoglycerate** (*phosphoglycerate kinase*)
- 3-phosphoglycerate → **2-phosphoglycerate** (*phosphoglycerate mutase*)
- 2-phosphoglycerate → **phosphoenol pyruvate** (*enolase* + Mg^{2+} & Mn^{2+})
- Phosphoenol pyruvate → **pyruvate [enol]** (*pyruvate kinase*) → **pyruvate [keto]** → **L-Lactate** *(lactate dehydrogenase)*


Glucose
ATP
ADP
Hexokinase
Glucose-6-phosphate
Phosphoglucoisomerase
Fructose-6-phosphate
ATP
ADP
Phosphofructokinase
Fructose-
1, 6-bisphosphate
Aldolase
Dihydroxyacetone
phosphate
Isomerase
Glyceraldehyde-
3-phosphate


2 NAD+
Triose phosphate dehydrogenase
2 NADH
+ 2 H+
2 Pi
2
1, 3-Bisphosphoglycerate
2 ADP
2 ATP
Phosphoglycerokinase
2
3-Phosphoglycerate
Phosphoglyceromutase
2
2-Phosphoglycerate
2 H2O
Enolase
2
Phosphoenolpyruvate
2 ADP
2 ATP
Pyruvate kinase
2
Pyruvate

Energy production of glycolysis:

ATP production = ATP produced - ATP utilized

	ATP produced	ATP utilized	Net energy
In absence of oxygen (anaerobic glycolysis)	4 ATP (Substrate level phosphorylation) 2ATP from 1,3 DPG. 2ATP from phosphoenol pyruvate	2ATP From glucose to glucose -6-p. From fructose -6-p to fructose 1,6 p.	2 ATP
In presence of oxygen (aerobic glycolysis)	4 ATP (substrate level phosphorylation) 2ATP from 1,3 BPG. 2ATP from phosphoenol pyruvate. + 4ATP or 6ATP (from oxidation of 2 NADH + H in mitochondria).	2ATP -From glucose to glucose -6-p. From fructose -6-p to fructose 1,6 p.	8 ATP / 6 ATP (Pyruvate dehydrogenase 2NADH,ETC, Oxidative phosphorylation)

CLINICAL ASPECT

1) Lactic acidosis

- Normal value – 4 to 15 mg/dl.
- Mild forms – strenous exercise, shock, respiratory diseases, cancers
- Severe forms – Impairment/collapse of circulatory system – myocardial infarction, pulmonary embolism, uncontrolled hemmorrhage and severe shock.

2) Cancer and glycolysis :

- Cancer cells – increased uptake of glucose and glycolysis.

- Blood vessels unable to supply adequate oxygen – HYPOXIC condition – Anaerobic glycolysis / hypoxic glycolysis – Involvement of Hypoxic inducible transcription factor (HIF).

- Treatment : Use drugs that inhibit vascularization of tumours

- ✓ Pasteur effect : Inhibition of glycolysis by oxygen (Phosphofructokinase) .

- ✓ Crabtree effect : The phenomenon of inhibition of oxygen consumption by the addition of glucose to tissues having high aerobic glycolysis.

RAPARPORT – LEUBERING CYCLE

- Supplementary pathway/ Shunt pathway to glycolysis .
- Erythrocytes
- Synthesis of 2,3-bisphosphoglycerate (2,3-BPG).
- Without the synthesis of ATP.
- Help to dissipate or waste the energy not needed by RBCs.
- Supply more oxygen to the tissues.

CITRIC ACID CYCLE

KREBS CYCLE / TRICARBOXYLIC ACID/ TCA CYCLE

Essentially involves the oxidation of acetyl CoA to CO_2 and H_2O.
This Cycle utilizes about two-third of total oxygen consumed by the body.

Brief History:

- Hans Adolf Krebs
- 1937
- Studies of oxygen consumptiom in pigeon breast muscle.

Location of TCA

- Mitochondrial matrix
- In close proximity to the electronic transport chain.

Overview

- 65-70% of the ATP is synthesized
- Name : TCA used because at the ouset of the cycle tricarboxylic acids participate.

Reactions of citric acid cycle

1) **Formation of citrate** : Condensation of acetyl CoA and oxaloacetate → catalysed by citrate synthase.

2) & 3) **Citrate is isomerized to isocitrate** → aconitase (two steps).

4) & 5) **Formation of ά-ketoglutarate** : enzyme isocitrate dehydrogenase.

6) **Conversion of ά-ketoglutarate to succinyl CoA** : through oxidative decarboxylation, catalysed by ά-ketoglutarate dehydrogenase complex.

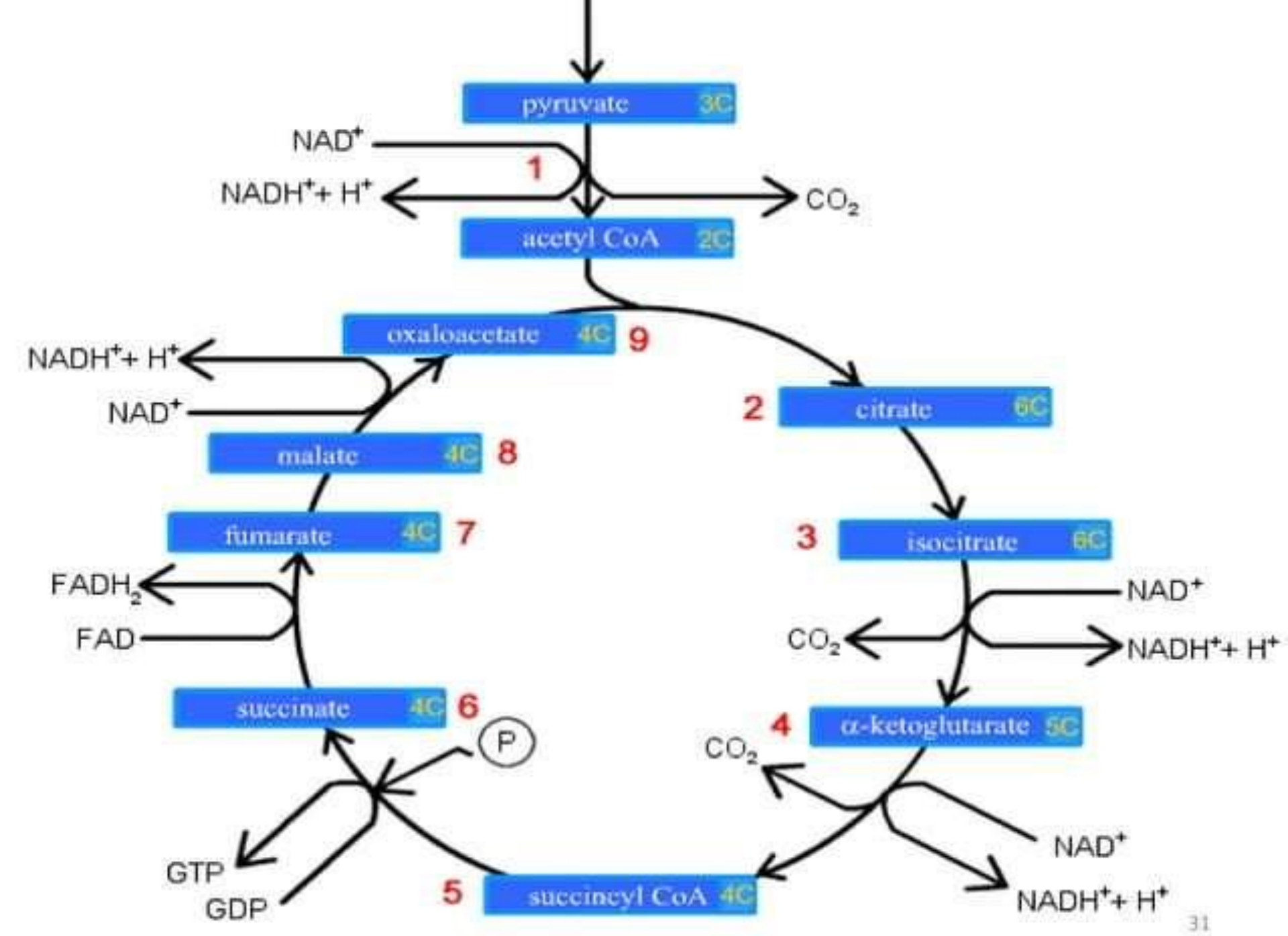
7) **Formation of succinate** : enzyme succinate thiokinase

GTP + ADP ←→ ATP + GDP (nucleoside diphosphate kinase)

8) **Conversion of succinate to fumarase** : enzyme succinate dehydrogenase

9) **Formation of malate** : enzyme fumarase

10) **Conversion of malate to oxaloacetate** : enzyme malate dehydrogenase.


pyruvate 3C
NAD+
1
NADH+ + H+
CO2
acetyl CoA 2C
oxaloacetate 4C
9
NADH+ + H+
NAD+
2
citrate 6C
malate 4C
8
fumarate 4C
7
3
isocitrate 6C
FADH2
FAD
NAD+
CO2
NADH+ + H+
succinate 4C
6
P
4
α-ketoglutarate 5C
CO2
GTP
GDP
5
succinyl CoA 4C
NAD+
NADH+ + H+

- TCA cycle is strictly **aerobic** in contrast to glycolysis.
- Total of **12 ATP** are produced from one acetyl CoA :-

✓ During the process of oxidation of acetyl CoA via citric acid cycle → 3 NADH & 1 FADH2.

✓ Oxidation of 3 NADH by electron transport chain coupled with oxidative phosphorylation results in 9 ATP, FADH2 → 2 ATP.

✓ One substrate level phosphorylation.

APPLIED ASPECTS OF TCA CYCLE

Mitochondrial encephalopathy occurs due to fumarase deficiency .

It is a mitochondrial myopathy affecting both the skeletal muscles and brain .

