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Class DT
SECTION. B
FINAL TERM
PAPER. BIOCHEMISTRY

Question no 1

- 1) Write down the 4 steps involve in beta oxidation?

- 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 FADH2 which are co-enzymes used in the electron transport chain.

Beta oxidation

Simple meaning of beta oxidation when fatty acids d break down to produce energy

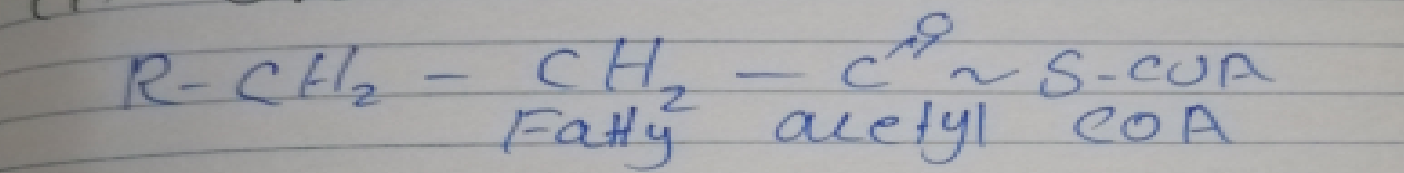
- In eukaryotic cell it takes place in mitochondria matrix
- In prokaryotic cell it happens in cytosol
- This oxidation takes place in beta carbon 2nd carbon of fatty acid

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 very long chain fatty acids are oxidized in peroxisomes.

Beta oxidation takes place in four steps: dehydrogenation, hydration, oxidation and thyolisis. Each step is catalyzed by a distinct enzyme. Briefly, each cycle of this process begins with an acyl-CoA chain and ends with one acetyl-CoA, one FADH2, one NADH and water, and the acyl-CoA chain becomes two carbons shorter

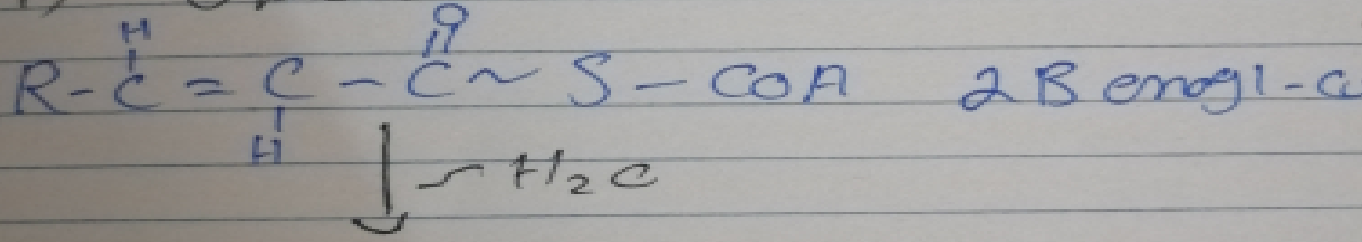
Beta oxidation takes place four steps

i) oxidation:

