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BIOCHEMISTRY

A Compilation of All India University Questions 2003–2021



2nd Edition

Highlights

- Compilation of Answers for University Questions of the past 19 years
- Categorized in Essays, Short Notes and Short Answers
- Complete with Figures and Flowcharts
- Comfortable to Read, Revise and Reproduce
- Compact Topic-wise University Questions appended.

Evangeline Jones | *Co-author* **Hariharan V**

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MBBS Examination 2006

ANSWER ALL QUESTIONS

I. Essay questions (10 Marks each)

1. Describe the process of glycogen synthesis and glycogenolysis.
2. What is normal serum cholesterol level? Describe the process of synthesis of cholesterol.
3. Describe the metabolism of tryptophan and add a note on inborn errors associated with it.
4. Describe the De novo synthesis of purine nucleotides.
5. Write detail about β-oxidation of fatty acids (FA) oxidation of FA under following headings:
   a. Definition b. Site
   c. Steps and d. Energetics
6. Discuss citric acid cycle under following headings:
   a. Location
   b. Reaction and
   c. Energetics
7. Write an essay on chemistry, functions, deficiency manifestations and hypervitaminosis state of vitamin A.
8. Describe the sources, requirement, absorption, transport, storage forms, functions, deficiency and toxic manifestations of Iron.
9. Discuss urea cycle under the following headings—(a) Site (b) Sources of amino group (c) Steps and (d) Regulation.
10. Discuss about nucleic acids under following headings—types, functions, components, chargaff's rule of DNA composition, different forms of DNA double helix and differences between DNA and RNA.

II. Short notes (5 Marks each)

1. Mutarotation.
2. Factors regulating the enzyme action.
3. Anaplerotic reactions of TCA cycle.
4. What are ketone bodies? Describe the formation of ketone bodies.
5. Chemiosmotic theory/oxidative phosphorylation.
6. Justify the statement that vitamin D is a hormone.
7. Thiamine.
8. Vitamin B12 (extrinsic factor of castle).
9. Acute intermittent porphyria (AIP).
10. Physiological jaundice (neonatal jaundice).
11. Isoelectric pH.
12. Thin layer chromatography.
13. Functions of plasma proteins.
14. Zinc.
15. Metabolic acidosis.
16. Gamma-aminobutyric acid (GABA).
17. Methylmalonic aciduria.
18. Structure of tRNA.
19. LAC operon.
20. Insulin.
21. Define and classify polysaccharides with examples.
22. Define and classify enzymes with examples.
23. Protein-energy malnutrition.
24. Acute intermittent porphyria.
25. Role of cytochromes in ETC.
26. Comparison between prokaryotic and eukaryotic cells.
27. Post-translational modifications with two examples.

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28. Alpha-helical structure of a peptide/ secondary structure of proteins.
29. Buffer system in the body.
30. Principles of electrophoresis and its clinical applications.
31. Applications of genetic engineering.
32. G proteins.

I. ESSAY QUESTIONS

1. Describe the process of glycogen synthesis and glycogenolysis.

Glycogen Metabolism (Fig. 1)

Glycogen is the storage form of glucose mainly in liver and in skeletal muscles.

Glycogen is a homopolysaccharide made up of glucose units linked by α1, 4 and α1,6 glycosidic linkages.

Glycogenesis: (Synthesis of Glycogen) (Fig. 1)

It is the synthesis of glycogen. It needs 2 ATP.

Glycogenolysis: It is the degradation of glycogento glucose but not the reversal of each.

Glycogenesis - Steps

1. Glucose is phosphorylated to glucose-6 phosphate (G6P) by hexokinase in muscle and glucokinase in liver
2. Glucose-6-phosphate is converted to glucose-1-phosphate by phospho-glucomutase enzyme
3. Synthesis of UDP glucose—glucose-1-phosphate reacts with UTP to form UDP-glucose by the enzyme UDP-glucose pyrophosphorylase
4. Glycogen primer—to initiate glycogen synthesis, a pre-existing glycogen may act as a primer. There is a specific protein-glycogenin which accepts glucose from UDP-glucose
5. Glycogen synthesis—by glycogen synthase enzyme: Glycogen synthase helps in the formation of 1, 4 glycosidic linkages by transferring the glucose from UDP-glucose to the nonreducing end of glycogen
6. Formation of branches—branching enzyme (glucosyl alpha 4, 6 transferase). This enzyme transfers a small fragment of 5 to 8 glucose residues from the non-reducing end of glycogen to another glucose residue where it is linked by alpha-1-6 linkage. This leads to the formation of another new nonreducing end. Further elongation of glycogen occurs with branches to form a fully formed glycogen.

