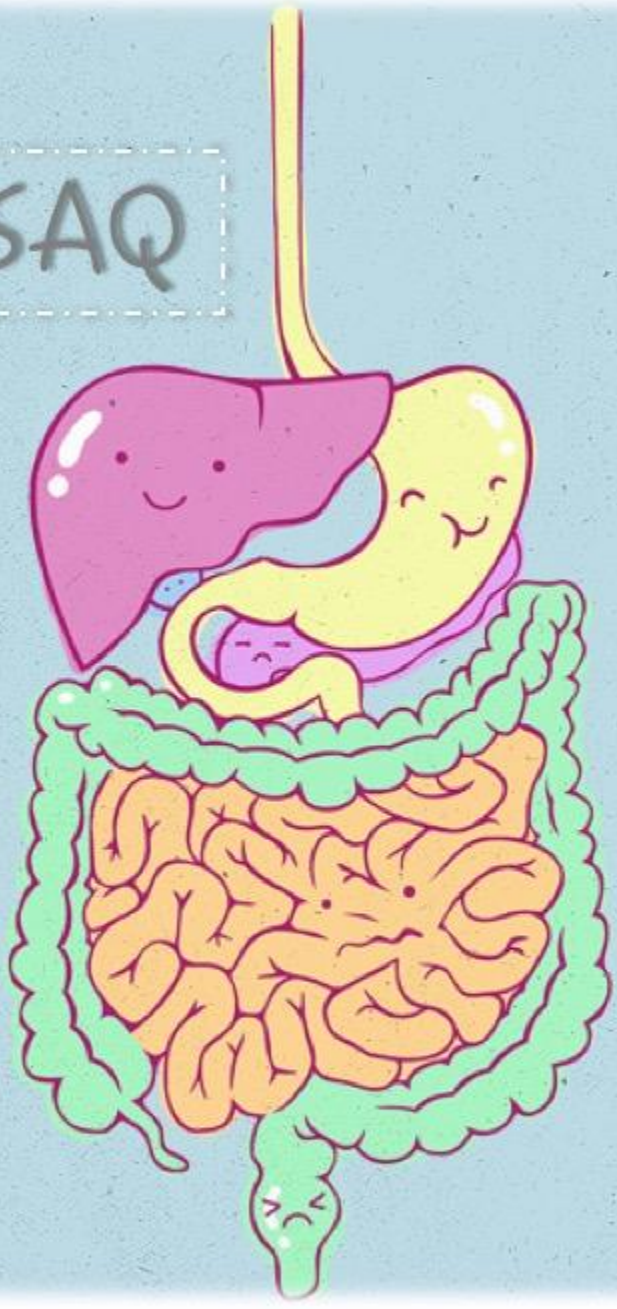
GIT SAQ



Biochemistry

Editing File

Role of Salivary Glands and Stomach in Digestion

	α-Amylase	Lingual lipase	Gastric lipase	pepsin	rennin
production	In the Parotid glands "In **Mouth**"	Dorsal surface of the tongue (Ebner's glands)	Chief cells in the stomach	Chief cells of stomach as pepsinogen	Chief cells of stomach of **Neonate and infant**
Substrate	**Starch and glycogen** "Carbohydrate"	**TAG** of short and medium Fatty acid chains "Lipid" as in milk fat		Denatured dietary **proteins** (HCL)	**Casein** of milk
Action	Hydrolysis of α (1,4) glycosidic bonds				
Product	**Oligosaccharides**	**Monoacylglycerols and fatty acids**		**Smaller polypeptides**	**Paracasein& milk clot**
Notes	1-Acid instable "inactivated by stomach acidity" 2-It's of little significance due to short time of it's action on the food 3-Found in **mouth** 4- Doesn't work on alpha (1,6) , beta (1,4) glycosidic bonds Nor Disacchrides .	Important in neonates & infants and patients with pancreatic insuffincy"B.c normally pancreas produce lipase but in this patient there is No pencreatic Lipase & they depend on these two types only "		Activated by HCL and autocatalytically *by pepsin *means it Activate itself	1-Prevents rapid passage of milk from stomach so allow more time for pepsin to act on milk protien

Mention the end producs of digestions :

- Carbohydrates → Monosaccharides
- Triacylglycerols (TAG) → Fatty acids & monoacylglycerols
- Proteins → Amino acids

Mention the role of saliva in digestions:

1- Acts as lubricant 2- Contains salivary α-amylase 3- Contains lingual lipase

Mention the role of stomach in digestions.

