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SUPPOSEDLY HEALTHY PLANT FATS
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Abstract
Nutritionists, dieticians and doctors have long considered plant fats to be the main source of omega-6 and omega-3 essential fatty acids (EFAs) in the human diet. The market offers a wide variety of plant oils which can be consumed raw or used for frying. In the past four decades, the consumption of vegetable fats increased several fold due to marketing campaigns advertising the health benefits of vegetable oils. Plant oils are a valuable source of n-6 polyunsaturated fatty acids whose excess supply can pose health risks for consumers. Analyses of oxidative stability of plant fats (during processing and storage) have shown that the consumption of oxidation products of fatty acids and sterols could cause various diseases. The results of epidemiological and clinical studies indicate that if plant fats (including oils, but not olive oil and margarines) were to deliver the alleged health benefits, the problem of atherosclerosis and its clinical complications (heart attack, stroke) could be mitigated through regular consumption of vegetable oils. Meanwhile, every second death in industrialized countries is caused by cardiovascular diseases. The incidence of cancer, neurological and neurodegenerative diseases is also increasing rapidly.

Key words: plant fats, essential fatty acids, oxidation, oxidative stability, artificial trans-isomers
Introduction

Despite high levels of consumption of supposedly healthy plant fats, health statistics in industrialized countries have not improved. The incidence of cancer and neurological diseases is growing rapidly, and the risk of atherosclerosis and its clinical complications, including heart attack and stroke, has not been mitigated [11].

According to the current state of knowledge, fat consumption levels are not the only risk factor in many diet-dependent metabolic diseases, such as atherosclerosis, obesity, type 2 diabetes, cancer and neurodegeneration. The composition and ratios between various groups of fatty acids are equally important considerations [4, 13, 25].

Vegetable oils as a source of essential fatty acids (EFAs)

Plant oils, excluding palm oil and coconut oil, contain high levels of unsaturated fatty acids (UFAs) and low concentrations (below 15%) of saturated fatty acids (SFAs). They differ with regard to the ratios between monounsaturated (MUFAs) and polyunsaturated fatty acids of omega-3 (n-3 PUFAs) and omega-6 (n-6 PUFAs) families. The n-6/n-3 PUFA ratio in sunflower, corn and grapeseed oils is 335, 141 and 173 to 1, respectively [4, 9].

Plant oils are a natural source of PUFAs: n-6 linoleic acid (LA, C18:2) and n-3 α-linolenic acid (ALA,C18:3). They are essential for the development and functioning of the human body, and a deficiency of those fatty acids can have adverse health consequences. EFAs are not synthesized by the human body (due to the inability to introduce double bonds in positions n-3 and n-6 of the carbon chain), therefore, they have to be supplied with food [9, 11, 13].

n-6 LA and n-3 ALA are not biologically active, whereas their long-chain derivatives produced in enzymatic reactions demonstrate biological activity. n-6 LA is synthesized to form γ-linolenic acid (GLA, C18:3), dihomo-γ-linolenic acid (DGLA, C20:3) and arachidonic acid (AA, C20:4). n-3 ALA is converted to eicosapentaenoic acid (EPA, C20:5) and docosahexaenoic acid (DHA, C22:6). Long-chain derivatives undergo synthesis if sufficient amounts of EFAs are supplied with food and if there are no enzyme defects in their metabolic pathway [9,11, 13].

Animal fats contain small amounts of n-6 and n-3 PUFAs in optimal proportions. Coldwater fish and sea mammals are the richest sources of biologically active long-chain PUFAs in the human diet [15].

n-6 and n-3 PUFAs are found in cell membrane phospholipids, and they are integral tissue components. When released from membrane phospholipids, they synthesize tissue hormones – eicosanoids. The results of many studies have shown that EPA and DHA differ in
their metabolic activity. n-3 EPA synthesizes eicosanoids (prostaglandins – e.g. PG₃, thromboxanes – e.g. TXA₃, leukotrienes – e.g. LT₅, and prostacyclins – e.g. PGI₃) which dilate blood vessels and demonstrate anti-inflammatory, antithrombotic, anti-allergic, anti-atherosclerotic and anticarcinogenic activity. Eicosanoids exert complex control over the body's metabolic system, and they prevent cardiovascular diseases. n-3 DHA is a vital component of nervous tissue (in particular the cerebral cortex) and the eye retina. n-3 DHA has a 25-35% share of all fatty acids in the human brain and up to a 60% share in neuron phospholipids. DHA is essential for brain development, and it is accumulated between weeks 25 and 40 of pregnancy. A DHA deficiency in the diet of small children may lead to vision deterioration, decrease in intelligence levels, attention deficit hyperactivity disorder (ADHD) and atopic dermatitis [9, 11, 13, 15, 25]. The only natural dietary sources of EPA and DHA are fish fats (mackerel, herring, salmon, tuna) and the fat of sea mammals [13, 15].