Glycogenolysis (Fig. 1)

It is the degradation of glycogen to glucose-6-phosphate in muscle and glucose in liver. It is not the reverse of glycogenesis but it is a separate pathway.

- **Action of glycogen phosphorylase: Rate-limiting enzyme**
  - **Glycogen phosphorylase** catalyzes the phosphorylytic cleavage by inorganic phosphate (phosphorylysis) of the 1→4 linkages of glycogen to yield glucose-1-phosphate
  - The terminal glucosyl residues from the outermost chains of the glycogen molecule are removed sequentially until approximately four glucose residues remain on either side of a 1→6 branch.
- **Action of glucan transferase: Debranching enzyme**
  - Another enzyme **glucan transferase** transfers a trisaccharide unit from one branch to the other, exposing the 1→6 branch point
  - **Hydrolysis** of the 1→6 linkages requires the **debranching enzyme.**
    The combined action of phosphorylase and these other enzymes leads to the complete breakdown of glycogen).
- **Phosphoglucomutase—reversible action**
  - The reaction catalyzed by phospho-glucomutase is reversible, so that glucose-6-phosphate can be formed from glucose-1-phosphate.
- **Glucose phosphatase**
  - Present only in **liver** and **kidney**, but not in muscle
  - A specific enzyme, **glucose-6-phosphatase,** that hydrolyzes glucose-6-phosphate yielding glucose that is

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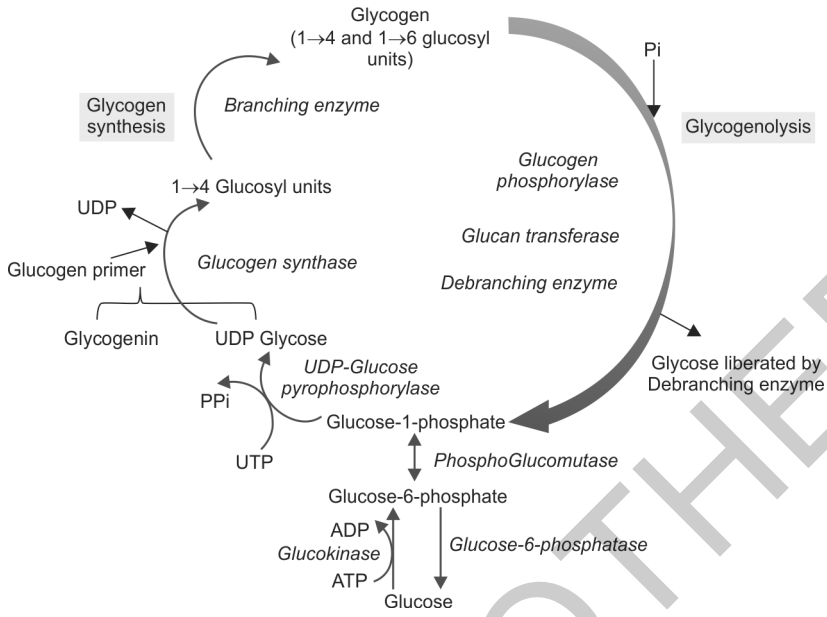

Fig. 1: Glycogen metabolism.

exported leading to an increase in the blood glucose concentration. **It is absent in muscle.**

Glycogenolysis

Regulation of Glycogen Metabolism

- **Allosteric regulation** of activation and deactivation of glycogen synthase and phosphorylase by phosphorylation or dephosphorylation.
- **Hormonal regulation:**
  - Glycogenesis—activated by insulin
  - Glycogenolysis—activated by glucagon, epinephrine
  - These regulations are mediated by cAMP in a cascade pathway.
- **Glycogen phosphorylase: Regulatory enzyme.**

  It exists in 2 forms.
  1. Glycogen phosphorylase a-active or phosphorylated form
  2. Glycogen phosphorylase b - inactive or dephosphorylated form
     - This activation and deactivation occur by stimulation by epinephrine (muscle) and glucagon (liver) via activation of adenylate cyclase by cascade pathway
     - **Allosteric regulation of phosphorylase:** It is allostearically inhibited by glucose-6-phosphate and ATP.

