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ONLINE COURSE WARE

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Module 1 8L

Lecture 1

Protein: Definition and Structure

1.1 Definition: The term "**protein**" derives from the Greek word "*proteios*", that means primary and was suggested for the first time by Jöns Jacob Berzelius, one of the fathers of modern chemistry. Proteinsarelarge biomolecules or macromolecules composed of one or more long chains of amino acids and are an essential part of all living organisms, especially as structural components of body tissues such as muscle, hair, etc., and as enzymes and antibodies.

1.2 Structure of Protein:

Protein structure is the three-dimensional arrangement of atoms in a protein molecule. Proteins are polymers — specifically polypeptides — formed from sequences of amino acids, the monomers of the polymer. To be able to perform their biological function, proteins fold into one or more specific spatial conformations driven by a number of non-covalent interactions such as hydrogen bonding, ionic interactions, Vander Waals forces, and hydrophobic packing. To understand the functions of proteins at a molecular level, it is often necessary to determine their three-dimensional structurewhich employs techniques such as X-ray crystallography, NMR spectroscopy, and dual polarisation interferometry.

Primary structure

The primary structure of a protein refers to the linear sequence of amino acids in the polypeptide chain. The primary structure is held together by covalent bonds such as peptide bonds, which are made during the process of protein biosynthesis. The two ends of the polypeptide chain are referred to as the carboxyl terminus (C-terminus) and the amino terminus (N-terminus) based on the nature of the free group on each extremity. Counting of residues always starts at the N-terminal end (NH_2-group), which is the end where the amino group is not involved in a peptide bond.

Secondary structure

Secondary structure refers to highly regular local sub-structures on the actual polypeptide backbone chain. Two main types of secondary structure, the α-helix and the β-sheets, were suggested in 1951 by Linus Pauling. These secondary structures are defined by patterns of hydrogen bonds between the main-chain peptide groups.

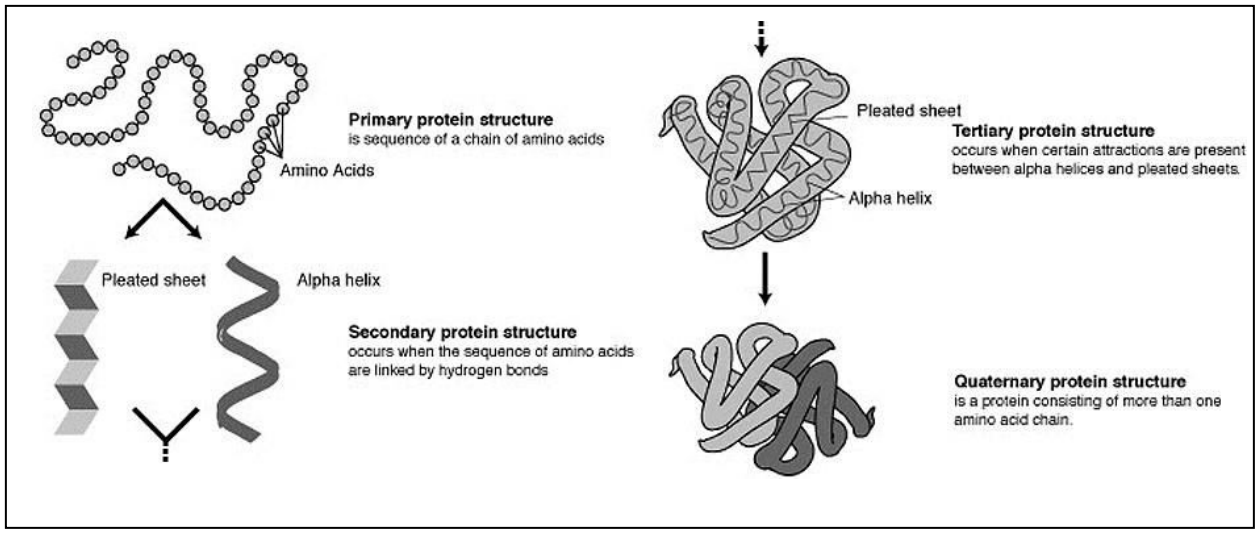
Tertiary structure

Tertiary structure refers to the three-dimensional structure of monomeric and multimeric protein molecules. The α-helixes and β-pleated-sheets are folded into a compact globular structure. The folding is driven by the non-specific hydrophobic interactions, the burial of hydrophobic residues from water, but the structure is stable only when the parts of a protein domain are

locked into place by specific tertiary interactions, such as salt bridges, hydrogen bonds, and the tight packing of side chains and disulfide bonds.

Quaternary structure

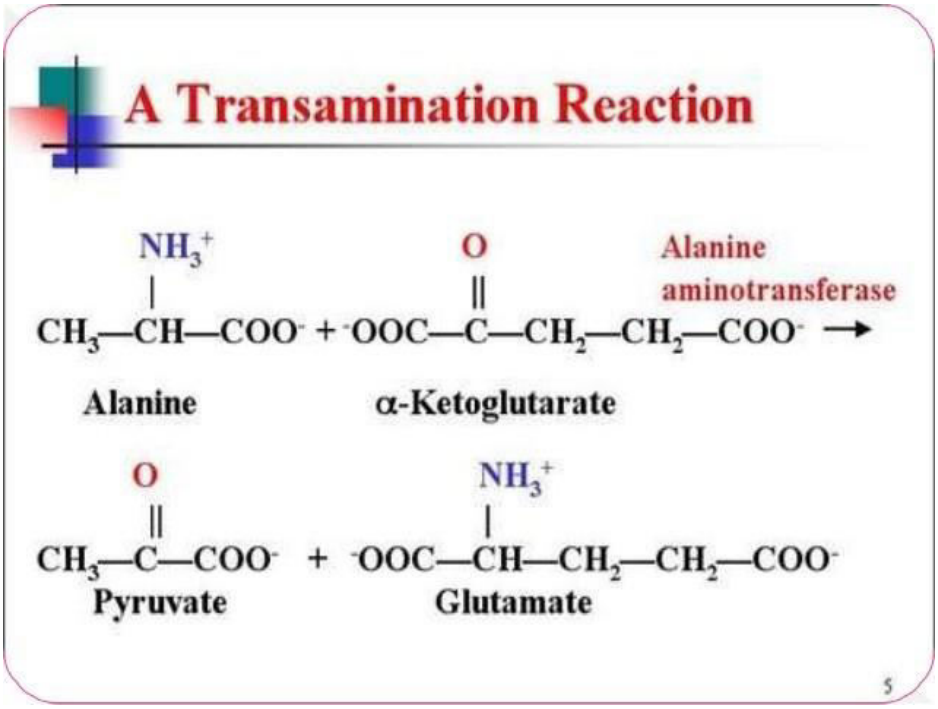
Quaternary structure is the three-dimensional structure of a multi-subunit protein and is stabilized by the non-covalent interactions and disulfide bonds. Complexes of two or more polypeptides (i.e. multiple subunits) are called multimers. Specifically it would be called a dimer if it contains two subunits, a trimer if it contains three subunits, a tetramer if it contains four subunits, and a pentamer if it contains five subunits. The subunits are frequently related to one another by symmetry operations, such as a 2-fold axis in a dimer. Multimers made up of identical subunits are referred to with a prefix of "homo-" (e.g. a homotetramer) and those made up of different subunits are referred to with a prefix of "hetero-", for example, a heterotetramer, such as the two alpha and two beta chains of hemoglobin.



Lecture 2

Transamination: Process description with examples

Transamination, a chemical reaction that transfers an amino group to a ketoacid to form new amino acids. This pathway is responsible for the deamination of most amino acids. This is one of the major degradation pathways which convert essential amino acids to nonessential amino acids.



Lecture 3

Nitrogen balance: Positive, negative, equilibrium

Nitrogen balance is a measure of nitrogen input minus nitrogen output.

Nitrogen Balance = Nitrogen intake - Nitrogen loss

Sources of nitrogen intake include meat, dairy, eggs, nuts and legumes, and grains and cereals.

Examples of nitrogen losses include urine, feces, sweat, hair, and skin.

Positive nitrogen balance is associated with periods of growth, hypothyroidism, tissue repair, and pregnancy. This means that the intake of nitrogen into the body is greater than the loss of nitrogen from the body, so there is an increase in the total body pool of protein.

Negative nitrogen balance is associated with burns, serious tissue injuries, fevers, hyperthyroidism, wasting diseases, and during periods of fasting. This means that the amount of nitrogen excreted from the body is greater than the amount of nitrogen ingested. A negative nitrogen balance can be used as part of a clinical evaluation of malnutrition.

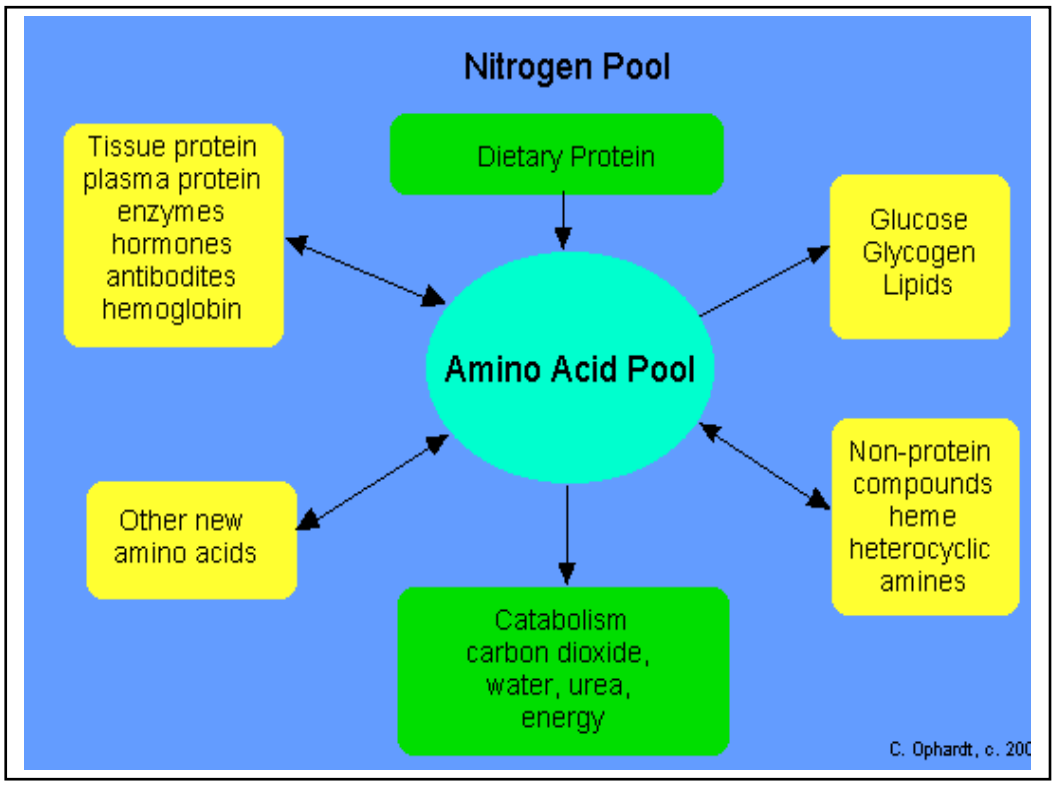
Equilibrium nitrogen balance is associated with state of healthy human where the intake of nitrogen into the body is equal to the loss of nitrogen from the body.

Lecture 4

Nitrogen pool: Occurrence and Importance

Amino acids are important compounds composed of different chains of four basic molecules: carbon, hydrogen, oxygen and nitrogen. About 500 different amino acids have currently been identified in cells. Each acid helps to maintain the functions of a living organism. For example, the amino acid glutamate, which is found in brain cells, is the main neurotransmitter responsible for conveying electrical impulses through the brain. An amino acid pool is the collection of amino acids available in an organism's cells at a given time, based on the proteins and fats recently consumed by the organism. Both plants and animals have amino acid pools, which are replenished whenever the organisms take in nourishment. Amino acids are not stored in cells over long period periods of time, so the makeup of an amino acid pool shifts regularly.

Organisms make use of the energy stored in amino acids through a process called catabolism. When an amino acid is catabolized, it breaks down into its component parts, releasing waste and energy that powers the functions of the organism. Once this amino acid has been used, it must be replenished through the introduction of new proteins. These proteins contain slightly different molecules, which leads to the production of slightly different amino acids and an altered amino acid pool.



Lecture 5

Evaluation of quality of proteins: BV, PER

Biological value (**BV**) is a measure of the proportion of absorbed protein from a food which becomes incorporated into the proteins of the organism's body. It captures how readily the digested protein can be used in protein synthesis in the cells of the organism. Proteins are the major source of nitrogen in food. BV assumes protein is the only source of nitrogen and measures the proportion of this nitrogen absorbed by the body which is then excreted. The remainder must have been incorporated into the proteins of the organisms body. A ratio of nitrogen incorporated into the body over nitrogen absorbed gives a measure of protein "usability" – the BV.

Biological value is determined based on this formula.[4][5]

$$BV = (N_r / N_a) * 100$$

Where:

N_a = nitrogen absorbed in proteins on the test diet

N_r = nitrogen incorporated into the body on the test diet

Protein efficiency ratio (PER) is based on the weight gain of a test subject divided by its intake of a particular food protein during the test period. From 1919 until very recently, the PER had been a widely used method for evaluating the quality of protein in food.

PER= Wt. gain of test group (gm) / total protein consumed (gm)

Lecture 6

NPU, Chemical Score

NPU: The net protein utilization, or NPU, is the ratio of amino acid converted to proteins to the ratio of amino acids supplied. This figure is somewhat affected by the salvage of essential amino acids within the body, but is profoundly affected by the level of limiting amino acids within a foodstuff.

NPU= Nitrogen retained x 100 / Nitrogen intake

Chemical Score: Protein digestibility-corrected amino acid score (PDCAAS) is a method of evaluating the protein quality based on both the amino acid requirements of humans and their ability to digest it. The PDCAAS rating was adopted by the US Food and Drug Administration (FDA) and the Food and Agricultural Organization of the United Nations/World Health Organization(FAO/WHO) in 1993 as "the preferred 'best'" method to determine protein quality.

A PDCAAS value of 1 is the highest, and 0 the lowest. The table shows the ratings of selected foods.

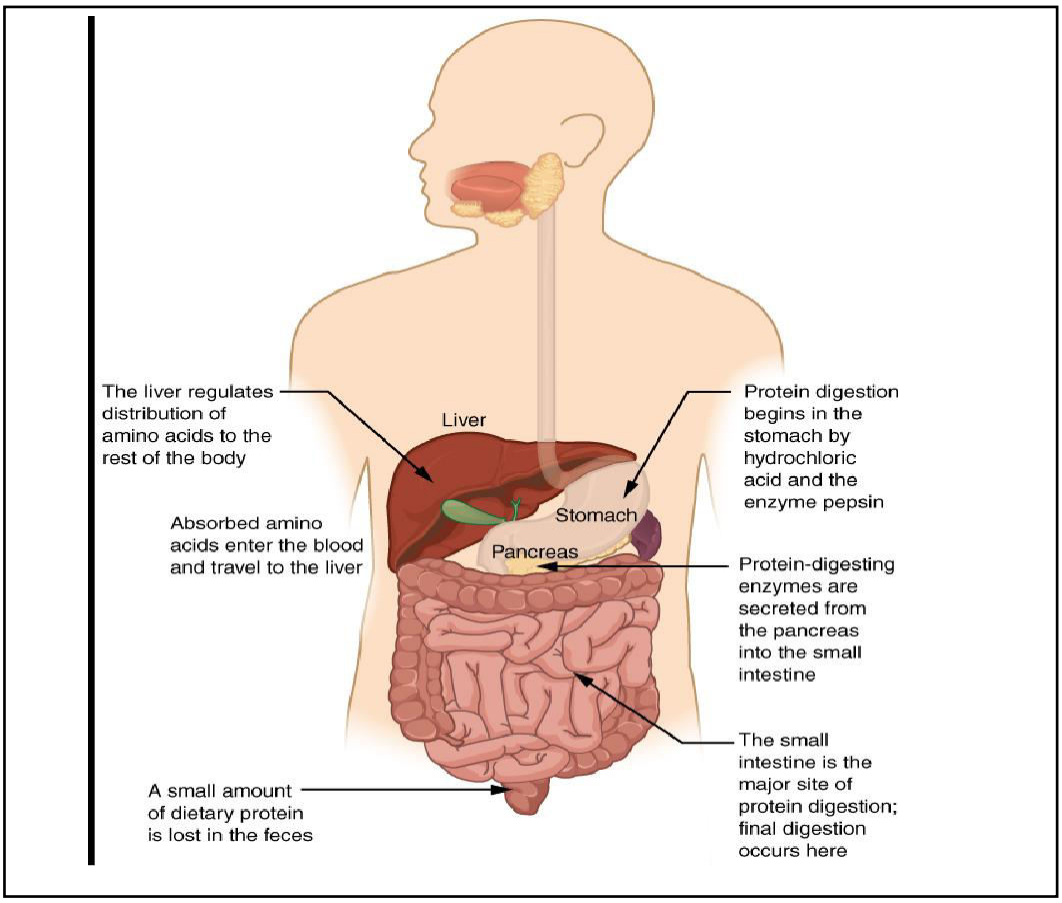
Food Stuffs	PDCAAS
Milk	1.0
Egg	1.0
Soy Protein	1.0
Beef	0.92
Black beans	0.75
Rice	0.5
Wheat	0.42

Lecture 7

Metabolism of proteins : digestion

Once protein is chewed and swallowed, hydrochloric acid and pepsin begin protein digestion in the stomach. HCl helps to kill bacteria in food that could cause infection. It also makes the stomach very acidic with a pH of 1.5. This acidic environment is necessary for HCl to react with pepsinogen to form pepsin so that it can break the central peptide bond in proteins. Rennin is an enzyme that is present in infants to help break down milk protein.