GLUCONEOGENESIS

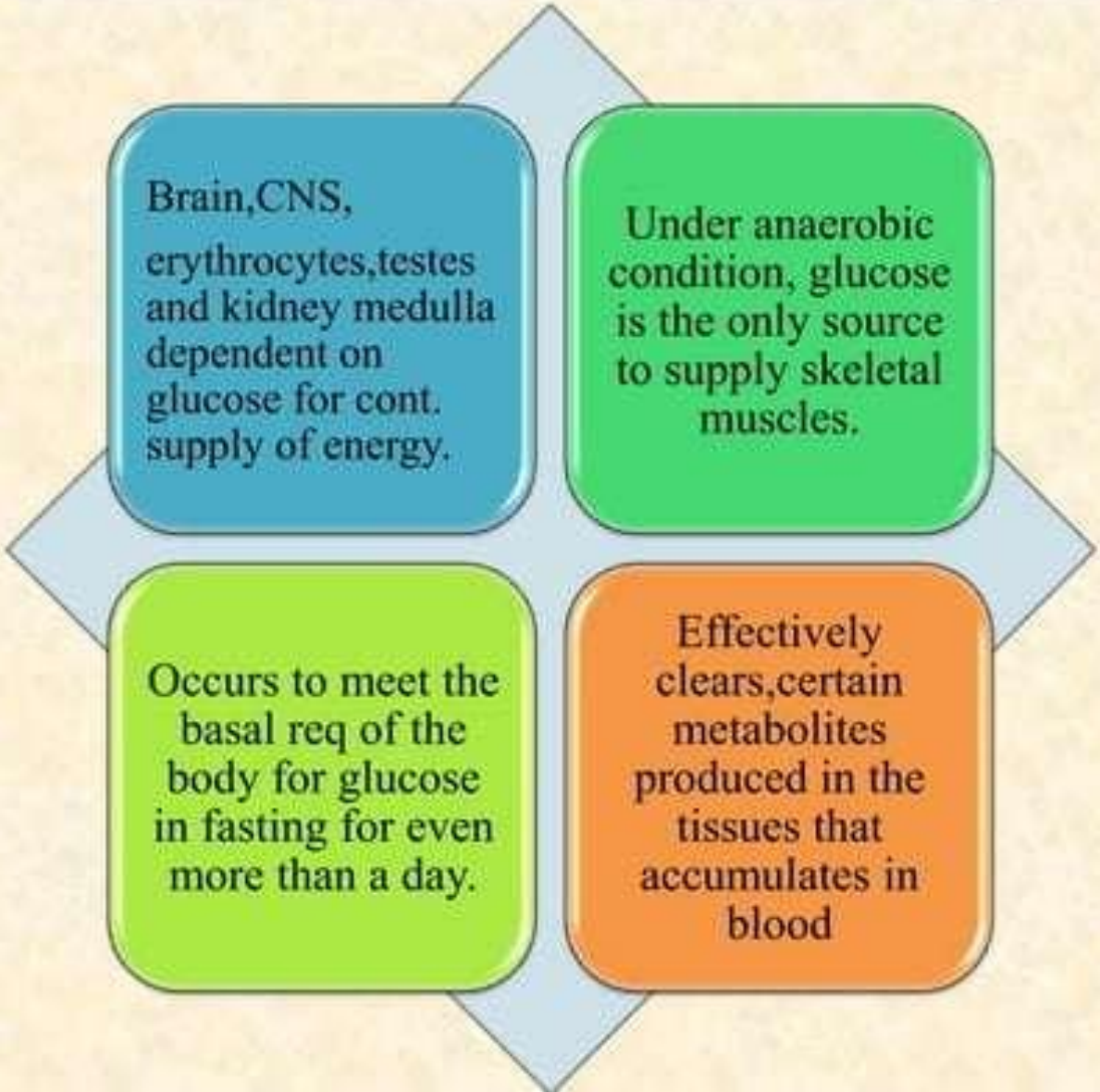
The synthesis of glucose from non-carbohydrate compounds is known as gluconeogenesis.

Major substrate/precursors : lactate, pyruvate, glycogenic amino acids, propionate & glycerol.

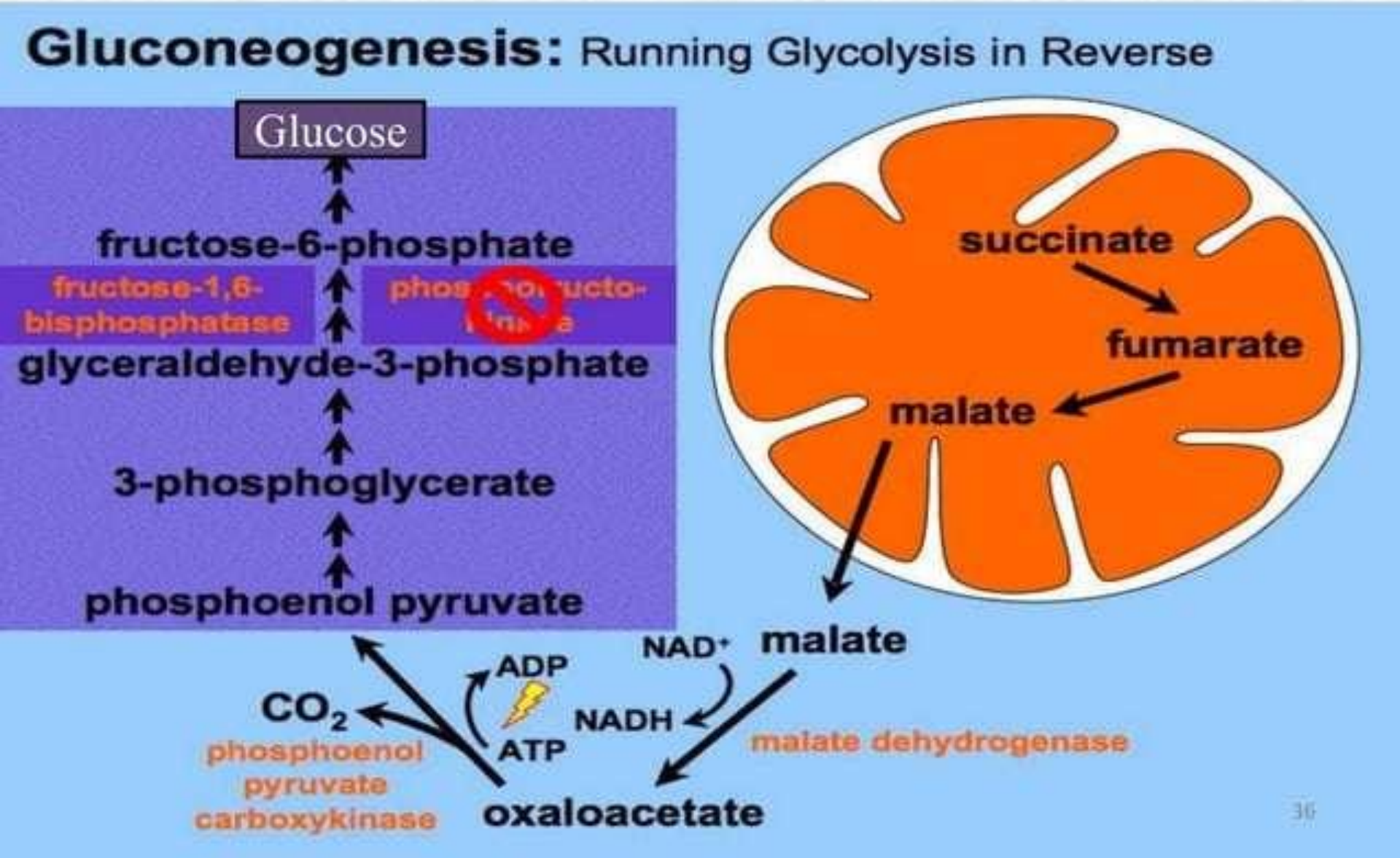
-Takes place in liver (1kg glucose) ; kidney matrix(1/3rd).

- Occurs in cytosol and some produced in mitochondria.

