



## OXIDATION OF BIOGENIC AMINES PYRIDOXINE DEFICIENCY

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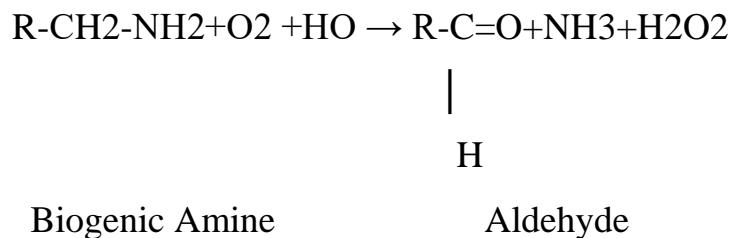
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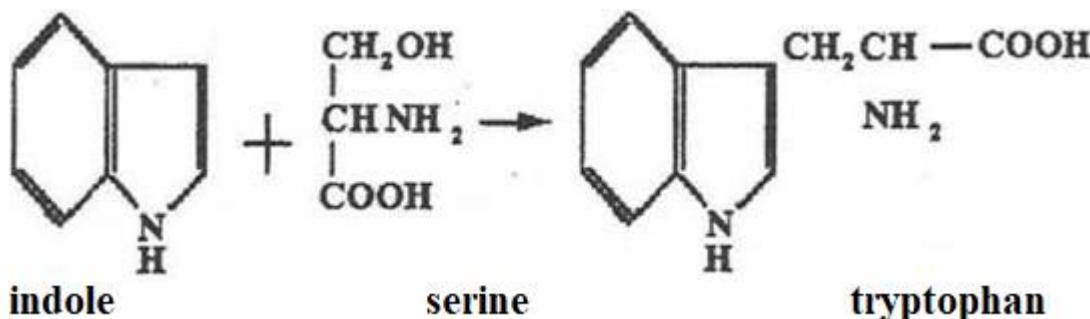
**Annotatsiya.** The main manifestation of vitamin B6 deficiency in adults is the development of seborrheic dermatitis, stomatitis, glossitis. More specific signs are hypochromic anemia and seizures. Pyridoxal-5-phosphate-containing enzymes play an important role in the conversion of tryptophan along the kynurenine pathway.

**Keywords:** Biogenic Amine, MAO, KF1434, Vitamin, GABA.

Oxidation of biogenic amines is carried out with the participation of (MAO, KF1434), which produces aldehyde, hydrogen monoamine oxidases and ammonia:

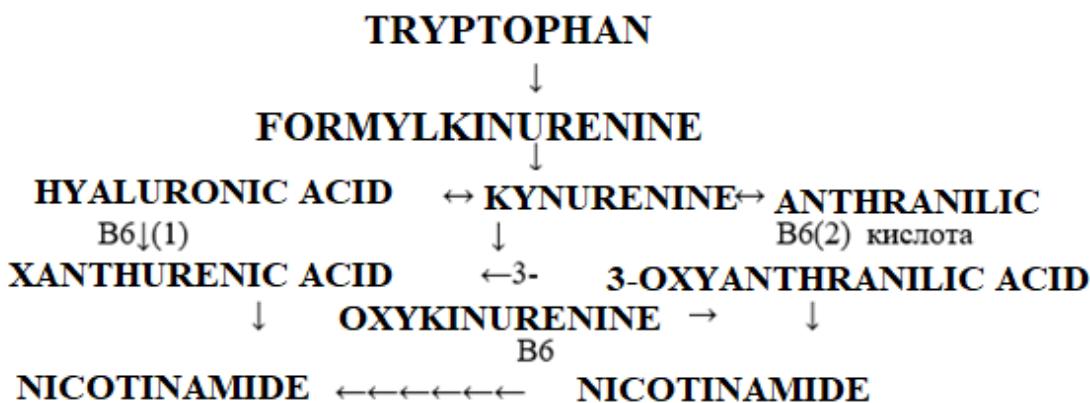


Pyridoxal-5-phosphate-containing enzyme is tryptophan synthase or L-serine hydrolase (attached indole) (CF 4.2.1.20), which involves the reaction of tryptophan synthesis:





Pyridoxal-5-phosphate-containing enzymes play an important role in the conversion of tryptophan along the kynurenine pathway (Figure 9).



### Scheme of tryptophan metabolism with the participation of pyridoxal enzymes

(designations: 1. - kynurenine transaminase; 2. – kynureninase),

With a lack of vitamin B6 in food, the metabolism of tryptophan along the kynurenine pathway is disrupted, which can lead to insufficient nicotinamide synthesis. In this case, unusually high amounts of kynurenine and xanthurenic acid appear in the urine. This is due to the fact that the conversion of kynurenine to alanine and anthranilic acid is impaired, as a result of which there is a spontaneous cyclization of kynurenine into xanthurenic acid, which is excreted in the urine.

Vitamin B6 is involved in the metabolism of carbohydrates. With a lack of vitamin B6 in the diet of animals, a decrease in the activity of glucose-6-phosphate dehydrogenase was found in the liver and adipose tissue (CF 1.1.1.49). - an enzyme that catalyzes the triggering reaction of the pentose phosphate pathway of carbohydrate metabolism - the process of direct oxidation of phosphorylated glucose.

50% of the total vitamin is in the form of pyridoxal phosphate as part of muscle phosphorylase, an enzyme that catalyzes the phosphorolytic breakdown of glycogen. It performs the functions of the glycogen phosphorylase coenzyme. The coenzyme is related to phosphorylase in the same way as transaminase, but its role is not clear



**Pyridoxine deficiency.** The main manifestation of vitamin B6 deficiency in adults is the development of seborrheic dermatitis, stomatitis, glossitis. More specific signs are hypochromic anemia and seizures.

Vitamin deficiency can be:

- the result of an intestinal infection, since it can be synthesized only with the participation of the intestinal microflora
- when its conversion into the active form is impaired
- during long-term treatment with the antituberculous drug isoniazid, which is an antagonist of pyridoxal. At the same time, increased excitability of the nervous system, polyneuritis and skin lesions characteristic of niacin deficiency also occur

Pyridoxine deficiency has also been described in children. This may be a consequence of vitamin deficiency during intrauterine life due to hypovitaminosis in a mother suffering from early toxicosis. It is accompanied by increased excitability of the central nervous system and periodic convulsions, which is associated with a reduced formation of gamma-aminobutyric acid, which is an inhibitory mediator of brain neurons, dryness, peeling of the skin, hypotrophy, hypochromic microcytic anemia, reduced resistance to infections.

Vitamin deficiency leads to impaired functions of pyridoxal-dependent enzymes, which creates the possibility of the development of enzymopathies, for example, homocystinuria develops when the enzyme cystathione synthetase is deficient.

In children, congenital pyridoxine-dependent seizure syndrome also occurs. This disease is inherited in an autosomal recessive pattern and is caused by a decrease in the activity of glutamic acid decarboxylase, which is responsible for the synthesis of GABA.



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