High consumption of plant fats rich in n-6 LA acid may attenuate the beneficial effects of EPA and DHA. Excessive synthesis of eicosanoids from arachidonic acid (e.g. PG₂, TXA₂, LT₄, PGI₂) intensifies inflammatory, allergic and thrombotic reactions, narrows the vascular lumen, contributes to the proliferation of neoplastic cells and promotes the growth of mammary gland, prostate and colorectal tumors [9, 11, 13].

n-3 ALA is not a metabolic equivalent of EPA and DHA, and n-6 LA is not a metabolic equivalent of n-6 AA. It is believed that 1 g of EPA and DHA is synthesized from 3-4 g of n-3 ALA [31]. n-3 ALA is enzymatically converted to long-chain derivatives (through elongation of the carbon chain and desaturation, i.e. the insertion of additional unsaturated bonds) in a cell's endoplasmic reticulum. Since elongase and desaturase (delta-6 desaturase) activity is conditioned by metabolic factors, ALA is not always converted to long-chain derivatives (EPA and DHA) in the human body. Zinc, magnesium and vitamin B₆ deficiencies, insulin secretion disorders and the use of anti-thrombotic drugs may inhibit the activity of elongase and desaturase. Low levels of enzymatic activity were observed in prematurely born infants, elderly people, diabetes and hypertension patients [13,17].

In most cases, the synthesis of n-3 ALA in the human body is blocked by excessive levels of n-6 PUFAs from plant oils. Most vegetable oils are characterized by a highly unfavorable n-6/n-3 PUFA ratio. The only exceptions are rapeseed oil and linseed oil. n-6 LA and n-3 ALA are converted to long-chain derivatives by the same enzymes. Omega-6 fatty acids are the first to be synthesized, therefore their excessive levels inhibit the transformation of n-3 ALA, which further deepens the imbalance between n-6 and n-3 PUFAs. Desaturase activity is also inhibited by artificial trans-isomer fatty acids which are prevalent in margarines, bakery fats, frying fats, sweets and fast foods. The above process blocks the
synthesis of n-6 AA, n-3 EPA and DHA. The conversion of EFAs supplied with plant oils into biologically active derivatives (n-6 AA, EPA and DHA) is limited and relatively ineffective in the human body [11, 13, 17].

Vegetable oils, including sunflower, corn, soybean and grapeseed oils, are characterized by an unfavorable n-6/n-3 PUFA ratio, therefore they should not be regarded as a healthy source of n-3 EFAs. Plant oils supply mainly n-6 LA which, if consumed in excessive amounts, converts n-6 AA to highly biologically active eicosanoids. Excessive levels of eicosanoids are potentially harmful [4, 25].

Health consequences of diets containing excessive levels of n-6 LA

High levels of dietary n-6 PUFAs may lead to health disorders for a variety of reasons. Long-chain PUFAs in cell membrane and organelle phospholipids determine their structure, liquidity and permeability. When long-chain PUFAs are released from cell structures, they are converted to tissue hormones – eicosanoids. Eicosanoids (prostaglandins, prostacyclins, thromboxanes, leukotrienes and lipoxins) synthesized from n-6 AA demonstrate high levels of activity. Excessive levels of eicosanoids stimulate inflammatory and allergic reactions, atherosclerotic and thrombotic processes, and carcinogenic changes. They have immunosuppressive effects [9, 11].

High levels of eicosanoids synthesized from n-6 AA pose a threat of metabolic diseases. The n-6/n-3 PUFA ratio determines healthy bodily function. Epidemiological studies have shown that the optimal n-6/n-3 PUFA ratio is 4-5:1. Excessive levels of n-6 PUFAs inhibit the conversion of n-3 ALA to n-3 EPA and DHA, which further deepens the imbalance between n-6 and n-3 PUFAs in the body [9, 13, 25, 26].