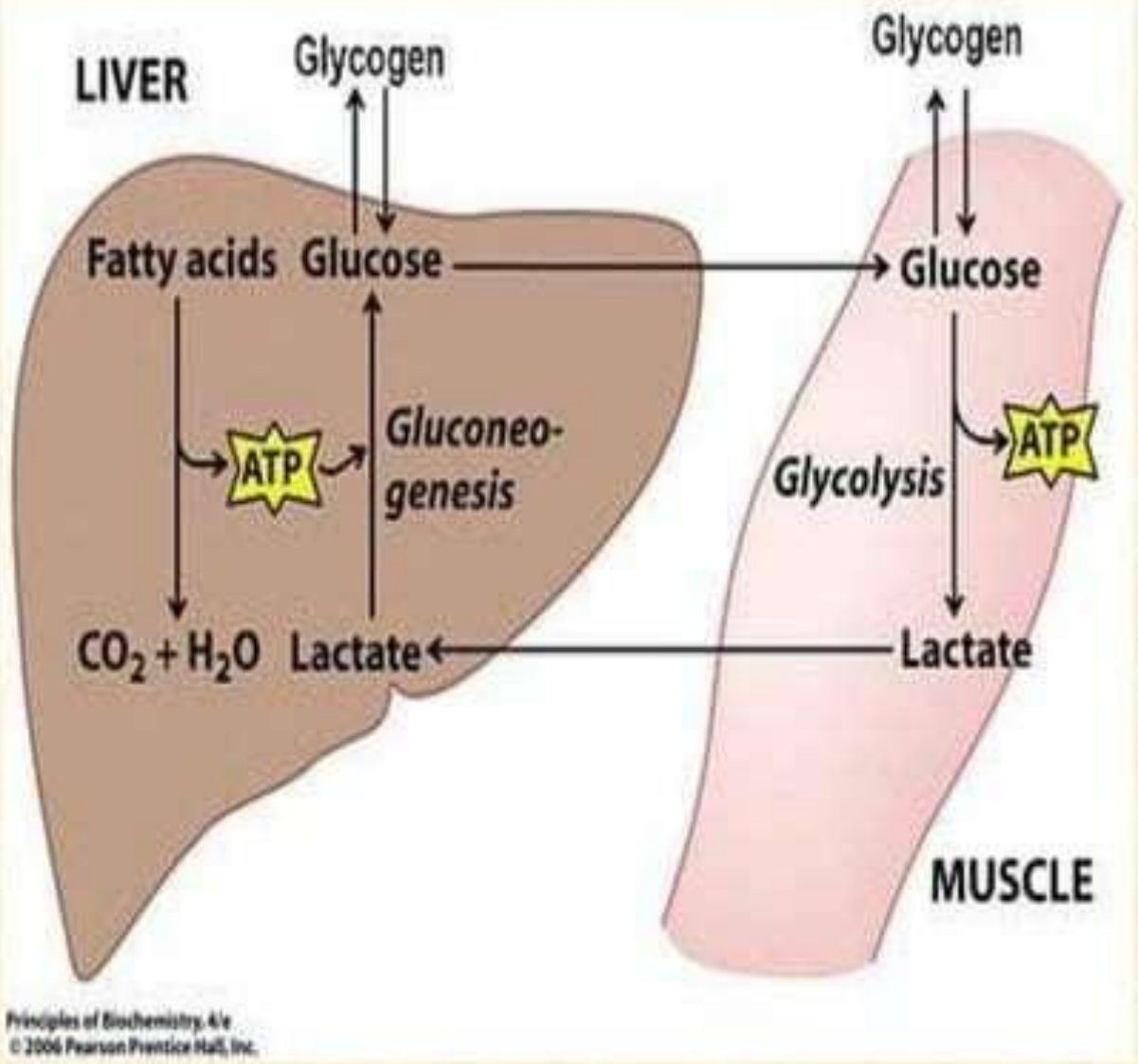
Importance of Gluconeogenesis



Reaction of Gluconeogenesis

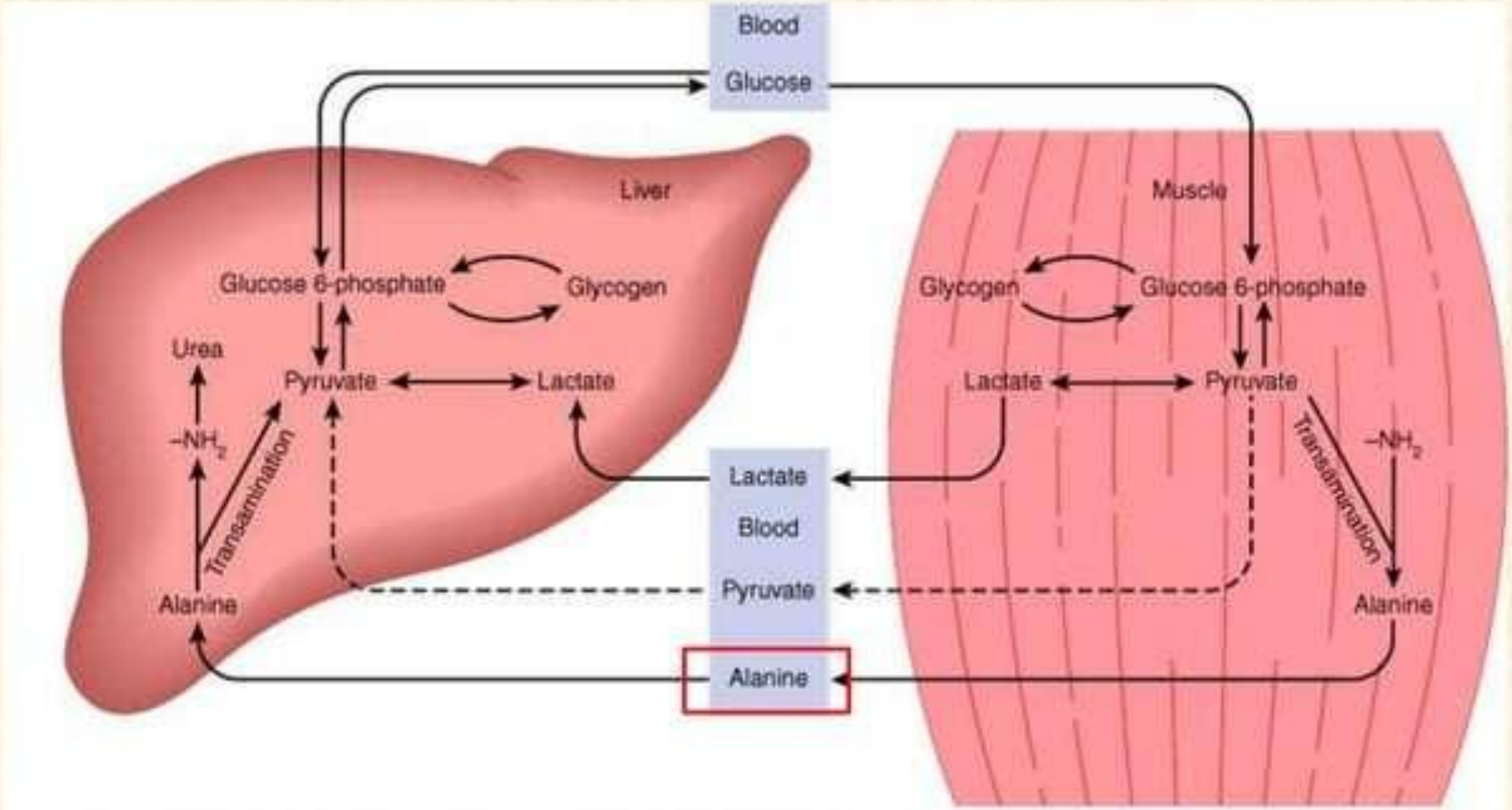


Cori Cycle



The cycle involveing the **synthesis of glucose in liver from the skeletal muscle lactate** and the reuse of glucose thus synthesized by the muscle for energy purpose is known as Cori cycle.

Glucose-Alanine Cycle



Source: Murray RK, Bender DA, Botham KM, Kennelly PJ, Rodwell VW, Weil PA: *Harper's Illustrated Biochemistry, 28th Edition*: http://www.accessmedicine.com

Clinical Aspects

* **Glucagon** stimulates gluconeogenesis:

1)Active pyruvate kinase converted to inactive form

2)Reduces the concentration of fructose 2,6-bisphosphate.

* **Glycogenic amino acids** have stimulating influence on gluconeogenesis.

* **Diabetes mellitus** where amino acids are mobilized from muscle protein for the purpose of gluconeogenesis.

Acetyl CoA promotes gluconeogenesis:

* **During starvation** – due to excessive lipolysis in adipose tissue –acetyl CoA accumulates in the liver.

* Acetyl CoA allosterically activates pyruvate carboxylase resulting in enhanced glucose production

* **Alcohol** inhibits gluconeogenesis

GLYCOGEN METABOLISM

- ✓Glycogen is a storage form of glucose in animals.
- ✓Stored mostly in liver (6-8%) and muscle (1-2%)
- ✓Due to muscle mass the quantity of glycogen in muscle = 250g and liver =75g
- ✓Stored as granules in the cytosol.
- ✓Functions : Liver glycogen – maintain the blood glucose level
  Muscle glycogen – serves as fuel reserve

GLYCOGENESIS

- Synthesis of glycogen from glucose.
- Takes place in cytosol.
- Requires UTP and ATP besides glucose.
- Steps in synthesis :

1) Synthesis of UDP- glucose
2) Requirement of primer to initiate glycogenesis
3) Glycogen synthesis by glycogen synthase
4) Formation of branches in glycogen

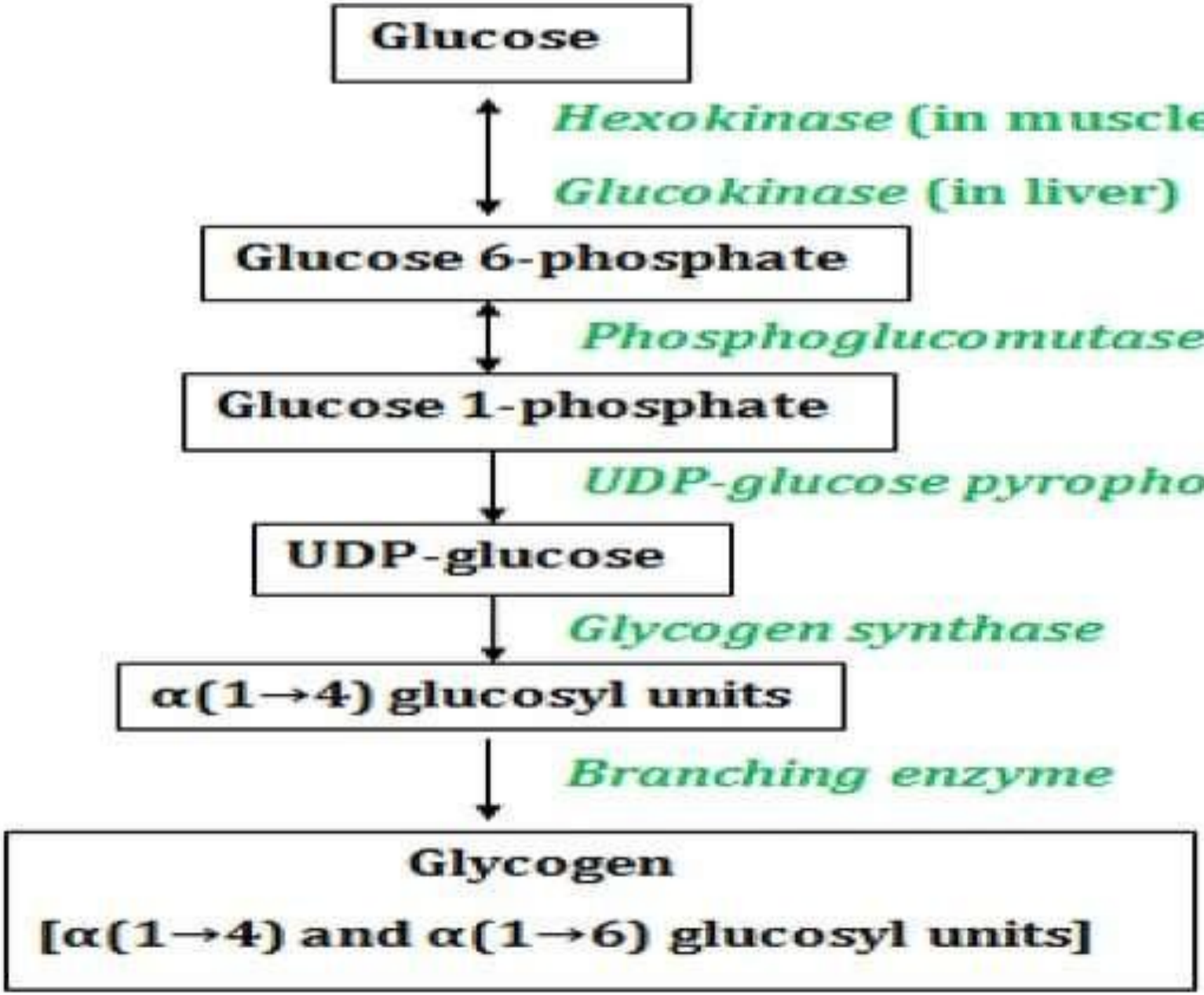


Diagram: Steps of glycogenesis

GLYCOGENOLYSIS

- Degradation of stored glycogen in liver and muscle constitutes glycogenolysis.

- Irreversible pathway takes place in cytosol.

- Hormonal effect on glycogen metabolism :

1) Elevated glucagon – increases glycogen degradation
2) Elevated insulin – increases glycogen synthesis

- Degraded by breaking majorly α-1,4- and α-1,6-glycosidic bonds.

