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## **INADEQUATE FAT DIET**

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### Abstract

Aim. To determine the main factors of inadequate fat diet (IAFD) and indicate the ways of their change.

Methods. Analysis and generalization of existing scientific literature data and the results of our own research.

**Results.** The following factors of IAFD have been identified:

1) excess fat in the diet;

2) an excess of palmitic acid in dietary fats;

3) an excess of linoleic acid in dietary fats;

4) consumption of thermoperoxide fats;

5) deficiency of  $\omega$ -3 polyunsaturated fatty acids (PUFA).

**Conclusion**. IAFD is a cause of metabolic and neuropsychiatric disorders underlying non-communicable diseases.

### Key words: fat nutrition; lipotoxicity; prevention of lipotoxicity.

Fats (triglycerides) are one of the parts of a large and very diverse in structure and functions class of organic compounds called lipids and united by only one common chemical property - the presence of a fatty acid [1, 2].

Biological classification of fatty acids (FA) [3]:

- 1. Bioenergetic FA
- 1.1. Oleic (C<sub>18:1</sub>)
- 1.2. Palmitic (C<sub>16:0</sub>)
- 1.3. Stearic acid (C<sub>18:0)</sub>
- 1.4. FA with C less than 16.
- 2. Irreplaceable structural PUFA (C<sub>18</sub>)
- 2.1. Linoleic acid (C<sub>18:2</sub>, ω-6)
- 2.2.  $\alpha$ -linolenic (C<sub>18:3</sub>,  $\omega$ -3)
- 2.3. γ-linolenic (C<sub>18:3</sub>, ω-6).
- 3. Irreplaceable structural and regulatory PUFA (C20 and C22)
- 3.1. Arachidonic (C<sub>20:4</sub>, ω-6)
- 3.2. Eicosapentaenoic acid (C<sub>20:5</sub>, ω-3)
- 3.3. Docosapentaenoic acid (C<sub>22:5</sub>,  $\omega$ -3 and  $\omega$ -6)
- 3.4. Docosahexaenoic acid ( $C_{22:6}$ ,  $\omega$ -3).
- 4. Anti-nutritional FA
- 4.1. Trans-isomers of FA
- 4.2. Oxygenated FA
- 4.3. High molecular weight FA
- 5. Toxic FA
- 5.1. Branched FA
- 5.2. Acetylene FA.

From a biological point of view, fats have two main functions: 1. Energy and 2. Source of essential (indispensable) fatty acids.

The energy function of fats is due to their high calorie content (9.1 kcal / g), by which they surpass all other nutrients by more than 2 times. The energy requirement of muscle tissue and heart is 80-90% satisfied due to the oxidation of fatty acids in mitochondria [4].

Being insoluble in water, fats are easily deposited in the subcutaneous adipose tissue, from where they enter the bloodstream in the form of non-esterified fatty acids during periods of fasting and thereby provide the energy requirements of organs and tissues. As for the second function of fats as a source of essential fatty acids, here we mean the need to enter the body for the synthesis of structural lipids (phospholipids, cholesterol esters, sphingomyelins) and the formation of regulatory substances (eicosanoids and docosanoids).

For humans and animals, such essential fatty acids are polyunsaturated fatty acids (PUFA), which include linoleic (C<sub>18:2</sub>,  $\omega$ -6),  $\alpha$ -linolenic (C<sub>18:3</sub>,  $\omega$ -3),  $\gamma$ -linolenic (C<sub>18:3</sub>,  $\omega$ -6), arachidonic (C<sub>20:4</sub>,  $\omega$ -6), eicosapentaenoic (C<sub>20:5</sub>,  $\omega$ -3), docosapentaenoic (C<sub>22:5</sub>,  $\omega$ -3 and C<sub>22:5</sub>,  $\omega$ -6) and docosahexaenoic (C<sub>22:6</sub>,  $\omega$ -3) acids [5, 6].

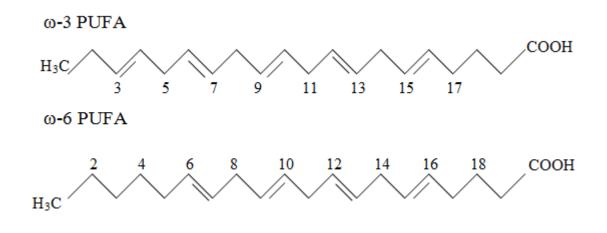


Fig. 1. The structure of  $\omega$ -3 PUFA (eicosapentaenoic) and  $\omega$ -6 PUFA (arachidonic)

From linoleic acid in humans and animals,  $\gamma$ -linolenic and arachidonic fatty acids are formed, and from  $\alpha$ -linolenic - eicosapentaenoic, docosapentaenoic ( $\omega$ -3) and docosahexaenoic fatty acids [7].

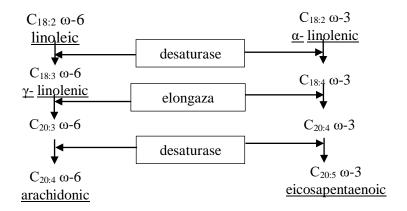


Fig. 2. Scheme of the conversion of  $C_{18}$  PUFA to  $C_{20}$  PUFA

Unfortunately, the process of conversion of  $\alpha$ -linolenic acid into long-chain fatty acids (C<sub>20</sub> and C<sub>22</sub>) in the human body is very limited, and the need for them is necessary for the

formation of structural lipids that are part of the cellular and intracellular membranes of all tissues, especially in the composition of the nervous tissue [8]. This circumstance makes it mandatory to introduce into the body with food fats with a high content of long-chain PUFA (for example, fish oil) [9, 10]. Lack of long-chain PUFA ( $C_{20}$  and  $C_{22} \omega$ -3 series) in food leads to impaired neuropsychic activity (only in the USA, 27% of the population suffers from neuropsychiatric disorders [11]) and weakened reproductive function (various forms of infertility [12].