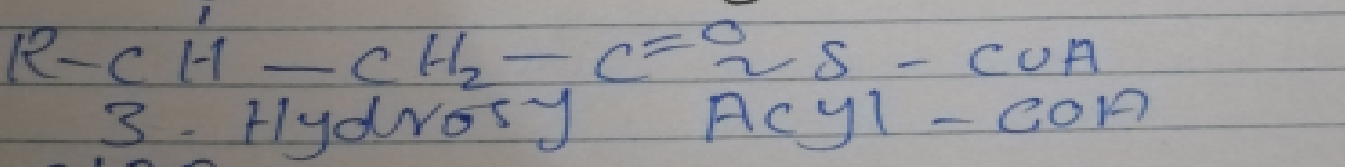


FAD
$NADH_2$

ii) oxidation

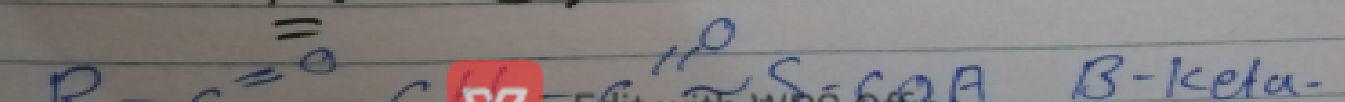


iii) Hydrolysis



NAD
NADH2

iv oxidation



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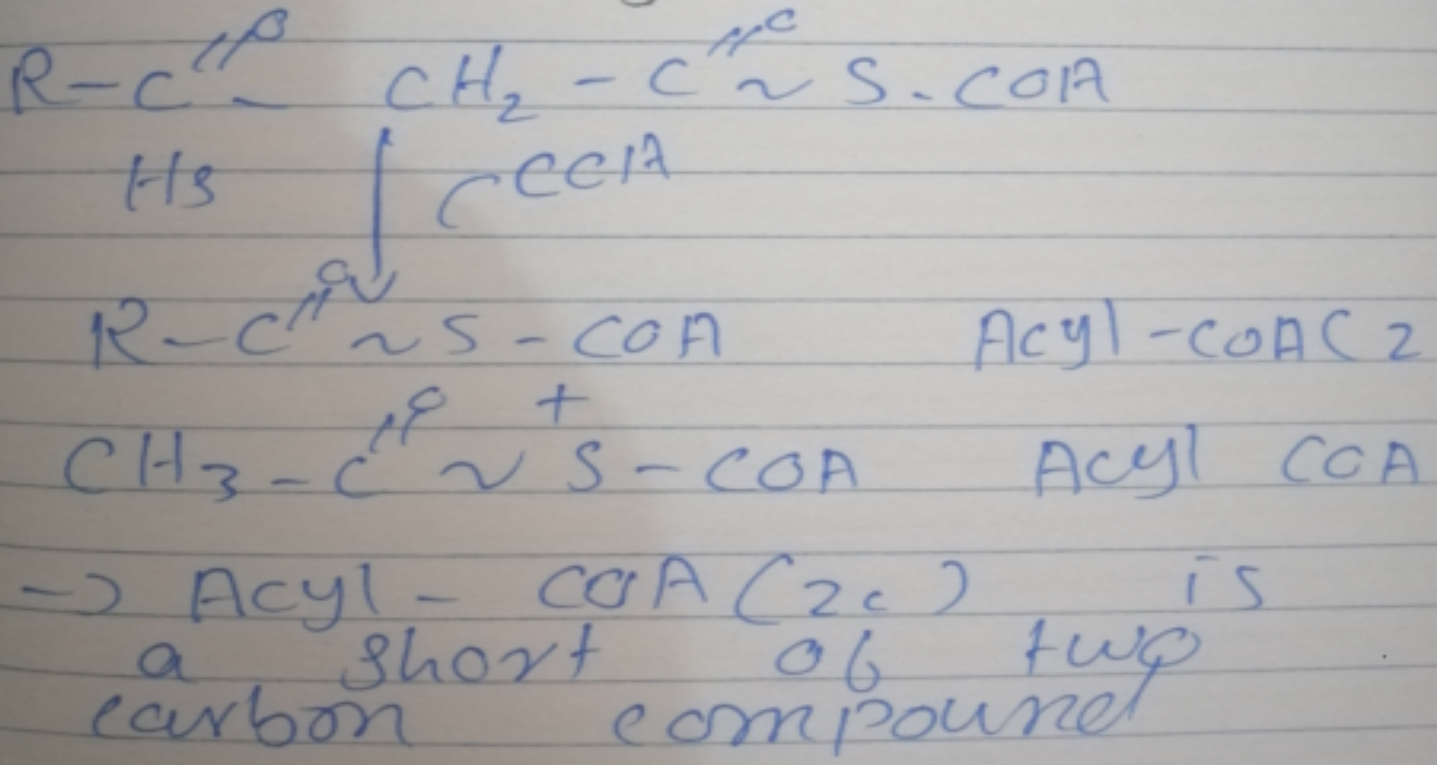
(iv) Thiolysis :->



