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SUPPOSEDLY UNHEALTHY ANIMAL FATS

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Abstract

Animal fats are generally considered to be a source of saturated fatty acids and cholesterol, the believed causes of atherosclerosis and its clinical complications (cardiovascular diseases, including heart attack, stroke, cerebral claudication). The real cause of atherosclerosis are inflammations of vascular endothelium resulting from oxidative stress, hyperhomocysteinemia, hypertriglyceridemia, the presence of artificial trans-isomers and excessive levels of eicosanoids originating from n-6 polyunsaturated fatty acids. According to the present status of knowledge, saturated fatty acids and cholesterol present in animal foods do not cause inflammations. Moreover, animal fats are a source of antioxidants which are active both in food and in the human body. Due to their high oxidative stability, animal fats do not pose a threat to human health. Despite a high content of saturated fatty acids and cholesterol, milk fat offers comprehensive protection against atherosclerosis and carcinogenesis

Key words: animal fats, atherosclerosis, LDL and HDL cholesterol, antioxidants

Introduction

Aggressive marketing campaigns of edible fats have made a scientifically unfounded distinction between supposedly healthy plant oils and supposedly unhealthy animal fats. The advertised health benefits of plant oils changed the fat consumption model in Poland. In the 1990s, the demand for vegetable fats increased rapidly with a simultaneous drop in the consumption of animal fats. At present, oils and margarines cater to 60% of domestic demand for fats, marking a significant increase from 30% in the previous decades. Despite expectations, this marked increase in the consumption of plant oils did not lead to an improvement in the consumers' health, and the observed trend was opposite to the predictions. In the past 40 years, the prevalence of atherosclerosis was not reduced, while the incidence of neoplastic, neurological and neurodegenerative diseases increased more than four-fold.

Contrary to popular belief that animal fats have atherogenic properties, saturated fatty acids (SFAs) and exogenous cholesterol do not increase the risk of atherosclerosis. The most common cause of hypercholesterolemia is the inhibition of transacetylase, an enzyme responsible for the esterification of cholesterol by artificial trans-isomer fatty acids present in margarines, which inhibits cholesterol metabolism in the body.

A dietary deficiency of essential unsaturated fatty acids may also lead to hypercholesterolemia. The first enzymatic reaction in cholesterol biosynthesis involves the esterification of cis-polyunsaturated fatty acids (PUFAs). Thus, cholesterol metabolism is

intensified by essential unsaturated fatty acids, and n-3 PUFAs are most effective in improving the blood lipid profile.

Unlike plant oils, animal fats have a positive effect on the prooxidant-antioxidant homeostasis. They are a source of bioactive components characterized by high levels of antioxidant activity. The antioxidants present in animal fats are active in foods as well as in the human body.

Biological functions of lipids

Lipids are essential for the healthy functioning of the human body and, at certain stages of development, they are more important than proteins. Dietary fats are not only a source of readily available energy. In the human body, they are the basic building blocks of cell membranes, the brain and the nervous system. The structure and functions of cell membranes are determined by the ratio of SFAs to monounsaturated (MUFAs) and polyunsaturated fatty acids, including n-6 PUFAs (linoleic acid, arachidonic acid, γ-linolenic acid and dihomo-γ-linolenic acid) and n-3 PUFAs (α-linolenic acid and its long-chain derivatives - eicosapentaenoic acid [EPA] and docosahexaenoic acid [DHA]) in the phospholipid profile. The human body contains smaller amounts of n-3 than n-6 PUFAs [30].

SFAs form simple and rigid chains in the lipid layer, whereas unsaturated fatty acids contribute to the relaxation of the membrane structure, thus increasing its liquidity and permeability. Cell membranes contain around 30% cholesterol which affects their rheological properties and supports changes in the shape of bodily organs [28, 30].