The pancreas releases digestive enzymes into the small intestine. In the duodenum, the first section of the small intestine, trypsin breaks down proteins into single amino acids by a process called hydrolysis. During hydrolysis, a water molecule is placed between two amino acids, breaking the bond. Trypsin also activates the enzymes chymotrypsin, carboxypeptidase and elastase that are released into the small intestine for amino acid chain breakdown.

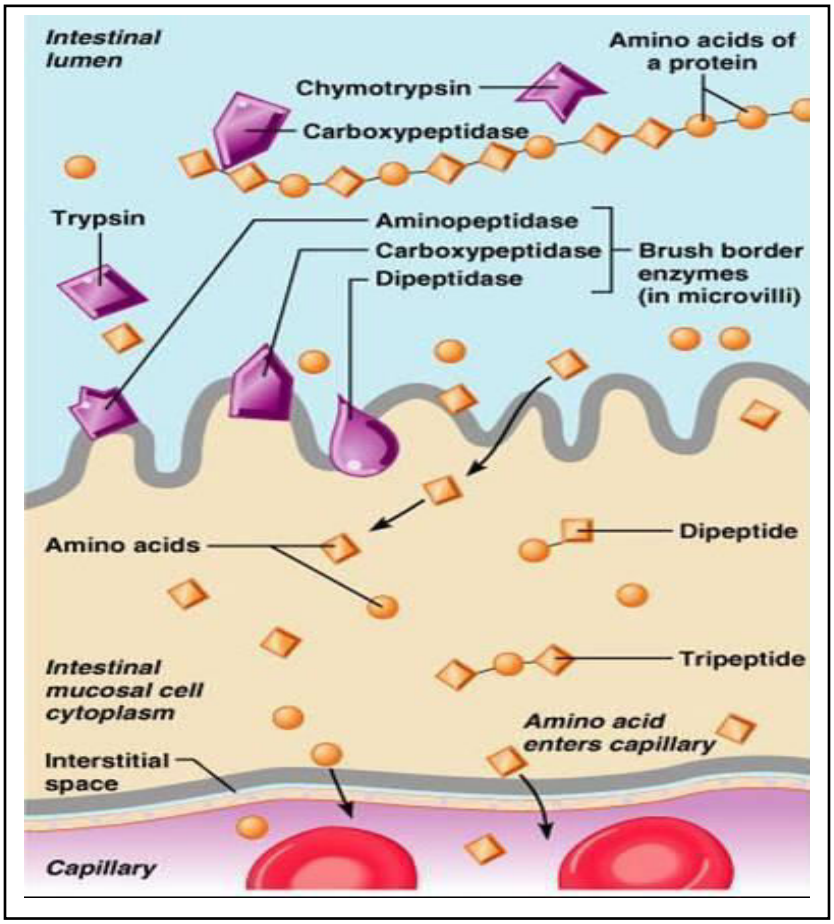


Lecture 8

Metabolism of proteins : absorption

Protein absorption takes place in the jejunum and ileum portions of the small intestine. This process requires energy. Adenosine triphosphate is the energy source the body utilizes during protein absorption. The body uses the carrier protein transport system to absorb amino acids. Each amino acid group has a carrier protein that is responsible for transporting it from the intestines to the mucosa cells. Sodium and potassium are minerals needed for the amino acids to pass from the intestines through the villi and into the bloodstream.

The single molecule amino acids, or free amino acids, that are absorbed through the wall of the small intestine are now used for the last part of protein metabolism, protein synthesis. The proteins ingested from animal and plant protein sources are made into new tissues or used for tissue repairs in the body (hair, skin, nails, muscle) or they are broken down and used for energy. When there is too much protein in the body, the excess gets converted into fat for storage.



Module 2 8L

Lecture 1

Enzymes; Definition, function, classification , nomenclature & structure, Co-enzymes and its Function

Enzymes are biologic polymers that catalyze the chemical reactions. With the exception of a few catalytic RNA molecules, or ribozymes, the vast majority of enzymes are proteins.

- The enzymes catalyze the conversion of one or more compounds (substrates) into one or more different compounds (products).
- They enhance the rates of the corresponding non-catalyzed reaction. Catalysts do not affect reaction equilibria.
- Like all catalysts, enzymes are neither consumed nor permanently altered as aconsequence of their participation in a reaction.
- Their catalytic activity depends on the integrity of their native protein conformation. If an enzyme is denatured or dissociated into its subunits, catalytic activity is usually lost. If an enzyme is broken down into its component amino acids, its catalytic activity is always destroyed.
- The primary, secondary, tertiary, and quaternary structures of protein enzymes are essential to their catalytic activity.

Enzymes are Highly Specific Catalysts

Enzymes are also extremely selective catalysts. Unlike most catalysts used in synthetic chemistry, enzymes are specific both for the type of reaction catalyzed and for a single substrate or a small set of closely related substrates.

- Enzymes are substrate specific or group specific Conformation of complex proteins and uniqueness of active site of enzymes make them substrate specific or absolute group specific. For example glucokinase recognize glucose as absolute substrate while hexokinase recognizes aldohexose (Glucose or mannose etc) as substrate. Similarly, trypsin, chymotrypsin and elastase cleaves proteins or polypeptides on carboxyl side of positively charged (Lysine, Arginine), aromatic amino acids (Tyrosine, Phenylalanine) and small group side chains (alanine, glycine) amino acids respectively.

- Enzymes are also stereospecific catalysts and typically catalyze reactions only of specific stereoisomers of a given compound—for example, D- but not L-sugars, L- but not D-amino acids.
- Enzymes show geometric specificity.
- Enzyme Since they bind substrates through at least "three points of attachment," enzymes can even convert nonchiral substrates to chiral products.

International Classification of Enzyme:

CLASSIFICATION OF ENZYMES

Group of Enzyme	Reaction Catalysed	Examples
1. Oxidoreductases	Transfer of hydrogen and oxygen atoms or electrons from one substrate to another.	Dehydrogenases Oxidases
2. Transferases	Transfer of a specific group (a phosphate or methyl etc.) from one substrate to another.	Transaminase Kinases
3. Hydrolases	Hydrolysis of a substrate.	Estrases Digestive enzymes
4. Isomerases	Change of the molecular form of the substrate.	Phospho hexo isomerase, Fumarase
5. Lyases	Nonhydrolytic removal of a group or addition of a group to a substrate.	Decarboxylases Aldolases
6. Ligases (Synthetases)	Joining of two molecules by the formation of new bonds.	Citric acid synthetase

Coenzymes: They are small molecules. They cannot by themselves catalyze a reaction but they can help enzymes to do so. In technical terms, coenzymes are organic nonprotein molecules that bind with the protein molecule (apoenzyme) to form the active enzyme (holoenzyme).

Examples of Coenzymes and Vitamins

reference; **Cooper GM; The Central Role of Enzymes as BiologicalCatalysts The Cell: A Molecular Approach. 2nd edition**

Coenzyme	Related vitamin	Chemical reaction
NAD^+, $NADP^+$	Niacin	Oxidation-reduction
FAD	Riboflavin (B_2)	Oxidation-reduction
Thiamine pyrophosphate	Thiamine (B_1)	Aldehyde group transfer
Coenzyme A	Pantothenate	Acyl group transfer
Tetrahydrofolate	Folate	Transfer of one-carbon groups
Biotin	Biotin	Carboxylation
Pyridoxal phosphate	Pyridoxal (B_6)	Transamination

Cofactor: Enzymes, like other proteins, have molecular weights ranging from about 12,000 to more than 1 million. Some enzymes require no chemical groups for activity other than their amino acid residues. Others require an additional chemical component called a cofactor— either one or more inorganic ions, such as Fe2+, Mg2+, Mn2+, or Zn2+, or a complex organic or metalloorganic molecule called a coenzyme. Some enzymes require both a coenzyme and one or more metal ions for activity. A coenzyme or metal ion that is very tightly or even covalently bound to the enzyme protein is called a prosthetic group. A complete, catalytically active enzyme together with its bound coenzyme and/or metal ions is called a holoenzyme. The protein part of such an enzyme is called the apoenzyme or apoprotein. Coenzymes act as transient carriers of specific functional groups. Most are derived from vitamins, organic nutrients required in small amounts in the diet.

cofactor	enzyme or protein
Zn++	carbonic anhydrase
Zn++	alcohol dehydrogenase
Fe+++ or Fe++	cytochromes, hemoglobin
Fe+++ or Fe++	ferredoxin
Cu++ or Cu+	cytochrome oxidase
K+ and Mg++	pyruvate phosphokinase

Lecture 2

Mechanism of enzyme action: Single, bi and multi substrate reactions; Lock and Key model, Induced fit model

Single substrate reaction: When an enzyme catalyzing a reaction involving sigle substrate and yielding single product it is called single substrate reactions. Enzymes with single-substrate mechanisms include isomerases such as triosephosphateisomerase or bisphosphoglycerate mutase, intramolecular lyases such as adenylate cyclase and the hammerhead ribozyme, an RNA lyase.

$$E+S \rightarrow ES \rightarrow E+ P$$

Bi- substrate reaction: When an enzyme catalyzing a reaction involving two substrates and yielding two products it is called bi-substrate reactions. It is account for 60% of the known enzymatic reactions.

$$E+S_1+S_2 \rightarrow ES_1S_2 \rightarrow E+ P_1+P_2$$

Multi substrate reaction: There are three general mechanisms which describe multi-substrate enzyme system.

- Ordered mechanism
- Random mechanism
- Ping-Pong mechanism

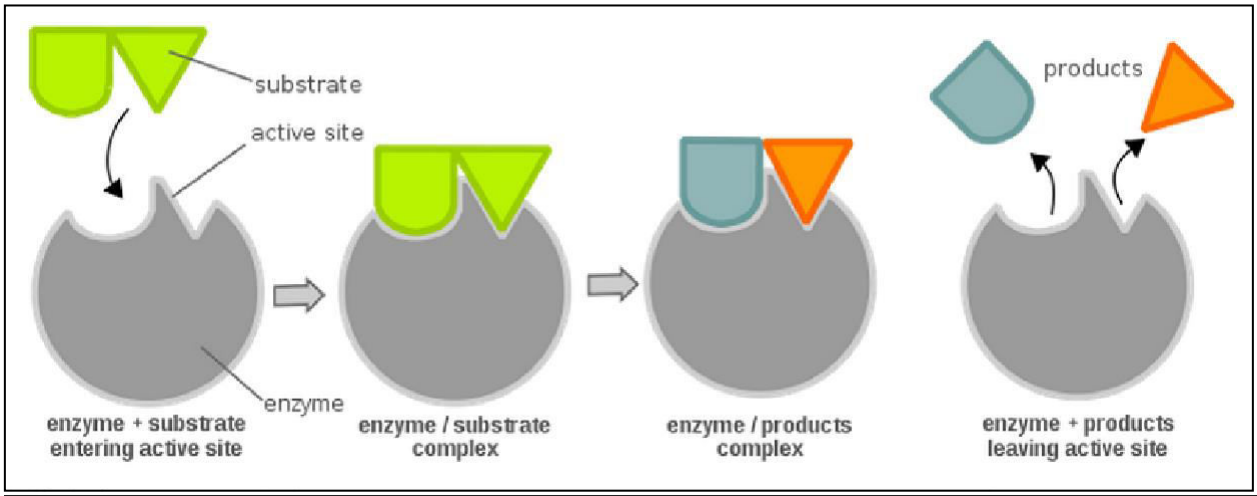
Ordered mechanism: In this type of reaction all substrates must bind to the enzyme before any product is released. Consequently, in a bisubstrate reaction, a ternary complex of the enzyme and both substrates forms. In ordered mechanism the substrates bind the enzyme in a defined sequence. Many enzymes that have NAD+ or NADH as a substrate exhibit the sequential ordered mechanism. Consider lactate dehydrogenase, an important enzyme in glucose metabolism. This enzyme reduces pyruvate to lactate while oxidizing NADH to NAD+. In the ordered sequential mechanism, the coenzyme always binds first and the lactate is always released first.

Random mechanism: In this mechanism also enzyme exists as a ternary complex: first, consisting of the enzyme and substrates and, after catalysis, the enzyme and products. In the random sequential mechanism, the order of addition of substrates and release of products is random. Sequential random reactions are illustrated by the formation of phosphocreatine and ADP from ATP and creatine, a reaction catalyzed by creatine kinase. Phosphocreatine is an important energy

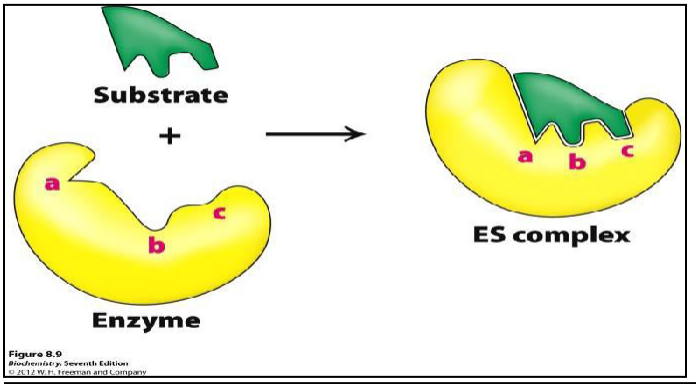
source in muscle. Sequential random reactions can also be depicted as below. Although the order of certain events is random, the reaction still passes through the ternary complexes including, first, substrates and, then, products.

Ping-Pong mechanism: In double-displacement, or Ping-Pong, reactions, one or more products are released before all substrates bind the enzyme. The defining feature of double-displacement reactions is the existence of a substituted enzyme intermediate, in which the enzyme is temporarily modified. Reactions that shuttle amino groups between amino acids and α-keto acids are classic examples of double-displacement mechanisms. The enzyme aspartate aminotransferase catalyzes the transfer of an amino group from aspartate to α-ketoglutarate. After aspartate binds to the enzyme, the enzyme removes aspartate's amino group to form the substituted enzyme intermediate. The first product, oxaloacetate, subsequently departs. The second substrate, α-ketoglutarate, binds to the enzyme, accepts the amino group from the modified enzyme, and is then released as the final product, glutamate. In this, the substrates appear to bounce on and off the enzyme analogously to a Ping-Pong ball bouncing on a table.

Lock and Key model: The specific action of an enzyme with a single substrate can be explained using a Lock and Key analogy first postulated in 1894 by Emil Fischer. In this analogy, the lock is the enzyme and the key is the substrate. Only the correctly sized key(substrate) fits into the key hole (active site) of the lock(enzyme).



Induced fit model: The **induced-fit model**, proposed by Daniel Koshland in 1958, attempts to explain how this is accomplished. His theory asserts that when the active site on the enzymes makes contact with the proper substrate, the enzyme molds itself to the shape of the molecule.