Our ancestors consumed far less fat and SFAs, and their diets were more balanced with regard to the content of n-6 and n-3 PUFAs [23]. High consumption of vegetable oils rich in n-6 PUFAs combined with low consumption of fish, the optimal source of n-3 PUFAs, produces excessive levels of n-6 PUFAs in contemporary diets. In a typical Western diet, the n-6/n-3 PUFA ratio is 20-30:1, and it is regarded as the major risk factor for metabolic diseases. The results of epidemiological and clinical research indicate that excessive consumption of n-6 PUFAs is more harmful than the ingestion of high levels of SFAs [13, 25].

The n-6 and n-3 families of PUFAs compete for the same enzymes (cyclooxygenase, COX, and 5-lipoxygenase, 5-LO) which participate in their synthesis to biologically active eicosanoids. Excessive levels of dietary n-6 PUFAs inhibit the transformation of n-3 PUFAs to compounds that have a beneficial influence on human health [11].
The majority of cancers, in particular in industrialized countries, are related to diet. Fat and fatty acids are regarded as risk factors for certain types of tumors, including breast, prostate and colorectal cancer [2, 9, 22, 24]. n-6 and n-3 PUFAs may have different effects on carcinogenesis. Subject to various mechanisms, they may exert both pro-carcinogenic and anti-carcinogenic effects [9, 26].

n-6 PUFAs, excluding GLA (C18:3, n-6), stimulate the proliferation of cancer cells and promote tumor growth. The effects of diets rich in n-6 PUFAs on the initiation of cancer have not yet been conclusively established. There is ample evidence to indicate that the secondary peroxidation products of n-6 PUFAs, present in excessive amounts in the diet, may induce DNA mutations and initiate carcinogenesis. n-6 LA, a substrate for the synthesis of prostaglandins which inhibit the production of interleukin 12 (angiogenesis inhibitor), stimulates angiogenesis, and it may weaken the anti-proliferative effects of n-3 EPA. n-3 PUFAs have an opposite effect, and they prevent the initiation, promotion and progression of cancer [9, 13].

Some indirect products of enzymatic transformations of n-6 LA also have anticarcinogenic properties. Delta-6 desaturase and LA synthesize GLA (C18:3, n-6) which is elongated to produce DGLA (C20:3, n-6). Since elongation takes place at a faster rate than desaturation, DGLA is generally produced in larger amounts than GLA [31].

Diets supplemented with GLA increase DGLA concentrations in body tissues (including serum lipids, blood platelets and red blood cells). They lower the activity of delta-5 desaturase, which inhibits the conversion of DGLA to AA. A balanced ratio of DGLA and AA in membrane phospholipids prevents excessive peroxidation. GLA regulates biochemical processes by inhibiting the production of AA and eicosanoids [31]. GLA is found in the seeds of evening primrose (5-10%), blackcurrant (15-20%) and borage plants (20-25%) [9, 20, 26].

In comparison with SFAs, PUFAs significantly increase the body's demand for antioxidants, and they are the number one cause of the prooxidant-antioxidant imbalance in the human body [5, 6].

Composition of fatty acids in vegetable oils and their susceptibility to oxidation

Plant oils differ with respect to their fatty acid profile (various proportions of SFAs, MUFAs, n-6 and n-3 PUFAs), the presence of conjugated bonds, antioxidant content and oxidative stability. The autoxidation of PUFAs produces free radicals which contribute to various diseases, mostly cancer. For this reason, the oxidative stability of vegetable and animal fats is a vital indicator of their safety for consumers [5, 17].
Fats containing high levels of oleic acid (olive oil, rapeseed oil, palm oil and lard) are characterized by greater oxidative stability than oils rich in PUFAs [6, 17]. Susceptibility to oxidation grows with an increase in the number of unsaturated bonds in different fatty acids (Table 1).