The quality of dietary fats, in particular the amount and proportions of n-6 and n-3 PUFAs, affect the function of the brain which is made up of lipids in 60%. High DHA concentrations in selected organs (30-35% in the cerebral cortex, 20-25% in retinal receptor phospholipids and cell membrane phospholipids) indicate that this fatty acid plays a vital role in their function [21]. DHA is also essential for optimal brain and nervous system development in fetal life, which is why it is accumulated between weeks 25 and 40 of pregnancy. The n-6/n-3 PUFA ratio determines the structure of nerve cells, the number of synapses and dendrites, and the effectiveness of information transfer between neurons [23].

PUFAs play an important role in lipid metabolism. HDL and LDL cholesterol transported in lipoproteins has the form of an ester with *cis*-PUFAs. PUFA ability to improve the blood lipid profile is determined directly by the number of unsaturated bonds. The highest effectiveness was reported in respect of n-3 α-linolenic acid, and the lowest – n-9 oleic acid

[8]. PUFAs inhibit triglyceride synthesis, they control insulin secretion and they are a source of eicosanoids, referred to as tissue hormones [14, 27].

Unlike PUFAs, long-chain SFAs are the basic building blocks of lipids, phospholipids and lipoproteins in all tissues and organs of the human body [30].

Supposedly atherogenic properties of saturated fatty acids

SFAs have been long believed to possess hypercholesterolemic and atherogenic properties, although convincing scientific evidence has never been proposed to back this claim. High levels of SFAs are found in milk fat (62.5%), followed by beef tallow and lard (47.8% and 40%, respectively). SFAs are associated mainly with animal fat, although palm oil and coconut oil also contain high levels of the discussed fatty acids (49.6% and 87%, respectively). Human adipose tissue is made up predominantly of monounsaturated oleic acid (46.9%), followed by SFAs (42.9%), whereas PUFA levels are low at approximately 10% (Fig. 1).