- Steps in glycogenolysis:

1) Action of glycogen phosphorylase
2) Action of debranching enzyme
3) Formation of glucose-6-phosphate and glucose

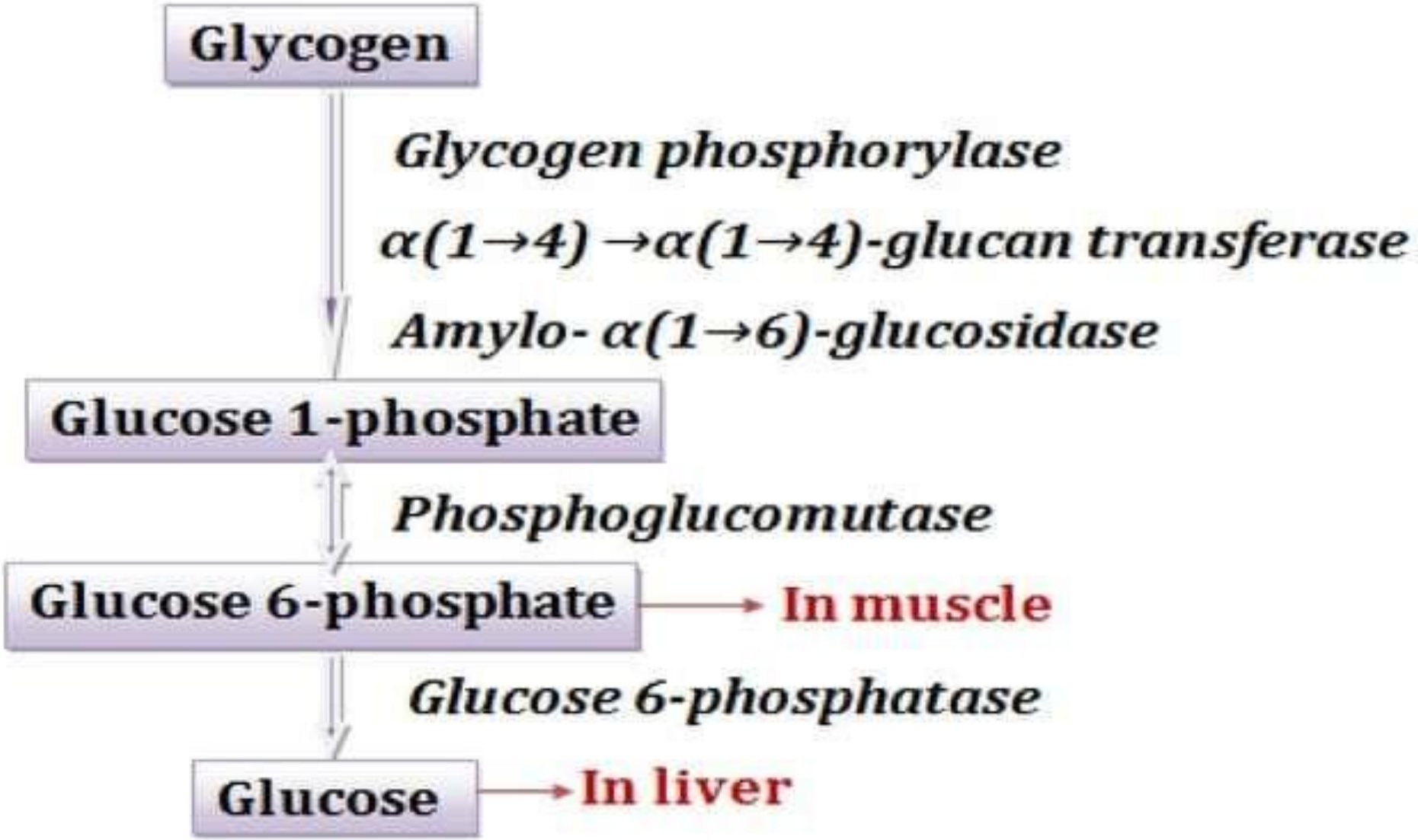
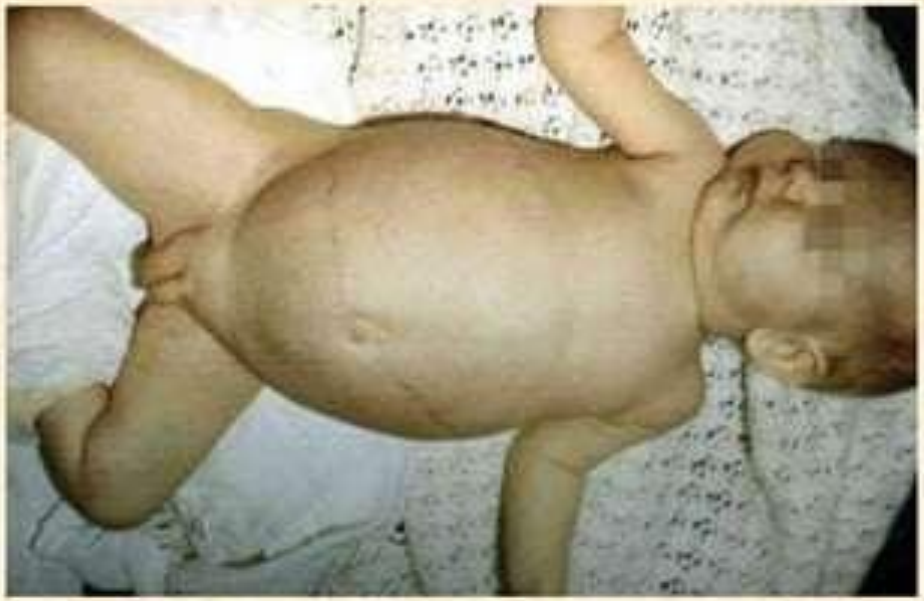


Diagram: Steps of glycogenolysis

GLYCOGEN STORAGE DISEASES

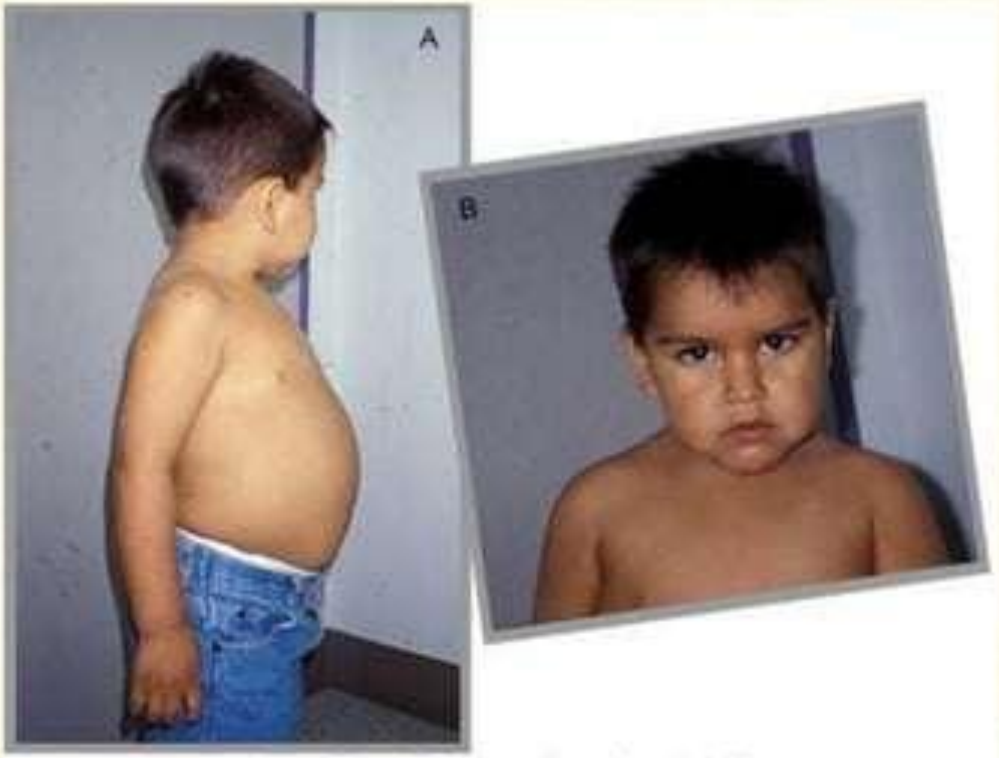
TYPE	ENZYME DEFECT	CLINICAL FEATURES
Type I (Von Gierke's disease)	Glucose-6-phosphatase deficiency.	Hypoglycemia, enlarged liver and kidneys, gastro-intestinal symptoms, Nose bleed, short stature, gout
Type II (Pompe's disease)	Acid maltase deficiency	Diminished muscle tone, heart failure, enlarged tongue
Type III (Cori's disease,Forbe disease)	Debranching enzyme deficiency	Hypoglycemia, enlarged liver, cirrhosis, muscle weakness, cardiac involvement
Type IV (Andersen's disease)	Branching enzyme deficiency	Enlarged liver & spleen, cirrhosis, diminished muscle tone, possible nervous system involvement
Type V (Mcardle's disease)	Muscle phosphorylase deficiency	Muscle weakness, fatigue and muscle cramps

TYPE	ENZYME DEFECT	CLINICAL FEATURES
Type VI (Her's disease)	Liver phosphorylase deficiency	Mild hypoglycemia, enlarged liver, short stature in childhood
Type VII (Tarui's disease)	Phosphofructokinase deficiency	Muscle pain, weakness and decreased endurance
Type VIII	Liver phosphorylase kinase	Mild hypoglycemia, enlarged liver, short stature in childhood, possible muscle weakness and cramps
Type 0	Liver glycogen synthetase	Hypoglycemia, possible liver enlargement



Von Gierke's disease)

Pompe's disease



Pictures 7A and 7B - Glycogen storage disease type III

Cori's disease, Forbe disease

HEXOSE MONOPHOSPHATE SHUNT

HMP Shunt/ Pentose Phosphate Pathway/
Phosphogluconate Pathway

* This is an alternative pathway to glycolysis and TCA cycle for the oxidation of glucose.

