SUBJECT.BIOCHAMISTRY

SECTION.B

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***Q no 1write steps involve in uric acid formation***

**Ans.**

**Eight natural ways to lower uric acid levels.**

1. **Limit purine-rich foods. ...**
2. **Eat more low-purine foods. ...**
3. **Avoid drugs that raise uric acid levels. ...**
4. **Maintain a healthy body weight. ...**
5. **Avoid alcohol and sugary drinks. ...**
6. **Drink coffee. ...**
7. **Try a vitamin C supplement. ...**
8. **Eat cherries.**

## **Uric acid is a waste product created during the normal breakdown of purines, naturally occurring substances found in foods such as liver, mushrooms, anchovies, mackerel and dried beans according to the NIAMS. Uric acids normally cleaned out of the blood by the kidneys, and passes out of the body along with urine.**

## **Q no 2write down clinical significance of the follwing enzymes.**

## **Alkaline phosphatase**

## **Definition**

**Alkaline phosphatase (ALP) refers to a group of phosphomonoesterases that hydrolyse phosphate esters with optimum in vitro activity at a pH of 10. Enzyme activity is expressed in international units (IU), the amount of enzyme that catalyses the conversion of 1 μmole of substrate per minute**

**Clinical Significance. The majority of sustained elevated ALP levels are associated with disorders of the liver or bone, or both. ... Since production is increased in response to cholestasis, serum ALPactivity provides a sensitive indicator of obstructive and space-occupying lesions of the liver.**

**Alkaline phosphatase (ALP) is an enzyme in a person's blood that helps break down proteins. The body uses ALP for a wide range of processes, and it plays a particularlyimportant role in liver function and bone development.**

**Creatine kinase.**

**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**

**Clinical Significance of the Creattin kinase**

**Creatine kinase (CK) or creatine phosphokinase (CPK) and myoglobin are present in muscle cells and are released intothe circulation in large quantities in thepresence of significant muscle injury. Themain role of CK is to catalyze the formation of phosphocreatine from creatine.**

**Gamma-glutamyl transfease**

**The gamma-glutamyl transferase(GGT) test may be used to determine the cause of elevated alkaline phosphatase (ALP). Both ALP and GGTare elevated in disease of the bile ducts and in some liver diseases, but only ALP will be elevated in bone disease.**

**GGT functions in the body as a transport molecule, helping to move other molecules around the body. It plays a significant role in helping the liver metabolize drugs and other toxins. GGT is concentrated in the liver, but it's also present in the gallbladder, spleen, pancreas, and kidneys**

**Q No 3.How many proteins are involve in electron transport chain and how do electrons move in the electron transferase.**

**There are four protein complexes (labeled complex I-IV) in the electron transport chain, which are involved in moving electrons from NADH and FADH2 to molecular oxygen. Complex I establishes the hydrogen ion gradient by pumping four hydrogen ions across the membrane from the matrix into the intermembrane space.**

**four protein**

**The electron transport chain is a series of fourprotein complexes that couple redox reactions, creating an electrochemical gradient that leads to the creation of ATP in a complete system named oxidative phosphorylation. It occurs in mitochondria in both cellular respiration and photosynthesis.**

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

**The electron transport chain and ATP synthase are embedded in the inner mitochondrial membrane. The electronsflow through the electron transport chain, causing protons to be pumped from the matrix to the intermembrane space. Eventually, theelectrons are passed to oxygen, which combines with protons to form water.**

***Q No 4.Write down the fore steeps involved in beta oxidation?***

**Ans**

**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 processbegins with an acyl-CoA chain and ends with one acetyl-CoA, one FADH2, one NADH andwater, and the acyl-CoA chain becomes two carbons shorter.**

### **Dehydrogenation**

### **Hydration**

### **Oxidation**

### **Thiolysis**

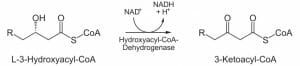
### **Dehydrogenation**

**In the first step, acyl-CoA is oxidized by the enzyme acyl CoA dehydrogenase. A double bond is formed between the second and third carbons (C2 and C3) of the acyl-CoA chain entering the beta oxidation cycle; the end product of this reaction is trans-Δ2-enoyl-CoA (trans-delta 2-enoyl CoA). This step uses FAD and produces FADH2, which will enter the citric acid cycle and form ATP to be used as energy. (Notice in the following figure that the carbon count starts on the right side: the rightmost carbon below the oxygen atom is C1, then C2 on the left forming a double bond with C3, and so on.)  
[Beta Oxidation Step 1](https://biologydictionary.net/wp-content/uploads/2017/06/Beta-Oxidation-Step-1.jpg)**

### **Hydration**

**In the second step, the double bond between C2 and C3 of trans-Δ2-enoyl-CoA is hydrated, forming the end product L-β-hydroxyacyl CoA, which has a**[**hydroxyl group**](https://biologydictionary.net/hydroxyl-group/)**(OH) in C2, in place of the double bond. This reaction is catalyzed by another enzyme: enoyl CoA hydratase. This step requires water.  
[Beta Oxidation Step 2](https://biologydictionary.net/wp-content/uploads/2017/06/Beta-Oxidation-Step-2.jpg)**

### **Oxidation**

**In the third step, the hydroxyl group in C2 of L-β-hydroxyacyl CoA is oxidized by NAD+ in a reaction that is catalyzed by 3-hydroxyacyl-CoA dehydrogenase. The end products are β-ketoacyl CoA and NADH + H. NADH will enter the citric acid cycle and produce ATP that will be used as energy.  
[](https://biologydictionary.net/wp-content/uploads/2017/06/Beta-Oxidation-Step-3.jpg)**

### **Thiolysis**

**Finally, in the fourth step, β-ketoacyl CoA is cleaved by a thiol group (SH) of another CoA**[**molecule**](https://biologydictionary.net/molecule/)**(CoA-SH). The enzyme that catalyzes this reaction is β-ketothiolase. The cleavage takes place between C2 and C3; therefore, the end products are an acetyl-CoA molecule with the original two first carbons (C1 and C2), and an acyl-CoA chain two carbons shorter than the original acyl-CoA chain that entered the beta oxidation cycle.  
[Beta Oxidation Step 4](https://biologydictionary.net/wp-content/uploads/2017/06/Beta-Oxidation-Step-4.jpg)**

### **End of Beta Oxidation**

**In the case of even-numbered acyl-CoA chains, beta oxidation ends after a four-carbon acyl-CoA chain is broken down into two acetyl-CoA units, each one containing two carbon atoms. Acetyl-CoA molecules enter the citric acid cycle to yield ATP.**

***Q no 5.How uric formation takes place in body?***

**Ans**

**Uric acid is a normal waste product that's made when the body breaks down chemicals called purines. Purines are substances found in your own cells and also in some foods. Foods with high levels of purines include liver, anchovies, sardines, dried beans, and beer.**

**Most of it is excreted (removed from your body) in your urine, or passes through your intestines to regulate "normal" levels. 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.**[**Careers**](https://biologydictionary.net/about/careers/)

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