1-No further digestion of carbohydrates 2- Lipid digestion begins by lingual and gastric lipases
3-Protein digestion begins by pepsin and rennin

Talk briefly about digestion of lipids in stomach :

In adults, no significant effects because of lack of emulsification that occurs in duodenum
In neonates and infants,digestion of milk TAG and production of short- and medium-chain fatty acids

Hemoglobin

What is the function of bohr effect ?

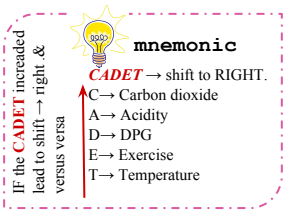
- The Bohr effect removes insoluble CO2 from blood stream
- Produces soluble bicarbonate

List the factor lead to increase affinity and decrease affinity to O2 ?

- **High O2 affinity occurs due to**
  - Hypoxia
  - High altitude
- **Low O2 affinity occurs due to**
  - Alkalosis
  - High levels of Hb F
  - Multiple transfusion of 2,3 DPG-depleted blood

What are the causes lead to shift Oxygen Dissociation Curve into right ?

Decrease in pH , increase in DPG , Temp , P50



What are the causes lead to shift Oxygen Dissociation Curve into left ?

Decrease in DPG , Temp , P50 , increase in pH , Abnormal Hb

What is the type of hemoglobin will find increase in diabetes mellitus? Hemoglobin A1c

Name three abnormal Hb with brief explain cause of the abnormality?

Carboxy-Hb : " found in smoker " CO replaces O_2 and binds 200X tighter than O_2

Met – Hb : Contains oxidized Fe3+ that cannot carry O_2

Sulf-Hb: Forms due to high sulfur levels in blood

What are the globin chains forming Hb F and Hb A2 ?

- Hb F : Tetramer with two α and two g chains
- Hb A2 : two α and two δ globin chains

What does the heme group group contain?

- A complex of protoporphyrin IX and ferrous iron (Fe^{2+})
- Fe^{2+} is present in the center of the heme & binds to four nitrogen atoms of the porphyrin ring
- Forms two additional bonds with: Histidine residue of globin chain & Oxygen

Name four types of the normal Hb. HbA , HbA_2 , HbF , HbA_{1c}

Name the Major Hb in adults and what it contains?

- Hb A and Composed of four polypetide chains(Two α and two β chains)
- Contains two dimers of ab subunits(non-covalent interactions)
- Each chain is a subunit with a heme group in the center that carries oxygen
- A Hb molecule contains 4 heme groups and carries 4 moelcules of O_2

Mention the two forms of Hb and describe them

Relaxed form	Taut form
Oxygenated form	Deoxy form of Hb
The dimers have more freedom of movement	The movement of dimers is constrained
High-oxygen-affinity form	Low-oxygen-affinity form

Name the factors affecting oxygen binding. pO_2 , pH , pCO_2 , Availability of 2,3-bisphosphoglycerate

Biochemical aspects of digestion of lipids

What is the site of Lipid digestion , and mention the required enzyme in each site ?
1-Stomach .. the enzyme required is Gastric Lipase
2- Small intestine .. the enzyme required is the Pancreatic enzymes (lipase & co-lipase , cholesterol esterase , Phospholipase A2 , Lysophospholipase)

Which one of fatty acid chain that does not require Micelle for absorption?
Short & Medium chain fatty acids

What are the end products of digestion the dietary lipids ?
- **TAG** by pancreatic lipase & colipase → *2-monoacylglycerol* with 2 Fatty acid
- **Cholesterol ester** by cholestrol estrase → *cholestrol* with fatty acid
- **Phospholipids** by Phospholipase A2 & Lysophospholipase.
First **Phospholipids** by Phospholipase A2 → *Lysophospholipids* with fatty acid
Then **Lysophospholipids** by Lysophospholipase → *Glycerolphosphoryl base* with fatty acid.