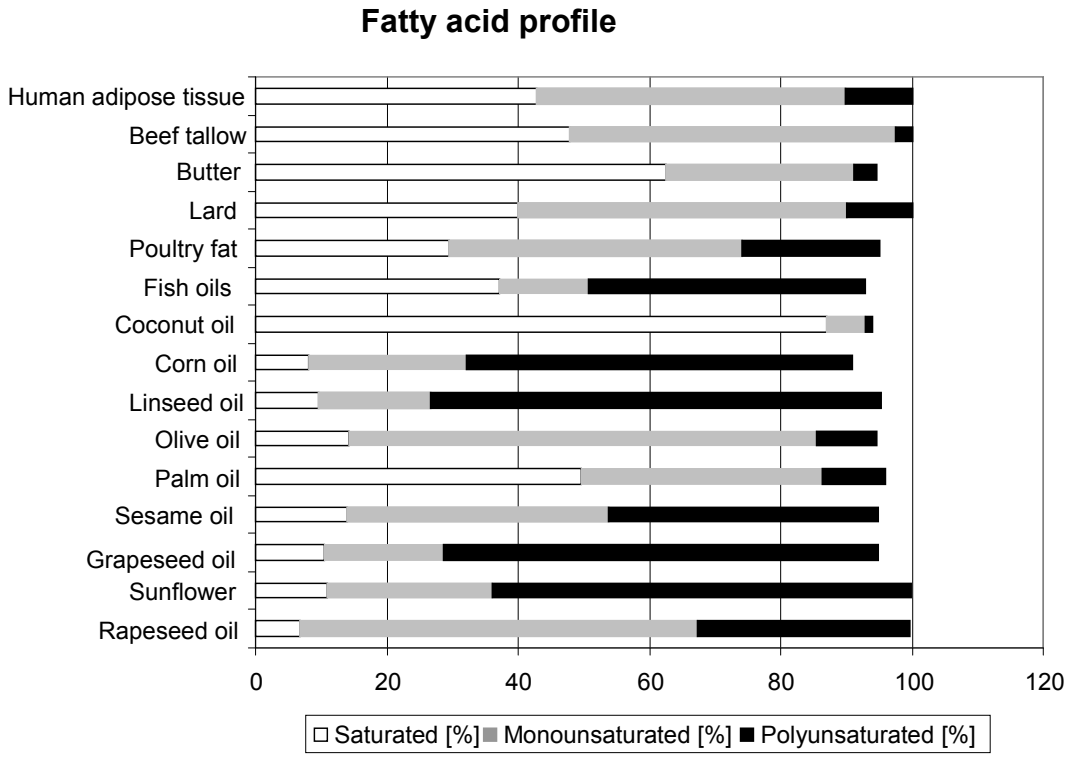

Fig. 1. Fatty acid profiles of various fats [5].

The consumption of SFAs is correlated with higher levels of total cholesterol and LDL-cholesterol. The above does not indicate, however, that SFAs have harmful effects [30].

Cholesterol metabolism is determined by the presence of n-6 and n-3 PUFAs which are not synthesized in the human body and have to be supplied with food. Due to their significant biological role, n-6 and n-3 PUFAs are considered essential fatty acids (EFAs). Diets deficient in EFAs have negative health consequences. The above is demonstrated by cholesterol transformation processes – the first enzymatic reaction which conditions cholesterol metabolism involves the esterification of *cis*-PUFAs [21, 28].

The findings of Keyes, which gave rise to the hypercholesterolemic theory of atherosclerosis, continue to be misinterpreted. When the results of his work are applied to the biosynthesis of cholesterol, it becomes evident that cholesterol metabolism is slowed down by a deficiency of PUFAs (in particular in the long-term) and not SFAs. It has been demonstrated that n-3 PUFAs are six times more effective in improving the blood lipid profile than n-6 PUFAs which, in turn, are two-three times more effective than n-9 oleic acid (due to differences in the number of unsaturated bonds) [8, 29]. A much greater improvement in the lipid profile is noted when larger quantities of n-3 PUFAs are consumed then when animal fats are eliminated from the diet.

The following facts stand in contradiction to the claim regarding the supposedly hypercholesterolemic properties of SFAs:

- the incidence of disease and death caused by atherosclerosis is low in populations consuming animal fats supplemented with n-3 PUFAs (Eskimo paradox);
- the presence of animal fats in low-calorie diets does not deteriorate the blood lipid profile;
- the elimination of animal fats from diets of heart attack patients had offered no visible health benefits, but the subjects' condition improved significantly when diets containing animal fats were supplemented with n-3 PUFAs [21].

The Polish Fat Consensus statement promotes a theory claiming that SFAs are as detrimental to consumer health as trans-isomer fatty acids present in margarines. It is true that an SFA-induced increase in LDL-cholesterol levels is similar to that caused by trans-isomer fatty acids. Yet unlike trans-isomer fatty acids, SFAs increase the levels of HDL-cholesterol which are reduced by the former. Therefore, the consumption of SFAs contributes to a healthy ratio between LDL-cholesterol and HDL-cholesterol. By contrast, trans-isomer fatty acids disrupt the LDL-cholesterol and HDL-cholesterol balance by increasing the LDL fraction and lowering HDL-cholesterol levels. In contrast to trans-isomer fatty acids, SFAs do not raise the levels of lipoproteins and triglycerides which are also atherosclerosis risk factors [16].

Artificial trans-isomers completely block cholesterol metabolism by inhibiting transacetylase, an enzyme responsible for cholesterol esterification. The consumption of

margarines containing artificial trans-isomers increases the risk of atherosclerosis ten-fold in comparison with an equivalent consumption of SFAs present in animal fats [16].

There are no justified grounds for equating animal fats with SFAs. Milk fat, lard and tallow contain also MUFAs (35% in milk fat, around 50% in lard and tallow) and PUFAs (2.6 – 10%), although their content is smaller than in vegetable oils (Fig. 1). In animal fats, n-6 and n-3 PUFAs are generally present at the 3.5:1 ratio which is optimal for human health [29].