-> Acyl - COA (2c) is a short of two carbon compound

Theory of four steps

- **Step no1**
- Oxidation means remove of hydrogen or addition of oxygen
- In first step beta oxidation from fatty acyl coA the hydrogen are removed from beta carbon and then FAD is converted to FADH2. So it is converted to beta B enoyl CoA
- **Step no 2**
- In 2nd step hydrolysis take place alpha B enol coA into 3 hydroxy coA

Step 3

- In the 3rd step again oxidation takes place of 3 hydroxy acyl coA and NADH formation take place
- **Step no 4**
- Finally B keto acyl-CoA are converted to acyl coA the process called thiolysis

Question no 2

- 2) Write down clinical significance of the following enzymes
- a) Alkaline phosphatase
- b) Creatine kinase
- c) gamma-glutamyl transferase

A). Alkaline phosphatase

- The normal serum alkaline phosphatase consist of many distinct isoenzymes found in the liver bone, placenta, and less commonly small intestine
- Alp is enxyme found in the liver and bone and it's important for breaking down proteins

Physiological variation

- Proteins over age 60 can have midly of elavted phosphate
- Individual with blood types O and B can have an elavation of serum alkaline phosphatase after eatingof fatty meals due to influx of intestinal alkaline phosphatase into the blood
- It is also non pathologically elavted into children andadolescent. Undergoing.rabid bone growth because alkalinephosphatase

B) Creatine kinase

- Creatine kinase is an enzyme found in the heart skeletal muscles and other tissues. Thus creatine kinase is an important enzyme in such tissues. Clinically, creatine kinase is assayed in blood tests as a marker of damage of CK-rich tissue such as in myocardial infarction (heart attack), rhabdomyolysis (severe muscle breakdown), muscular dystrophy, autoimmune myositides, and acute kidney injury.

Gamma-glutamyl transfuse

- Gamma-glutamyl transferase (GGT) is an enzyme that is found in many organs throughout the body, with the highest concentrations found in the liver. GGT is elevated in the blood in most diseases that cause damage to the liver or bile ducts. This test measures the level of GGT in a blood sample.
- Normally, GGT is present in low levels, but when the liver is injured, the GGT level can rise. GGT is usually the first liver enzyme to

Question no 3

- 3) How many proteins are involve in electron transport chain and how do electrons move in the electron transport chain?

- In electrone transport chain we have protein which is involved electron transport chain.
- The electron transport chain is a series of four protein complexes that couple redox reactions, creating an electrochemical gradient that leads to the creation of ATP in a complete system named oxidative phosphorylation

- Four protein involved in electrone transport chain
- FMN. (flavin protien)
- F. es. (iron Sulphur protein)
- Ubiquinone
- Cytochrome
- These are called complex they are divided into 4 proteins

How electrone move in electrone transport chain

- There are many ways to define hwo electrone moves in electrone transport chain.
- But we have to define electrone movement in the different way
- There are four complex through that electrone can move and make a motion
-

- The electron transport chain (ETC) is a series of complexes that transfer electrons from electron donors to electron acceptors via redox (both reduction and oxidation occurring simultaneously) reactions, and couples this electron transfer with the transfer of protons (H+ ions) across a membrane

Complex 1

- To start, two electrons are carried to the first complex aboard NADH. This complex, labeled I, is composed of flavin mononucleotide (FMN) and an iron-sulfur (Fe-S)-containing protein. FMN, which is derived from vitamin B2, also called riboflavin, is one of several prosthetic groups or co-factors in the electron transport chain

- A prosthetic group is a non-protein molecule required for the activity of a protein. Prosthetic groups are organic or inorganic, non-peptide molecules bound to a protein that facilitate its function; prosthetic groups include co-enzymes, which are the prosthetic groups of enzymes.