What is a characteristic of dietary lipids? Hydrophobic
What is the first site where lipids are digested is: Stomach
Where is TAG re-synthesized? Intestinal Mucosal Cells
Intestinal digestion of lipids is done by: Pancreatic Enzymes
Mention 3 causes that lead to incomplete absorption of fat & protein?
1-Liver Disease 2-Pancreatic insufficiency 3-Intestinal Disease

Mention enzymes is not essential for adults butimportant for infants: Gastric and lingual Lipase
What is the effect of Secretin? Stimulates the pancreas to neutralize the pH of the intestinal contents.

What are the steps in Re-synthesis of lipids by intestinal mucosal cells ?
1.Activation of long chain fatty acids into acyl CoA
2.Synthesis of - TAG from monoacylglycerol
- Cholesterol ester from cholesterol
- Phospholipids from glycerolphosphoryl base
3.Short- and medium-chain fatty acids are not converted into their CoA derivatives. Instead, they are released into portal circulation, carried by serum albumin.

Talk about the metabolism (synthesis & secretion) and the fate of CHYLOMICRON?
Synthesis: Newly synthesized TAG and cholesterol ester are packaged as lipid droplets surrounded by thin layer of: Apolipoprotein B-48 (apo B-48) , Phospholipids & Free cholesterol.
Secretion: By exocytosis into lymphatic vessels around villi of small intestine (lacteals) then enter into systemic circulation.
Fate: Uptake by the liver as chylomicron remnant via apoE receptor on the *liver (endocytosed)*

What are the clinical manifestations of diseases that involve defective lipid digestion? Liver diseases, pancreatic insufficiency, or intestinal diseases → incomplete digestion and absorption of fat & protein → abnormal appearance of lipid (steatorrhea) & undigested proteins in the feces (Malabsorpton syndrome).

Biochemical Aspects of of digestion of proteins & CHO

What are the source of proteolytic enzymes responsible for degrading dietary proteins?
Stomach, pancreas and small intestine.

The gastric juice contains 2 components for protein digestion? 1- hydrochloric acid. 2- Pepsin.

Two small peptide hormones are released from cells of the upper part of small intestine?
1- Cholecystokinin. 2- secretin.

What are the functions of cholecystokinin?
1- secretion of pancreatic enzymes. 2- Bile secretion. 3- Slow release of gastric contents.

What is the function of secretin? Release of watery solution rich in bicarbonate by pancreas.

What does enteropeptidase do? It converts trypsinogen to trypsin.

What is celiac disease?
It is a disease of malabsorption resulting from immunemediated damage to the villi of small intestine in response to ingestion of gluten which found in whet, ray and barley.

Sites for digestion of dietary carbohydrates? 1- The mouth . 2- The intestinal lumen.

What are enzymes for digestion of dietary carbohydrates?
a-amylase, disaccharidases and isomaltase and a(1,6) glucosidase.

What is the important of secreting of HCl by partial cell in stomch ?
1. kills bacteria “
2. Denatures proteins → denatured proteins are more susceptible to hydrolysis by proteases.
3. activation of Pepsinogen into pepsin.

Why there are many enzymes for digestion of protein ?
Because Each of these enzymes has different specificity for the cleavage sites

Why there is No dietary carbohydrate digestion occurs in the stomach ?
Due to the high acidity of the stomach inactivates the salivary a–amylase

Name a condition will lead to increase serum level of a-amylases with brief explain the cause ?
acute pancreatitis (damage of pancreatic cells → release & activation of the intracellular enzymes into the blood)

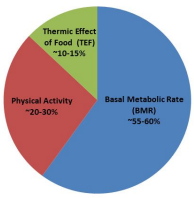
Why is serum level of a-amylases is not specific diagnosis tool ?
because it’s level starts to rise within few hours ,reaches a peak within 12- 72 hours, then returns to normal within few days

Describe the mechanisms of absorption of Glucose & Galactose and fructose in small intenstin ?
1. Fructose will absorbed at brush border by Facilitated diffusion (GLUT5-mediated) and then from basolateral membrane transfer to blood stream by diffusion (GLUT-2 – mediated)
2. Glucose & Galactose will absorbed at brush border by Active transport Co-transport with Na^+ and then from basolateral membrane transfer to blood stream by diffusion (GLUT-2 – mediated)