Supposedly atherogenic properties of exogenous cholesterol

Atherosclerotic plaques contain mostly oxidized cholesterol, which is why it is popularly associated with atherosclerosis. This does not imply, however, that cholesterol present in food poses a health risk. In addition to exogenous (dietary) cholesterol, the human body also contains endogenous cholesterol [28, 30].

Cholesterol, an essential nutrient, is synthesized mainly in the liver as well as in the skin and intestines in the daily amount of around 1500 mg (in healthy adults). Healthy nervous tissue is composed of minimum 5% cholesterol, and 60% of brain matter consists of lipids. Cell membranes contain 30% cholesterol which affects their rheological properties and supports rapid changes in organ shape (heart, lung, intestinal, vocal cord function). The transmembrane movement of cholesterol molecules relaxes membrane tension, and it is known as the flip-flop mechanism. Cholesterol is also required for the synthesis of vitamin D_3, sex hormones (estrogen, testosterone), stress hormones (cortisol) and bile acids [28]. Around 95% of cholesterol present in the human body is endogenously synthesized. A decrease in the levels of dietary cholesterol leads to only a minor improvement in the blood lipid profile which is determined mainly by dietary essential unsaturated fatty acids, in particular of the n-3 family. Cholesterol present in animal fats poses a risk of atherosclerosis only after it is oxidized.

Animal fats contain various components with antioxidant properties which effectively prevent the oxidation of cholesterol and unsaturated fatty acids [17, 20]. Plant sterols found in oils, margarines and highly processed foods are easily oxidized. Oxidized sterols (oxysterols) induce oxidative changes in both exogenous and endogenous cholesterol [12].

The real causes of atherosclerosis

Atherosclerosis, the main cause of ischemic heart disease, heart attack, stroke and peripheral vascular disease, is a condition with complex pathogenesis [27, 30]. The results of clinical and epidemiological research indicate that inflammations are a causative agent in

atherosclerosis and its complications. In combination with other genetic and environmental factors, inflammations significantly affect the function of endothelial cells, arterial smooth muscle cells and blood platelets. Pro-inflammatory agents, including oxLDL, homocysteine, secondary products of lipid oxidation as well as viral and bacterial infections, enhance the secretion of cytokines (e.g. IL-1, IL-6, TNF-α), free oxygen radicals and reactive oxygen species which contribute to atherosclerotic changes, including stronger monocyte adhesion to vascular walls and the risk of atherosclerotic plaque rupture. Atherogenesis involves local inflammations inside vessels and a systemic inflammatory response [1, 19]. Oxidative stress resulting from excessive levels of reactive oxygen species plays a very important role in the pathogenesis of atherogenesis. ROS-induced peroxidation of unsaturated fatty acids in phospholipids and cholesterol esters leads to endothelial damage and dysfunction [1, 7, 31].

Another important factor in atherogenesis are plasma low density lipoproteins (LDL) which are relatively easily oxidized by ROS [14]. The uptake of LDL (oxLDL) by macrophages leads to the formation of foam cells, a major component of atherosclerotic plaques. The growth of foam cells intensifies with an increase in plasma oxLDL levels. In addition to the peroxidation of cell membrane lipids and plasma lipoproteins, also platelet aggregation plays an important role in the pathogenesis of atherogenesis, and it often leads to thrombosis [1, 28, 30].

Homocysteine contributes to the degradation of vascular endothelium. This cytotoxic component damages the elastin layer, and it speeds up vascular fibrosis and calcification. Homocysteine disturbs clotting processes, and it contributes to higher platelet aggregation which may accelerate the onset of atherosclerosis [13].

