# ORGANIC AND MINERAL FORMS OF SELENIUM, THEIR METABOLISM, BIOLOGICAL AVAILABILITY AND ROLE IN THE BODY

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**Abstract.**The concept of bioavailability of organic and mineral forms of selenium is considered. The influence of the domestic drug selenopyran on the functional activity of the antioxidant-antiradical defense system of the body is described. The role and significance of selenopyran in increasing productivity and nonspecific resistance in animals and poultry is discussed.
**Keywords:** selenium, selenopyran, organic and mineral forms, metabolism, bioavailability, antioxidant properties.

## INTRODUCTION

The problem of selenium metabolism in animals and the closely related problem of the bioavailability of its organic and mineral forms remain not only confusing but also often misinterpreted. It is known that selenium-containing proteins present in the body of vertebrates contain only one selenium-containing amino acid, selenocysteine. In 1980, the leading specialist in selenium metabolism R.A. Sunde (University of Missouri, USA) showed on perfused rat liver that the incorporation of 75Se from selenocysteine into glutathione peroxidase is effectively inhibited by the presence of a 9-fold excess of unlabeled selenite or sodium selenide in the perfusate, while a 100-fold excess of unlabeled selenomethionine was less effective. Based on these experiments, it was concluded that inorganic selenite and selenide are better metabolized and are more preferable as precursors of selenoproteins than selenium from methionine and selenocysteine [1].

## MATERIALS AND METHODS

Selenium, which is part of selenite and selenides, is included in the amino acid serine, which then serves as the only direct precursor of selenocysteine, i.e. the body itself is able to synthesize selenocysteine using only the carbon skeleton of serine and mineral selenium. In turn, selenocysteine and selenomethionine are not direct precursors for the synthesis of selenium-containing proteins. Selenite (Se4+) and selenate (Se6+) in the body of higher animals are natural metabolic, and therefore the most accessible precursors of selenium for inclusion in all selenium-containing proteins. In plants and microorganisms, selenium is found mainly in the form of selenomethionine, but can also be found in the form of mineral and other non-metabolizable organic compounds. For example, astragalus is considered one of the best selenium accumulators. They are able to accumulate hundreds of times more of this element than it is contained in the soil and other plants growing in the same areas. The issue of including selenium-concentrating plants in the composition of dietary selenium supplements was considered, but did not find a positive solution, since these plants contain a large number of mineral (in particular, selenates) and organic forms (mainly in the form of various derivatives of selenocysteine, which are not natural products of the metabolism of this amino acid and are not capable of being included in proteins).

## RESULTS AND DISCUSSION

Selenocysteine is not found in plants and microorganisms in practically significant quantities. At the same time, with the participation of methionine tRNA, selenomethionine can be mistakenly included in proteins instead of methionine (they do not acquire any new properties, but simply serve as its carriers). However, the practical significance of this possibility is minimal. Selenomethionine in higher animals can only be included in albumin non-covalently, based on van der Waals forces, electrostatic and hydrophobic interactions, that is, without the formation of a true chemical bond. According to

G.F. Combs, a member of the US National Committee on Nutrition, the selenomethionine pool in albumin is estimated at 1-2% of the total selenium content in the body (4-6), since this amino acid performs sorption and transport functions. All known vertebrate selenoproteins contain only selenocysteine; neither a codon nor tRNA has been found for selenomethionine. Moreover, even the selenocysteine consumed by animals does not have metabolic pathways for direct inclusion in selenopeptides: for this, it first undergoes a phase of transformation into mineral selenium, after which selenocysteine is formed through serine, which serves as a precursor.

In animal tissues, there is currently only one known metabolic pathway for all selenium-containing compounds (both organic and mineral) that ultimately results in hydrogen selenide (hydroselenide anion HSe-) — a highly toxic compound that the body gets rid of in two ways. The first (catabolic pathway) involves its sequential enzymatic methylation to trimethylselenonium — the main metabolic product of all selenium-containing compounds. This pathway is reversible only at the first stage of selenium methylation. Consequently, when the conditions of selenium nutrition change, anabolic processes of selenium incorporation into endogenous proteins can begin. Trimethylselenonium is excreted from the body through the kidneys with urine. If there is an excess in the diet, selenium does not have time to turn into trimethylselenonium and is excreted with sweat and through the lungs as dimethyl selenide, giving the secretions a garlic smell, which usually serves as a qualitative criterion for such an excess. In the second case (anabolic pathway), hydrogen selenide undergoes sequential enzymatic transformations. It is activated (phosphorylated) by selenophosphate synthetase. Serine is attached to its specific transport ribonucleic acid to form the corresponding complex, serine-acyl-tRNA-adenylate. Then the activated mineral selenium is enzymatically attached to the serine-tRNA complex. The reaction is catalyzed by the enzyme selenocysteine synthetase. As a result of this reaction, selenocysteine is formed. Only selenocysteine obtained from hydrogen selenide and serine is capable of being included in selenium-containing proteins in vertebrates.

**CONCLUSION**

Thus, the domestic drug selenopyran compares favorably with all existing selenium-containing substances. Its metabolism in the body occurs along one of two main pathways. With a selenium deficiency in the diet, the drug can serve as a normal source of mineral selenium. With sufficient selenium nutrition, selenopyran undergoes a complex metabolic pathway of transformations without the release of selenium (with the possibility of recycling at a certain stage), performing the function of an antioxidant.

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