2. What is normal serum cholesterol level? Describe the process of synthesis of cholesterol.

Refer 2004 Essay Question 2.

Normal blood cholesterol levels—150–200 mg/dL.

3. Describe the metabolism of tryptophan and add a note on inborn errors associated with it?

- The aromatic amino acids are phenylalanine, tyrosine, tryptophan
- Tryptophan is an essential amino acid containing indole group
- It is both glucogenic and ketogenic amino acid.

Metabolism of Tryptophan

- **Major pathway (Kynurenine-Anthralinate pathway)—97% (Fig. 2)**

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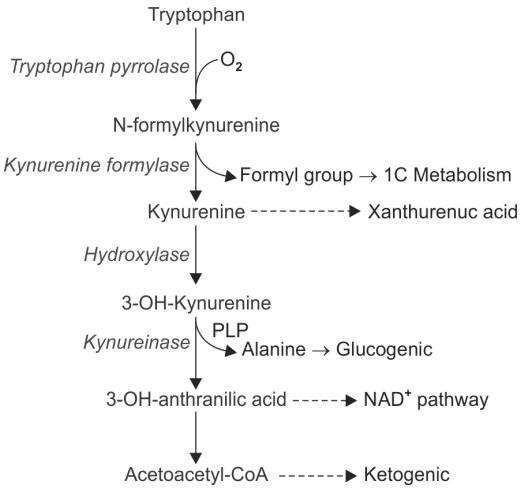

Fig. 2: Kynurenine-anthralinate pathway.

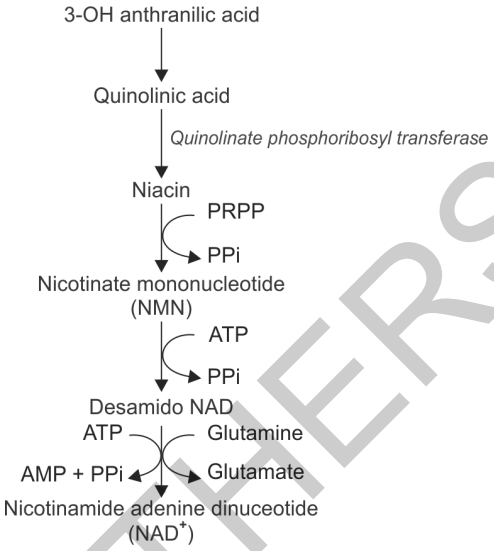

Fig. 3: Niacin pathway.

- Tryptophan is oxidized by **tryptophan pyrrolase**—a dioxygenase enzyme to form N-formylkynurenine. This enzyme is a heme containing enzyme induced by glucocorticoids
- Formylkynurenine is converted to kynurenine by the enzyme **kynurenine formylase**
- Kynurenine is then hydroxylated to 3–hydroxy kynurenine by a **hydroxylase enzyme**
- 3-hydroxykynurenine is converted to 3-hydroxyanthranlinate and alanine by a PLP dependent enzyme **kynureinase**. Alanine enters into glucogenic pathway after converted to pyruvate
- In **B6 deficiency** hydroxykynurenine is converted to xanthurenic acid which gets excreted in urine
- 3-hydroxyanthranilate undergoes either decarboxylation to form niacin or it is catabolized to ketoadipic acid and decarboxylated to Acetoacetyl-CoA which enter to the ketogenic pathway.

Niacin Pathway—3% (Fig. 3)

- 3-hydroxyanthranlinate undergoes decarboxylation to form nicotinic acid by the action of the rate limiting enzyme quinoline phosphoribosyl transferase (QPRTase) NAD and NADP are formed
- 60 mg of tryptophan will give rise to 1mg of niacin.

Serotonin Pathway—1% (Fig. 4)

This pathway occurs in brain, mast cells, platelets and gastrointestinal tract.