Hypertriglyceridemia is yet another risk factor associated with carbohydrate-rich diets. The elimination of fats and increased consumption of carbohydrates to supplement energy reserves leads to a minor drop in total cholesterol and LDL-cholesterol concentrations, but it also decreases HDL-cholesterol levels, it significantly increases the levels of VLDL-cholesterol (atherosclerosis risk factor), triglycerides and blood coagulation indicators [24].

Atherosclerosis is a vascular disease which develops as a progressive dysfunction of vascular wall cells, endothelial cells and smooth muscle cells with accompanying disorders of the monocyte-macrophage system. Endothelial damage induces inflammations and the release of inflammatory mediators. Eicosanoids metabolized from n-6 PUFAs play a crucial role in this process [15, 29]. The disease progresses as more atherosclerotic plaques are produced in vascular walls. With time, atherosclerotic plaques undergo calcification and hardening, which

narrows down the vascular lumen, reduces the supply of nutrients and oxygen to bodily tissues and deteriorates organ function [28].

Numerous studies have demonstrated that lipid profile disturbances are not the decisive cause of atherosclerosis. The key mechanisms of atherosclerosis initiation are oxidative stress (excess ROS levels) and chronic inflammations. A disturbed balance between oxidative stress and antioxidant defense promotes the initiation of atherosclerosis. Atherosclerotic plaques (containing oxLDL cholesterol) which are formed only in the absence of prooxidant-antioxidant homeostasis are the effect, and not the cause of the discussed disease.

According to the current state of knowledge, SFAs and cholesterol found in animal fats are unrelated to vascular inflammations. Animal fats are a source of antioxidants which are highly active in food and in the human body [11].

The effect of animal fats on the prooxidant-antioxidant homeostasis

Excessive dietary levels of n-6 PUFAs consumed with plant oils increase antioxidant utilization and disrupt the prooxidant-antioxidant balance in the body [9, 10]. Unlike vegetable oils, animal fats lower the demand for antioxidants. They are a source of bioactive compounds characterized by high levels of antioxidant activity which protect cell membranes, tissues and organs exposed to oxidative stress (brain, nervous system, respiratory system, circulatory system).

The presence of active antioxidants, high levels of SFAs and oleic acid (30% in milk fat, up to 50% in lard and beef tallow) and significantly lower PUFA levels than in vegetable oils contribute to the high oxidative stability of animal fats [11]. Chilled pork lard remains oxidatively stable for 12 months, but its oxidative stability is reduced at higher temperatures [22]. Lard does not contain trans-isomers whose content in hydrogenated vegetable oils ranges from 4.7% to more than 50%. The most oxidatively stable substance is refined beef tallow which contains fat-soluble vitamins (including α-tocopherol) and conjugated linoleic acid (CLA) [11, 17].

UHT milk and milk powder do not contain oxysterols, products of cholesterol oxidation, which further attests to the high oxidative stability of milk fat. The above can be attributed to the presence of lipophilic antioxidants which are characterized by high thermal stability. Milk fat antioxidants support the body's antioxidant defense mechanisms. It should

also be noted that milk fat is susceptible to hydrolysis, but the resulting butyric acid which is perceptible to the human sense of smell, does not pose a health risk [10].

Milk fat antioxidants are highly effective although they are present in small quantities. They create a synergistic system (antioxidant network) which supports their regeneration – some antioxidants are regenerated at the expense of their synergists [2, 25, 32]. Vitamin E is the most active antioxidant in the group of lipophilic compounds present in the human body. It is accumulated in tissues that are most exposed to oxidative stress: pulmonary alveoli and erythrocytes. The combined effects of vitamin E, selenium and sulfur-containing amino acids in cell membranes prevent structural lipid oxidation. Vitamin E also exerts a protective effect on vitamin A, and it contributes to the regeneration of β-carotene.