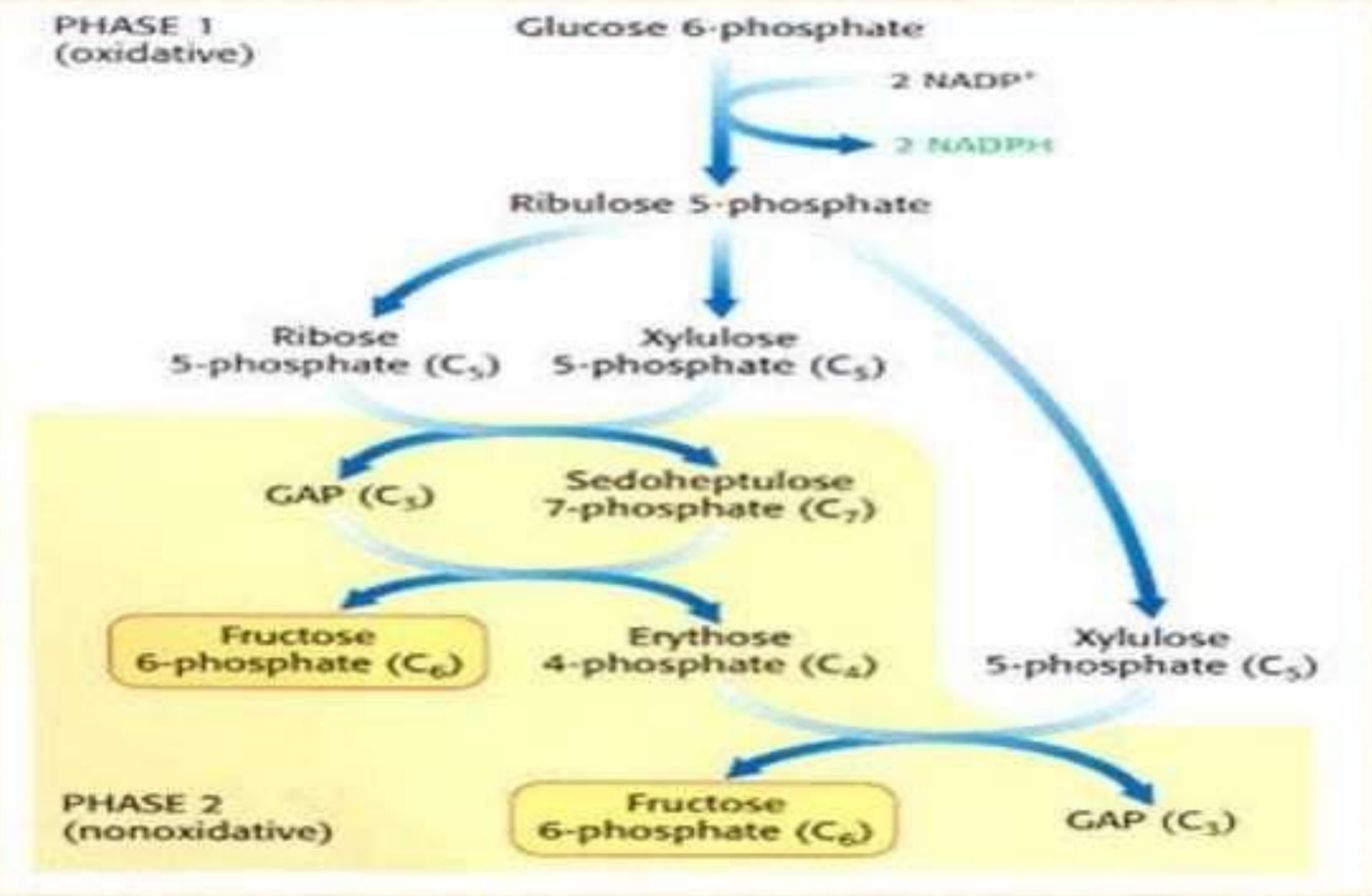
* Anabolic in nature, since it is concerned with the biosynthesis of NADPH and pentoses.

* Unique multifunctional pathway

* Enzymes located – cytosol

* Tissues active – liver, adipose tissue, adrenal gland, erythrocytes, testes and lactating mammary gland.

Reactions of the HMP Shunt Pathway



Significance of HMP Shunt

- Pentose or its derivatives are useful for the synthesis of nucleic acids and nucleotides.

- NADPH is required :
  - For reductive biosynthesis of fatty acids and steroids.
  - For the synthesis of certain amino acids.
  - Anti-oxidant reaction
  - Hydroxylation reaction– detoxification of drugs.
  - Phagocytosis
  - Preserve the integrity of RBC membrane.

Clinical Aspects

- Glucose-6-Phosphate dehydrogenase deficiency :
  - Inherited sex-linked trait
  - Red blood cells
  - Impaired synthesis of NADPH
  - hemolysis , developing hemolytic anemia

✓ Resistance towards malaria [Africans]

Clinical Aspects

- Wernicke-Korsakoff syndrome :
  - Genetic disorder
  - Alteration in transketolase activity
  - Symptoms : mental disorder, loss of memory, partial paralysis

- Pernicious anemia : transketolase activity increases.

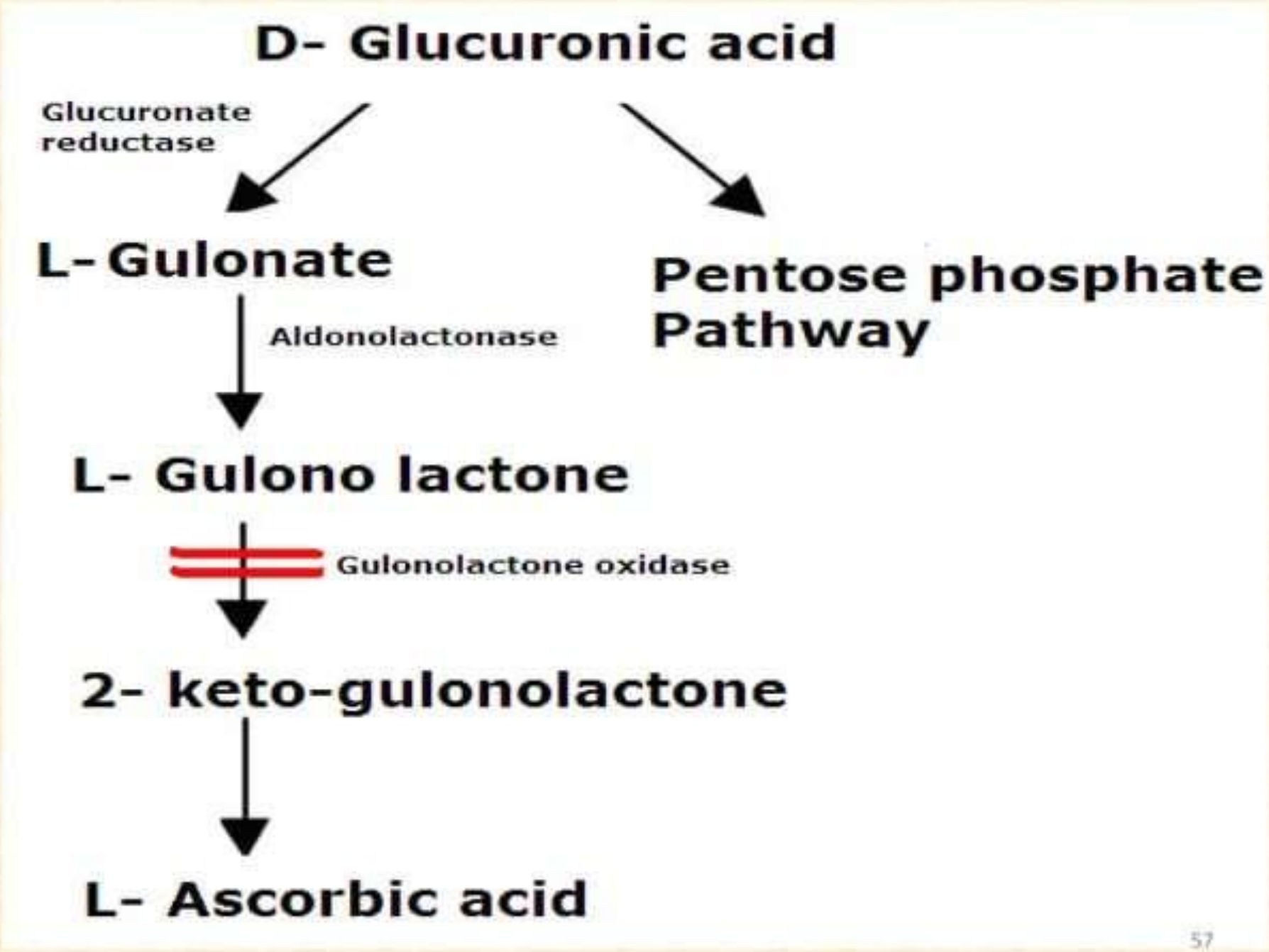
URONIC ACID PATHWAY

(OR)

GLUCORONIC ACIS PATHWAY

- ✓ Alternative oxidative pathway for glucose.
- ✓ synthesis of glucorinc acid,pentoses and vitamin (ascorbic acid).
- ✓ Normal carbohydrate metabolism ,phosphate esters are involved – but in uronic acid pathway free sugars and sugar acids are involved.
- ✓ Steps of reactions :

1) Formation of UDP-glucoronate
2) Conversion of UDP- glucoronate to L-gulonate
3) Synthesis of ascorbic acid in some animals
4) Oxidation of L-gulonate


D- Glucuronic acid
Glucuronate reductase
L- Gulonate
Pentose phosphate Pathway
Aldonolactonase
L- Gulono lactone
Gulonolactone oxidase
2- keto-gulonolactone
L- Ascorbic acid

Clinical Aspects

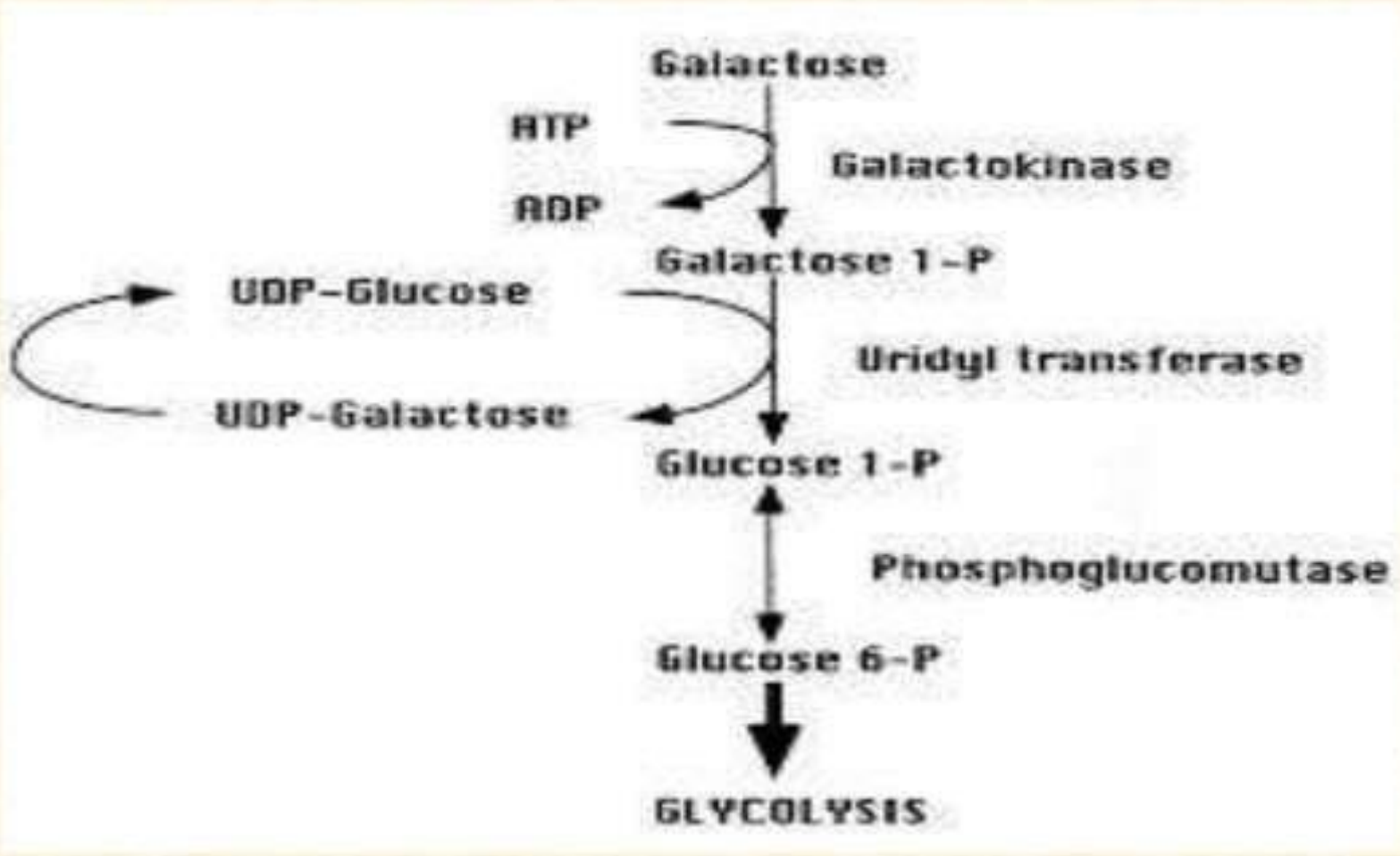
- Effects of drugs : increases the pathway to achieve more synthesis of glucaronate from glucose .