- **Step-1 (hydroxylation of tryptophan):** Tryptophan is hydroxylated to 5-hydroxytryptophan by tryptophan hydroxylase which needs a cofactor tetrahydrobiopterin and NADPH
- **Step-2 (decarboxylation):** 5-hydroxytryptophan is decarboxylated to serotonin which is 5-hydroxytryptamine (5-HT). Decarboxylase enzyme requires pyridoxal phosphate as coenzyme
- **Degradation of serotonin: Serotonin undergoes** acetylation by acetyl-CoA and methylation by S-adenosylmethionine to form **Melatonin** (N-acetyl-5-methoxy-serotonin)
- **Serotonin—**It is also oxidized by mono amine oxidase to form 5-hydroxyindole-acetic acid (5-HIA) which is excreted in urine.

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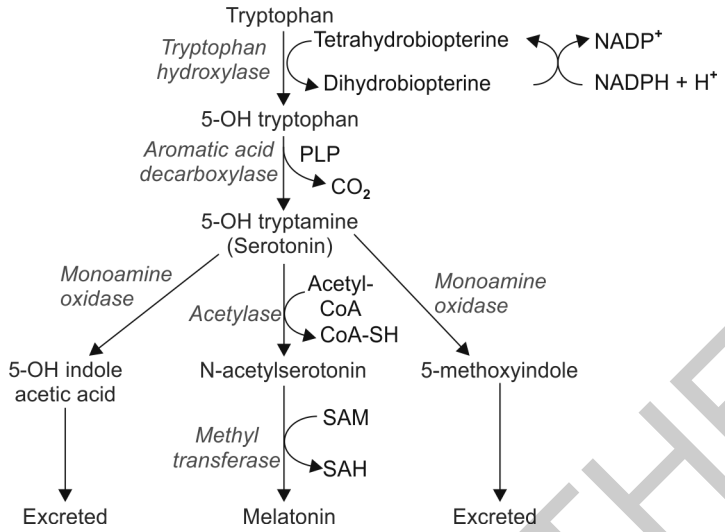

Fig. 4: Serotonin pathway.

Melatonin

- It is the acetylated and methylated product of serotonin secreted by Pineal gland
- It is involved in diurnal variations, sleep wake cycles and biological rhythm
- It is also a neurotransmitter.

Functions of Serotonin

- Serotonin is synthesized by neurons and by the argentaffin cells of gastrointestinal tract
- It is a neurotransmitter in the brain and an antidepressant
- It is a vasoconstrictor and induces smooth muscle contraction
- Serotonin level is low in cases of depressive psychosis and it is involved in inducing sleep, appetite, mood and temperature regulation
- It decreases the sensitivity to pain.

Products formed from Tryptophan

- Serotonin
- Melatonin
- Vitamin Niacin and its coenzymes – NAD, NADP
- Alanine
- Formyl group (one carbon unit)
- 5 -HIAA
- Indican

Disorders Associated with Tryptophan Metabolism

Malignant Carcinoid syndrome (Carcinoid tumors)

- Serotonin is synthesized by the argentaffin cells of gastrointestinal tract and it is necessary for GI motility
- Carcinoid tumors are malignant tumors of argentaffinomas in small intestines and appendix
- There will be increased secretion of serotonin from these tumors (> 40 mg/dL)
- Oat cell carcinoma of lung also produce increased serotonin secretion
- Flushing, diarrhea, sweating and hypertension will be present as symptoms
- Pellagra may be associated with this condition due to niacin deficiency
- Estimation of urinary 5-HIAA is diagnostic of malignant carcinoid tumor. Normal level of urinary-5-HIAA is < 5 mg/day and in carcinoid tumor the level is increased more than that.

Hartnup disease—autosomal recessive disorder.

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- It is named after the family of Hartnup in whom the disorder was described first
- It is due to defective amino acid transport during absorption of amino acids from the intestines and also during reabsorption of amino acids from renal tubules
- This leads to the deficiency of tryptophan and nicotinic acid and NAD
- Neurological and pellagra like symptoms are present
- Aminoaciduria will be present due to failure of amino acid transport in renal tubules
- Increased excretion of indole compounds will be detected by Obermeyer test

Treatment: Supplementation of niacin and high protein diet.

4. Describe the De novo synthesis of purine nucleotides.

Site of synthesis: Liver, erythrocytes, polymorphs—not in brain.