In food, vitamin E is present in the form of eight compounds: four tocopherols (α, β, γ, δ) and four tocotrienols (α, β, γ, δ), but only α-tocopherol shows high levels of antioxidant activity at bodily temperature. The predominant compound in milk fat is α-tocopherol, and its antioxidant activity is 50- to 100-fold higher in comparison with other tocopherols present in plant oils. The antioxidant properties of vitamin E are enhanced by vitamin A which, due to its higher singlet oxygen quenching ability than vitamins E and C, minimizes the peroxidation of PUFAs in the cell. Vitamin D_3 also inhibits lipid peroxidation [2, 11, 32].

Milk fat also contains other bioactive components with high levels of antioxidant activity. Conjugated linoleic acid is found only in milk fat (2.9-11.3 mg/g fat) and the meat and fat of ruminants (3.1-8.5 mg/g fat). Its antioxidant activity levels are around 100-fold higher in comparison with α-tocopherol. Studies of animals have demonstrated that CLA has anticarcinogenic properties, and it contributes to the treatment of osteoporosis, obesity and atherosclerosis [4, 6, 18].

Phospholipids may also act as antioxidants due to their PUFA content and the ability to bind cations. Selected phospholipids, such as sphingomyelin, have anticarcinogenic properties [3, 33].

Coenzyme Q_{10} (ubiquinone) is a highly active antioxidant which participates in energy generation in cells. Milk fat contains around 3.5 mg ubiquinone per 100 g. Coenzyme Q_{10} delivers a broad spectrum of health benefits: it reduces glucose levels, controls blood pressure, decreases the frequency of chest pain and contributes to the treatment of arrhythmia. The heart is particularly vulnerable to coenzyme Q_{10} deficiency [33].

Antioxidants present in milk fat significantly contribute to the maintenance of prooxidant-antioxidant homeostasis in the body. Unlike the antioxidants found in fruit and

vegetables, they are active in a lipophilic environment (cell membranes, brain and the central nervous system) [32].

Milk fat contains a complex mixture of bioactive components which intensify cholesterol metabolism (n-9 oleic acid, n-6 and n-3 PUFAs, phospholipids) and inhibit the synthesis of endogenous cholesterol in the liver. For this reason, milk fat cannot be regarded as the key contributor to atherosclerotic changes or the cause of cardiovascular diseases [9, 10].

Anti-atherosclerotic properties of milk fat

Milk fat contains the highest levels of cholesterol (230 mg/100 mg) and SFAs (up to 65%) in comparison with other edible fats. Around 25% of its SFAs are short-chain and medium-chain fatty acids which do not increase the risk of obesity or hypercholesterolemia. They are a source of energy for maintaining stable bodily temperature and supporting organ functions. Short-chain and medium-chain fatty acids play a host of important biological roles [10]. Milk fat prevents atherosclerosis due to the presence of various bioactive components which:

- limit the synthesis of hepatic cholesterol and triglycerides (short-chain SFAs, n-9 oleic acid, PUFAs, in particular of the n-3 family),
- intensify cholesterol esterification and metabolism (phospholipids, n-9 oleic acid, optimal n-6/n-3 PUFA ratio),
- prevent cholesterol oxidization (CLA, α-tocopherol, coenzyme Q_{10}, vitamins A, D_3, C and phospholipids),
- reduce plasma LDL-cholesterol levels (n-3 linolenic acid, n-6 linoleic acid and n-9 oleic acid).

The majority of components with anti-atherosclerotic effects also have anticarcinogenic properties [26].

Although animal fats, in particular milk fat, deliver a variety of health benefit, the human diet should also contain adequate amounts of n-3 PUFAs which can be readily found in coldwater fish and sea mammals. Fish fats contain biologically active long-chain PUFAs which improve the blood lipid profile as well as brain, nervous system and immune system function. The daily demand for fish oils is only ca. 1 g. Regular consumption of two servings of high-fat sea fish per week fully satisfies the requirements for n-3 PUFAs in adults [29].