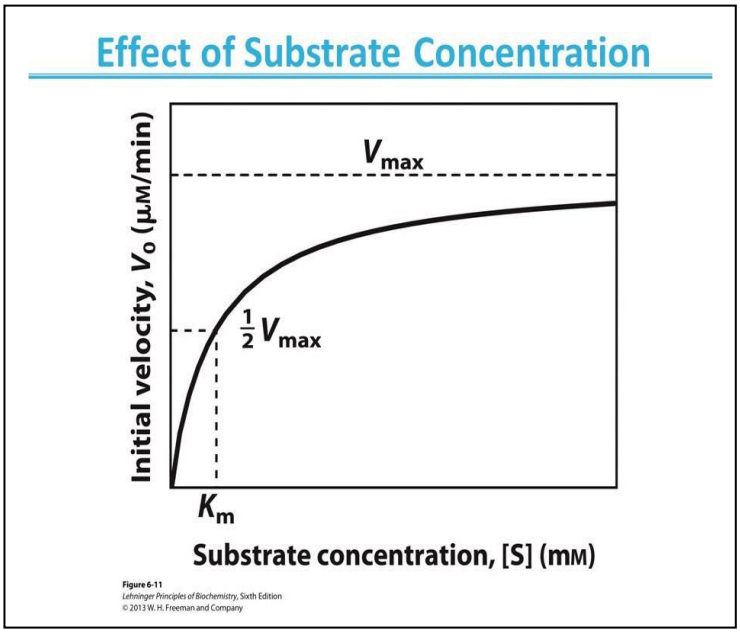


Lecture 3

Enzyme kinetics: MME, Significance of MM Constant

The Michaelis-Menten Equation: The primary function of enzymes is to enhance rates of reactions so that they are compatible with the needs of the organism. To understand how enzymes function, we need a kinetic description of their activity. For many enzymes, the rate of catalysis V_0, which is defined as the number of moles of product formed per second, varies with the substrate concentration [S]. The rate of catalysis rises linearly as substrate concentration increases and then begins to level off and approach a maximum at higher substrate concentrations.

$$V_0 = \frac{V_{max}[S]}{(K_M + [S])}$$



The Significance of Km and Vmax Values:

- The Km value for an enzyme depends on the particular substrate and on environmental conditions such as pH, temperature, and ionic strength.
- Km is the concentration of substrate at which half the active sites are filled. Thus, Km provides a measure of the substrate concentration required for significant catalysis to occur.
- Km is equal to the dissociation constant of the ES complex if k2 is much smaller than k-1 .
- High Km indicates weak binding; a low Km indicates strong binding. Km indicates the affinity of the ES complex only when k-1 is much greater than k2.
- The maximal rate, V max, reveals the turnover number of an enzyme, which is the number of substrate molecules converted into product by an enzyme molecule in a unit time when the enzyme is fully saturated with substrate.

Lecture 4

Enzyme inhibition: Reversible, Irreversible

Enzyme inhibition:

- Inhibitors are molecules that often resemble the substrate(s) or product(s) and bind to the active site thus they interfere with catalysis, slowing or halting enzymatic reactions.

- Many drugs are reversible enzyme inhibitors. They have their physiological effect by decreasing the activity of a specific enzyme. For example, aspirin (acetylsalicylate) inhibits the enzyme that catalyzes the first step in the synthesis of prostaglandins, compounds involved in many processes, including some that produce pain.
- The concentration of inhibitor needed to inhibit the enzyme depends on how tightly the inhibitor binds to the enzyme.
- The inhibition constant (Ki) is used to describe how tightly an inhibitor binds to an enzyme. The bigger the Ki, the weaker the binding.

Types of Inhibitors: There are two broad classes of enzyme inhibitors

- Irreversible
- Reversible

Irreversible: The irreversible inhibitors are those that bind covalently with or destroy a functional group on an enzyme that is essential for the enzyme's activity, or those that form a particularly stable noncovalent association. Formation of a covalent link between an irreversible inhibitor and an enzyme is common. For example reaction of chymotrypsin with diisopropylfluorophosphate (DIFP) irreversibly inhibits the enzyme by binding with Ser195 in the active-site of chymotrypsin.

Reversible: This type of inhibition involves equilibrium between enzyme and the inhibitor, the equilibrium constant (ki) being the measure of affinity of the inhibitor for the enzyme. This inhibition is further classified into three categories

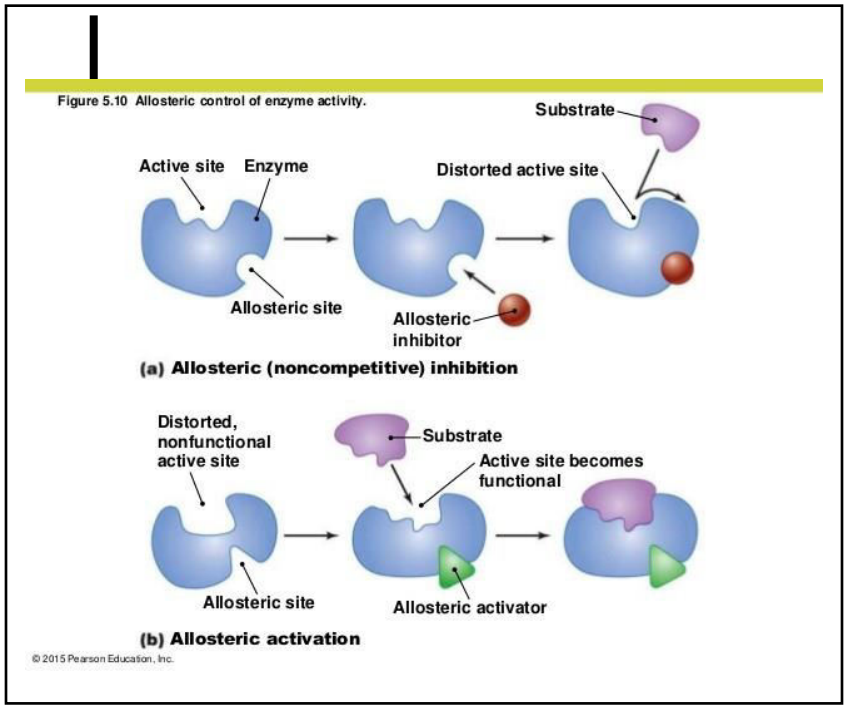
- Competitive
- Uncompetitive
- Noncompetitive

Competitive Inhibition: Competitive inhibitors bind only to the free enzyme and to the same site as the substrate. Competitive inhibitors are molecules that usually look like the substrate but can't undergo the reaction. At an infinite concentration of the substrate the competitive inhibitor cannot bind to the enzyme since the substrate concentration is high enough that there is virtually no free enzyme present. At low concentrations of substrate ([S] << Km), the enzyme is

predominantly in the E form. The competitive inhibitor can combine with E, so the presence of the inhibitor decreases the velocity when the substrate concentration is low. Under competitive inhibition Vmax remains unchanged ; Km increases

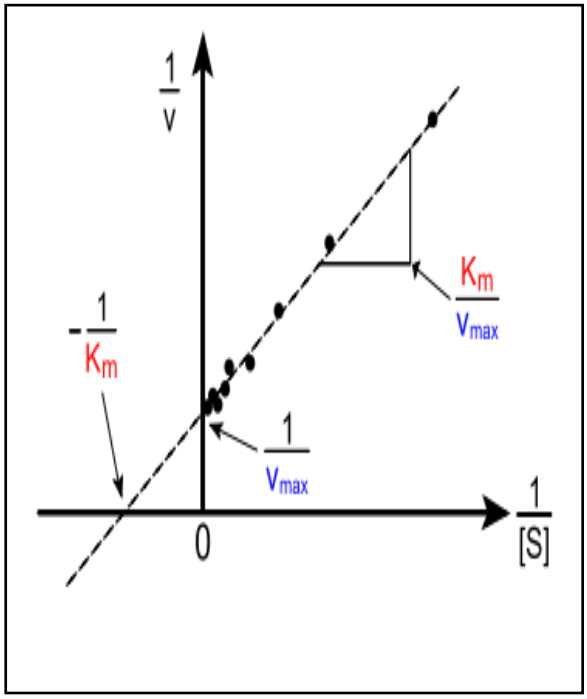
Noncompetitive Inhibition:

Compounds that reversibly bind with either the enzyme or the enzyme substrate complex are designed as noncompetitive inhibitors. Noncompetitive inhibition therefore differs from competitive inhibition in that the inhibitor can combine with ES, and S can combine with EI to form in both instances EIS. This type of inhibition is not completely reversed by high substrate concentration since closed sequence will occur regardless of the substrate concentration. Since inhibitor binding site is not identical to nor does it modify the active site directly, the Km is not altered but Vmax is decreased.

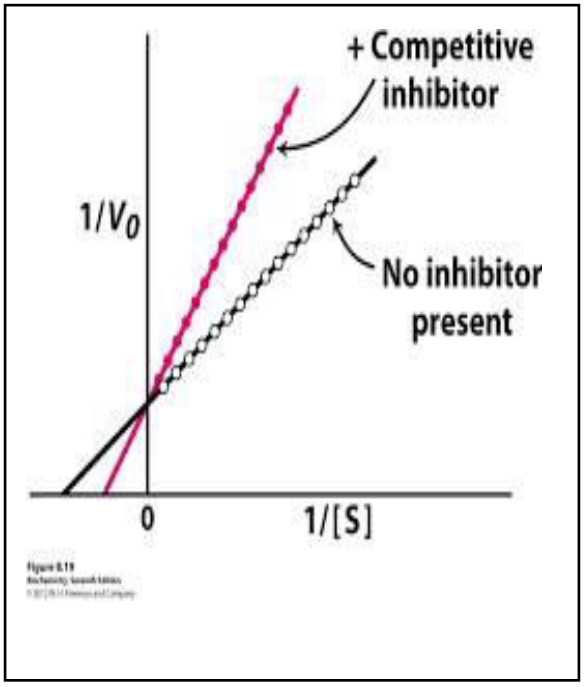


Lecture 5

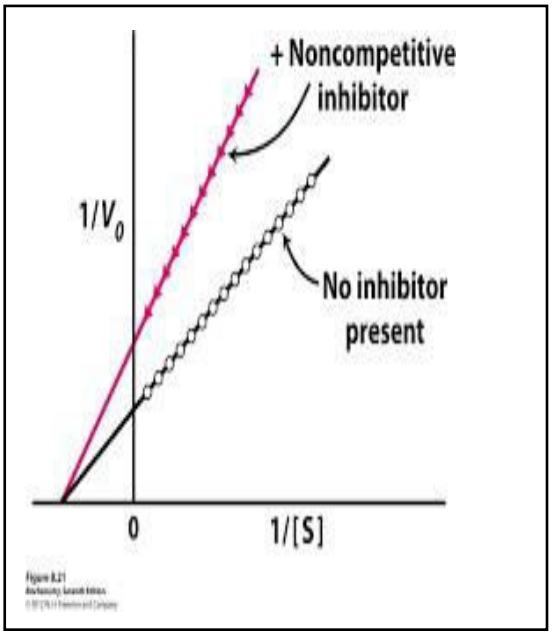
Lineweaver-Burk Plot



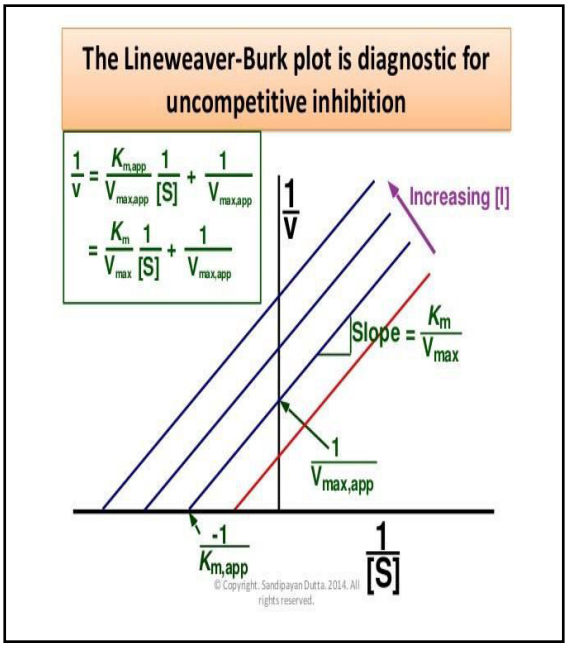
LB plot for No Inhibition



LB plot for Competitive Inhibition



LB plot for Noncompetitive Inhibition



LB plot for Uncompetitive Inhibit

Lecture 6

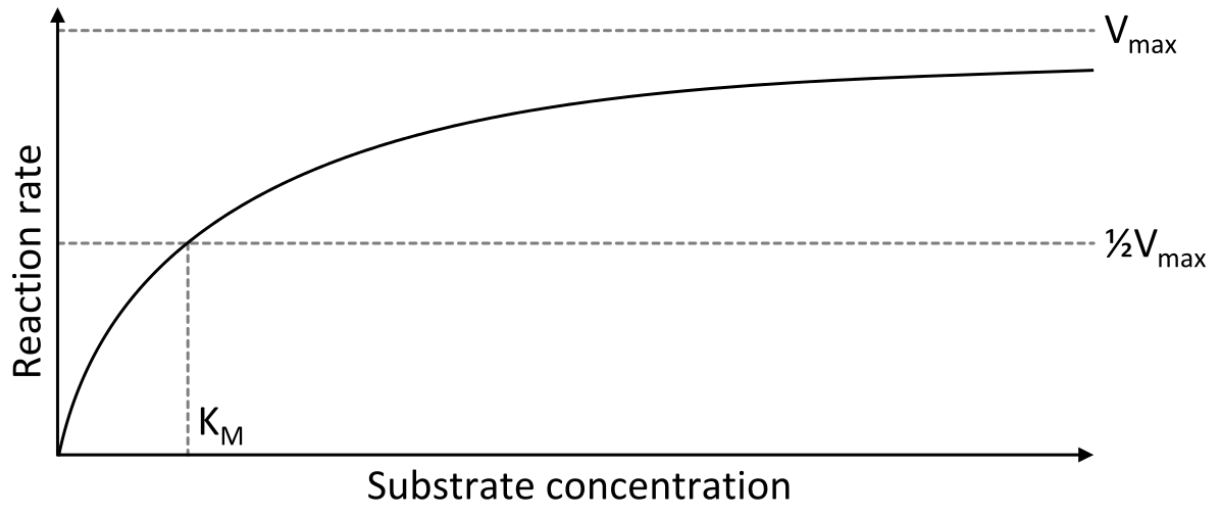
MME and Allosteric enzyme kintics, Allosteric Regulation

Michaelis-Menten Kinetics:

In biochemistry, *'Michaelis–Menten' kinetics* is one of the best-known models of enzyme kinetics. It is named after German biochemist Leonor Michaelis and Canadian physician Maud Menten. The model takes the form of an equation describing the rate of enzymatic reactions, by relating reaction rate V to [S], the concentration of a substrate *S*. Its formula is given by:

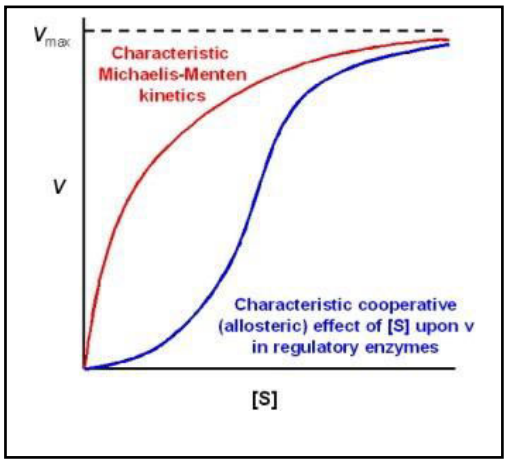
$$V_0 = \frac{V_{\max}[S]}{(K_M + [S])}$$

This equation is called the **Michaelis–Menten equation**. Here, V_{max} represents the maximum rate achieved by the system, at saturating substrate concentration. The Michaelis constant K_M is the substrate concentration at which the reaction rate is half of V_{max}.