Nutritional Requirements

Dietary Reference Intakes (DRIs): Quantitative estimates of nutrient intakes required to prevent deficiencies & maintain optimal health in population			
Estimated Average Requirement (EAR)	**Recommended Dietary Allowance (RDA)**	**Adequate Intake (AI)**	**Tolerable Upper Intake Level (UL)**
The amount of nutrient intake estimated to meet the **nutritional requirement of *half of the healthy individuals*** *(50%)* in an age and gender group	The amount of nutrient intake that is sufficient to meet the **nutritional requirement of *nearly all (97-98%) healthy individuals*** in a group RDA = EAR + 2 SD	Instead of EAR and RDA if: A nutrient is considered essential but the **experimental data are inadequate** for determining EAR and RDA AI covers the nutritional requirement of *all individuals in a group with approximation* due to insufficient data	The **highest level** of daily nutrient intake that **has no adverse effects** or toxicity in almost all individuals.

Components of Energy Expenditure



Which level of food pyramid have the small serving size ? fats -oils - sweet

Mention two of dietary guidelines and goals ?
1-control calorie intake to manage body wight 2- be physicualy active everyday

Energy balance of the body is maintaned by ? 1-calorie intake 2-energy expenditure

Which minerals or substances that usually can be decreased in vegetarian people ?
Iron - calcium - vitamine (D & B_{12}) and fats

Basic energy expenditure depends on :
1- resting metabolic rate(RMR) 2-physical activety 3-thermic effect of food

What dose total parenteral nutrition (TPN) indicate ?
It's particulary indecated in severe inflammatory bowel disease,coma,cachexia, prolonged ileus & extensive burns

Vegetarians and nutrient intake	
Advantage	Disadvantage
1-lower body mass index (BMI) 2-lower death rate of ischmic heart disease	1- lower intake of iron , calicium and vitamine D 2-most consum enough protein

Macro and micro nutrients

What does Essential amino acids mean and mention 3 of them ?

It's the amino acid that's the Body can't synthesize,must be supplied in diet.eg: Pheylalanine, Valine, Tryptophan

What is the difference between positive and negative nitrogen balance ?

Positive nitrogen balance	**Negative** nitrogen balance
When nitrogen intake is more than loss	When nitrogen loss is more than intake
Occurs in growth, pregnancy, lactation, recovery from illness	Occurs in burns, trauma, illness, metabolic stress

A 3 year-old child was presented by his mother to the general practitioner with Extreme muscle wasting, arrested growth , Weakness ,Weight loss and No edema or changes in plasma proteins what is diagnosis ? and mention the cause of this condition ?

Marasmus , The cause is due to Inadequate intake of energy with adequate protein intake.

An infant (at about 1 year) the doctor examine him and found that he has Edema, Distended abdomen , Diarrhea , Dermatitis / thin hair , Enlarged fatty liver and Low plasma albumin, what is his diagnosis? and mention the cause of this condition ?

Kwashiorkor , The cause is due to Inadequate intake of proteins with adequate energy intake.

Mention the results of the following?

Exssesive diatery fat: Atherosclerosis, Obesity **Fat deficincy:** scaly skin, Dermatitis, Reduced growth

Healthy Patient is suffering from omega-3 deficincy, what is your recomindition for his Omega-3 Fatty Acid Intake? Fatty fish twice a week, Include oils and foods rich in a-linolenic acid

Unsaturated fatty acids, behaving more like saturated fatty acids in the body, found in baked food like cookies? Trans fatty acid

35 year old female complaining from wrinkles under her eyes and anemia, what vitamen supplemt should you give her? Vit E(α - tocopherol)⇒ Anit-aging+anti-oxidant(prevent RBCs lysis due to oxidative damage)

Mention 2 disease that are associated with vit B_{12} deficiency?

Beriberi⇒ chronic peripheral neuritis causes Neuropathy affects glial cells (astrocytes) of the brain and spinal cord causing neuron death

Wernicke-Korsakoff syndrome⇒ Common in alcoholics due to defective intestinal absorption of thiamin or dietary insufficiency. Causes apathy, loss of memory.

What is Scurvy disease and its symptoms?

It is a disease due to vit c deficiency.

symptoms:Abnormal collagen production,painful gums, The pulp is separated and the teeth are lost

What are the forms of stored iron in the body? Ferritin, hemosiderin and transferrin

Plasma Protiens

Give 4 Functions of plasma proteins?