Conclusions

Despite extensive campaigns that popularize healthy lifestyle options and programs for the primary and secondary prevention of cardiovascular diseases, epidemiological data indicate that reduced consumption of animal fats in favor of vegetable oils not only fails to effectively prevent atherosclerosis, but also increases the risk of cancer and neurological diseases.

Despite popular belief, SFAs and exogenous (dietary) cholesterol in animal fats do not contribute to the risk of atherosclerosis. The blood lipid profile is determined mainly by sufficient amounts of biologically active n-3 PUFAs in the diet which intensify cholesterol metabolism in the human body. High-fat fish and sea mammals, in particular coldwater species, are the most valuable sources of n-3 PUFAs. Artificial trans-isomer fatty acids present in margarines block transacetylase, an enzyme responsible for cholesterol esterification, inhibit cholesterol metabolism and increase the risk of hypercholesterolemia 10-fold in comparison with SFAs.

Antioxidant components are active both in the animal fat source and in the human body. They support endogenous defense systems in protecting the cell membrane, a vital component of every cell, as well as respiratory, nervous and circulatory systems which are particularly prone to oxidative stress.

References

[1]. Banach M., Markuszewski L., Zasłonka J., GrzegorczykJ., Okoński P., Jegier B.: Rola zapalenia w patogenezie miażdżycy, Przegl. Epidemiol. 2004, **58** 663-670.

[2]. Bandarra, N. M., Campos R. M., Batista I., Nunes M. L., Empos J. M.: Antioxidant synergy of alpha-tocopherol phospholipids, J. AOCS, 1999, **76**, 905–913.

[3]. Barłowska J., Litwińczuk Z.: Właściwości odżywcze i prozdrowotne tłuszczu mleka Med. Wet., 2009, **65 (3**), 171-174.

[4]. Bartnikowska E., Obiedziński M.W., Grześkiewicz S.: Sprzężone dieny kwasu linolowego – niedawno wykryte związki o działaniu antykancerogennym występujące w mleku i jego przetworach, Przegl. Mlecz. 1999, **3**, 86-91.

[5]. Bettelheim F.A., Brown W.H., Campbell M.K., Farrell S.O.: Study Guide for Bettelheim/Brown/Campbell/Farrell's Introduction to Organic and Biochemistry, 8th. Ed. Brooks Cole, 2006.

[6]. Białek A., Tokarz A.: Źródła pokarmowe oraz efekty prozdrowotne sprzężonych dienów kwasu linolowego (CLA), Biul. Wydz. Farm., WUM, 2009, **1**,1-12.

[7]. Chopra M., Thurnham D. I.: Antioxidants and lipoprotein metabolism, Proc. Nutr. Soc. 1999, **58(3),** 663-667.

[8]. Cichon R.: Kwasy tłuszczowe n-3 i n-6 w fizjologii i patologii człowieka; Mat. III Symp. Nauk. nt. Olej z nasion wiesiołka i inne oleje zawierające kwasy n-6 lub n-3 w profilaktyce i terapii, Sulejów, 1998, 116-129.

[9]. Cichosz G.: Aterogenne właściwości tłuszczu mlekowego – rzeczywistość czy mit? Przegl. Lek., 2007, **12,**32-34. [http://www.slideworld.org/slideshow.aspx/ATHEROGENIC-PROPERTIES-OF-MILK-FAT-TRUTH-OR-MITH-ppt-60095]

[10]. Cichosz G.: Tłuszcz mlekowy – nierozpoznany nutraceutyk, Mat. Konf. Nauk. Żywność wzbogacona i nutraceutyki, Kraków 2009.

[11]. Cichosz G., Czeczot H.: Tłuszcz mlekowy – źródło antyoksydantów w diecie człowieka, Bromat. Chem. Toksykol. Xl IV, 2011, 1:8-16.