Complex 2

- Complex II directly receives FADH2, which does not pass through complex I. The compound connecting the first and second complexes to the third is ubiquinone (Q). The Q molecule is lipid soluble and freely moves through the hydrophobic core of the membrane. Once it is reduced, (QH2), ubiquinone delivers its electrons to the next complex in the electron transport chain

- Q receives the electrons derived from NADH from complex I and the electrons derived from FADH2 from complex II, including succinate dehydrogenase. This enzyme and FADH2 form a small complex that delivers electrons directly to the electron transport chain, bypassing the first complex. Since these electrons bypass and thus do not energize the proton pump in the first complex

Complex 3

- The third complex is composed of cytochrome b, another Fe-S protein, Rieske center (2Fe-2S center), and cytochrome c proteins; this complex is also called cytochrome oxidoreductase. Cytochrome proteins have a prosthetic group of heme. The heme molecule is similar to the heme in hemoglobin, but it carries electrons, not oxygen.

- As a result, the iron ion at its core is reduced and oxidized as it passes the electrons, fluctuating between different oxidation states: Fe++ (reduced) and Fe+++ (oxidized). The heme molecules in the cytochromes have slightly different characteristics due to the effects of the different proteins binding them, giving slightly different characteristics to each complex

- Complex III pumps protons through the membrane and passes its electrons to cytochrome c for transport to the fourth complex of proteins and enzymes (cytochrome c is the acceptor of electrons from Q; however, whereas Q carries pairs of electrons, cytochrome c can accept only one at a time

Complex 4

- The fourth complex is composed of cytochrome proteins c, a, and a3. This complex contains two heme groups (one in each of the two cytochromes, a, and a3) and three copper ions (a pair of CuA and one CuB in cytochrome a3). The cytochromes hold an oxygen molecule very tightly between the iron and copper ions until the oxygen is completely reduced.

- The reduced oxygen then picks up two hydrogen ions from the surrounding medium to make water (H2O). The removal of the hydrogen ions from the system contributes to the ion gradient used in the process of chemiosmosis.

Question no 4

- Q 4. Write steps involve in uric acid formation

- Uric acid production and metabolism are complex procces are involved various factors that regulates production. As well as renal and get excretion of this compound
- Uric acid ie the end product of an exogenous purine metabolism
- The exogenous pool various significantly with diet protein contribute significantlyto this purine pool the endogenous productions of uric acid

Date:

Step involved in Uric Acid Formation

Adenosine mono Phophate

AMP

| Nucleotide

Adenosine

Guanine mono Phosphate | Adenosine deminse

GMP inosine

| nucleticase | Nucleosidose

Guanosine Hypoxanthine

| Nucleoticase | xanthone oxidase

Guanine xanthone

Guanine deamicase | xantine oxidase

Uric Acid

- Is mainly from liver, intestine and some other tissues
- Like muscle kidney and the vascular endothelium1
- 1 AMP and GMP are purin
- 2.Amp are change into adenosine by the action of enzyme in the liver
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- **3.**Then adenosine are converted into inosine and these inosine are converted into ipoxantine
- Ipoxantine are converted into xantine
- And finally these xantine change into uric acid By the action enzyme
- **4**
- GMP are changed.Into guanosine these into guinine and these guanine change into ranthine and finally.Into uric acid

Question no 5

- The compound from which uric acid form is called purine
- Uric acid is a waste product created during the normal breakdown of purines, naturally occurring substances found in foods such as liver, mushrooms
- Uric acid is the last product purine metabolism in humans
- The formation of uric acid is through the enzyme ranthane oxidase which oxidase xoypurines

Normal range of uric acid

- Normal Uric acid levels are 2.4-6.0 mg/dL (female) and 3.4-7.0 mg/dL (male). Normal values will vary from laboratory to laboratory.
- Also important to blood uric acid levels are purines.

- Uric acid formation may occur when the blood uric acid level rises above 7 mg/dL. Problems, such as kidney stones, and gout (collection of uric acid crystals in the joints, especially in your toes and fingers), may occur.

Causes of high uric acid

- Primary hyperuricemia
- Increased production of uric acid from purine
- Your kidneys cannot get rid of the uric acid in your blood, resulting in high levels
- Secondary hyperuricemia
- Certain cancers, or chemotherapy agents may cause an increased turnover rate of cell death. This is usually due to chemotherapy, but high uric acid levels can occur before chemotherapy is administered

- Kidney disease - this may cause you to not be able to clear the uric acid out of your system, thus causing hyperuricemia.
- Medications - can cause increased levels of uric acid in the blood

THE END