1-**Transport** (Albumin, prealbumin, globulins) 2-**Maintain plasma oncotic pressure** (Albumin)

3-**Defense** (Immunoglobulins and complement) 4-**Clotting and fibrinolysis** (Thrombin and plasmin)

How could we measure Plasma Protiens "give 2 ways"?

A)Quantitative measurement of a specific protein: Chemical or immunological reactions

B) Semiquantitative measurement by electrophoresis: Proteins are separated by their electrical charge in electrophoresis Five separate bands of proteins are observed These bands change in disease

Prealbumin (Transthyretin) **What is the function of it?** transport protein of:Thyroid hormones&Retinol (vitamin A)

How is it migrate in Electrophoresis ? It migrates faster than albumin

Where can we find low levels of Prealbumin?

liver disease, nephrotic syndrome, acute phase inflammatory response, malnutrition.

Albumin **Where is it synthesis ?and How?** Synthesized in the **liver** as **preproalbumin** and **secreted as albumin.**

When is it decrease Rapidly?in injury, infection and surgery

What're the functions of albumin? (mention 2)

1) Maintains oncotic pressure 2) A non-specific carrier of hormones, calcium, free fatty acids, drugs, etc.

What causes Hyperalbuminemia? Dehydration.

What are the causes of Hypoalbuminemia?

- **Decreased albumin synthesis** as in (liver cirrhosis, malnutrition)
- **Increased losses of albumin** as in Increased catabolism in infections or Excessive excretion by the kidneys (nephrotic syndrome) or Excessive loss in bowel (bleeding) or severe burns (plasma loss in the absence of skin barrier)

What is the effect of Hypoalbuminemia & what it'll result in?

Effect is "Edema" because of low oncotic pressure due to low Albumin it'll result in

1-Reduced transport of drugs and other substances in plasma.

2-Reduced protein-bound calcium : A–Total plasma calcium level drops B–Ionized calcium level may remain normal

a_1-Antitrypsin

Where is it synthesis ?What it's function? liver and macrophages, Is An acute-phase protein that inhibits proteases "which is synthesized by leukocytes and bacteria & release during infection"

How many types & which is most common? >30 types ,M is the most common

Explain how Genetic deficiency of a_1-Antitrypsin Happens? there is Synthesis of the defective a_1-Antitrypsin in the liver but it cannot secrete it so a_1-Antitrypsin accumulates in hepatocytes and is deficient in plasma.

What are the clinical Consequences of a_1-Antitrypsin Deficiency?

1-Neonatal jaundice with evidence of cholestasis 2-Childhood liver cirrhosis.

3-In young adults Pulmonary emphysema.

What is your finding from the Laboratory Diagnosis? the electrophoresis show Lack of a_1-globulin band in protein

What is the function of Haptoglobin ? Limits iron losses by preventing Hb loss from kidneys

In which cases dose b_2–Microglobulin serum levels elevation as found. Overproduction in disease

Liver Function Test

What are the markers of hepatocellular injury?

- Alanine aminotransferase (ALT) specific
- Aspartate aminotransferase (AST)

In which conditions high bilirubin levels are observed ? Gallstones, acute and chronic hepatitis

What dose biliary passages obstruction cause?

- Leakage of bile salts into circulation
- Excretion in urine

Serum albumin Synthesis depends on what? It depends on extent of functioning liver cell mass

What is the type of bilirubin will be raised in case of pre-hepatic jaundice? Unconjugated bilirubin

Where are a and b-globulins mainly synthesized ? In the liver

In which cases high serum levels of Aspartate aminotransferase are observed?

Chronic hepatitis, cirrhosis and liver cancer

What is the best marker to determine if someone is drinking alcohol or not ?

- Gamma-glutamyltransferase

The underlying cause of increased Globulin levels in patients with Hypoalbuminemia is.