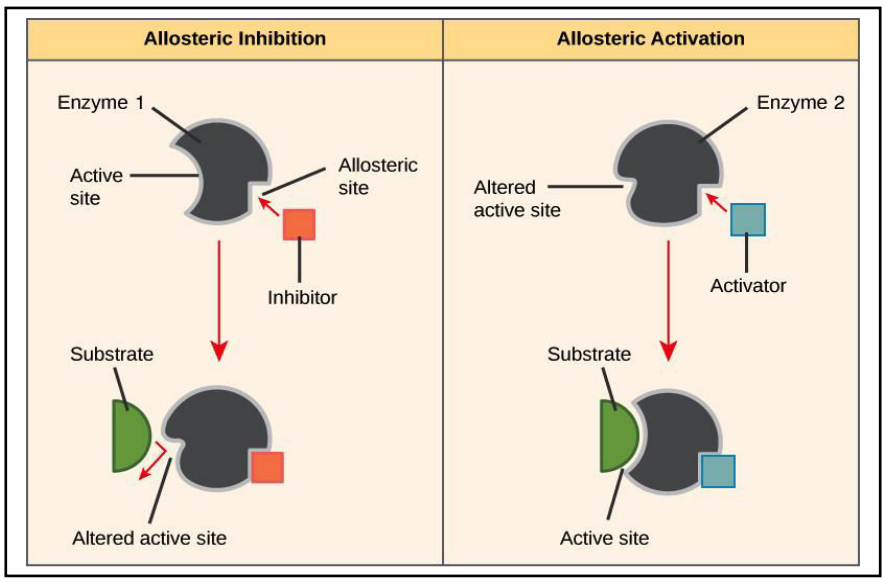


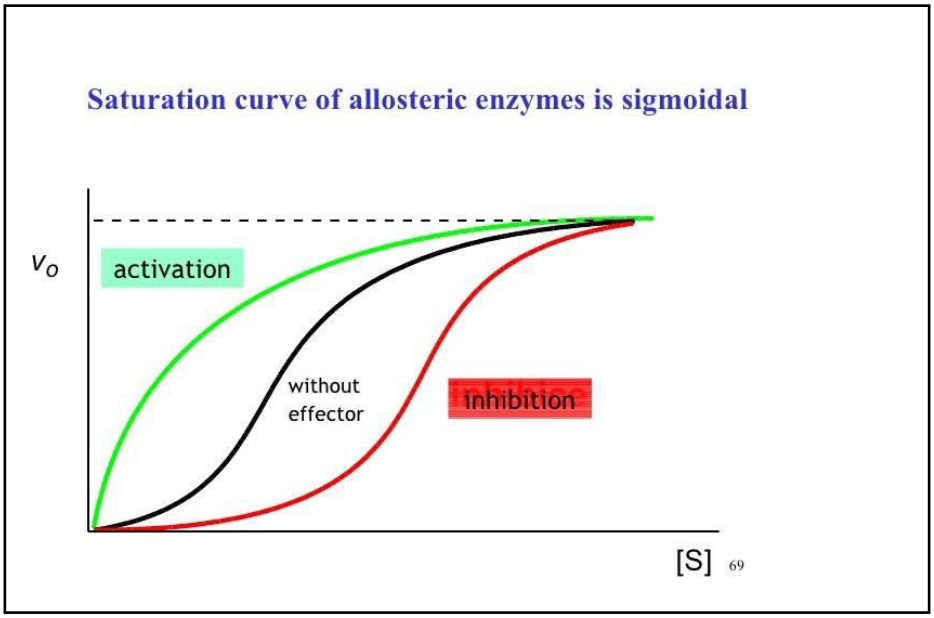
Allosteric enzyme kintics:

Allosteric enzymes are enzymes that change their conformational ensemble upon binding of an effector, which results in an apparent change in binding affinity at a different ligand binding site. This "action at a distance" through binding of one ligand affecting the binding of another at a distinctly different site, is the essence of the allosteric concept. Allostery plays a crucial role in many fundamental biological processes, including but not limited to cell signaling and the regulation of metabolism. The site to which the effector binds is termed the allosteric site. Allosteric sites allow effectors to bind to the protein, often resulting in a conformational change involving protein dynamics. Effectors that enhance the protein's activity are referred to as allosteric activators, whereas those that decrease the protein's activity are called allosteric inhibitors.



Allosteric Regulation:

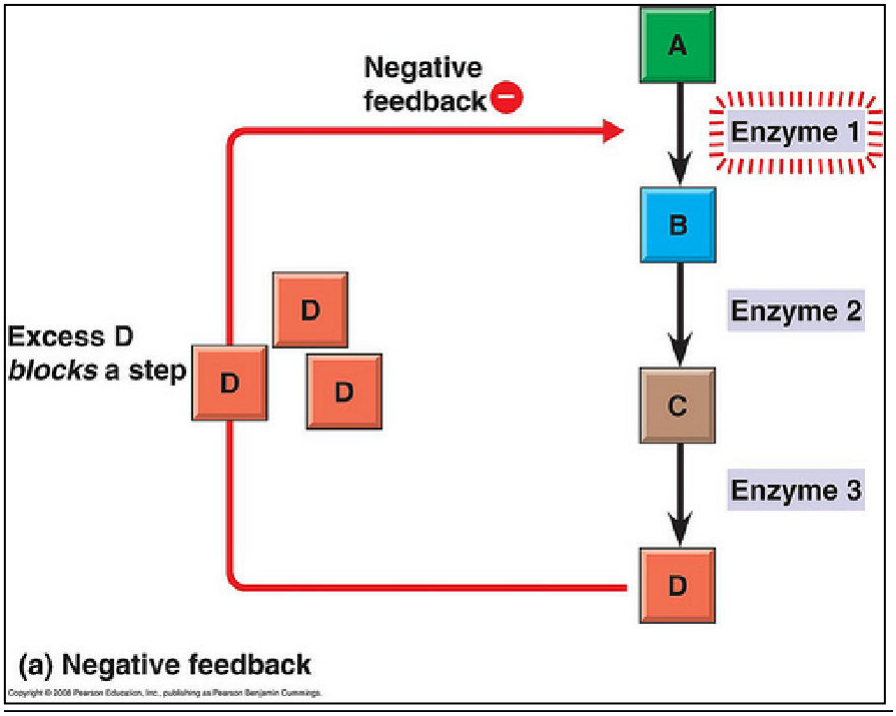




Lecture 7

Feedback inhibition

Feedback inhibition is the phenomenon where the output of a process is used as an input to control the behavior of the process itself, oftentimes limiting the production of more product. Many enzyme catalyzed reactions are carried out through a biochemical pathway. In these pathways, the product of one reaction becomes the substrate for the next reaction. At the end of the pathway, a desired product is synthesized. In order to tightly regulate the concentration of that product, the biochemical pathway needs to be shut down. This is done through feedback inhibition. The product of the final reaction in that pathway reacts with an enzyme somewhere along the pathway at the enzyme's allosteric site, changing the conformation of the enzyme. That enzyme can no longer bind to its substrate as effectively due to the conformational change, closing down that pathway and stopping the final product from synthesizing. The higher the concentration of the final product, the more likely that product will bind to the allosteric site of the enzyme, shutting down that pathway.



Lecture 8

Substrate acts as inhibitor, Turn over number

Substrate acts as inhibitor:

Phosphofructokinase-1 (PFK-1) is one of the most important regulatory enzymes of glycolysis. It is an allosteric enzyme made of 4 subunits and controlled by many activators and inhibitors. PFK-1 catalyzes the important step of glycolysis, the conversion of fructose 6-phosphate and ATP to fructose 1,6-bisphosphate and ADP. Glycolysis is the foundation for respiration, both anaerobic and aerobic. Because phosphofructokinase (PFK) catalyzes the ATP-dependent phosphorylation to convert fructose-6-phosphate into fructose 1,6-bisphosphate and ADP, it is one of the key regulatory and rate limiting steps of glycolysis. PFK is able to regulate glycolysis through allosteric inhibition, and in this way, the cell can increase or decrease the rate of glycolysis in response to the cell's energy requirements. For example, a high ratio of ATP to ADP will inhibit PFK and glycolysis. The key difference between the regulation of PFK in eukaryotes and prokaryotes is that in eukaryotes PFK is activated by fructose 2,6-bisphosphate. The purpose of fructose 2,6-bisphosphate is to supersede ATP inhibition, thus allowing eukaryotes to have greater sensitivity to regulation by hormones like glucagon and insulin.

Turn over number:

In enzymology, turnover number (also termed k_{cat}) is defined as the maximum number of chemical conversions of substrate molecules per second that a single catalytic site will execute for a given enzyme concentration [E_T]. It can be calculated from the maximum reaction rate V_{max} and catalyst site concentration [E_T]. as follows:

$$k_{cat} = V_{max} / [E_T]$$

For example, carbonic anhydrase has a turnover number of 400,000 to 600,000 s^{-1}, which means that each carbonic anhydrase molecule can produce up to 600,000 molecules of product (bicarbonate ions) per second.

Module 3 8L

Lecture 1

Carbohydrates; Definition & classification; General chemistry of carbohydrates

Definition: Carbohydrate is an organic molecule found in animals, plants etc., and composed of carbon, hydrogen and oxygen.Sometimes it is called hydrate of carbon.

EmpiricalFormula :$C_x(H_2O)_y$

Sources:

- Cereals
- Pulses
- Fruits and Vegetables
- Nuts and oilseeds
- Miscellaneous: Sugar, jiggery, honey, dates, skim milk powder etc.

Classification of Carbohydrates:

Carbohydrates can be classified in two ways:

I. Classification depending on their digestibility:

- **Available Carbohydrates**: The available carbohydrates, that is sugar plus starch, were defined as those that are digested and absorbed by the human small intestine and which are glucogenic.
- **Unavailable Carbohydrate:** The unavailable carbohydrates were defined as those that are not digested by the endogenous secretions of the human digestive tract. These are now generally referred to as dietary fiber.

II. Chemical classification of carbohydrates:

- **Monosaccharides:** Monosaccharides are simple sugars, which possess a free ketone or aldehyde group. Being the simplest of sugars, they cannot be further hydrolyzed. Their chemical formula is $C_nH_{2n}O_n$ or $C_n(H_2O)_n$. Monosaccharides are classified into tiroses, tertroses, pentoses, etc., and as ketoses or aldoses, depending on their ketone or aldehyde group.

Examples include glucose, fructose, galactose, glycerose, ribose, and ribulose.

- **Oligosaccharides:**"Oligo" meaning few, oligosaccharides are sugars that break down into two to 10 molecules of monosaccharides when hydrolyzed.

An oligosaccharide that yields two monosaccharide molecules on hydrolysis is a

disaccharide, while those that break down into three or four monosaccharides are called trisaccharides, tetrasaccharides, and so on. Disaccharides have a chemical formula of $C_n(H_2O)_{n-1}$ while trisaccharides and others are $C_n(H_2O)_{n-2,}$ etc.

Oligosaccharide examples include sucrose, maltose, lactose, raffinose, and stachyose.

- **Polysaccharides:** "Poly" meaning many, polysaccharides are compound molecules that yield more than ten monosaccharide molecules on hydrolysis. They are also classified depending on the type of molecules hydrolyzed. These include homopolysaccharides (with several monosaccharides of one type) or heteropolysaccharides (with different types of monosaccharides). $(C_6H_{10}O_5)_x$ is their chemical formula.

Polysaccharide examples include starch, cellulose, pectin, glycogen, inulin, and hyaluonic acid.

Lecture 2

Metabolic pathways for breakdown of carbohydrates: glycolytic pathway; importance, energy yield

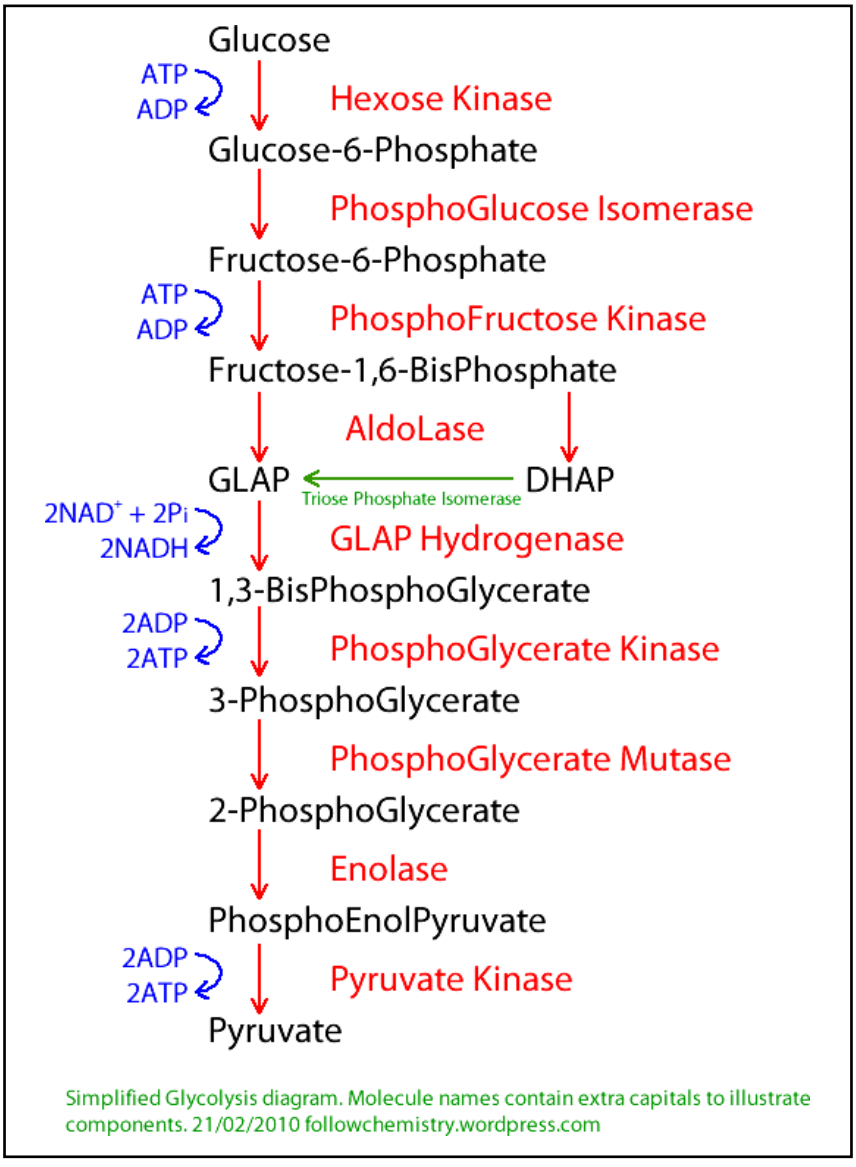
Glycolysis is a set of reactions that take place in cytoplasm of prokaryotes and eukaryotes.Glycolysis is an almost universal central pathway of glucose catabolism .The major roles of glycolysis are to produce energy and to produce intermediates for biosynthetic pathways.