[12]. Derewiaka D., Obiedziński M.W.: Modelowe badania nad utlenianiem steroli, Żywn. Nauka Tech. Jakość 2007, **5(54),**337-345.
[13]. Finkelstein J. D., Martin J. J.: Homocysteine, Int. J. of Bioch. and Cell Biol., 2000, **32**, 385-389.
[14]. Heller F.R., Descamps O., Hondekijn J.C.: LDL oxidation therapeutic perspectives. Atherosclerosis, 1998, **137**, Supp., 25-31.
[15]. Jelińska M.: Kwasy tłuszczowe – czynniki modyfikujące procesy nowotworowe, Biul. Wydz. Farm. AMW, 2005, **1,** 1-9.
[16]. Katan M.B., Zock P.L.: Kwasy tłuszczowe typu *trans* w pożywieniu a ryzyko choroby niedokrwiennej serca, Czyn. Ryz. 1996, (**1**), 45-48
[17]. Kawahara S., Takenoyama S., Nagato Ch., Muguruna M., Ito T., Yamauchi K.: Evaluation of beef tallow as a natural source of conjugated linoleic acid, Animal Sci. J., 2002, **73**,533-539.
[18]. Kritchevsky D.: Antimutagenic and some other effects of conjugated linoleic acid, Br. J. Nutr., 2000, **83**, 459-465.
[19]. Kromhout D.: Fatty acids antioxidants, and coronary heart disease from an epidemiological perspective, Lipids, 1999, **34**, 27-31.
[20]. Landmark- Mansson H., Akesson B.: Antioxidative factors in milk, British J. of Nutr., 2000, **84(1**), 103-110.
[21]. Liebke F.: Omega-3 to co w rybach jest najlepsze,Wyd. Interspar, 2001.
[22]. Madhavi D.L., Deshpande S.S., Salunkhe D.K.: Food Antioxidants Technological, Toxicological and Health Perspectives, Marcel Dekker Inc., 1995.
[23]. Nettleton J. A.: Omega-3 fatty acids and health; Champan and Hall, New York, 1995.
[24]. Oberman A.: Hypertriglyceridemia and Coronary Heart Disease, AM J. Physiol-Endoc. M.., 2000, **85(6**), 2098-2105.
[25]. Palozza P., Krinsky N.: Beta-carotene and alpha –tocopherol are synergistic antioxidants, Arch. Biochem. Piophys., 1992, **15**, 184-187.
[26]. Przybojewska B., Rafalski H.: Kwasy tłuszczowe występujące w mleku a zdrowie człowieka. Krótkołańcuchowe nasycone kwasy tłuszczowe SCFA (cz.1), Przeg. Mlecz., 2003, **4**, 148-151.
[27]. Rose D.P., Connoly J.M.: Regulation of tumor angiogenesis by dietary fatty acids and eicosanoids, Nutr. Cancer, 2003, **37 (2**), 119-127.
[28]. Schneider Z.: Molekularne aspekty miażdżycy; Post. Biol. Kom., 1998, **25(10),** 157-190.
[29]. Simopoulos A.P.: The importance of the ratio of omega-6/omega-3 essential fatty acids, Biomed Pharmacother, 2002, **56**, 365–379
[30]. Skoczyńska A.: Rola lipidów w powstawaniu miażdżycy, Post. Hig.. Med. Dośw., 2005, **59**, 346-375.
[31]. Skrzydlewska E., Łuczaj W.: Współczesne spojrzenie na peroksydację lipidów Post. Bioch., 2006, **52(2**):173-178.
[32]. Ziemlański Ś., Wartanowicz M.: Rola antyoksydantów żywieniowych w stanie zdrowia i choroby. Ped. Wsp. Gastroenter. Hepat. Żyw. Dzieci, 1999, **1**, 97-105.
[33]. Żegarska Z.: Składniki tłuszczu mlekowego o potencjalnym działaniu przeciwnowotworowym, Przegl. Mlecz., 2005, **6**, 4-6.