- Compensatory mechanism

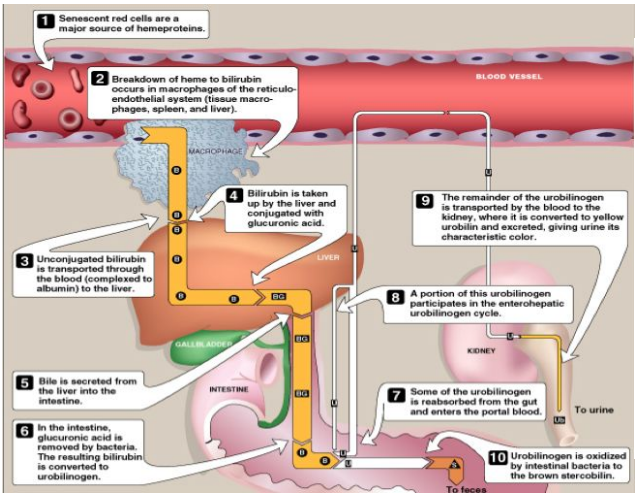
List some of the Major Metabolic Functions of the Liver?

•Synthetic Function : Plasma proteins (albumin, globulins), cholesterol, triglycerides and lipoproteins

•Detoxification and excretion :Ammonia to urea (urea cycle), bilirubin, cholesterol, drug metabolites

•Storage Function : Vitamins A, D, E, K and B12

•Production of bile salts : Helps in digestion



Class of Jaundice	Type of Bilirubin raised	Causes
Pre-hepatic or hemolytic	Unconjugated	Abnormal red cells; antibodies; drugs and toxins; thalessemia Hemoglobinopathies
Hepatic or Hepatocellular	Unconjugated and conjugated	Viral hepatitis, toxic hepatitis, intrahepatic cholestasis, Gilbert's, Crigler-Naajjar syndrome
Post-hepatic	Conjugated	Extrahepatic cholestasis; gallstones; tumors of the bile duct, carcinoma of pancreas

Biochemical aspects of bile acids and salts

What are bile salts ?
they are bile acids which are conjugauted with either a molecule of glycine or taurine
What is the precursor of bile acid and salt ? cholesterol

Where is the synthesis of bile acids located ? in the liver

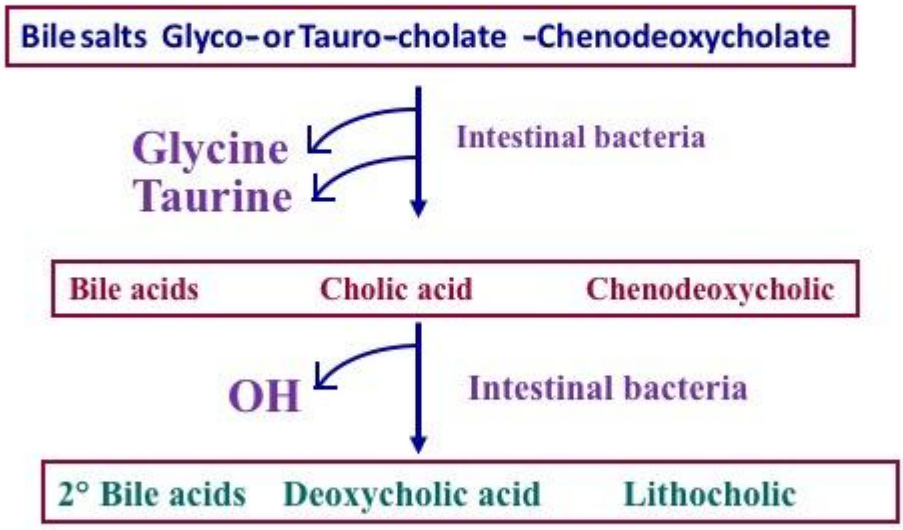
What is the ratio of glycin/taurine form of bile salts ? 3:1

Mention the factores that lead to inhibiton and induction of the activity of (cholestrol 7- alpha - hydroxylase)?
inhibiton → cholic acid
induction → cholesterol

primary bile acid and salts

chenodeoxycholic acid	BILE ACIDS	cholic acid
↓		↓
GLYCOchenodeoxycholic TAUROchenodeoxycholic	BILE SALTS	GLYCOcholic TAUROcholic

Secondary bile acid and salts



Functions of bile salts

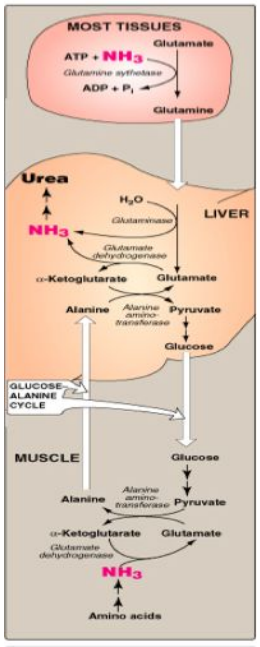
important for cholestrol excertion : 1-as a metablic products of cholestrol. 2-sollubilizer of cholestrol in bile.	Emulsifying factors for dietary lipids , a prerequisits step for efficient lipid digestion .	cofactor for pancreatic lipase and PLA2	facilitate intestinal lipid absorption by formatiom of mixed micells