Glycolysis has two Phases

- **Preparatory phase**
- **Pay-off Phase**

In the preparatory phase of glycolysis, two molecules of ATP are invested and the hexose chain is cleaved into two triose phosphates. The payoff phase of glycolysis includes the energy-

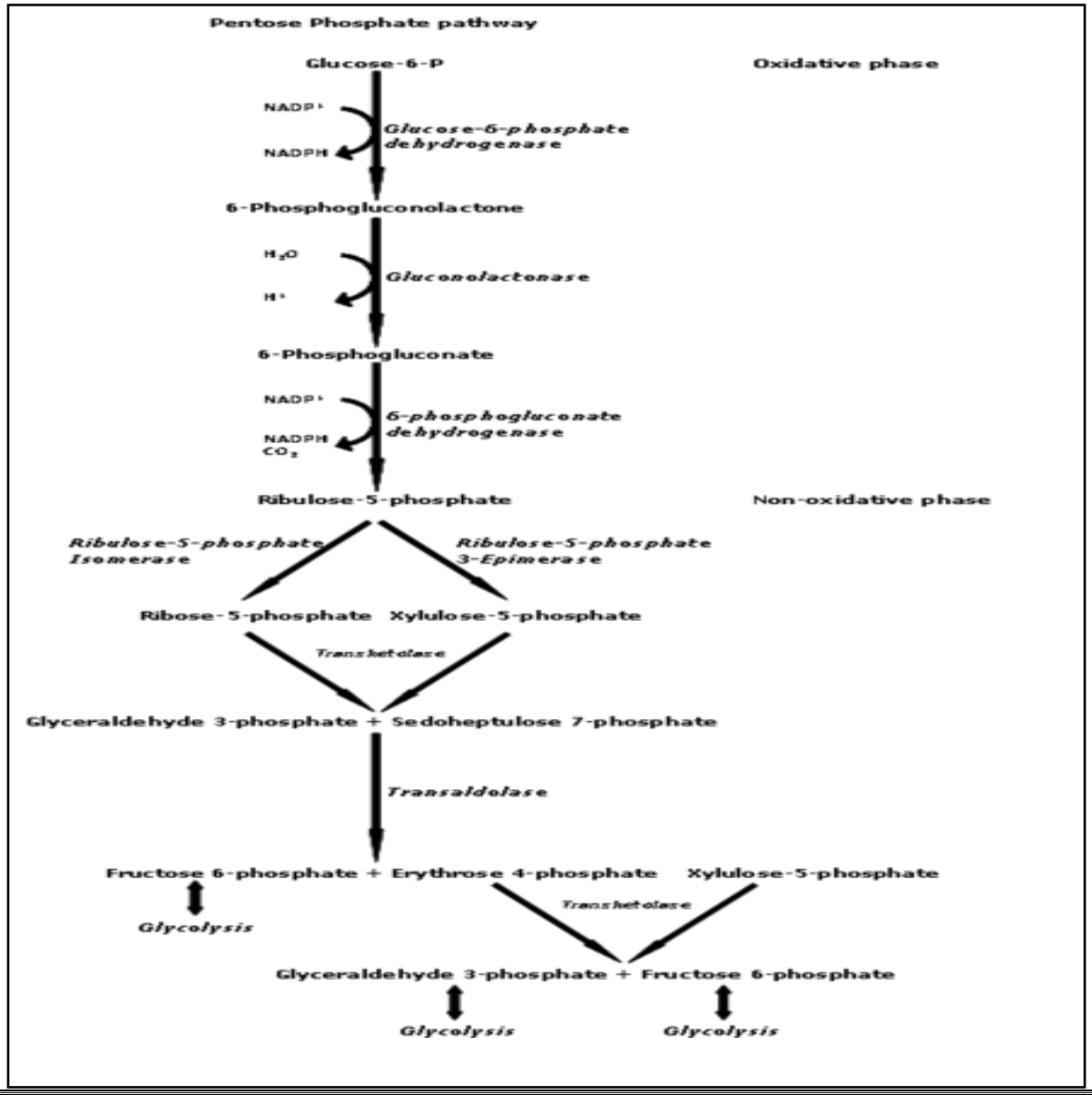
conserving phosphorylation steps in which some of the free energy of the glucose molecule is conserved in the form of ATP. One molecule of glucose yields two molecules of glyceraldehydes 3-phosphate; both halves of the glucose molecule follow the same pathway in the second phase of glycolysis. The conversion of two molecules of glyceraldehyde 3-phosphate to two molecules of pyruvate is accompanied by the formation of four molecules of ATP from ADP. However, the net yield of ATP per molecule of glucose degraded is only two, because two ATP were invested in the preparatory phase of glycolysis to phosphorylate the two ends of the hexose molecule. For each molecule of glucose degraded to pyruvate, two molecules of ATP are generated from ADP and Pi.



Lecture 3

Pentose phosphate pathway, importance, energy yield

The pentose phosphate pathway (also called the phosphogluconate pathway and the hexose monophosphate shunt) is a metabolic pathway parallel to glycolysis.It generates NADPH and pentoses (5-carbon sugars) as well as ribose 5-phosphate, the last one a precursor for the synthesis of nucleotides. While it does involve oxidation of glucose, its primary role is anabolic rather than catabolic. There are two distinct phases in the pathway. The first is the oxidative phase, in which NADPH is generated, and the second is the non-oxidative synthesis of 5-carbon sugars. For most organisms, the pentose phosphate pathway takes place in the cytosol; in plants, most steps take place in plastids.



Lecture 4

Citric acid cycle: Pathway, importance, energy yield

The citric acid cycle, also known as the TCA (tricarboxylic acid) cycle or Krebs cycle (after its discoverer in 1937), is used to oxidize the pyruvate formed during the glycolytic breakdown of glucose into CO2 and H2O. The cycle is a major energy source in the form of ATP and also produces precursors for many biosynthetic pathways. The citric acid cycle operates in the mitochondria of eukaryotes and in the cytosol of prokaryotes. Succinate dehydrogenase, the only membrane-bound enzyme in the citric acid cycle, is embedded in the inner mitochondrial membrane in eukaryotes and in the plasma membrane in prokaryotes.

The cycle

The cycle forms the central part of a three-step process which oxidizes organic fuel molecules into CO_2 with the concomitant production of ATP.

Step 1 – Oxidation of fuel molecules to acetyl CoA

A major source of energy is glucose which is converted by glycolysis into pyruvate. Pyruvate dehydrogenase (a complex of three enzymes and five coenzymes) then oxidizes the pyruvate (using NAD+ which is reduced to NADH) to form acetyl CoA and CO_2. Since the reaction involves both an oxidation and a loss of CO_2, the process is called oxidative decarboxylation.

Step 2 – The citric acid cycle

The cycle carries out the oxidation of acetyl groups from acetyl CoA to CO_2 with the production of four pairs of electrons, stored initially in the reduced electron carriers NADH and $FADH_2$.

The cycle has eight stages:

1. Citrate (6C) is formed from the irreversible condensation of acetyl CoA (2C) and oxaloacetate (4C) – catalyzed by citrate synthase.

2. Citrate is converted to isocitrate (6C) by an isomerization catalyzed by aconitase. This is actually a two-step reaction during which cis-aconitate is formed as an intermediate. It is the cis-aconitate which gives the enzyme its name.

3. Isocitrate is oxidized to α-ketoglutarate (5C) and CO2 by isocitrate dehydrognase. This mitrochondrial enzyme requires NAD+, which is reduced to NADH.

4. A-Ketoglutarate is oxidized to succinyl CoA (4C) and CO2 by the α-ketoglutarate dehydrogenase complex. Like pyruvate dehydrogenase, this is a complex of three enzymes and uses NAD+ as a cofactor.

5. Succinyl CoA is converted to succinate (4C) by succinyl CoA synthetase. The reaction uses the energy released by cleavage of the succinyl-CoA bond to synthesize either GTP (mainly in animals) or ATP (exclusively in plants) from Pi and, respectively, GDP or ADP.

6. Succinate is oxidized to fumarate (4C) by succinate dehydrogenase. FAD is tightly bound to the enzyme and is reduced to produce FADH2.

7. Fumerate is converted to malate (4C) by fumarase; this is a hydration reaction requiring the addition of a water molecule.

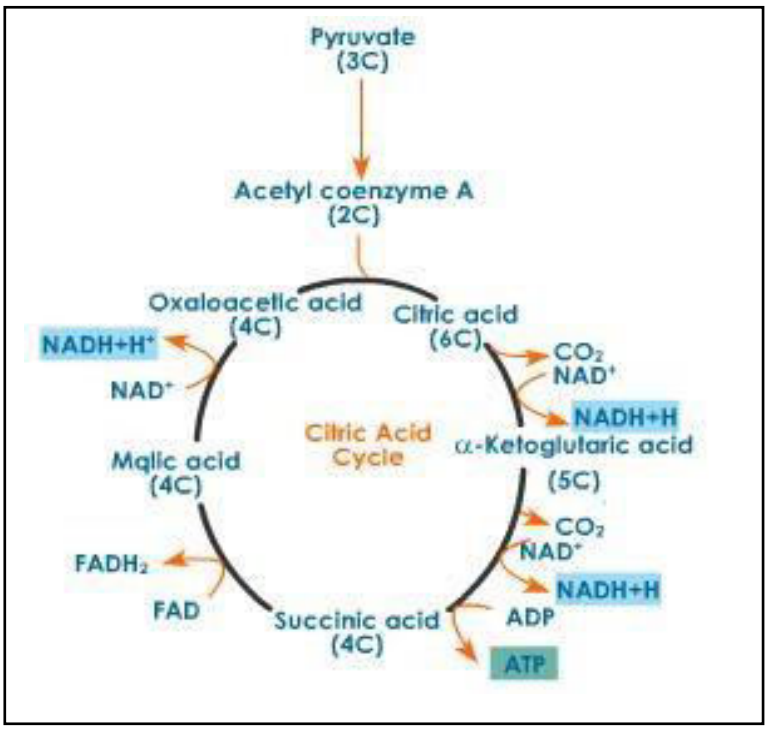
8. Malate is oxidized to oxaloacetate (4C) by malate dehydrogenase. NAD+ is again required by the enzyme as a cofactor to accept the free pair of electrons and produce NADH.

Step 3 – Oxidation of NADH and FADH2 produced by the citric acid cycle

The NADH and FADH2 produced by the citric acid cycle are reoxidized and the energy released is used to synthesize ATP by oxidative phosphorylation.

Energy Yield

Each of the three NADH molecules produced per turn of the cycle yields three ATPs and the single $FADH_2$ yields two ATPs by oxidative phosphorylation. One GTP (or ATP) is synthesized directly during the conversion of succinyl CoA to succinate. Thus the oxidation of a single molecule of glucose via the citric acid cycle produces 12 ATP molecules.



Lecture 5

Electron transport chain: Pathway, importance, Energy yield, Oxidative phosphorylation

- In eukaryotes, electron transport and oxidative phosphorylation occur in the inner membrane of mitochondria.
- These processes re-oxidize the NADH and FADH2 that arise from the citric acid cycle (located in the mitochondrial matrix), glycolysis (located in the cytoplasm) and fatty acid oxidation (located in the mitochondrial matrix) and trap the energy released as ATP.
- Oxidative phosphorylation is by far the major source of ATP in the cell. In prokaryotes, the components of electron transport and oxidative phosphorylation are located in the plasma membrane.
- NADH oxidation and ATP synthesis do not occur in a single step. Electrons are not transferred from NADH to oxygen directly. Rather the electrons are transferred from NADH to oxygen along a chain of electron carriers collectively called the electron transport chain (also called the respiratory chain).

Electron Transport Chain:

Electron transport chain consists of three large protein complexes embedded in the inner mitochondrial membrane,

- NADH dehydrogenase complex (Complex I)
- Succinate Q reductase (Complex II)
- The cytochrome bc1 complex (Complex III)
- Cytochrome oxidase (Complex IV)

Each complex contains several electron carriers that work sequentially to carry electrons down the chain. Two free electron carriers are also needed to link these large complexes;

- Ubiquinone also called as coenzyme Q (CoQ)
- Cytochrome c

ATP Synthesis (Oxidative Phosphorylation)

- Oxidative phosphorylation is also the name given to the synthesis of ATP (phosphorylation) that occurs when NADH and FADH2 are oxidized (hence oxidative) by electron transport through the respiratory chain. Unlike substrate level phosphorylation, it does not involve phosphorylated chemical intermediates.
- This proposes that energy liberated by electron transport is used to create a proton gradient across the mitochondrial inner membrane and that is used to drive ATP synthesis (chemiosmotic hypothesis). Thus the proton gradient couples electron transport and ATP synthesis, not a chemical intermediate as in substrate level phosphorylation.

- The actual synthesis of ATP is carried out by an enzyme called ATP synthase located in the inner mitochondrial membrane.

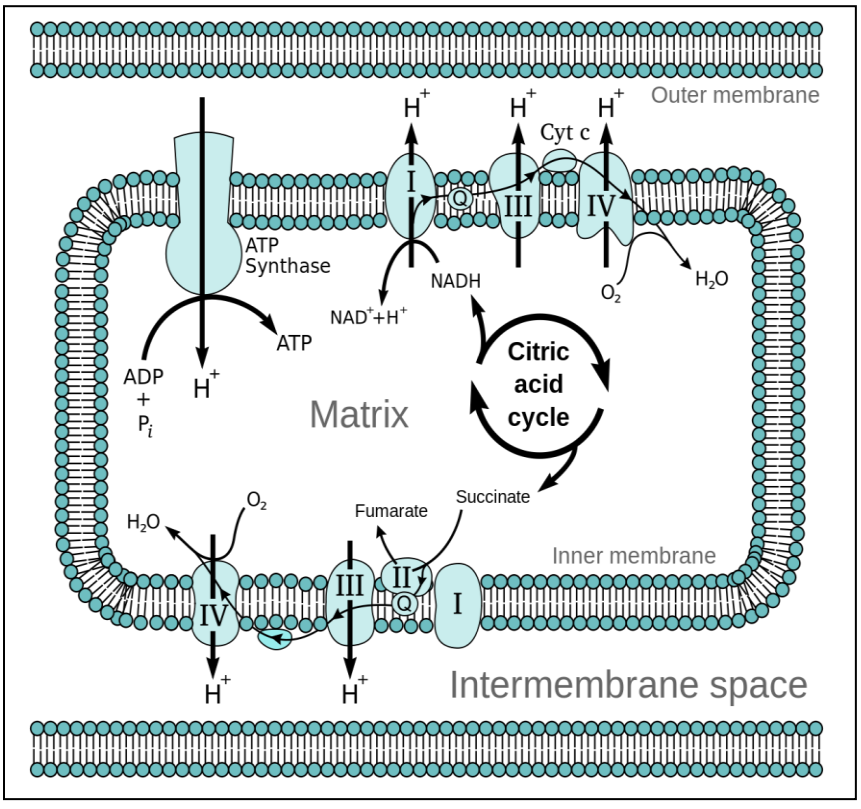
Summary:

- In brief, Electron transport down the respiratory chain from NADH oxidation causes H+ ions to be pumped out of the mitochondrial matrix across the inner mitochondrial membrane into the intermembrane space by the three H+ pumps; NADH dehydrogenase, the cytochrome bc1 complex and cytochrome oxidase.

- The free energy change in transporting an electrically charged ion across a membrane leads to the formation of electrochemical proton gradient. The pumping out of the H+ ions generates a higher concentration of H+ ions in the intermembrane space and an electricalnpotential. Thus, the side of the inner mitochondrial membrane facing the intermembrane space being positive.

- The protons flow back into the mitochondrial matrix according to electrochemical gradient through the ATP synthase and this drives ATP synthesis. The ATP synthase is driven by proton-motive force, which is the sum of the pH gradient (i.e. the chemical gradient of H+ ions) and the membrane potential (i.e. the electrical charge potential across the inner mitochondrial membrane).