  - barbital,chloro-butanol etc.

- Essential pentosuria : deficiency of xylitol-dehydrogenase

- Rare genetic disorder
- Asymptomatic
- Excrete large amount of L-xylulose in urine
- No ill-effects

METABOLISM OF GALACTOSE



✓ Disaccharide lactose present in milk – principle source of of galactose.

✓ Lactase of intestinal mucosal cells hydrolyses lactose to galactose and glucose.

✓ Within cell galactose is produced by lysosomal degradation of glycoproteins and glycolipids.

✓ CLINICAL ASPECTS :

- **Classical galactosemia** : deficiency of galactose-1-phosphate uridyltransferase. Increase in galactose level.

- **Galactokinase deficiency** : Responsible for galactosemia and galactosuria.

- **Clinical symptoms** : loss of weight in infants, hepatosplenomegaly, jaundice, mental retardation , cataract etc.

- **Treatment** : removal of galactose and lactose from diet.

METABOLISM OF FRUCTOSE

Sorbitol/Polyol Pathway:

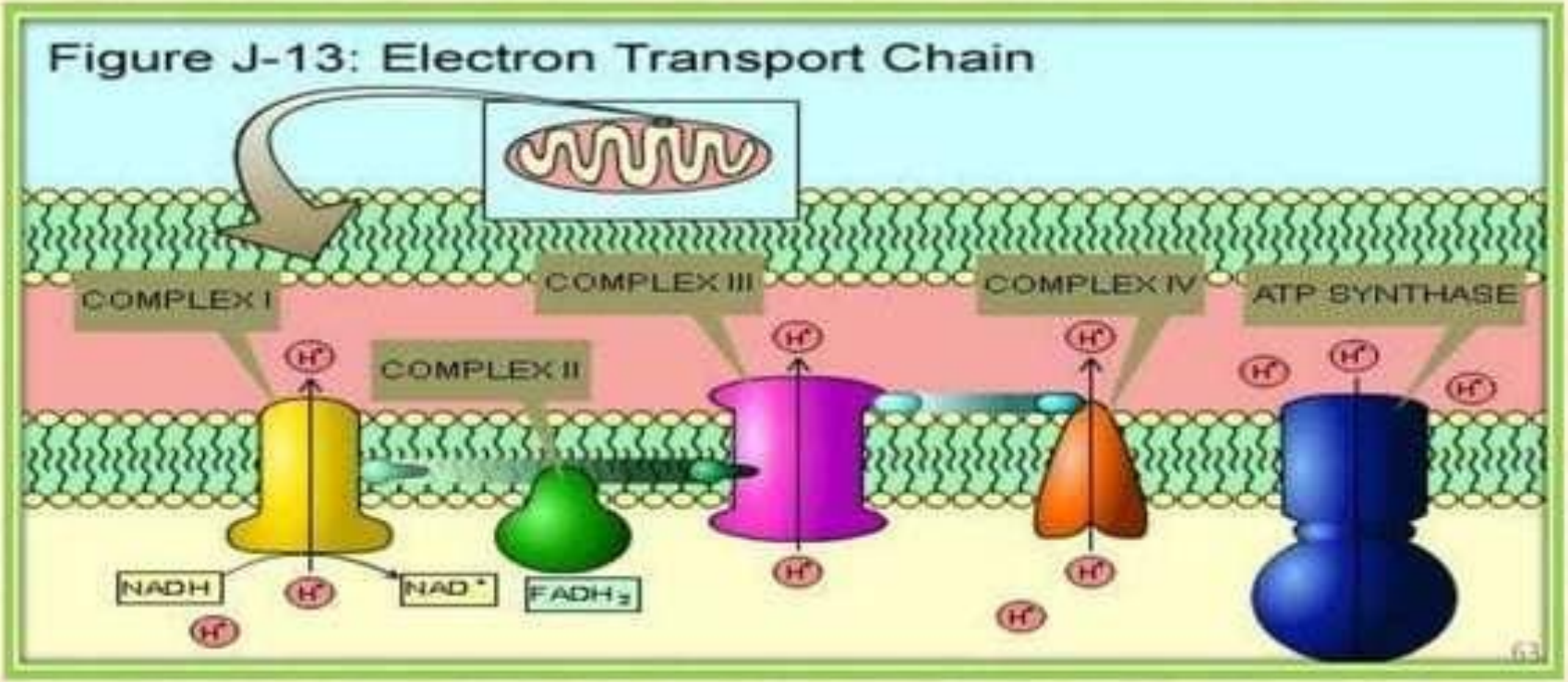
- ✓ Conversion of glucose to fructose via sorbitol.
- ✓ Glucose to Sorbitol reduction by enzyme aldolase (NADPH).
- ✓ Sorbitol is then oxidized to fructose by sorbitol dehydrogenase and NAD^+.
- ✓ Fructose is preferred carbohydrate for energy needs of sperm cells due to the presence of sorbitol pathway.
- ✓Pathway is absent in liver.
- ✓Directly related to glucose : higher in uncontrolled diabetes.

METABOLISM OF AMINO SUGARS

- When the hydroxyl group of the sugar is replaced by the amino group, the resultant compound is an amino sugar.
- Eg. Glucosamine,galactosamine,mannosamine,sialic acid etc.
- Essential components of glycoproteins, glycosaminoglycans, glycolipids.
- Found in some antibiotics.
- 20% of glucose utilized for the synthesis of amino sugars – connective tissues.

ELECTRON TRANSPORT CHAIN REACTIONS

- Electron transport chain is a series of protein complexes located in the inner membrane of mitochondria .



POLYSACCHARIDES
&
CLINICAL ASPECTS

Proteoglycans & Glycosaminoglycans

✓ Seven glycosaminoglycans :

1) Hyaluronic acid
2) Chondriotin sulfate
3) Keratan sulfate I
4) Keratan sulfate II
5) Heparin
6) Heparan sulfate
7) Dermatan sulfate

Functions of glycoaminoglycans

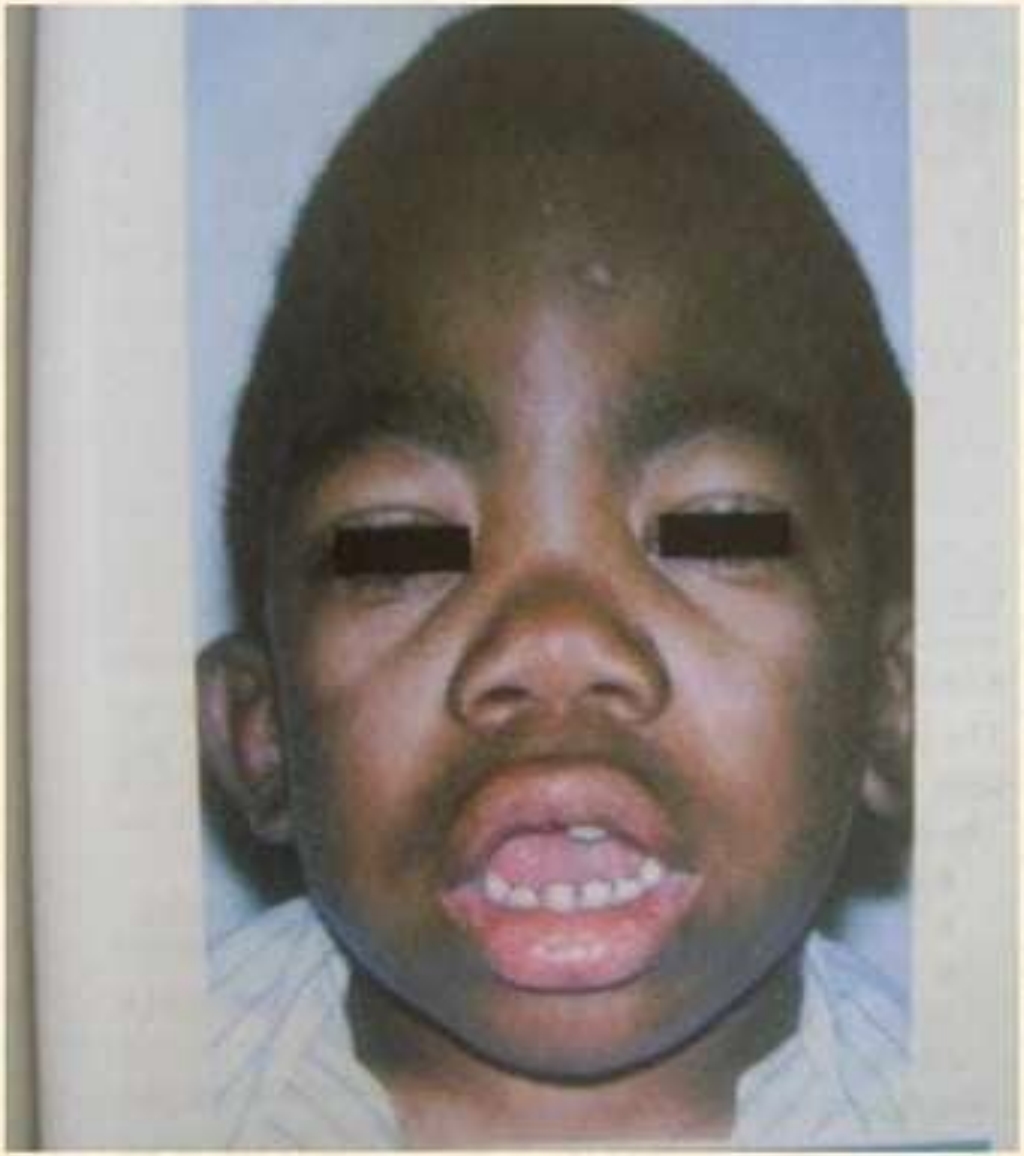
- Structural components of extracellular matrix.
- Act as sieves in extracellular matrix.
- Facilitate cell migration.
- Corneal transparency.
- Anticoagulant (Heparin).
- Components of synaptic & other vesicles.