Sources of Purine Ring

- **N1**—aspartate; **N3, N9**—glutamine
- C4, C5 and N7—glycine
- **C2**—formyltetrahydrofolic acid
- **C8**—methenyl-tetrahydrofolic acid
- **C6—**respiratory CO_2

Steps of purine nucleotides synthesis: De novo synthesis (Fig. 5)

- **Synthesis of PRPP:** Ribose 5-phosphate becomes phosphoribosyl pyrophosphate (PRPP) by the enzyme PRPP synthetase using ATP
- **Formation of N_9: The rate-limiting enzyme PRPP amidotransferase** catalyses addition of ammonia group from glutamine to PRPP forming 5-phosphoribosyl amine liberating glutamate
- **Addition of C_4,C_5 and N_7:** Next a whole molecule of glycine (C4,5 and N**7**) is inserted forming glycinamide ribosyl 5- phosphate by the enzyme **PR-glycinamide synthase**
- **Addition of C_8**: **Formyl transferase** enzyme inserts a formyl group from N5, N10-methenyl-THF to form **formylglycinamide R5P**
- **Formation of N_3:** By the action of the enzyme **formylglycinamidine synthase o**ne molecule of ammonia is added from one more molecule of glutamine to form **formylglycinamidine R5P**

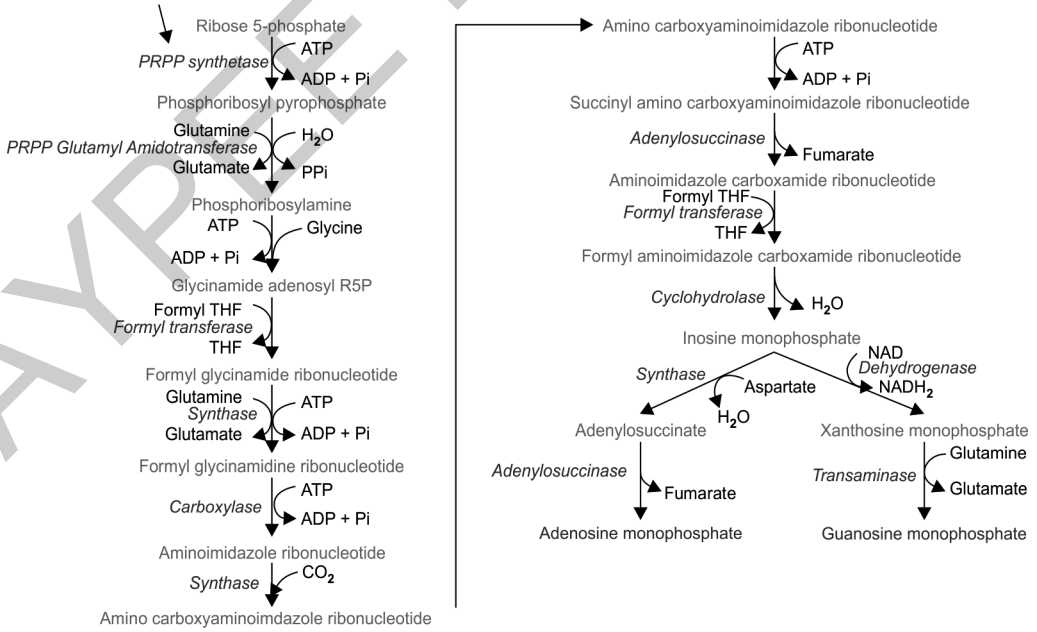

Fig. 5: Synthesis of purine nucleotides.

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- **Ring closure:** By the enzyme **amino-imidazole synthase,** one molecule of water is removed and ring is closed to form **aminoimidazole R5P**
- **Addition of C_6: Carboxylase** enzyme adds CO_2 to form **aminoimidazole carboxylate R5P**
- **Addition of N_1:** By the enzyme succiny-carboxamide R5P synthase aspartate is added with the removal of water to form **aminoimidazole succinyl carboxamide R5P**
- **Removal of Fumarate: Adenylosuccinase enzyme** removes fumarate to form **amino-imidazole carboxamide R5P (**AICAR**)**
- **Addition of C_2:** One formyl group obtained from N10-formyl-THF is added to form formimino imidazole carboxamide R5P by the enzyme **formyl transferase**
- **Complete closure of purine ring:** By the enzyme **cyclohydrolase** the ring is closed to form the **first purine nucleotide — the parent nucleotide—**inosine monophosphate (IMP)
- **Conversion of IMP to AMP and GMP:**
  - **Inosine 5'-monophosphate (IMP)** undergoes dehydrogenation by **dehydrogenase** using NAD and becomes xanthosine monophosphate (XMP). XMP take ammonia from Glutamine to form **guanosine mono-phosphate (GMP)**
  - It takes succinate from aspartate by the **adenylosuccinate synthase** enzyme to form **adenylosuccinate** which liberates fumarate to form **adenosine monophosphate (AMP).**