Urea cycle

	Glutamine	Alanine
Site of formation	most peripheral tissue	muscles
Transamination	α-ketoglutarate + NH3 → Glutmate	
Formation	NH3 + Glutamate→ Glutamine by Glutamine synthetase	Pyurvate + Glutamate → Alanine + α-ketoglutarate by ALT(Alanine aminotransferase)
Inside the liver	converted into Glutamate by Glutaminase	Alanine + α-ketoglutarate → Pyurvate + Glutamate by ALT
	Glutamate →α-ketoglutarate+NH3 by glutamate dehydrogenase	

Why does amino group of amino acid is funnled to glutamate?

Because it's the only amino acid that undergoes rapid oxidative deamination.



Enumerate some features of Urea cycle.

- Major disposal form of amino group -occurs in the liver
- one N from NH3 and one from aspartate -Urea excreteed in kidneys

The five enzymes of urea cycle:

1_Carbamoyl phosphate synthetase I (CPSI) Rtae-limiting enzyme
2-Ornithine transcarbamoylase (OCT) 3-Argininosuccinate synthase
4-Argininosuccinate lyase 5- Arginase

*Hyper*ammonemia:

	Acquired	Inherited
Causes	➔ liver disease acute : viral hepatites chronic : cirrhosis ➔ kidneys disease	genitic deficiencies of any 5 enzymes or NAGS OTC deficency: • X-linked recessive • most common
Clinical presintaion	-lethargy -somnolence -tremors -vomiting -cerebral edema -convulsions -coma and death	
Management	1-protein restriction 2-volume depletion 3-hemodialysis $-avoid drugs that catabolite protein	

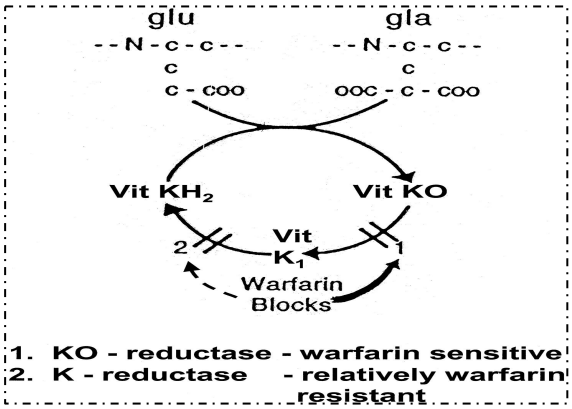
Vitamin K

Forms	Sources
Vitamin K1 (phylloquinone)	Cabbage, kale, spinach.
Vitamin K2 (menoquinone)	Intestinal bacteria (deficiency is rare)
Vitamin K3 (menadione)	Synthetic form that will be converted to menaquinone.

What is the main function of vitamin K?

Coenzyme for the synthesis of prothrombin and blood clotting factors in the liver (VII,IX,X). This requires carboxylation of their glutamic acid residues (Glu) → gamma-carboxyglutamate (Gla) containing tqo carboxylate groups → bind to calcium → calcium-prothrombin complex → binds to platelets → convert prothrombin to thrombin and proceeds in blood clot formation.

Complete the cycle of vitamin K?



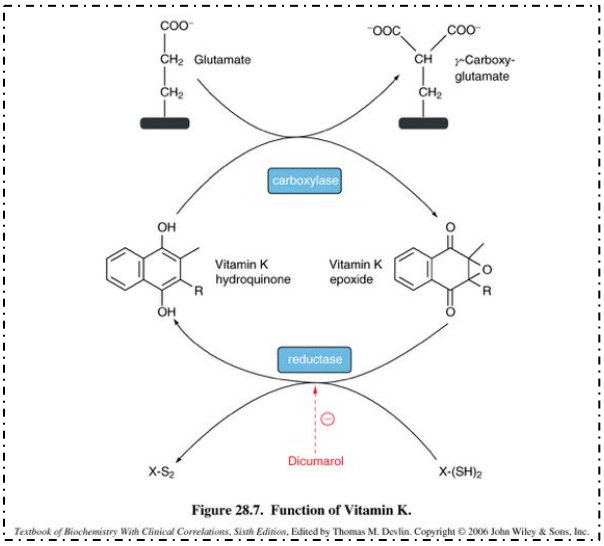


Figure 28.7. Function of Vitamin K.