Mucopolysaccharidoses

MPS	Defect	Symptoms
MPS I (Hurler syndrome)	Alpha-L-Iduronidase	Mental retardation, micrognathia, coarse facial features, macroglossia, retinal degeneration, corneal clouding, cardiomyopathy, hepatosplenomegaly
MPS II (Hunter syndrome)	Iduronate sulfatase	Mental retardation (similar, but milder, symptoms to MPS I). This type exceptionally has X-linked recessive inheritance
MPS IIIA (Sanfilippo A)	Heparan sulfate N sulfatase	Developmental delay, severe hyperactivity, spasticity, motor dysfunction, death by the second decade
MPS IIIB (Sanfilippo B)	Alpha-Acetylglucosaminidase	
MPS IIIC (Sanfilippo C)	Acetyl transferase	

MPS	Defect	Symptoms
MPS IVA (Morquio A)	Galactose-6-sulfatase	Severe skeletal dysplasia, short stature, motor dysfunction
MPS IVB (Morquio B)	Beta galactosidase	
MPS VI (Maroteaux Lamy syndrome)	N acetylgalactosamine 4 sulfatase	Severe skeletal dysplasia, short stature, motor dysfunction, kyphosis, heart defects
MPS VII (Sly)	Beta glucoronidase	Hepatomegaly, skeletal dysplasia, short stature, corneal clouding, developmental delay
MPS IX (Natowicz syndrome)	Hyaluronidase deficiency	Nodular soft-tissue masses around joints, episodes of painful swelling of the masses, short-term pain, mild facial changes, short stature, normal joint movement, normal intelligence

Hunter's syndrome



- Short and broad mandible
- Localized radiolucent lesions of the jaw
- Flattened temporomandibular joints
- Macroglossia
- Conical peg-shaped teeth with generalized wide spacing
- Highly arched palated with flattened alveolar ridges
- Hyperplastic gingiva

ROLE OF HORMONES IN CARBOHYDRATE METABOLISM

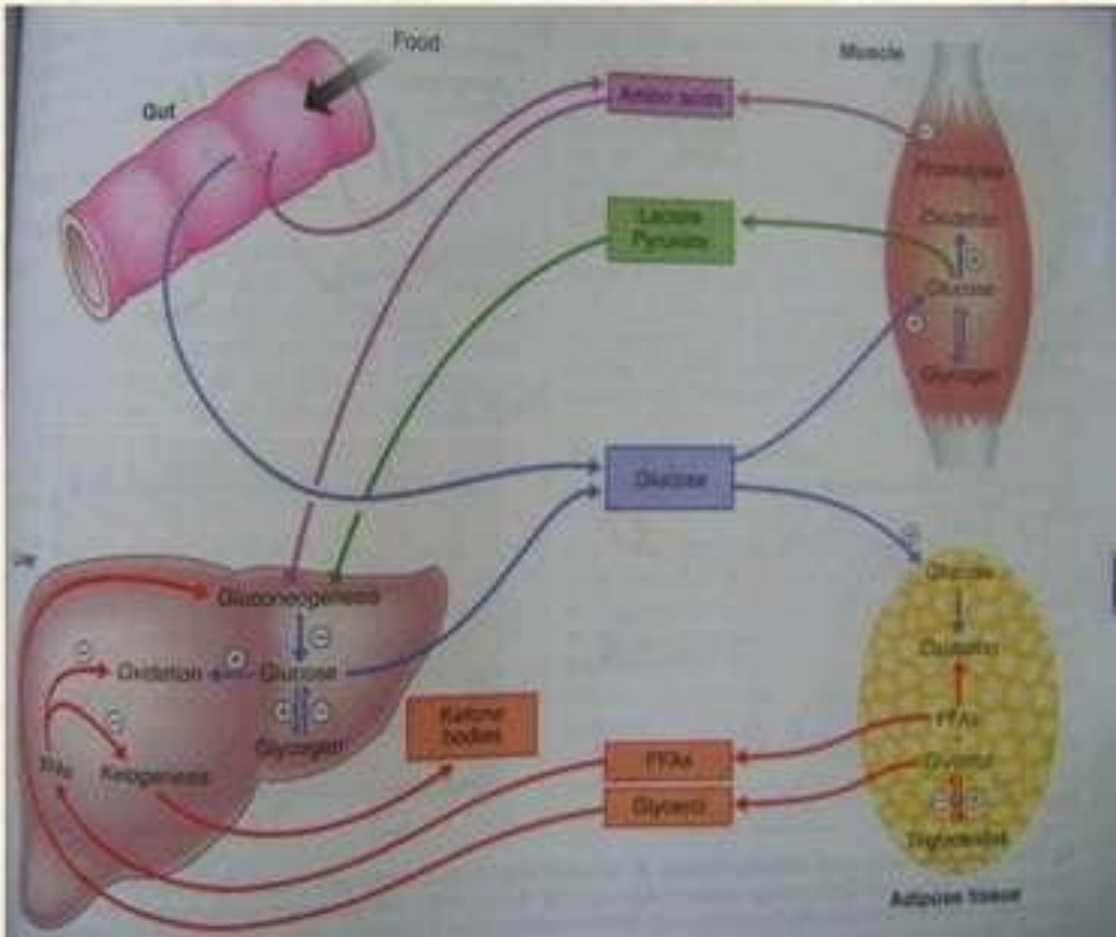
Regulation of Blood glucose

- Postabsorptive state: Blood glucose is 4.5-5.5mmol/L.
- After carbohydrate meal: 6.5-7.2mmol/L
- During fasting : 3.3-3.9mmol/L

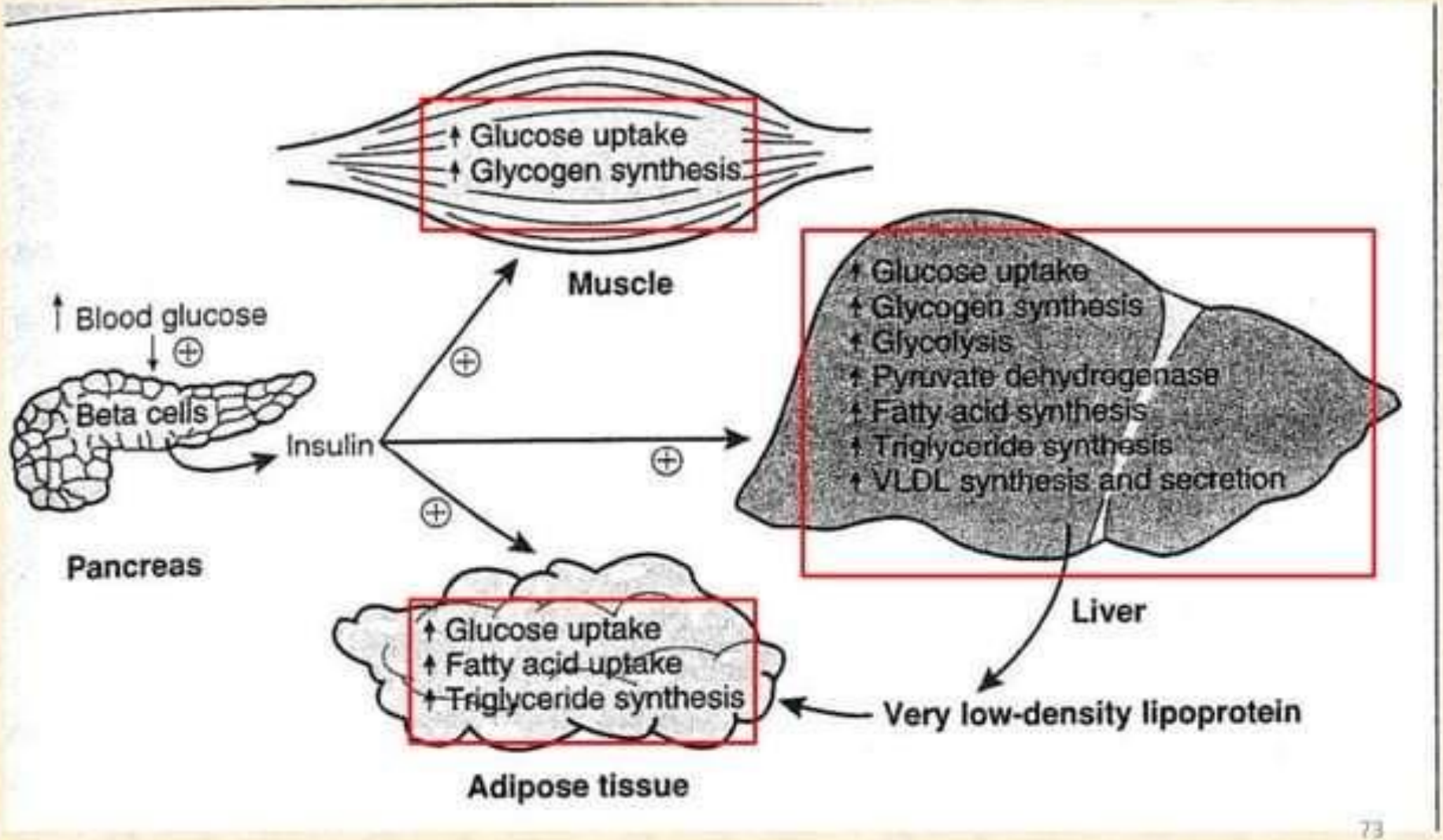
Metabolic & hormonal mechanisms regulate blood glucose level