Regulation of Purine Synthesis

- The rate-limiting step of purine synthesis is amidotransferase (step 1). It is inhibited by AMP and GMP
- The availability of PRPP—depends on availability of Ribose 5-P (HMP pathway)
- The activity of PRPP synthetase is regulated by:
  - Negative modifiers like purines and pyrimidine (↑Pi) (↓ - ATP, GTP)
  - Feedback inhibition by products—ADP and AMP, GMP and GDP.
- PRPP glutamyl amidotransferase—key enzyme inhibited by purine nucleotides.

5. Write detail about β-oxidation of fatty acid (FA) under following headings
a. Definition b. Site
c. Steps and d. Energetics

Beta-oxidation of palmitic acid (16 C fatty acid) and its regulation is discussed below:

Definition: Palmitic acid is 16 carboned saturated long-chain fatty acid

Beta-oxidation is the process of oxidation and splitting of 2 carbon units which are sequentially removed at the beta carbon of fatty acids

- **Site of occurrence**: All tissues predominantly in liver and skeletal muscle
- **Intracellular location**: Mitochondria.
- **Substrate**: Palmitic acid
- **Product**: Acetyl-CoA, NADH, $FADH_2$.

Steps

- Preparative steps—activation of fatty acids (palmitic acid) and transport of fatty acids across mitochondrial membranes into mitochondrial matrix
- Beta-oxidation of fatty acids.

Preparative Step 1: Activation of Palmitic Acids (Fig. 6)

Palmitic acids are activated by ATP to form their acyl-CoA derivatives by the enzyme thiokinase or fatty acyl-CoA synthetase.

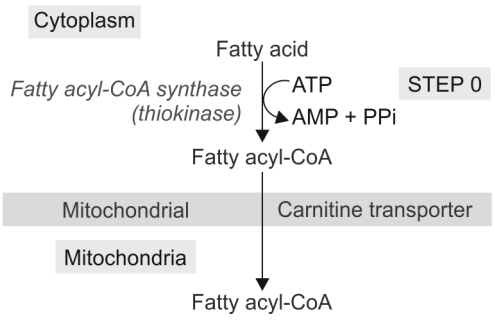

Fig. 6: Activation of fatty acid (palmitic acid).

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Two high-energy bonds are utilized in this step. This step occurs at cytoplasm.

Fatty acid + ATP + CoA Acyl-CoA + PPi + AMP

Preparative Step 2: Role of Carnitine as a Carrier (Fig. 7)

- Palmitic acyl-CoA cannot pass through inner mitochondrial membrane. Hence activated fatty acids are transported to the mitochondria by carnitine shuttle
- Carnitine is synthesized from lysine and methionine in liver and kidney and it is beta-hydroxy-gamma-trimethyl ammonium butyrate
- Carnitine carries fatty acyl groups across the inner mitochondrial membrane. Transfer of acyl group to carnitine occurs by the enzyme carnitine acyltransferase (CAT-1) at the cytosolic side of inner mitochondrial membrane to form acylcarnitine
- Translocase enzyme carries acyl-carnitine across the membrane to the matrix of mitochondria where carnitine acyltransferase II (CAT-II) transfers acyl group to coenzyme. Free carnitine will return back to cytosol by translocase.

Beta-Oxidation Steps

Four steps are involved in Beta-oxidation. They are ***(Fig. 8)****:*

1. FAD dependent dehydrogenation
2. Hydration
3. NAD dependent dehyrogenation
4. Thiolytic cleavage.

FAD dependent dehydrogenation: By FAD linked fatty acyl-CoA dehydrogenase: Fatty acyl-CoA is dehydrogenated to α,β unsaturated fatty acyl-CoA (trans-enoyl-CoA) by FAD dependent dehydrogenase. FAD is converted to $FADH_2$ to produce 1.5 ATPs at electron transport chain.

Hydration: Enoyl-CoA hydratase enzyme helps in adding one molecule of water to α, β unsaturated fatty acyl-CoA to form β-hydroxy fatty acyl-CoA.