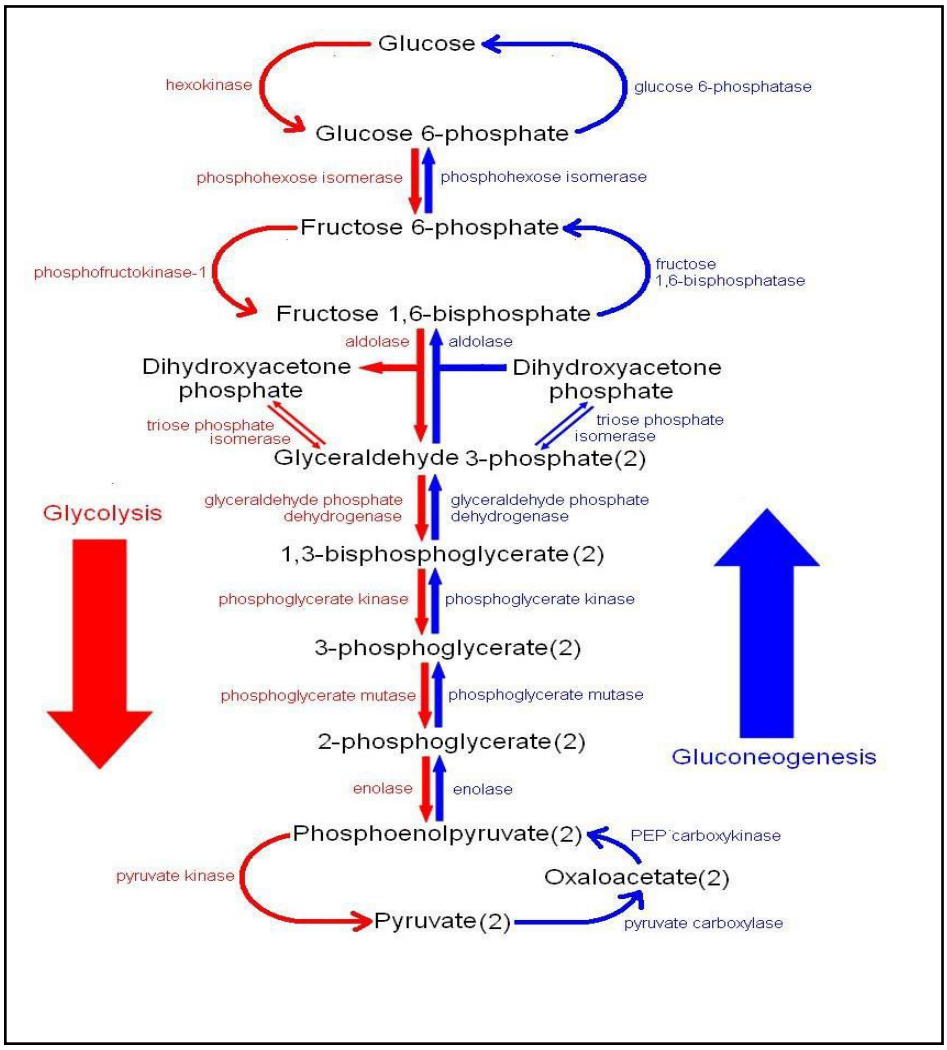
- FADH2 is reoxidized via ubiquinone , its oxidation causes H+ ions to be pumped out only by the cytochrome bc1 complex and cytochrome oxidase and so the amount of ATP made from FADH2 is less than from NADH. Measurements made have shown that 2.5 ATP molecules are synthesized per NADH oxidized whereas 1.5 ATPs are synthesized per FADH2 oxidized.



Lecture 6

Gluconeogenesis;Pathway, importance, energy used, Cori cycle

- Gluconeogenesis is especially important in periods of starvation or vigorous exercise.
- During starvation, the formation of glucose via gluconeogenesis particularly uses amino acids from protein breakdown and glycerol from fat breakdown.
- During exercise, the blood glucose levels required for brain and skeletal muscle function are maintained by gluconeogenesis in the liver using lactate produced by the muscle.
- Gluconeogenesis synthesizes glucose from noncarbohydrate precursors, including lactate and pyruvate, citric acid cycle intermediates, the carbon skeletons of most amino acids and glycerol.
- This is extremely important since the brain and erythrocytes rely almost exclusively as their energy source under normal conditions. The store of liver glycogen is sufficient to supply the brain with glucose for only about half a day during fasting.
- The main site of gluconeogenesis is the liver, although it also occurs to a far lesser extent in the kidneys. Very little gluconeogenesis occurs in brain or muscle.



Energy used

As would be expected, the synthesis of glucose by gluconeogenesis needs the input of energy. Two pyruvate molecules are required to synthesize one molecule of glucose. This compares with only two ATPs as the net ATP yield from glycolysis. Thus an extra four ATPs per glucose are required to reverse glycolysis.

In fact, the glyceraldehydes 3-phosphate dehydrogenase reaction also consumes NADH, equivalent to two molecules of NADH for each molecule of glucose synthesized. Since each cytosolic NADH would normally be used to generate approximately three ATP molecules via the glycerol 3-phosphate shuttle and oxidative phosphorylation, this is equivalent to the input of another six ATPs per glucose synthesized.

Cori cycle:

The Cori cycle (also known as the Lactic acid cycle), named after its discoverers, Carl Ferdinand Coriand Gerty Cori, refers to the metabolic pathway in which lactate produced by anaerobic glycolysis in the muscles moves to the liver and is converted to glucose, which then returns to the muscles and is metabolized back to lactate.

The Cycle:

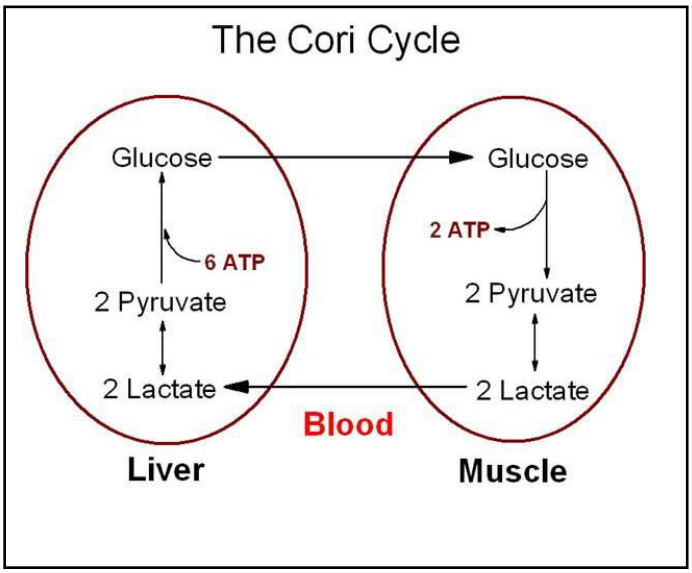
Muscular activity requires ATP, which is provided by the breakdown of glycogen in the skeletal muscles. The breakdown of glycogen, a process known as glycogenolysis, releases glucose in the form of glucose-1-phosphate (G-1-P). The G-1-P is converted to G-6-P by the enzyme phosphoglucomutase. G-6-P is readily fed into glycolysis, (or can go into the pentose phosphate pathway if G-6-P concentration is high) a process that provides ATP to the muscle cells as an energy source. During muscular activity, the store of ATP needs to be constantly replenished. When the supply of oxygen is sufficient, this energy comes from feeding pyruvate, one product of glycolysis, into the Krebs cycle.

When oxygen supply is insufficient, typically during intense muscular activity, energy must be released through anaerobic metabolism. Lactic acid fermentation converts pyruvate to lactate by lactate dehydrogenase. Most importantly, fermentation regenerates NAD^+, maintaining the NAD^+ concentration so that additional glycolysis reactions can occur. The fermentation step oxidizes the NADH produced by glycolysis back to NAD^+, transferring two electrons from NADH to reduce pyruvate into lactate. Refer to the main articles on glycolysisand fermentation for the details.

Instead of accumulating inside the muscle cells, lactate produced by anaerobic fermentation is taken up by the liver. This initiates the other half of the Cori cycle. In the

liver, gluconeogenesis occurs. From an intuitive perspective, gluconeogenesis reverses both glycolysis and fermentation by converting lactate first into pyruvate, and finally back to glucose. The glucose is then supplied to the muscles through the bloodstream; it is ready to be fed into further glycolysis reactions. If muscle activity has stopped, the glucose is used to replenish the supplies of glycogen through glycogenesis.

Overall, the glycolysis part of the cycle produces 2 ATP molecules at a cost of 6 ATP molecules consumed in the gluconeogenesis part. Each iteration of the cycle must be maintained by a net consumption of 4 ATP molecules. As a result, the cycle cannot be sustained indefinitely. The intensive consumption of ATP molecules indicates that the Cori cycle shifts the metabolic burden from the muscles to the liver.



Lecture 7

General chemistry of lipids; classification, Essential fatty acids, Metabolism of ketonebodies, alpha, beta and omega oxidation of fatty acids

In biology, a lipid is a substance of biological origin that is soluble in nonpolar solvents.[1] It comprises a group of naturally occurring molecules that include fats, waxes, sterols, fat-soluble vitamins (such as vitamins A, D, E, and K), monoglycerides, diglycerides, triglycerides, phospholipids, and others. The main biological functions of lipids include storing energy, signaling, and acting as structural components of cell membranes.

Classification:

1. Fatty acids
2. Glycerolipids
3. Glycerophospholipids
4. Sphingolipids
5. Sterol lipids
6. Prenol lipids
7. Saccharolipids
8. Polyketides

Essential Fatty Acids:

The human body is capable of producing all the fatty acids it needs, except for two: linoleic acid (LA), an omega-6 fatty acid, and alpha-linolenic acid (ALA), an omega-3 fatty acid. These have to be consumed from the diet and are therefore termed "essential fatty acids". Both of these fatty acids are needed for growth and repair, but can also be used to make other fatty acids (e.g. arachidonic acid (AA) is formed from LA). However, as conversion to the omega-3 fatty acids eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) is limited, it is recommended that sources of these are also included in the diet. ALA and LA are found in plant and seed oils. Although the levels of LA are usually much higher than those of ALA, rapeseed oil and walnut oil are very good sources of the latter. EPA and DHA are found in oily fish (e.g., salmon, mackerel, and herring). AA can be obtained from animal sources, such as meat and egg yolk.

Metabolism of ketonebodies:

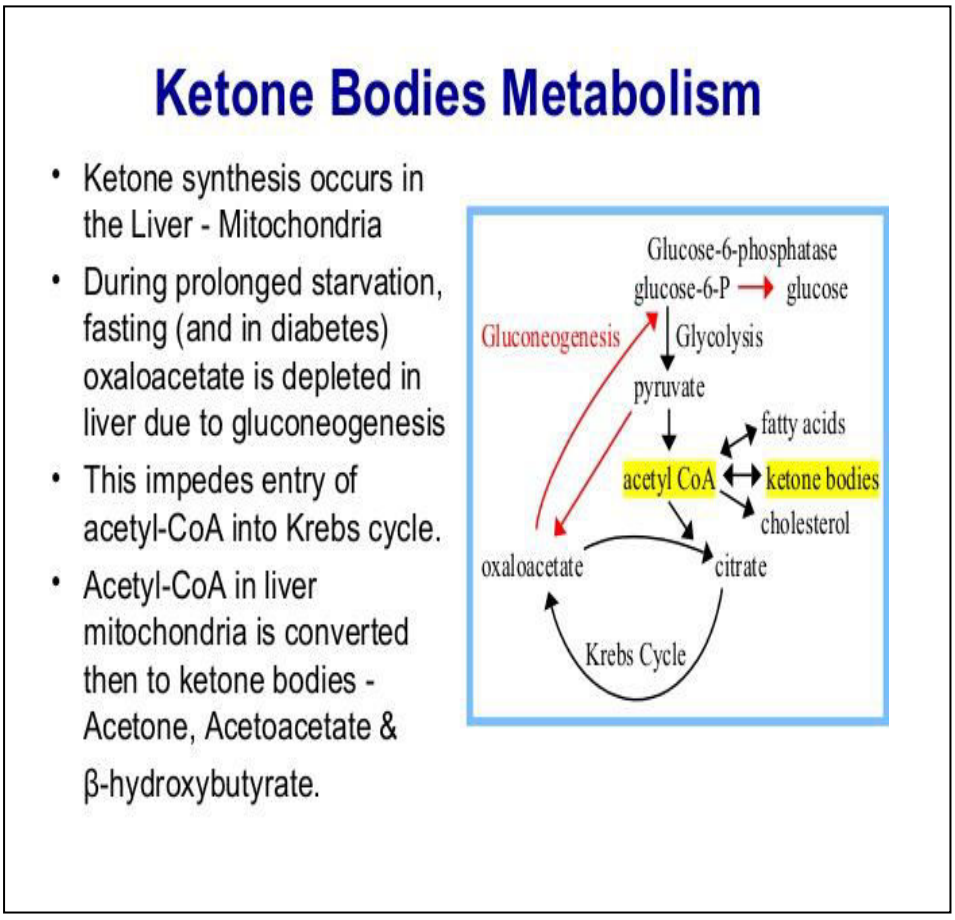
Ketone bodies are three water-soluble molecules (acetoacetate, beta-hydroxybutyrate, and their spontaneous breakdown product, acetone) that are produced by the liver from fatty acids during periods of low food intake (fasting), carbohydrate restrictive diets, starvation, prolonged intense exercise, or in untreated (or inadequately treated) type 1 diabetes mellitus. These ketone bodies are readily picked up by the extra-hepatic tissues, and converted into acetyl-CoA which then enters the citric acid cycle and is oxidized in the mitochondria for energy. In the brain, ketone bodies are also used to make acetyl-CoA into long-chain fatty acids.

Ketone bodies are produced by the liver under the circumstances listed above (i.e. fasting, starving, low carbohydrate diets, prolonged exercise and untreated type 1 diabetes mellitus) as a result of

intense gluconeogenesis, which is the production of glucose from non-carbohydrate sources (not including fatty acids). They are therefore always released into the blood by the liver together with newly produced glucose, after the liver glycogen stores have been depleted.

When two acetyl-CoA molecules lose their -CoAs, (or Co-enzyme A groups) they can form a (covalent) dimer called acetoacetate. Beta-hydroxybutyrate is a reduced form of acetoacetate, in which the ketone group is converted into an alcohol (or hydroxyl) group. Both are 4-carbon molecules, that can readily be converted back into acetyl-CoA by most tissues of the body, with the notable exception of the liver. Acetone is the decarboxylated form of acetoacetate which cannot be converted back into acetyl-CoA except via detoxification in the liver where it is converted into lactic acid, which can, in turn, be oxidized into pyruvic acid, and only then into acetyl-CoA.

Ketone bodies have a characteristic smell, which can easily be detected in the breath of persons in ketosis and ketoacidosis. It is often described as fruity or like nail polish remover (which usually contains acetone or ethyl acetate).



Ketosis:

In normal individuals, there is a constant production of ketone bodies by the liver and their utilization by extrahepatic tissues. The concentration of ketone bodies in blood is maintained around 1 mg/dl. Their excretion in urine is very low and undetectable by routine urine tests (Rothera's test).

When the rate of synthesis of ketone bodies exceeds the rate of utilization, their concentration in blood increases; this is known as *ketonemia*. This is followed by *ketonuria* – excretion of ketone bodies in urine. The overall picture of ketonemia and ketonuria is commonly referred as ketosis. The smell of acetoacetate and/or acetone in breath is a common feature in ketosis.

Alpha, beta and omega oxidation of fatty acids:

alpha-Oxidation is a process in which fatty acids are shortened by one carbon atom. It is occur in peroxisomes. Substrates are Phytanic acid, 3-methylfatty acids and their alcohol and aldehyde derivatives, metabolites of farnesol, geranylgeraniol, and dolichols.

Alpha-oxidation of phytanic acid is believed to take place entirely within peroxisomes.

1. Phytanic acid is first attached to CoA to form phytanoyl-CoA.
2. Phytanoyl-CoA is oxidized by phytanoyl-CoA dioxygenase, in a process using Fe^{2+} and O_2, to yield 2-hydroxyphytanoyl-CoA.
3. 2-hydroxyphytanoyl-CoA is cleaved by 2-hydroxyphytanoyl-CoA lyase in a TPP-dependent reaction to form pristanal and formyl-CoA (in turn later broken down into formate and eventually CO_2).
4. Pristanal is oxidized by aldehyde dehydrogenase to form pristanic acid (which can then undergo beta-oxidation).

In biochemistry and metabolism, beta-oxidation is the catabolic process by which fatty acid molecules are broken down in the cytosol in prokaryotes and in the mitochondria in eukaryotes to generate acetyl-CoA, which enters the citric acid cycle, and NADH and $FADH_2$, which are co-enzymes used in the electron transport chain. It is named as such because the beta carbon of the fatty acid undergoes oxidation to a carbonyl group. Beta-oxidation is primarily facilitated by the mitochondrial trifunctional protein, an enzyme complex associated with the inner mitochondrial membrane, although some fatty acids are oxidized in peroxisomes.

Fatty acid catabolism consists of:

1. Activation and membrane transport of free fatty acids by binding to coenzyme A.
2. Oxidation of the beta carbon to a carbonyl group.
3. Cleavage of two-carbon segments resulting in acetyl-CoA.
4. Oxidation of acetyl-CoA to carbon dioxide in the citric acid cycle.
5. Electron transfer from electron carriers to the electron transport chain in oxidative phosphorylation.

Omega oxidation (ω-oxidation) is a process of fatty acid metabolism in some species of animals. It is an alternative pathway to beta oxidation that, instead of involving the β carbon, involves the oxidation of the ω carbon (the carbon most distant from the carboxyl group of the fatty acid). The process is normally a minor catabolic pathway for medium-chain fatty acids (10-12 carbon atoms), but becomes more important when β oxidation is defective.