Table 1
The rate of fatty acid oxidation subject to the degree of unsaturation [5, 6]

<table>
<thead>
<tr>
<th>Fatty acid characteristic of the product</th>
<th>Product</th>
<th>Number of double bonds</th>
<th>Oxidation rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stearic acid</td>
<td>tallow, lard</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Oleic acid (n-9)</td>
<td>olive oil</td>
<td>1</td>
<td>10</td>
</tr>
<tr>
<td>Linoleic acid (n-6)</td>
<td>sunflower oil</td>
<td>2</td>
<td>100</td>
</tr>
<tr>
<td>α-linolenic acid (n-3)</td>
<td>linseed oil</td>
<td>3</td>
<td>250</td>
</tr>
<tr>
<td>Eicosapentaenoic acid (EPA, n-3)</td>
<td>fish oil</td>
<td>5</td>
<td>350</td>
</tr>
<tr>
<td>Docosahexaenoic acid (DHA, n-3)</td>
<td>fish oil</td>
<td>6</td>
<td>350</td>
</tr>
</tbody>
</table>

Rapeseed oil and linseed oil have the highest total content of unsaturated fatty acids (more than 90%). The main linseed oil component is n-3 ALA which is 25-times more susceptible to oxidation than oleic acid, the predominant component in rapeseed oil (more than 50%). Soybean, corn, sunflower and grapeseed oils contain high levels of n-6 LA (55.07% to 65.90%), which makes them highly susceptible to oxidation. Fish oils, the most valuable source of biologically active long-chain n-3 PUFAs (EPA and DHA), are characterized by the highest rate of oxidation due a high degree of unsaturation (iodine value 202) [3].

Although they are regarded as oxidatively stable, plant oils containing oleic acid are also susceptible to oxidation. In comparison with other oils, however, olive oil and palm oil are characterized by significantly higher oxidative stability due to low levels of n-3 ALA and high concentrations of n-9 oleic acid. Animal fats which contain high levels of oleic acid, SFAs and active antioxidants are least vulnerable to oxidation. The highest relative oxidative stability of edible fats, determined based on the content of fatty acids and their susceptibility to oxidation, is noted in beef tallow (0.86), palm oil (1.3), olive oil (1.5) and lard (1.7). Soybean oil (7.0) and sunflower oil (6.8) are characterized by significantly lower oxidative stability [16].

Vegetable oils undergo intensified oxidation during frying [17]. The impact of high temperature on highly reactive double bonds in unsaturated fatty acids leads to isomeration and the production of trans-isomers. Food products have a high water content which
accelerates fat hydrolysis, and similarly to fatty acids with conjugated bonds, free fatty acids are much more susceptible to oxidation. Cold-pressed oils contain antioxidants, and the synthesis of primary and secondary oxidation products takes place at a much slower rate than in refined oils [1, 8].

Primary oxidation products, hydroperoxides and peroxides, are metabolized by intestinal microflora or converted to hydroxy acids. Secondary oxidation products, such as aldehydes, ketones, hydrocarbons, epoxides, polymers and free radicals synthesized from primary oxidation products, pose a health risk. They are characterized by very high levels of biological activity, they damage cell membranes and organelles (such as mitochondria), inhibit enzyme activity and exert cytotoxic effects. Most secondary oxidation products have mutagenic potential, and they are involved in neoplastic transformation [17, 19].

Thermal processing of oils leads to the production of cyclic monomers and polymers which cause gastrointestinal disorders. Cyclic monomers are effectively absorbed from the digestive tract, and they may damage internal organs. They are also characterized by mutagenic and carcinogenic activity [21].

High processing temperatures contribute to the oxidation of sterols in plant oils [30]. Oxysterols exhibit cytotoxic, mutagenic, carcinogenic and immunosuppressive effects. The presence of oxyphytosterols accelerates cholesterol oxidation, both in food and in the human body. The involvement of oxysterols in the pathogenesis of atherosclerosis has been well documented [7, 10]. The use of vegetable oils in thermal food processing poses evident health risks proportionally to the content of harmful aldehydes and ketones, including malondialdehyde (MDA) [8, 27], cyclic monomers [21], oxyphytosterols [10], and at temperatures higher than 200°C – also acrylamide [18]. The oxidation products of PUFAs and phytosterols supplied by plant oils are the main initiators of atherosclerotic and carcinogenic changes in the human body. They speed up degenerative changes and cell ageing processes.