Maintenance of stable levels of glucose in blood is by

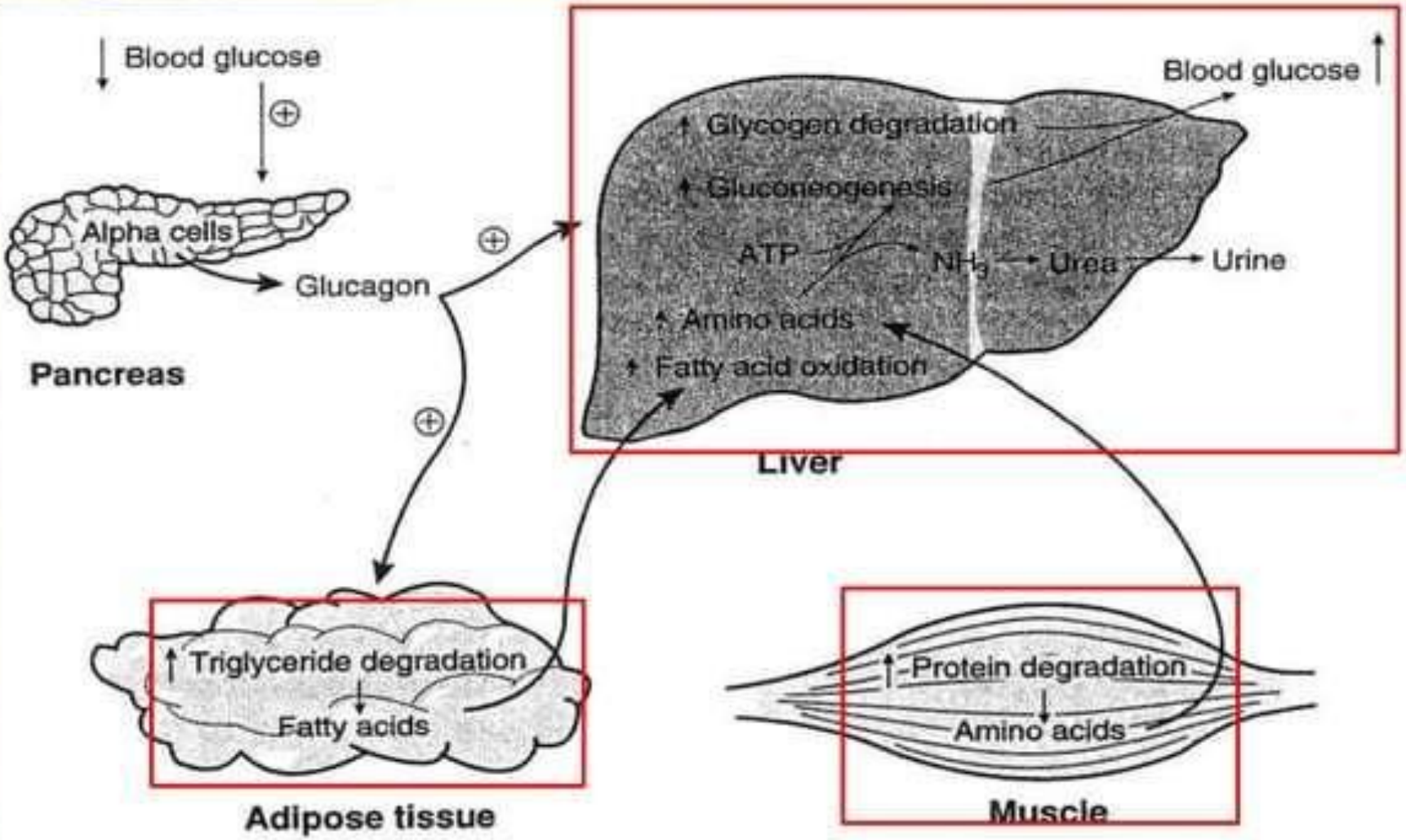
- ✓ Liver.
- ✓ Extrahepatic tissues.
- ✓ Hormones .



Regulation of blood glucose levels
Insulin



Role of glucagon



Role of thyroid hormone

- ✓ It stimulates glycogenolysis & gluconeogenesis.

Hypothyroid

- ➢ Fasting blood glucose is lowered.
- ➢ Patients have decreased ability to utilise glucose.
- ➢ Patients are less sensitive to insulin than normal or hyperthyroid patients.

Hyperthyroid

- ➢ Fasting blood glucose is elevated
- ➢ Patients utilise glucose at normal or increased rate

Glucocorticoids

- ✓ Glucocorticoids are antagonistic to insulin.
- ✓ Inhibit the utilisation of glucose in extrahepatic tissues.
- ✓ Increased gluconeogenesis .

Epinephrine

- ✓ Secreted by adrenal medulla.
- ✓ It stimulates glycogenolysis in liver & muscle.
- ✓ It diminishes the release of insulin from pancreas.

Other Hormones

- Anterior pituitary hormones

Growth hormone:

- ✓ Elevates blood glucose level & antagonizes action of insulin.
- ✓ Growth hormone is stimulated by hypoglycemia (decreases glucose uptake in tissues)
- ✓ Chronic administration of growth hormone leads to diabetes due to B cell exhaustion.

SEX HORMONES

- ✓Estrogens cause increased liberation of insulin.
- ✓Testosterone decrease blood sugar level.

Hyperglycemia

- Thirst, dry mouth
- Polyuria
- Tiredness, fatigue
- Blurring of vision.
- Nausea, headache,
- Hyperphagia
- Mood change

Hypoglycemia

- Sweating
- Trembling,pounding heart
- Anxiety, hunger
- Confusion, drowsiness
- Speech difficulty
- Incoordination.
- Inability to concentrate

Clinical aspects

- ✓ Glycosuria: occurs when venous blood glucose concentration exceeds 9.5-10.0mmol/L

- ✓ Fructose-1,6-Biphosphatase deficiency causes lactic acidosis & hypoglycemia..

Diabetes Mellitus

A multi-organ catabolic response caused by insulin insufficiency

Muscle

- Protein catabolism for gluconeogenesis

Adipose tissue

- Lipolysis for fatty acid release

Liver

- Ketogenesis from fatty acid oxidation
- Gluconeogenesis from amino acids and glycerol

Kidney

- Ketonuria and cation excretion
- Renal ammoniagenesis.

DENTAL ASPECTS OF CARBOHYDRATES METABOLISM

Role of carbohydrates in dental caries

- Fermentable carbohydrates causes loss of caries resistance.
- Caries process is an interplay between oral bacteria, local carbohydrates & tooth surface

Bacteria + Sugars+ Teeth ⟶ Organic acids

↓

Caries

Role of carbohydrates in periodontal disease

Abnormal glucose metabolism

↓

Diabetes Mellitus

↓

Periodontal disease

Excessive carbohydrate intake

↓

Obesity

↓

Periodontal disease

RECENT CLINICAL ISSUES RELATED TO CARBOHYDRATES METABOLISM

Cystic Fibrosis

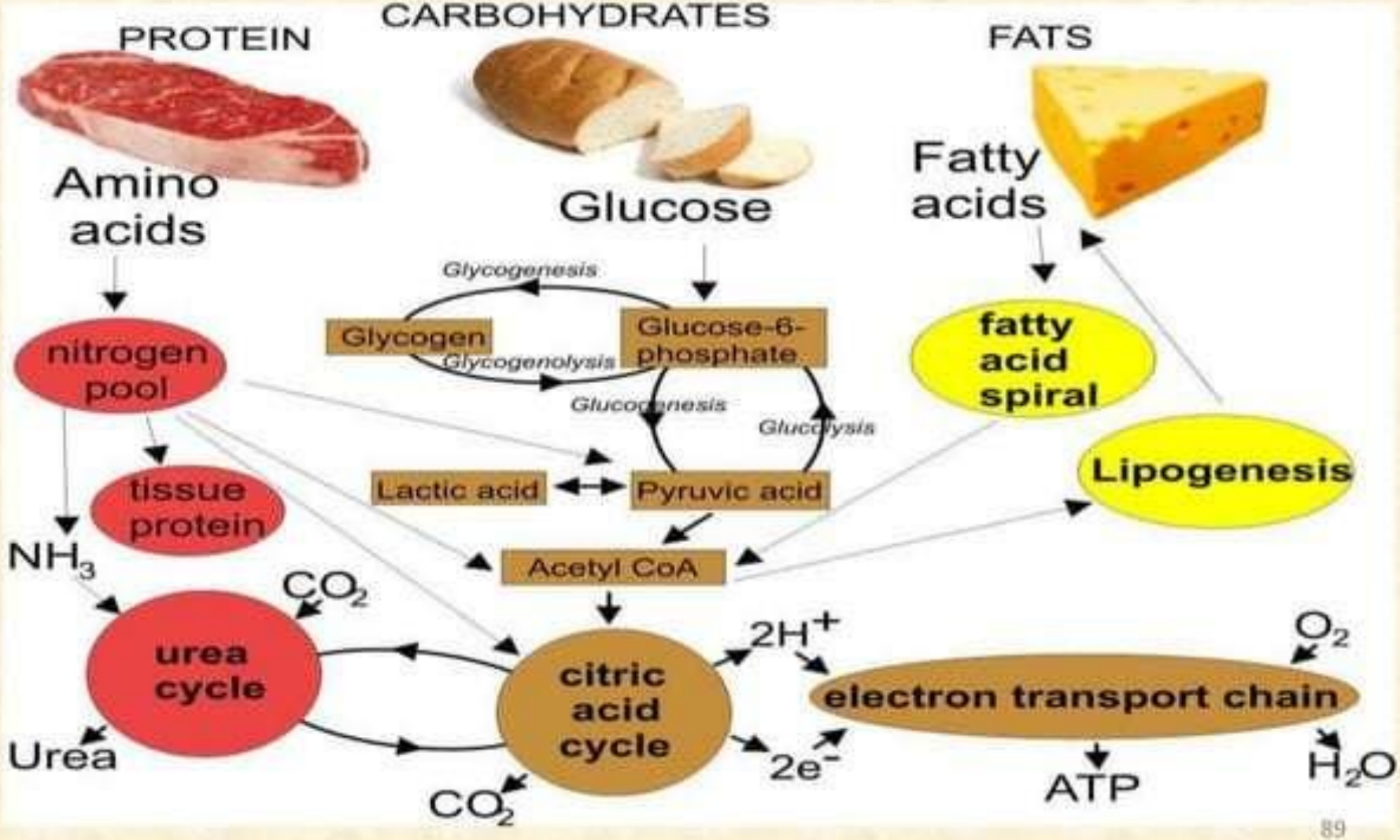
- CMD in Cystic Fibrosis is characterized by its high rates and latent course.
- The patients with CMD have retarded physical development, more pronounced morphofunctional disorders in the bronchopulmonary system, lower lung functional parameters, and more aggressive sputum microbial composition. (Samoĭlenko VA et al.)

CMD in Gout

- OGTT causes a 34% increase in the detection rate of T2D in patients with gout.

- Carbohydrate metabolic disturbances are revealed in the majority of patients with gout and associated with obesity, hypertriglyceridemia, high serum UA levels, chronic disease forms, the high incidence of CHD and arterial hypertension.(Eliseev MS et al.)

SUMMARY OF CARBOHYDRATE METABOLISM



PER DAY INTAKE OF CARBOHYDRATE

- Carbohydrate Calculator http://www.calculator.net/carbohydrate-calculator.html?ctype=metric&cage=25&csex=f&cheightfeet=5&cheightinch=10&cpound=160&cheightmeter=163&ckg=74&cactivity=1.375&x=85&y=10#

CONCLUSION

- Carbohydrate are the measure source of energy for the living cells. Glucose is the central molecule in carbohydrate metabolism, actively participating in a number of metabolic pathway.
- One component of etiology of dental caries is carbohydrate which act as substrate for bacteria. Every effort should be made to reduce sugar intake for healthy tooth.

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THANK
YOU