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Other functions of vitamin K:

Synthesis of gamma-carboxy glutamate in osteocalcin which is an important bone protein. Also called Bone Gla Protien (BGP)

Mention some Clinical manifestations of Vit.K deficiency ?

- Hemorrhagic disease of the newborn.
- Bleeding tendency of all types
- Prolongation of prothrombin time.

List some Causes of Vit.K deficiency ?

1-Most commonly in infants due to lack if intestinal flora + insufficient vit K in mother's milk. Solution? → prophylactic single IM dose of vit K to all newborns.
2- Malabsorption of lipids.
3- Prolonged use of antibiotics. Specially second-generation cephalosporin may act like warfarin → inhibits the vitamin K epoxide reductase → hypoprothrombinemia

What is the cause of Toxicity and mention the Clinical manifestation ?

Due to prolonged administration of large doses of synthetic vit K in infants.

Clinical manifestations: - Hemolytic anemia. - Jaundice.

Glucose-6-Phosphate Dehydrogenase (G6PD) Deficiency Anemia

What is the alternative oxidative pathway for glucose

Hexose monophosphate pathway (HMP) or Pentose Phosphate Pathway (PPP)

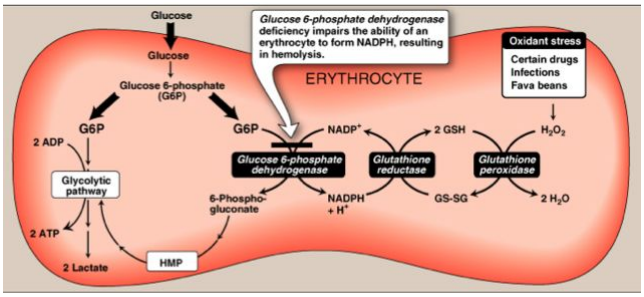
List some features of NADPH .

- Reductive biosynthesis e.g., fatty acid biosynthesis
- Antioxidant (part of glutathione system)
- Oxygen-dependent phagocytosis by WBCs
- Synthesis of nitric oxide (NO)

Mention some diseases that caused by Oxidative Stress

- Inflammatory conditions e.g., Rheumatoid arthritis
- Atherosclerosis and coronary heart diseases
- Obesity
- Cancers
- G6PD deficiency hemolytic anemia

Biochemical Basis of G6PD Deficiency Hemolytic Anemia



Why it is most severe in RBCs Although G6PD deficiency affects all cells,

Because other cells have other sources for NADPH production such as malic enzyme that converts malate into pyruvate

List some Precipitating Factors for G6PD Deficiency Hemolytic Anemia.

1- Intake of oxidant drugs (AAA):
Antibiotics e.g., sulfa preparation
Antimalarial: e.g., Primaquine
Antipyretics

2- Exposure to infection

3- Ingestion of fava beans (favism, Mediterranean variant)
Chronic nonspherocytic anemia: Hemolytic attack in absence of precipitating factors. Severe form due to class I mutation

What are the Different Classes of G6PD Deficiency Hemolytic Anemia

I (Very severe) associated with chronic nonspherocytic hemolytic anemia.
II (Severe, e.g. Mediterranean) Enzyme with normal stability but low activity (severe). Affect all RBCs (both young and old)
III: (Moderate: G6PD A-) Moderate, young RBCs contain enzymatic activity. Unstable enzyme, but kinetically normal
IV: (Normal)

Describe Diagnosis of G6PD Deficiency Hemolytic Anemia

Diagnosis of hemolytic anemia : Complete Blood Count (CBC) & reticulocytic count
Screening: Qualitative assessment of G6PD enzymatic activity (UV-based test)
Confirmatory test: Quantitative measurement of G6PD enzymatic activity
Molecular test: Detection of G6PD gene mutation

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