Lecture 8

Digestion & absorption of lipids, Rancidity in fats

Digestion of lipids:

The first step in the digestion of triacylglycerols and phospholipids begins in the mouth as lipids encounter saliva. Next, the physical action of chewing coupled with the action of emulsifiers enables the digestive enzymes to do their tasks. The enzyme lingual lipase, along with a small amount of phospholipid as an emulsifier, initiates the process of digestion. These actions cause the fats to become more accessible to the digestive enzymes. As a result, the fats become tiny droplets and separate from the watery components. In the stomach, gastric lipase starts to break down triacylglycerols into diglycerides and fatty acids. Within two to four hours after eating a meal, roughly 30 percent of the triacylglycerols are converted to diglycerides and fatty acids. The stomach's churning and contractions help to disperse the fat molecules, while the diglycerides derived in this process act as further emulsifiers. However, even amid all of this activity, very little fat digestion occurs in the stomach.

As stomach contents enter the small intestine, the digestive system sets out to manage a small hurdle, namely, to combine the separated fats with its own watery fluids. The solution to this hurdle is bile. Bile contains bile salts, lecithin, and substances derived from cholesterol so it acts as an emulsifier. It attracts

and holds on to fat while it is simultaneously attracted to and held on to by water. Emulsification increases the surface area of lipids over a thousand-fold, making them more accessible to the digestive enzymes.

Once the stomach contents have been emulsified, fat-breaking enzymes work on the triacylglycerols and diglycerides to sever fatty acids from their glycerol foundations. As pancreatic lipase enters the small intestine, it breaks down the fats into free fatty acids and monoglycerides. Bile salts envelop the fatty acids and monoglycerides to form micelles. Micelles have a fatty acid core with a water-soluble exterior. This allows efficient transportation to the intestinal microvillus. Here, the fat components are released and disseminated into the cells of the digestive tract lining.

Absorption of lipids:

Lipid absorption involves the digestion products of triglycerides, phospholipids, cholesterol esters, and fat-soluble vitamin esters, that is, free fatty acids, small amounts of 2-monoglycerides, lysophospholipids (mainly lysophosphatidylcholine), cholesterol, fat-soluble vitamins and glycerol, molecules that, with the exception of short-chain and medium-chain fatty acids and glycerol, have a poor solubility in aqueous medium.

The luminal surface of enterocytes is covered by a layer of water called “unstirred water layer”, whose thickness depends inversely on how vigorously the luminal content has been mixed, and which represents the main barrier that the less soluble lipids must cross to be absorbed. The water barrier is overcome by the formation of mixed micelles that are made up of bile salts (pure micelles consist of only bile salts), and products of lipid digestion. Their formation begins as the pH of luminal content rises, and the concentration of bile salts reaches or exceeds the “critical micellar concentration”, a value that in the intestinal lumen is almost always exceeded.

Triacylglycerols, cholesterol, and phospholipids form lipoproteins when joined with a protein carrier. Lipoproteins have an inner core that is primarily made up of triacylglycerols and cholesterol esters (a cholesterol ester is a cholesterol linked to a fatty acid). The outer envelope is made of phospholipids interspersed with proteins and cholesterol. Together they form a chylomicron, which is a large lipoprotein that now enters the lymphatic system and will soon be released into the bloodstream via the jugular vein in the neck. Chylomicrons transport food fats perfectly through the body’s water-based environment to specific destinations such as the liver and other body tissues.

Cholesterols are poorly absorbed when compared to phospholipids and triacylglycerols. Cholesterol absorption is aided by an increase in dietary fat components and is hindered by high fiber content. This is the reason that a high intake of fiber is recommended to decrease blood cholesterol.

Rancidity in fats:

Rancidity is a term generally used to denote unpleasant odours and flavours in foods resulting from deterioration in the fat or oil portion of a food. Three different mechanisms of rancidity may occur. These are oxidative, hydrolytic, and ketonic.

- **Oxidative rancidity** of fats such as lard, shortenings, salad and cooking oils refers to the undesirable odors and flavors which develop when such products are exposed to the oxygen in the air. Products containing these fats, including but not limited to food products such as fish, poultry, meat, frozen vegetables and dry milk can become rancid as the fats in the products react to air. The poly-unsaturated fatty acid portions of these foods react with oxygen to form peroxides. The peroxides decompose to yield a complex of mixtures, including aldehydes, ketones, and other volatile products. These products are responsible for "rancid" odors and flavors. It is important to note that fish contain highly unsaturated (poly-unsaturated and mono-unsaturated) fatty acids which make some fish products particularly susceptible to oxidative deterioration. Highly saturated products, such as butter, are not as prone to oxidative rancidity due to the absence of polyunsaturated fatty acid compounds. These products also tend to be more solid at room temperature.

- **Hydrolytic rancidity** refers to the odor that develops when triglycerides are hydrolyzed and free fatty acids are released. This reaction of lipid with water sometimes requires a catalyst, but results in the formation free fatty acids and salts from free fatty acids (soaps). In particular, short chain fatty acids, such as common butter fats, are odorous.

- **Ketonic rancidity:** Some moulds (*Penicillium* and *Aspergillus* spp.) attack fats containing short-chain fatty acids and produce ketones with a characteristic odour and taste. Butter, coconut, and palm kernel oils are most susceptible.

MODULE 4 **8L**

Lecture 1

Vitamins: occurrence, physiological functions (Fat Soluble Vitamins)

Lecture 2

Vitamins: occurrence, physiological functions (Water Soluble Vitamins)

Vitamin: food sources, functions, deficiency diseases

Vitamin	Food Source	RDA	Function	Deficiency diseases
Vitamin A (Retinol or Beta-carotene)	Liver, egg yolk, dairy products, margarine. Beta carotene (pro-vitamin A) is found in dark green and deep yellow fruits and vegetables.	5,000 IU	Keeps eyes healthy; develops bones; protects linings of respiratory, digestive and urinary tracts; maintains healthy skin and hair. Beta carotene fights free radicals (chemicals that damage cells).	Night blindness
Vitamin B_1 (Thiamine)	Whole grains, cereals and enriched grain products; also legumes (dried beans, peas, and nuts), organ meats, lean pork and eggs.	1.1 - 1.5 mg	Promotes healthy functioning of the nerves, muscles and heart. Metabolizes carbohydrates.	Beri-beri
Vitamin B_2 (Riboflavin)	Organ meats, enriched breads and cereals, legumes, almonds, cheese and eggs; also meat, fish and dark green vegetables.	1.3 - 1.7 mg	Metabolizes carbohydrates, fats and proteins, produces hormones; promotes eye and skin health.	Slow growth, sore eyes
Vitamin B_3 (Niacin)	Meat, organ meats, whole grains and cereals, and legumes; also eggs, milk, green leafy vegetables and fish.	15 - 19 mg	Metabolizes carbohydrates and fats; helps functioning of digestive system; maintains health skin.	Pellagra
Vitamin B_5 (Pantothenic Acid)	Organ meats, yeast, raw vegetables, eggs and dairy products.	None; 4 - 7 mg suggested	Produces hormones and maintains body's immune system.	Paresthesia
Vitamin B_6 (Pyridoxine)	Whole-grain products, poultry, fish, and nuts; also meat, most fruits and vegetables, eggs and dairy products	1.6 - 2 mg	Metabolizes protein; helps produce hemoglobin; promotes functioning of	Anemia

			digestive and nervous systems, and healthy skin.	
Vitamin B_{12} (Cyanocobalamin)	Primarily organ meats; also fish, lean meats, poultry, cheese, and eggs.	2 µg	Builds genetic material of cells and produces blood cells.	Megaloblastic anemia
Vitamin C (Ascorbic Acid)	Almost exclusively fruits and vegetables (especially citrus fruits, tomatoes, peppers, strawberries, and cantaloupe) although breast milk and organ meats contain small amounts.	100 - 200 mg	An antioxidant, fights and resists infection; heals wounds; promotes growth and maintenance of bones, teeth, gums, ligaments and blood vessels.	Scurvy
Vitamin D (Cholecalciferol)	For most people, sun exposure is the primary source of vitamin D. Food sources include Vitamin D-fortified milk, eggs, fish-liver oils and fatty fish such as herring, mackerel and salmon.	400 IU	Builds strong bones and teeth and maintains the nervous system.	Rickets
Vitamin E (Tocopherol)	vegetable oils, nuts, wheat germ and whole-wheat products, egg yolks and green leafy vegetables.	Women 8 mg; Men 10 mg	Protects the lungs, nervous system, skeletal muscle and the eye's retina from damage by free radicals; may reduce risk of heart disease by protecting against atherosclerosis.	Hemolysis and sterility
Vitamin H (Biotin)	Oats, organ meats, yeast and eggs (cooked); also whole-wheat products, dairy products, fish and tomatoes.	None; 30 - 200 µg suggested	Metabolizes proteins and carbohydrates; breaks down fatty acids.	Loss of appetite, depression, fatigue, dermatitis, nausea, weakness, hair loss
Vitamin K	Dark green leafy vegetables, eggs, cheese, pork and liver.	60 - 80 mg	Promotes normal blood-clotting.	Hemorrhage
Vitamin M (Folic Acid)	vegetables (especially dark-green ones), organ meats, whole-	180 - 200 µg	Synthesis of protein and genetic materials; may help	Megaloblastic anemia, neurologic and

	wheat products, legumes and mushrooms.		prevent some cancers, heart disease and stroke; when taken during pregnancy, protects against some birth defects.	psychiatric problems

Lecture 3

Minerals: occurrence

Lecture 4

Minerals: physiological functions

Minerals: food sources, functions, deficiency diseases

Mineral	Food Source	RDA	Function	Deficiency diseases
Calcium (Ca)	Primarily in milk and dairy products; also dark-green vegetables, legumes, shellfish, fish with edible bones and tofu; also calcium-fortified orange juice.	800 - 1,200 mg	Builds bones and teeth; promotes blood clotting, contraction of muscles and nerve impulses.	Osteoporosis
Chromium (Cr)	Whole wheat and other whole grains and molasses.	None; 50 - 200 µg suggested	An essential nutrient required for normal sugar and fat metabolism; may also help prevent high cholesterol and atherosclerosis.	Fatigue, anxiety, stunted growth, poor blod glucose control, heart complications due to high cholesterol
Copper (Cu)	Organ meats, shellfish, whole-grain products, legumes and dried fruits.	None; 2 - 3 mg suggested	Builds bones, red blood cells and hemoglobin; metabolizes iron, maintains connective tissue and blood vessels; may play a role in cancer prevention.	neurologic and psychiatric disorder, heart enlargement, progressive difficulty in walking, low appetite, retarded growth
Fluoride (F)	Seafood, tea, coffee	None	Promotes bone and tooth	Dental decay

	and soybeans; sodium fluoride is often added to the water supply to prevent tooth decay.		formation; prevents tooth decay.	
Iodine (I_2)	Saltwater fish, shellfish, sea kelp and iodized salt.	150 µg	Helps produce thyroid hormones; adequate iodine intake during pregnancy is crucial to normal fetal development.	Goitre
Iron (Fe)	Iron is poorly absorbed from food. The richest sources are red meat and organ meats; other sources include whole-wheat products, shellfish, nuts and dried fruit. Many breads and cereals are enriched with iron. Vitamin C aids absorption of iron and is often added to iron supplements.	Women 15 mg; Men 10 mg	Helps produce hemoglobin and red blood cells; delivers oxygen to muscles and other body tissues; protects against effects of stress	Anemia, fatigue, irritability, weakness
Magnesium (Mg)	Legumes, whole-grain cereals, nuts and dark-green vegetables; also meat, seafood and dairy products.	Women 280 mg; Men 350 mg	Builds bones and teeth; involved in functioning of muscular and nervous systems and hear and circulatory system.	Anxiety, fatigue, insomnia, headache, irritability
Manganese (Mn)	Tea, green vegetables, legumes, oats and rice.	2 - 5 mg	Involved in reproductive processes, sex hormone formation; essential for normal brain function and bone development.	Defective ovulation, ovarian degeneration, testicular degeneration
Molybdenum (Mo)	Dairy products, legumes, whole-grain cereals and organ meats.	75 - 250 mg	Involved in enzyme activities.	Impairment in growth, diarrhea, anemia
Phosphorus (P)	Meat, fish, eggs, legumes and dairy products; also whole wheat, corn and rice.	1 gram	Builds bones and teeth.	Bad teeth and bones
Potassium (K)	Potatoes, dried fruits, bananas, legumes, raw vegetables,	None; 3.5 grams suggested	Helps nerves and muscles function; regulates heart's rhythm;	Muscle weakness, paralysis

	avocados and mushrooms; also lean meat, milk and fish.		regulates bodily fluids.	
Selenium (Se)	Whole-grain cereals, fish and shellfish, meat and dairy products.	Women 55 µg; Men 70 µg	An antioxidant, helps protect cells and tissues from damage by free radicals; may also protect against some cancers.	Cirrhosis of lever, cardiac failure, loss of appetite, nausea
Sodium (Na)	Naturally in many foods and is added to many prepared foods.	2,400 mg	Maintains body's fluid balance; important for nerve function and muscle contraction; controls heart's rhythm.	Dehydration, weakness
Zinc (Zn)	Shellfish (particularly oysters), organ meats and lean red meat, yeast, whole-grain cereals, and legumes.	Women 12 mg; Men 15 mg	Involved in growth, skin health and wound healing, development of the reproductive organs, protein metabolism and energy production.	Folate deficiency, diabetes mellitus

Lecture 5

Introduction to human nutrition; Nutritive values of foods

Nutritive value of some common foodstuffs

Ingredients	Calories/100 gms	Ingredients	Calories/100 gms
Rice	345	Mutton	194
Wheat	341	Fish	87
Pulses	35	Egg	173
Leafy vegetables	34	Milk – Buffalo's	67
Orange	40	Butter milk	15
Banana	115	Sugar of sweets	400

Lecture 6

Basal metabolic rate; RDA

Basal metabolic rate (**BMR**) is the minimal **rate** of energy expenditure per unit time by endothermic animals at rest. It is reported in energy units per unit time ranging from watt (joule/second) to ml O_2/min or joule per hour per kg body mass J/(h·kg).

Factors that affect BMR and metabolism:

1. **Muscle mass.** The amount of muscle tissue on your body. Muscle requires more energy to function than fat. So the more muscle tissue you carry, the more energy your body needs just to exist. (Resistance or strength training is most effective for building and maintaining mass.)
2. **Age.** As you get older, your metabolic rate generally slows. This is because of a loss of muscle tissue and changes to hormonal and neurological processes. During development children go through periods of growth with extreme rates of metabolism.
3. **Body size.** Those with bigger bodies have a larger BMR because they have larger organs and fluid volume to maintain.
4. **Gender.** Men generally have faster metabolisms than women.
5. **Genetics.** Some families have faster BMR than others with some genetic disorders also affecting metabolism.
6. **Physical activity.** Exercise increases muscle mass and powers up your metabolic engines burning kilojoules at a faster rate, even when at rest.
7. **Hormonal factors.** Hormonal imbalances such as hypo & hyperthyroidism can affect your metabolism.
8. **Environmental factors.** Environmental changes such as increased heat or cold forces the body to work harder to maintain its normal temperature and increases BMR.
9. **Drugs.** Caffeine and nicotine can increase your BMR whilst medications such as antidepressants and steroids increase weight gain regardless of what you eat.
10. **Diet.** Food changes your metabolism. What and how you eat has a big influence on your BMR.