For various reasons, in recent decades, the consumption of fish oil and seafood from northern latitudes, the richest in the content of long-chain PUFAs, has sharply decreased [9, 10]. Therefore, most of the Earth's population (except for residents of northern latitudes and some other countries in which fish consumption has not decreased, for example, Japan) is in a state of hypovitaminosis and often vitamin F deficiency with all the ensuing health consequences [13].

Returning to the question of the energy function of fats, here, too, not everything is clear. First, in civilized countries, most of the population has significantly reduced muscle load as a result of the mechanization of almost all technology processes. However, the habit of consuming fatty foods, remained, moreover, it even intensified due to taste preferences and the expansion of the range of fat-containing foods. As a result of all this, the excess intake of fat in the body of a modern person leads to an increase in its deposition, i.e., ultimately, to obesity, which affects almost 20% of the world's population (almost 1.5 billion people) [14, 15]. And in this case, the record holder is the United States, in which almost 50% of the population is overweight [16].

It should be noted that not only the consumption of fats has increased, but the set of fats in the diet has also changed due to the wider use of animal fats and palm oil, rich in saturated fatty acids (primarily palmitic acid) [17, 18]. At the recommended consumption rate of palmitic acid (no more than 10% of the total fatty acids), animal fats contain 25-30% of this acid, and palm oil up to 50% [1, 3]. The rate of oxidation of palmitic acid in mitochondria is significantly lower than the rate of oxidation of other fatty acids (for example, linoleic and, especially, oleic) [19]. Fats with a high content of palmitic acid contribute to the development of atherosclerosis and other diseases of the cardiovascular system [20].

The ideal fats for the energy function of the body are fats with a high-oleic acid content (olive oil, high oleic sunflower oil). Oleic acid is more readily oxidized than other fatty acids in mitochondria [21]. It does not inhibit the formation of long-chain PUFA [22] and does not contribute to the development of atherosclerosis and fatty liver disease [23].

Excessive consumption of dietary fats with a high content of linoleic acid (C<sub>18:2</sub>,  $\omega$ -6) is also undesirable. These are, first of all, ordinary sunflower oil (linoleic acid content 55-60%), corn and soybean oils (50-55% linoleic acid). The need for linoleic acid is 5-6 g per day [3]. An excess of linoleic acid leads to an increased formation of arachidonic acid (C<sub>20:4</sub>  $\omega$ -6), from which pro-inflammatory eicosanoids are formed [24]. As a result, a situation is created in the body that is favorable for the development of systemic inflammation [25].

Another factor in inadequate fat nutrition is the consumption of thermoperoxide fats, which are formed when using high-temperature fat cooking technologies [26]. At high temperatures, PUFA (especially linoleic acid) undergo oxidation with the formation of lipid peroxides, epoxy compounds, trans-fatty acids, aldehydes and ketones [27]. All these compounds are toxic to the body [28].

Given the widespread use of high-temperature fat cooking methods, it is necessary to take measures to reduce the formation of toxic products of thermal peroxidation (use of olive or high oleic sunflower oil instead of sunflower oil, introduction of antioxidants into the oil before heat treatment). Methods for preventing complications of the consumption of thermoperoxide fats by taking preparations containing bioflavonoids are also effective [29].

Thus, to summarize all of the above, inadequate dietary fat is the following:

1. High-fat food (the fat content in the diet is more than 30% of calories).

High-fat nutrition causes the development of not only obesity, but is the cause of the development of dysbiotic syndrome [25]. If it is not possible to reduce excessive consumption of fats, then there are two recommendations: first - replace, if possible, dietary fats with a high content of palmitic or linoleic acid for high oleic sunflower oil (for example, oil "Olivka"); the second is to use antidisbiotic agents containing antioxidants and prebiotics (for example, dietary supplements "Kvertulin", "Lekvin" or "Lysozyme-Forte") [22, 25, 29].

2. High content of palmitic acid in edible fats (over 10%). Recommendations in this case: as with a high-fat diet.

3. High content of linoleic acid in edible fats. Here the recommendations are similar to those indicated in paragraphs 1 and 2.

4. Deficiency of essential PUFA due to low consumption of fish oil and seafood with a high content of  $\omega$ -3 PUFA. The daily consumption rate of PUFA is: linoleic acid – 5-6 g,  $\alpha$ -linolenic acid – 1-2 g, eicosapentaenoic acid – 0.5-0.7 g, docosahexaenoic acid – 0.4-0.6 g.

We have proposed a dietary supplement "Liposan-forte" (oil solution), which contains all essential PUFA. One tablespoon of Liposan-Forte provides the daily requirement for PUFA, and the  $\omega$ -6 /  $\omega$ -3 PUFA ratio is significantly less than 1 [30].

5. Consumption of thermal peroxide fats of fat-containing products, which must be limited by using fats that are more resistant to thermal peroxidation (for example, high oleic sunflower oil) for high-temperature fat cooking. A method of preventing the undesirable effect of thermoperoxide fats by taking bioflavonoid preparations (for example, "Kvertulin") is recommended.

The transition from inadequate to adequate fatty nutrition will prevent the development of a large number of diseases (atherosclerosis, obesity, type 2 diabetes mellitus, neuropsychic and metabolic disorders), which are the main cause of high mortality in the population.

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