**Plant oils and the prooxidant-antioxidant imbalance**

The consumption of vegetable fats leads to a logarithmic increase in the demand for antioxidants, proportionally to the content of PUFAs and the number of unsaturated bonds. Excessive consumption of vegetable oils weakens antioxidant defense mechanisms. Antioxidants do not always effectively counteract oxidation due to disproportions between the kinetics of free radical and reactive oxygen species (ROS) production and elimination. The kinetics of ROS generation is several orders of magnitude higher than that of reactions involving antioxidants. Disturbances in prooxidant-antioxidant homeostasis lead to metabolic diseases, and ROS play an important role in their pathogenesis (inflammations,
atherosclerosis, hypertension, diabetes, cancer). Diets rich in antioxidant vitamins (A, E and C) and natural antioxidants (polyphenols) counteract those health risks [4, 11, 13].

The Mediterranean diet is a prime example of the above. Its health benefits can be attributed to the composition of olive oil: 68% monounsaturated oleic acid (less susceptible to oxidation), 10% n-6 LA and 0.7% n-3 ALA. Olive oil contains phenolic compounds which protect oleic acid and PUFAs against oxidation (but their effectiveness is reduced considerably under exposure to high temperatures) [1]. Other ingredients of the Mediterranean diet, including vegetables, fruit and red wine, are also rich sources of antioxidants. People who follow a Mediterranean diet consume high levels of biologically active n-3 PUFAs (EPA and DHA) in fish and sea food, and they maintain a healthy n-6/n-3 PUFA ratio by eating fewer foods which are a source of n-6 PUFAs. Low consumption of artificial trans-isomers is also a characteristic feature of the Mediterranean diet [12, 28].

The prooxidant-antioxidant balance can be maintained by eating a diet which caters to the real demand for EFAs. In healthy adults, the reference daily intake for LA and ALA is as low as 4.5 g and 1.0 g, respectively [15]. Only 1 g of fish oils, which contain biologically active long-chain PUFAs, supplies the daily requirement of EFAs.

Very high levels of PUFAs can enhance pro-oxidative processes and disrupt the prooxidant-antioxidant balance. The lipid peroxidation chain reaction affects the key properties of cell membranes – cohesion, liquidity and permeability. Structural lipid peroxidation not only disrupts a cell's life functions, but it can also lead to cell death. Excessive levels of n-6 and n-3 PUFAs contribute to the peroxidation of membrane lipids in cells and LDL molecules (production of oxLDL). The oxidation of long-chain PUFAs and AA metabolites is a major factor in the pathogenesis of atherosclerosis [19, 27]. Proteins are also highly susceptible to oxidative change. The accumulation of oxidatively modified proteins leads to a gradual loss of a cell's biochemical and physiological functions. Fragmentation, cross-linking and the production of protein aggregates contribute to cell and organ dysfunctions [11].

The highest health risk is posed by ROS-induced DNA damage. Oxidative modifications of DNA (oxidation of nitrogen bases, single- and double-strand breaks, adduct formation) may have carcinogenic effects [20].

Artificial trans-isomer fatty acids – the main cause of metabolic diseases

The findings of numerous research studies have demonstrated that trans-isomer fatty acids pose a health hazard. During the hydrogenation process in the production of margarines, PUFA double bonds become completely saturated with hydrogen which leads to 100% loss of
LA and ALA. The resulting geometric and positional isomers of unsaturated fatty acids contribute to the risk of cardiovascular diseases, cancer, obesity, and they impair immune functions [12, 28].

Trans-isomers present in solid margarines disrupt lipid metabolism and promote atherosclerotic changes. Trans-isomer fatty acids block transacetylase, an enzyme responsible for cholesterol esterification, and inhibit cholesterol metabolism. The melting point of trans-isomers is around 20°C higher than that of cis-isomers, therefore, they have a solid consistency in the human body. The above, combined with the Z configuration of trans-isomers, supports cholesterol crystallization and contributes to the formation of atherosclerotic plaques [23].

Trans-isomer fatty acids in hydrogenated vegetable oils induce a similar increase in the concentrations of LDL-cholesterol as SFAs. Yet unlike SFAs, trans-isomer fatty acids decrease the levels of HDL-cholesterol and produce an unhealthy LDL/HDL ratio. Diets rich in trans-isomers also increase the levels of lipoproteins (a) and triglycerides, potential risk factors for atherosclerosis and ischemic heart disease [12, 17].