Recommended Dietary Allowance (RDA):

RDA (per day) for Indians (ICMR)

Group	Particulars	Body Wt. kg	Net Energy kC	Protein gm	Fat gm	Ca mg	Fe mg
Man	Sedentary	60	2425	60	20	400	28
	Moderate	"	2875	"	"	"	"
	Heavy work	"	3800	"	"	"	"
Woman	Sedentary	50	1875	50	"	"	30
	Moderate	"	2225	"	"	"	"
	Heavy work	"	2925	"	"	"	"
	Pregnancy	"	+300	+15	30	1000	38
	Lactation (0-6 m)	"	+550	+25	45	"	30
	Lactation (6-12 m)	"	+400	+18	"	"	"

Lecture 7

Dietary requirements and deficiency diseases of different nutrients

Vitamin, minerals, fibre and water

Nutrient	Function	Deficiency	Food sources
Vitamin C	Maintain healthy **skin** and **gums**	**Scurvy**- bleeding under skin, bleeding gums	Citrus fruits, cabbage, blackcurrants, guava, mango, tomato
Vitamin D	-Maintain hard bones -Help to absorb **calcium** from small intestine	**Rickets** - soft bones that become deformed (e.g. bow legs)	-Milk, butter, cheese, egg yolk, fish-liver oil. -Made by **skin** when exposed to sunlight
Calcium	-Formation of healthy **bones** and teeth - Normal **blood clotting**	-Rickets, brittle bones and teeth -Slow blood clotting	Milk, cheese, fish
Iron	-Formation of **haemoglobin** in red blood cells	**Anaemia** (not enough red blood cells → not enough O_2 delivered to tissues): constant tiredness, lack of energy	Red meat, liver, kidney, eggs, vegetables (spinach, cabbage…) , chocolate
Fibre	**Cellulose** adds bulk (mass) to undigested food passing through the intestines, maintaining **peristalsis** (constriction and relaxation)	-**Constipation** -Long-term deficiency leads to **bowel cancer**	Vegetables, fruit, whole meal bread
Water	-Formation of **blood**, **cytoplasm** -Solvent for transport of nutrients and removal of wastes (**urine**) -**Enzymes** only work in solution	**Dehydration**	Drinks, fruit, vegetables

Lecture 8

Micronutrients

Vitamins and minerals are the two types of micronutrients. While only needed in small amounts, they play important roles in human development and well-being, including the regulation of metabolism, heartbeat, cellular pH, and bone density. Lack of micronutrients can lead to stunted growth in children and increased risk for various diseases in adulthood. Without proper consumption of micronutrients, humans can suffer from diseases such as rickets (lack of vitamin D), scurvy (lack of vitamin C), and osteoporosis (lack of calcium).

Type of micronutrients:

Vitamins are available in two forms: water-soluble and fat-soluble. Water-soluble vitamins are easily lost through bodily fluids and must be replaced each day. Water-soluble vitamins include the B-complex vitamins and vitamin C. Vitamins B6 and B12 are two of the most well-known B-complex vitamins. Since they are not lost as easily as their water-soluble counterparts, fat-soluble vitamins tend to accumulate within the body and are not needed on a daily basis. The fat-soluble vitamins are A, D, E and K.

Minerals are also available in two forms: macrominerals and microminerals.

Macrominerals are needed in larger amounts and include the following:

- Calcium
- Magnesium
- Phosphorus
- Sodium
- Potassium

Microminerals are only needed in trace amounts and include the following:

- Iron
- Copper
- Iodine
- Zinc
- Fluoride

Micronutrients in Food

All foods contain micronutrients. Here's a list of important micronutrients and common foods where they can be found:

- Calcium - milk, yogurt, spinach, and sardines
- Vitamin B12 - beef, fish, cheese, and eggs
- Zinc - beef, cashews, garbanzo beans, and turkey
- Potassium - bananas, spinach, potatoes, and apricots
- Vitamin C - oranges, peppers, broccoli, and bananas

Foods containing many micronutrients are considered nutrient dense. This ratio compares the amount of calories the food provides to the amount of nutrients it contains. Low calorie foods with many micronutrients, such as fruits and vegetables, have higher nutrient densities.

Question Bank

1. Multiple Choice Question (MCQ)

i. Non-competitive inhibitor of an enzyme catalyzed reaction

A. decreases Vmax

B. binds to Michaelis complex (ES)

C. both (a) and (b)

D. can actually increase reaction velocity in rare cases

ii. In a Lineweaver-Burk Plot, competitive inhibitor shows which of the following effect?

A. It moves the entire curve to right

B. It moves the entire curve to left

C. It changes the x-intercept

D. It has no effect on the slope

iii. The enzyme inhibition can occur by

A. reversible inhibitors

B. irreversible inhibitors

C. Both (a) and (b)

D. None of these

iv. Which of the following common drugs is not a specific enzyme inhibitor?

A. Iodine

B. Methotrexate

C. Sulfbnilamide

D. Penicillin

v. A competitive inhibitor of an enzyme is usually

A. a highly reactive compound

B. a metal ion such as Hg2+ or Pb2+

C. structurally similar to the substrate

D. water insoluble

vi. The types of inhibition pattern based on Michaelis-Menten equation are

A. competitive

B. non-competitive

C. uncompetitive

D. all of the above

vii. The effect of non-competitive inhibition on a Lineweaver-Burk Plot is that

A. it can move the entire curve to the right

B. it can change the y-intercept

C. it can change the x-intercept

D. all of these

viii. The rate-determining step of Michaelis-Menten kinetics is

A. the complex formation step

B. the complex dissociation step to produce product

C. the product formation step

D. Both (a)and(c)

ix. Which of the following statements is not true?

A. Enzymes are proteins that bind to specific substrates and increase the velocity of reactions involving those substrates

B. Enzymes function by overcoming the activation energy barrier of a reaction

C. Enzymes make thermodynamically favorable reactions to proceed; they cannot make unfavorable reactions to occur

D. Enzymes only function when they are in intact cells

x. Which of these enzymes contains a Zinc (Zn) ion?

A. Carboxypeptidase A

B. Phosphorylase B kinase

C. Tyrosine hydroxylase

D. Phosphodiesterase

xi. An enzyme and a reactant molecule maintain relationship as

A. a temporary association

B. an association stabilized by a covalent bond

C. one in which the enzyme is changed permanently

D. non complementary binding

xii. A noncompetitive inhibitor of an enzyme-catalyzed reaction

A. increases K_M and increases Vmax

B. increases K_M and reduces Vmax

C. reduces K_M and increases Vmax

D. reduces K_M and reduces Vmax

xiii. An allosteric inhibitor of an enzyme usually

A. participates in feedback regulation

B. denatures the enzyme

C. is a hydrophobic compound

D. causes the enzyme to work faster

xiv. A classical uncompetitive inhibitor is a compound that binds

A. reversibly to the enzyme substrate complex yielding an inactive ESI complex

B. irreversibly to the enzyme substrate complex yielding an inactive ESI complex

C. reversibly to the enzyme substrate complex yielding an active ESI complex

D. irreversibly to the enzyme substrate complex yielding an active ESI complex

xv. Enzyme Papain is used with success to

A. Increase meat production, B. Ripen papaya fruit, C. Leaven bread, D. Tenderize meat

xvi. Which one of the following reactions used for the purpose of recycling enzymes in bioprocesses

A. Isomerization, B. Phosphorylation, C. Immobilization, D. Polymerization

xvii. Most industrial enzymes are obtained from

A. Plants, B. Microbes, C. Insects, D. Animal tissues

xviii. Out of the total enzymes present in cell, mitochondria alone has_____% enzymes

A. 40%, B. 70%, C. 90%, D. 95%

xix. Which is a typical example of feedback inhibition?

A. cyanide and cytochrome reaction, B. sulpha drugs and folic acid synthesizer bacteria, C. allosteric inhibition of hexokinase by glucose 6 phosphate, D. reaction between suucinic dehydrogenase and succinic acid

xx. Competitive inhibition overcome by adding substrate show that

A. enzymes are pH dependent, B. enzymes are made up of protein,

C. enzyme are biocatalysts, D. enzymes are specific in nature

xxi. The order n for a given substrate concentration in an enzyme catalyzed reaction following Michaelis Menten Kinetics, is

A. n=1, B. n=0, C. n is not defined, D. $0 \leq n \leq 1$

xxii. Which of the following is an unsaturated fatty acid ?

A. lauric acid, B. acetic acid, C. arachidonic acid, D. valeric acid

xxiii. TCA cycle operates in the

A. golgi bodies, B. mitochondria, C. ribosome, D. lysosome .

xxiv. Reducing sugars have

A. Free aldehyde, B. Bound aldehyde, C. Free aldehyde or ketone, D. Bound ketone

xxv. Glycosidic bond is

A. C-O-C, B. CONH, C. >C==O , D. CHO

xxvi. Each cycle of oxidation produces

A. 1 FAD, 1 NADH and 1 acetyl-CoA

B. 1 $FADH_2$, 1 NADH and 1 acetyl-CoA

C. 1 FAD, 1 NAD and 2 CO_2 molecules

D. 1 $FADH_2$, 1 NAD and 1 Acetyl-CoA

xxvii. Example of a cobalt containing Vitamin is

A. Vit-A, B. Vit-C, C. Vit-B_6, D. Vit- B_{12}

xxviii. The molecular weight of β-Casein is

A. 23400, B. 21000, C. 24000, D. 66000

xxix. Fatty Liver is formed due to the deficiency of

A. Alanine, B. Bile Salt, C. Choline, D. Cholesterol.

xxx. The chief constituent of ketone bodies will be

A. Ethyl alcohol, B. Ethylacetoacetate, C. Acetic acid, D. None of these

xxxi. Example of sulpher contamining amino acid is –

A. leucine , B. isolecine , C. lysine , D. metheonine

xxxii. In saliva the nature of enzyme present is

A. Protein degrading , B. Carbohydrate degrading ,C. Lipid degrading , D. Crude fiber degrading

xxxiii. Calcium absorption in the human system is enhanced by-

A. vit-A , B. vit-D , C. vit-K , D. None of these .

xxxiv. Precursor of glycogen is –

A. glucose , B. fructose-1-phossphate, C. glucose-1-phosphate , D. UDP-glucose

xxxv. The physiological fuel value (Kcal/gm) of protein is –

A. 5.65, B. 4.05, C. 5.25, D. 5.01

xxxvi. The principal donor of methyl group in the synthesis of choline is-

A. tryptophan , B. histidine , C. valine , D. methionine .

xxxvii. Non essential amino acids can be formed in the body by a process known as-

A. decarboxylation , B. transamination , C. carboxylation , D. dehydration .

xxxviii. Negative nitrogen balance can occur during-

A. growth , B. pregnancy , C. malnutrition, D. all of these

xxxix. The most abundant organic molecule in nature is

A. protein, B. iron, C. calcium, D. carbohydrate.

xl. Which of the following is not a disaccharide?

A. galactose, B. glucose, C. lactose, D.both a &b

xli. The water insoluble fraction of starch has a share of

A. 65-90%, B. 55-65%, C. 80-85%, D. 2-4%

xlii. The glycosidic bonds present in cellulose structure is

A. α(1:4), B. β(1:4), C. α(1:6), D. none

Short Question (SQ)

2. Mention the role of tocopherol in human body. What happens in case of vitamin E deficiency ? (2.5+2.5=5)

3. Write the natural sources, functions and daily requirement of thiamine in human body. (2+2+1=5)

4. Enumerate the differences between competitive and non-competitive inhibition with suitable examples. (5)

5. Explain briefly the factors that affect enzyme activity. (5)

6. Distinguish between: (any two) (2.5x2=5)

a) Cofactor and Coenzyme

b) Holoenzyme and Apoenzyme

c) Positive N-Balance and Negative N-Balance

d) Lyases and Lygases

7. Justify "nutrition plays an important role in promotion of health". What are anti-nutritional factors? (2.5+2.5=5)

8. Write the physiological role of carbohydrates. Mention the sources of plant carbohydrate. (3+2=5)

9. What is nicotinic acid? Mention the sources and role of nicotinic acid in human body. (1+4=5)

10. What is the significance of N value with reference to BV? Explain the regulatory functions of proteins. (2+3=5)

11. What do you mean by BV and PER? Animal Proteins have high BV- comment on the statement. (2+2+1)

12. What are heteroglycans? Give examples. Mention the role of glycoproteins. (2+1+2=5)

13. Distinguish between simple and complex lipids. Give examples of both. (4+1=5)

14. Write Short notes on: (3+ 2=5)

a) Essential fatty acids, b) Respiratory quotient (RQ)

15. What are derived lipids? Distinguish between SAFA, MUFA and PUFA. Mention the role of EFAs. (1+3+1=5)

16. Write down the non-oxidative stages involved in PP pathway. Give two example of the water soluble vitamins? (4+1=5)

17. Explain the following: (1+2+1+1=5)

a) BMR, b) Nitrogen Balance, c) Calcium control of glycogen metabolism, d) Chemical score

18. What do you mean by Feedback Inhibition in an enzyme catalyzed reaction- explain briefly. (5)

19. Draw the double reciprocal plot for competitive inhibition. The plot of MME follow 1st order and zero order kinetics explain. (2.5+2.5= 5)

Long Question (LQ)

20. (a) What do you mean by Allosteric Modulation? Discuss about competitive, uncompetitive and non-competitive enzyme inhibition reaction. (5+4=9)

(b) Enumerate with example that substrate acts as inhibitor in an enzyme catalyzed reaction. Name one of each of activator and inhibitor in enzyme catalyzed reaction. (4+2=6)

21. a) Define fatty Liver? Give the mechanism of fatty liver formation. (1+4=5)

b) Write the functions of phospholipids. What are the natural sources of riboflavin? (3+2=5)

c) Write short note on digestion and absorption of lipids. (5)

22. Briefly enumerate the Induced Fit model of enzyme activity. What do you know about feedback inhibition of an enzyme-catalyzed reaction? Explain the "hyperbolic substrate saturation kinetics" of an enzyme-catalyzed reaction. Give the significance of Km in enzyme catalyzed reactions? Give one example each of fibrous protein and globular protein. (3+3+5+2+2=15)

23. Explain transition state theory of enzyme activity. What is K_{cat}? What do you mean by enzyme activity and specific activity? Explain the terms Amino acid Pool and Chemical score briefly. Name the substrates and products formed when the following enzymes act,

(a) invertase, (b) pepsin, (c) lipase, (d) xanthine oxidase (3+2+3+3+4=15)

24. Derive the Michaelis- Menten equation for an enzyme reaction. Mention the significance of M.M. graph. What are the units of K_m and V_{max}? The plot of MME follow 1st order and zero order kinetics explain. (5+5+2+3=15)

25. (a) Explain the glycolytic pathway along with total ATP production. (7)

(b) What is the significance of conversion of pyruvate to lactate? Write down the metabolic importance of pyruvate. What is cori cycle? (3+2+3=8)

26. (a) Why Kreb's cycle is called TCA cycle? Calculate the total energy yield of TCA cycle. "TCA cycle provides precursors for many biosynthetic pathways"- Explain. (3+ 1+ 2=6)

(b) Briefly describe the allosteric control of glycogen metabolism. Write down the oxidative stage involved in PP pathway. Why PP pathway is called Hexose Metaphosphate shunt? (4+3+2=9)

27.(a)What are the three factors that determine the energy needed by the body?(3)

(b) Write the significance of SDA. (3)

(c)What is glycemic index ? Explain in detail with some values of glycemic index of food stuffs. (3+3=6)

(d)What is meant by 'RDA'? Write the factors affecting RDA. (1.5+1.5=3)

28.(a) What are the functions of Protein in our diets? Write down the properties of protein. Give the examples of two of each type of acidic and basic amino acids. (3+2+4=9)

(b) What is zwitterion? Explain how peptide linkage formed. Give exples of each of the following:

a) fibrous protein, b) globular protein (2+2+2=6)

29. (a) What is BMR ? Write the factors affecting BMR. (3+4)

(b)Write the physiological role of iron in human body. (4)

(c)Mention the sources, functions and requirements of ascorbic acid. (4)

30. Write short notes on (any three) : (3x5=15)

(a) Amino acid pool

(b)Gluconeogenesis

(c)PER, BV

(d)Hexose monophosphate shunt

(e)Ketosis

(f) Allosteric control by epinephrine and glucagon.

(g) NPU and Chemical score.

31.What is the fate of pyruvate under anaerobic condition? Describe the process of oxidative phosphorylation mentioning the total number of ATP generated per molecule of glucose. What is BMR? (3 + 10 + 2=15)

32. (a) What are Ketone bodies?. Mention the importance of Ketone bodies formation. What is Ketosis? (2+2+2=6)

(b) Name two essential fatty acids. What are triglycerides? What is lipotropic agent? Write down the general structure of triglyceride. What do you mean by transamination? (2+2+1+2+2=9)

33. Write short note on TCA cycle. How does the liver Hexokinase play important role in Glycolysis? What is the overall reaction in Gluconeogenesis? (8 + 5 + 2=15)

34. Mention the scientific name, natural occurrence, biological function and deficiency diseases for the vitamins E, K, B_2, B_{12}, C. (3 x5 = 15)