Vitamin B2

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Lec. 4 Cont.

Vitamin B2 is a **water-soluble** vitamin just like vitamin B1 and is also active in the breakdown of carbohydrate, protein and fat.(Molecular formula: C17H20O6N4)

It is the central component of the cofactors **FAD and FMN** and is therefore required by all flavoproteins .

It plays a key role in energy metabolism, and for the metabolism of fats , ketone bodies,carbohydrates , and proteins

The name **"riboflavin"** comes from **"ribose" (the sugar whose reduced form, ribitol, forms part of its structure) and "flavin", the ring-moiety** which imparts the yellow color to the oxidized molecule.

Vitamin B2 helps **break down proteins, fats, and carbohydrates**. It plays a vital role in maintaining the body's energy supply

Riboflavin helps convert carbohydrates into adenosine triphosphate (ATP). The human body produces ATP from food, and ATP produces energy as the body requires it. The compound ATP is vital for storing energy in muscles

Maintaining the mucous membranes in the digestive system

Maintaining a healthy liver

Converting tryptophan into niacin, an amino acid

Keeping the eyes, nerves, muscles and skin healthy

Absorbing and activating iron, folic acid, and vitamins B1, B3 and B6

Hormone production by the adrenal glands

Preventing the development of cataracts

Fetal development, especially in areas where vitamin deficiency is common

Biological Active Forms•

FMN **flavin mononucleotide**•

FAD **flavin Adenine dinucleotide**•

They serve **as prosthetic groups of oxidoreductase enzymes** (flavoproteins).•

If these enzymes contain one or more metals e.g. Fe or Mb as essential cofactor, they are known as metalloflavoproteins.•

(FMN) and (FAD) function as coenzymes for a wide variety of oxidative enzymes and remain bound to the enzymes during the oxidation-reduction reactions. •

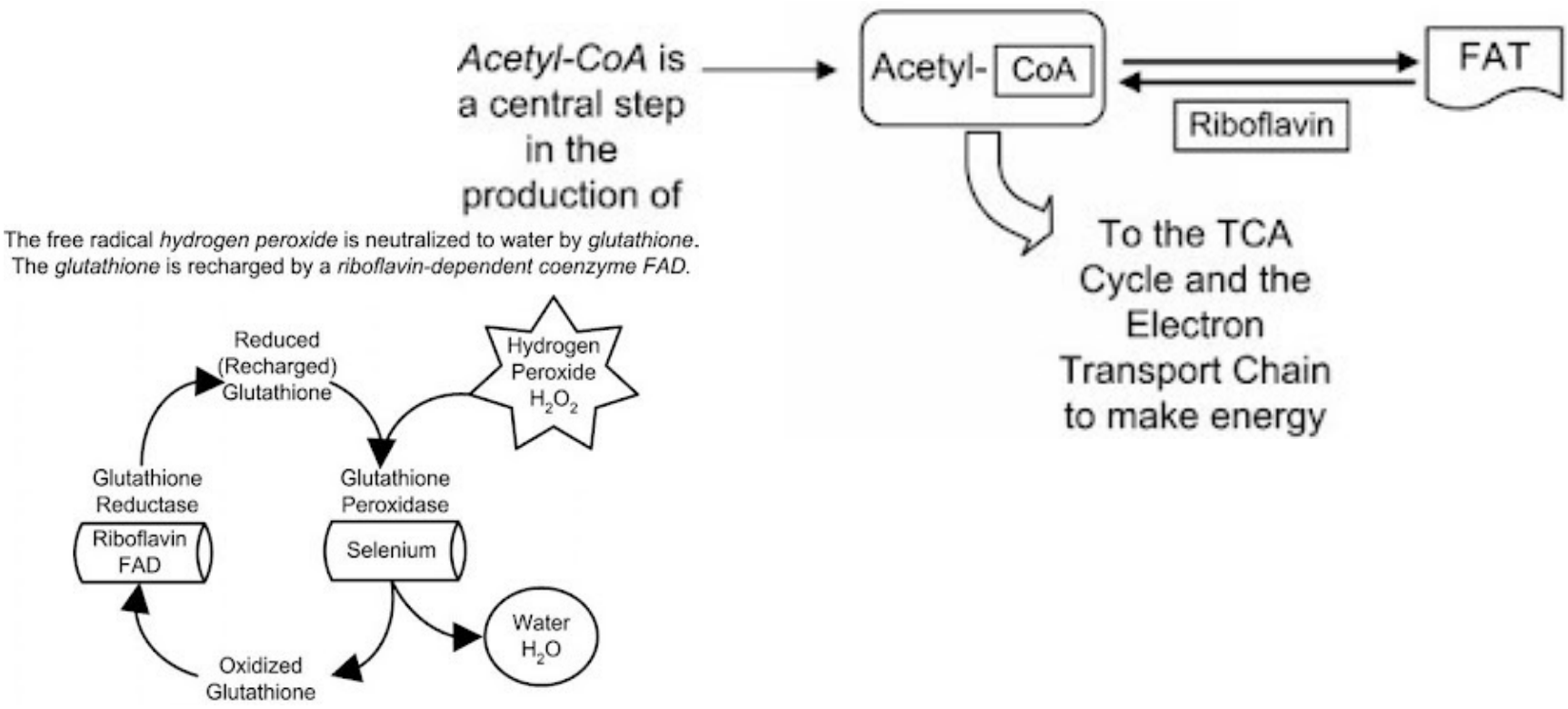
Flavins can act as oxidizing agents because of their ability to accept a pair of hydrogen atoms•

Two main derivatives are:

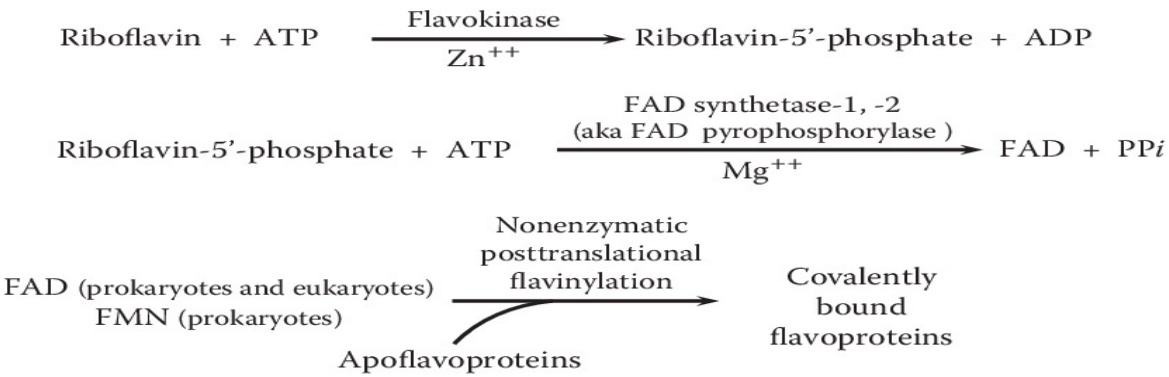
1-**FMN** (Flavin mononucleotide): in this the phosphoric acid is attached to ribityl alcoholic group in position 5.

2-**FAD** (Flavin adenine nucleotide): It may be linked to an adenine nucleotide through a pyrophosphate linkage to from FAD.

Thus, FMN and FAD are two coenzymes of this vitamin.



Riboflavin's role in antioxidant protection



Conversion of riboflavin to riboflavin-5′-phosphate, FAD, and covalent attachment. Flavokinase catalyzes phosphorylation of riboflavin to form riboflavin-5′-phosphate (FMN).

FAD is formed from the action of FAD synthetase (FAD pyrophosphorylase) on FMN after combination with a second molecule of adenosine triphosphate. Flavinylation of specific client apoenzymes occurs posttranslationally through the covalent attachment of either FAD or FMN

Mechanism of action as cofactors and flavoproteins•

Flavoproteins exhibit a wide range of **redox potential** and therefore can play a wide variety of roles in intermediary • metabolism. Some of these roles are:

-1Flavoproteins play very important roles in the **electron transport chain.** •

2-Decarboxylation of **pyruvate and α-ketoglutarate** requires FAD. •

3-Fatty acyl CoA dehydrogenase requires FAD in fatty acid oxidation.•

FAD is required to the production of pyridoxic acid from pyridoxal (vitamin B6 •

FAD is required to convert **retinal (vitamin A) to retinoic acid.•**

FAD is required to convert **tryptophan to niacin (vitamin B3.•**

Reduction of the oxidized form of **glutathione (GSSG)** to its reduced form **(GSH) is also FAD dependent.**•

Metabolism

Riboflavin itself and flavin mononucleotides are absorbed from **the proximal small bowel**, where as flavin adenine dinucleotide must be degraded to flavin mononucleotide prior to absorption.

Absorption occurs mainly in the upper GI tract.

- Gastric acid is responsible for releasing B-2 from noncovalently bonding in foods so that it may be absorbed.
- Riboflavin is also found in high amounts in the retina of the eye.

Excretion

Mainly in free form, up to 50‰ as nucleotides in urine. Daily urinary excretion(0.1 to 0.4) mg(10-20‰ of intake).

Deficiency

A shortage of this vitamin may manifest itself as cracks and sores at the corners of the mouth, eye disorders, Sore tongue, bloodshot eyes; sensitivity to light. Dermatitis, hair loss, poor digestion, retarded growth, burning feet can also be indicative of a shortage

Vitamin B2 deficiency is a significant risk when diet is poor, because the human body excretes the vitamin continuously, so it is not stored.

There are two types of riboflavin deficiency

Primary riboflavin deficiency happens when the person's diet is poor in vitamin B2

Secondary riboflavin deficiency happens for another reason, maybe because the intestines cannot absorb the vitamin properly, or the body cannot use it, or because it is being excreted too rapidly

Riboflavin deficiency is also known as ariboflavinosis

Assessment of riboflavin status

Biochemical tests are essential for confirming clinical cases of riboflavin deficiency and for establishing subclinical deficiencies. Among these tests:

1-Erythrocyte glutathione reductase activity:

Glutathione reductase is a nicotinamide adenine dinucleotide phosphate **(NADPH),** a FAD-dependent enzyme, and the major flavoproteins in erythrocyte. The measurement of the activity coefficient of erythrocyte glutathione reductase (EGR) is the preferred method for assessing riboflavin status.

2-Urinary riboflavin excretion:

Urinary riboflavin excretion rates increase slowly with increasing intakes, until intake level approach 1.0 mg/d, when tissue saturation occurs. At higher intakes, the rate of excretion increases dramatically.

Vitamin B1 and B2-mediated immunometabolism in B cell differentiation in the intestine.

Vitamin B1 acts as a cofactor for enzymes such as pyruvate dehydrogenase and α-ketoglutarate dehydrogenase that are involved in the TCA cycle.

Vitamin B2 acts as a cofactor for enzymes such as succinate dehydrogenase in the TCA cycle and acyl-CoA dehydrogenase in fatty acid oxidation (FAO β-oxidation). Naïve B cells preferentially use the TCA cycle for efficient energy generation.

Once B cells are activated to differentiate into IgA-producing plasma cells, they utilize glycolysis for the production of IgA antibody.