Trans-isomers in cell membrane structures disrupt ion channel function, which leads to cell stiffness and dysfunction. Membrane function deteriorates with an increase in the concentrations of trans-isomers. The above is observed in the inner layer of blood vessels which prevents thrombosis and regulates blood flow. Vascular dysfunctions trigger atherosclerosis [14]. Elevated levels of trans-isomers in cardiac and nervous system cells contribute to arrhythmia.

According to a report on the effects of hydrogenated plant oils on human health, published by the Danish Nutrition Council in 1994, margarine consumption increases the risk of heart disease ten-fold in comparison with an equivalent consumption of SFAs. The correlation between the consumption of trans fatty acids and increased risk of heart disease has been validated by the results of epidemiological studies (three research projects where 150,000 patients were monitored over a period of 6-14 years).

The Euramic research program of 1997 and the Transfair study of 2000 demonstrated a correlation between trans-isomer levels in adipose tissue and the incidence of breast, prostate and colorectal cancer. A positive correlation was also reported between the consumption of trans fatty acids and the prevalence of allergy and asthma in 13-14-year-old children undergoing treatment in 155 medical centers around the world (ISAAC, 1998) [24, 29].

The data accumulated by the Nurses' Health Study over a period of 14 years indicate that the consumption of artificial trans-isomers contributes to the risk of type 2 diabetes. It
was found that elaidic acid (9 \textit{trans} 18:1) promotes insulin resistance which causes the metabolic syndrome, an imbalance in lipid metabolism, obesity and type 2 diabetes. A study of prematurely born children revealed a negative correlation between birth weight and trans-isomer levels in the blood plasma of newborns [26].

Polish food products contain high levels of trans-isomer fatty acids whose daily consumption reaches 2.8 to 6.9 g. According to research data, a 2% increase in dietary energy supplied by trans-isomers (5 g/day) increases the risk of coronary artery disease by around 23%. In view of the harmful health effects of trans-isomer fatty acids, urgent measures are needed to control their levels in food products [12].

\textbf{Conclusions}

For several decades, vegetable oils were regarded as the main source of EFAs, although most of them do not contain significant amounts of n-3 PUFAs. Soybean, corn, sunflower and grapeseed oils supply mostly LA (55 to 66%), and their n-6/n-3 PUFA ratio equals 8, 140, 355 and 173 to 1, respectively [9].

Populations which are dependent on a traditional Western diet and which consume increasing amounts of plant oils rich in omega-6 PUFAs are characterized by higher prevalence of atherosclerosis with the accompanying complications, including heart attack and stroke, as well as growing incidence of cancer. Cancer rates are low among the inhabitants of Greenland and Alaska who eat high-fat diets (fat supplies up to 50% of energy). In Greece, where 42% of dietary energy comes from fat, mostly olive oil, the incidence of cancer is significantly lower than in the US where fat supplies 35% of dietary energy. The results of epidemiological research indicate that increased consumption of margarines and plant oils rich in LA is a risk factor for cancer [2, 9, 25, 26].

Incidence rates of cardiovascular diseases, cancer and neurological disorders increase proportionally with higher consumption of vegetable oils due to:

- high levels of LA in plant fats,
- unhealthy n-6/n-3 PUFA ratio in the diet and excessive levels of eicosanoids synthesized from AA,
- prooxidant-antioxidant imbalance caused by excessive dietary levels of n-6 PUFAs,
- high, uncontrolled levels of artificial trans-isomers in convenience foods,
- thermal processing of vegetable fats which are highly susceptible to oxidation,
- accumulation of mutagenic and carcinogenic substances in fats (not only animal fats), including dioxins, PCB, PAH and heavy metals.
The growing incidence of cardiovascular diseases, cancer, neurological and neurodegenerative disorders disproves the supposed health benefits of vegetable fats (sunflower, corn, soybean and grapeseed oils, hydrogenated margarines) and challenges the decades-old dietary guidelines.

References


[15]. MacKay S. Techniques and Types of Fat used in Deep-Fat Frying - A policy statement and background paper. Heart Foundation of New Zealand 2000
[30]. Ziemlański Š.: Fizjologiczna rola kwasów tłuszczowych n-6 i n-3 w ustroju człowieka ze szczególnym uwzględnieniem profilaktyki cywilizacyjnych chorób metabolicznych., Mat. Symp. Olej z wiesiołka i inne oleje zawierające kwasy n-6 lub n-3 w profilaktyce i terapii. Sulejów 1998