NAD dependent dehydrogenation: β-hydroxy fatty acyl-CoA is oxidized to form β-keto fatty acyl-CoA with NAD as coenzyme to generate 2.5 ATP.

Thiolytic cleavage: β-keto fatty acyl-CoA undergoes thiolytic cleavage to split one molecule of acetyl-CoA. A fatty acid with 2 carbon atom less (14 C) is produced.

Further cycles of step 1,2,3 and 4 of β-oxidation are repeated till the fatty acid

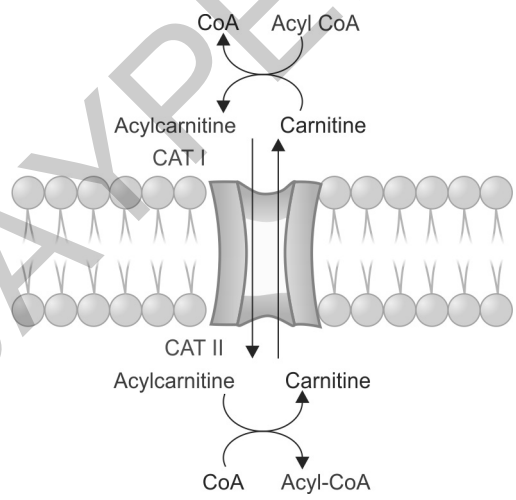

Fig. 7: Carnitine shuttle.

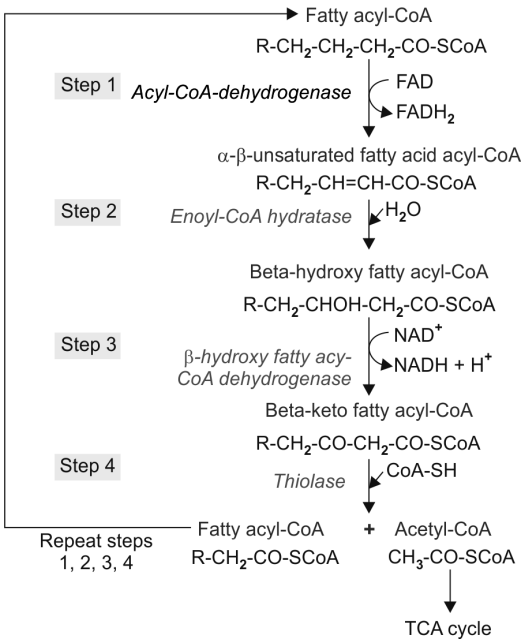

Fig. 8: Beta-oxidation of fatty acid.

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- This *Uniquest Series—Biochemistry* is a compilation of university questions of past 19 years and the relevant answers for them in a compact format.
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- Multiple Choice Questions and Answers have been included in this edition for the year 2021 as well.
- Figures and flowcharts have been added wherever required to help students to understand the answers and easily reproduce in the examination.
- References for the repeated questions and answers have been indicated in an easy to locate format.
- For a quick reference and revision, a Topic-wise University Questions chapter has been appended at the end.

Evangeline Jones MBBS MD (Biochemistry) had worked as Professor and Head, Department of Biochemistry, Government Mohan Kumaramangalam Medical College, Salem and subsequently as Head of Department of Biochemistry in Vinayaka Mission's Kirupananda Variyar Medical College and Hospital, Salem, India. Her teaching experience in Biochemistry spans over 30 years, teaching undergraduate and postgraduate students in Biochemistry, and also serving as a guide for many research projects. She had earlier worked as Dean, Kanyakumari Government Medical College, Tamilnadu. She has many research publications to her credit and had been the Chief Editor of the National Journal of Basic Medical Sciences for three years. She is Member of Association of Clinical Biochemists of India and Association of Medical Biochemists of India. In recognition of her outstanding teaching career, she was awarded "Life time achievement award" by the Tamil Nadu Dr MGR Medical University, Chennai.

Hariharan V MBBS MD (Biochemistry) is currently Associate Professor in the Department of Biochemistry, Karpagam Faculty of Medical Sciences and Research, Coimbatore, Tamil Nadu, India. He has nearly ten years of teaching experience in Biochemistry. He did his MD Biochemistry from Rajah Muthiah Medical College, Chidambaram, Tamil Nadu.

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